The Role of Income and Gender Inequalities in the Spread of the HIV/AIDS Epidemic: Evidence from Sub-Saharan Africa

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THE ROLE OF INCOME AND GENDER INEQUALITIES IN THE SPREAD OF THE HIV/AIDS EPIDEMIC: EVIDENCE FROM SUB-SAHARAN AFRICA

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A mes parents

Summary

List of Illustrations

Boxes

Figures

Tables

INTRODUCTION GÉNÉRALE

Découvert à l'Institut Pasteur de Paris en 1983[1], le Virus de l'Immunodéficience Humaine (VIH) est de nos jours l'un des plus grands meurtriers de la planète. Il s'est tout d'abord manifesté dans les communautés homosexuelles et toxicomanes des pays riches avant de s'étendre aux pays pauvres, bien que ce soit dans ces régions qu'il ait le plus causé de ravages. En effet, la maladie est devenue la quatrième cause de mortalité des adultes dans le monde et la deuxième en Afrique subsaharienne (OMS, 2002); ainsi le VIH/Sida est responsable de vingt-trois millions de décès dans le monde depuis le début de l'épidémie dont un peu plus de dix-neuf millions pour la seule Afrique sub-saharienne. Trente trois millions de personnes vivent avec le virus et plus de deux millions de nouvelles infections ont eu lieu en 2007 (ONUSIDA, 2008).

Cependant, les effets de la maladie ne s'arrêtent pas au secteur sanitaire. Le VIH/Sida exacerbe la pauvreté au niveau individuel et familial; de plus il contribue à l'augmentation des inégalités de santé entre le monde développé et le monde en développement (Cornia, 2005). On estime que lorsque le taux de prévalence du VIH/Sida dans la population adulte atteint 8%, le coût au niveau macroéconomique peut atteindre 0,5 de point de pourcentage de croissance (Banque Mondiale, 2001). Des calculs macro-économiques ont permis d'évaluer les conséquences du Sida sur l'économie des pays en développement : un à deux points de croissance du PIB (produit intérieur brut) sont perdus quand 10 % de la population est contaminée (Couderc et Ventelou, 2005). C'est énorme pour l'Afrique sub-saharienne dont la moitié de la population vit en dessous du seuil de pauvreté et qui doit réaliser un taux de

[1] Les symptômes de la maladie ont été découverts chez les premiers sujets en Juin 1981 par le Center for Disease Control and Prevention (CDC) aux Etats-Unis, le nom SIDA a été adopté en 1982, mais ce n'est qu'en 1983 que le virus responsable du syndrome a pu être identifié par l'équipe du Professeur Montagnier de l'Institut Pasteur de Paris.

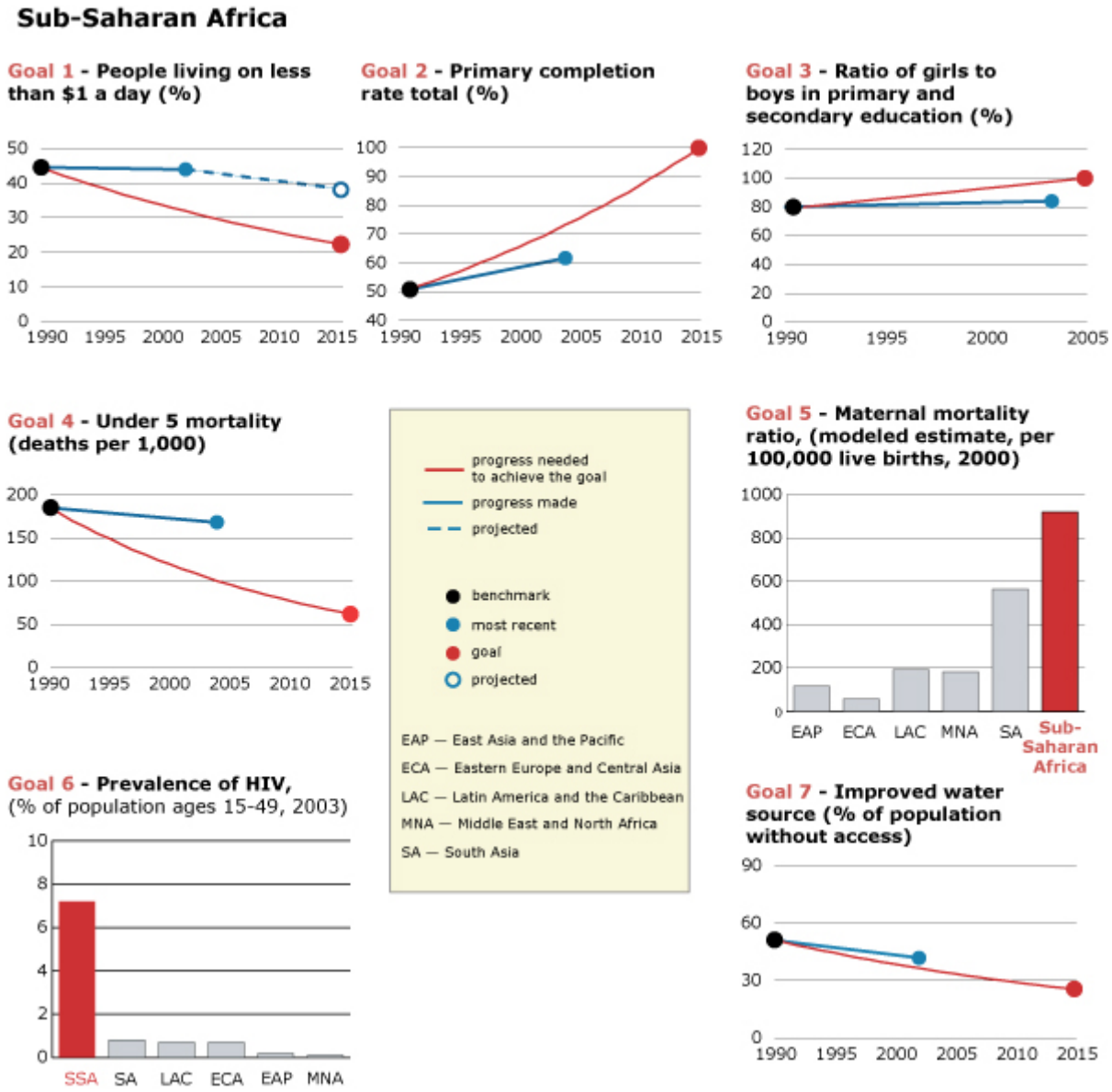
croissance économique de 5% par an juste pour maintenir le *statu quo*. En termes de performances économiques, le continent est toujours à la traîne de par sa contribution au produit mondial, même s'il ne se démarque pas des autres régions en développement en matière d'évolution. En effet, sa part dans le produit mondial est demeurée constante à 2% alors que celle de certaines autres régions en développement a augmenté (Banque Mondiale, 2007). Les autres indicateurs de développement complètent ce tableau sombre car l'Afrique sub-saharienne est la région du monde qui a le plus d'efforts à réaliser pour atteindre les Objectifs de Développement du Millénaire, comme l'illustre la Figure 1.

Au-delà du secteur sanitaire, ce qui fait la particularité du VIH/Sida c'est son impact potentiel sur le développement. En effet, la maladie affecte les adultes dans leurs années les plus sexuellement actives qui coïncident malheureusement avec leurs années les plus productives. Sans l'épidémie du VIH/Sida, la probabilité que ces adultes meurent serait beaucoup moins élevée[2]. Ainsi, le VIH/Sida affecte le développement par l'intermédiaire de la main-d'œuvre qui est le facteur de production-clé en Afrique. A travers elle, la maladie mine trois piliers du développement : la croissance économique, le capital humain et le climat des investissements. Il n'est donc pas surprenant que sur les huit objectifs définis pendant le sommet du Millénaire comme étant les défis du développement à affronter dans le premier quart du 21ème siècle, six soient liés à l'épidémie du VIH/Sida, dont un, l'objectif 6 clairement défini : « combattre le Sida, le paludisme et d'autres maladies »[3]. Cette prise de conscience de l'impact de l'épidémie du VIH/Sida sur la santé et le développement a ensuite conduit à la création du Fonds Global pour la Lutte contre le VIH/Sida, la Tuberculose et le Paludisme lors de la session spéciale de l'Assemblée Générale des Nations Unies consacrée au Sida (l'UNGASS-AIDS) en Juin 2001.

[2] D'après l'annexe statistique du rapport mondial sur la santé 2004 publié par l'OMS, plus de la moitié (56%) de la mortalité en Afrique Sub-Saharienne est due aux maladies infectieuses et parasitaires et le VIH/Sida est responsable de 47 % de la mortalité des maladies infectieuses, il est donc responsable de 27 % de la mortalité totale et l'essentiel de ces décès se produit chez les adultes. Les autres causes majeures de décès chez les adultes en Afrique Sub-Saharienne sont les maladies cardiovasculaires (qui se déclarent en général après 50 ans et qui ne concernent donc pas la classe d'âge des personnes comptabilisées dans le taux de prévalence du VIH/SIDA) qui totalisent 9% des décès, les tumeurs malignes et le diabète avec respectivement 3,5% et moins de 1 % des décès totaux.

[3] L'épidémie touche également de manière moins directe, mais toute aussi importante les objectifs 1 : « réduire l'extrême pauvreté et la faim », 2 : « assurer l'éducation primaire pour tous », 3 : « promouvoir l'égalité et l'autonomisation des femmes », 4 : « réduire la mortalité infantile » et 5 : « améliorer la santé maternelle ».

Figure 1 : Les Objectifs du Millénaire pour le Développement en Afrique sub-saharienne



Source : World Bank, 2007.

Du fait de ses conséquences désastreuses, l'épidémie du VIH/Sida a profondément contribué à modifier la perception de la santé dans le développement ; cette dernière est de moins en moins perçue comme une conséquence du développement et beaucoup plus comme un facteur de développement à part entière, d'où la nécessité de repenser à la santé en termes de développement et non plus seulement sous l'angle des maladies (Audibert 2006, Hsiao et Heller 2006). Le secteur

de la santé qui était auparavant considéré comme un secteur non productif, non profitable et essentiellement source de dépenses est de plus en plus perçu comme un secteur prioritaire pour le développement. L'épidémie du VIH/Sida a également contribué à la perception de l'accès à la santé comme un droit humain. De plus comme l'a souligné Michel Kazatchkine[4], l'épidémie du VIH/Sida constitue un catalyseur puissant pour révéler les dysfonctionnements dans une société, notamment dans les relations entre riches et pauvres.

La plupart des déterminants de la pandémie interagissent entre eux et ont un lien de causalité inverse avec elle. En effet, le virus se transmet parce que les conditions économiques, socioculturelles et épidémiologiques lui sont favorables. A son tour, il aggrave ces conditions, notamment par son impact sur la main-d'œuvre, les institutions et même les maladies[5]. On aboutit alors à un cercle vicieux de l'épidémie du VIH/Sida dans les pays pauvres. En tant que maladie infectieuse, le VIH/Sida est une maladie de la pauvreté et de ses corollaires. Comme le souligne Stillwaggon (2006), les conditions étaient réunies en Afrique sub-saharienne pour que le VIH/Sida y atteigne des proportions pandémiques. Ces mêmes conditions se retrouvent dans d'autres régions du monde comme dans les Caraïbes qui sont la deuxième région la plus affectée, dans les pays d'Asie du Sud gravement touchés par la pauvreté comme le Cambodge et dans les communautés marginalisées des pays riches comme aux Etats-unis. Pour beaucoup d'Africains en effet, le Sida constitue juste une menace de plus à leur survie, et même pas la plus apparente comparée aux guerres, à la famine, à la sécheresse, à la pauvreté et aux autres maladies endémiques telles que le paludisme. La transmission du virus d'un individu à un autre dépend à la fois des caractéristiques du virus et d'un ensemble de facteurs de risque dont certains sont les conséquences des choix des individus et d'autres malheureusement pas. Le contexte dans lequel ont lieu les relations sexuelles est influencé par des facteurs tels que les guerres, les déplacements de populations réfugiées, le travail des migrants, la culture et la religion. Cependant, au-delà de ces causes évidentes de l'épidémie, c'est le contexte sociétal plus large composé entre autres

[4] Michel Kazatchkine, Discours de clôture lors de la 7ème conférence AIDSIMPACT, Marseille 2007.
[5] On observe actuellement une recrudescence des maladies qu'on croyait bientôt éradiquées comme la tuberculose car l'affaiblissement du système immunitaire laisse la voie libre aux « infections opportunistes » qui en temps normal seraient bénignes.

d'un faible accès aux soins de santé, de la pauvreté et du statut subordonné des femmes qui détermine l'épidémie. En réalité, beaucoup de personnes pourraient être considérées à risque du simple fait des conditions de vie difficiles auxquelles elles font face.

Malgré les progrès réalisés par la recherche médicale, il n'existe à l'heure actuelle aucun vaccin contre le VIH/Sida. Les traitements les plus efficaces qui consistent en une combinaison de médicaments anti-rétroviraux sont de plus en plus disponibles, mais toujours inaccessibles à l'immense majorité des populations les plus exposées à l'infection. Ceci signifie hélas que le seul moyen de lutter contre l'évolution de l'épidémie pour ces pays réside dans la prévention. Malheureusement, les gains acquis par la trithérapie dans la lutte contre la pandémie sont plus que compensés par le nombre de nouvelles infections. La prévention est donc en train d'essuyer un échec car avec en moyenne six nouvelles infections pour une personne mise sous traitement (ONUSIDA, 2007), on ne s'achemine pas encore vers un ralentissement de l'épidémie. Cet échec de la prévention dans l'épidémie du VIH/Sida tire ses racines dans une erreur d'identification; le problème a d'abord été catégorisé comme une question de santé publique uniquement alors qu'une réponse efficace supposait qu'on le définisse comme un problème de développement à part entière. En ce sens, une réponse efficace devait être composée de politiques de développement pertinentes et effectives. Et c'est précisément l'échec des politiques de développement qui a été à l'origine des conditions dans lesquelles l'épidémie a prospéré. Paul Farmer (1999) rappelle que le VIH se transmet dans un contexte qui est celui de toutes les maladies infectieuses.

En effet, les pays les plus touchés par le virus sont les plus pauvres de la planète. Les deux tiers des malades vivent dans les pays en développement et les trois quarts des nouvelles infections ont également lieu dans ces pays. Si au niveau international, l'épidémie du VIH/Sida est une bien maladie de la pauvreté[6], curieusement les pays les plus affectés en Afrique Sub-saharienne ne sont pas les plus pauvres, mais les plus riches. Un début de compréhension à ce paradoxe émerge quand on regarde la répartition des revenus de ces pays. Les pays d'Afrique australe qui sont ceux qui font face à l'épidémie la plus sévère sont également ceux qui ont les répartitions de revenu

[6] En fait, après la découverte du VIH/SIDA aux Etats-Unis, cette affection s'est plus développée dans les pays du Sud qui n'ont pas pu mettre en place des moyens de prévention adéquats (sensibilisation, éducation, mobilisation du corps médical, mobilisation de la société civile, etc…).

les plus inégales. De fait, l'inégalité crée une synergie avec la pauvreté pour propager l'épidémie et ce, dans les pays riches comme dans les pays pauvres. Les Etats-Unis, pays de l'OCDE avec la prévalence du VIH/Sida la plus élevée (0,6 % de la population adulte), affichent un coefficient de Gini relativement élevé (40,8) pour un pays développé[7]. Au lendemain du passage du Cyclone 'Katrina' durant l'été 2005, le monde entier a d'ailleurs découvert avec consternation des poches de pauvreté nichées au cœur de la Louisiane, traduisant ainsi l'ampleur des inégalités dans le pays le plus riche du monde. Deux autres pays aux écarts de revenu importants, le Brésil et l'Afrique du Sud affichent des indicateurs de santé moins bons que ne le suggérerait leur seul niveau de revenu. Par contre, certains pays moins riches, mais avec un faible niveau d'inégalité présentent de bons résultats sanitaires comme l'Etat de Kerala en Inde et Cuba[8].

L'inégalité ne reçoit toujours pas toute l'attention qui lui est due en tant que problème de politique économique, alors même qu'elle apparaît de moins en moins acceptable aux yeux du monde et que la communauté scientifique semble lui accorder un intérêt croissant et que ses conséquences sont si importantes (Cornia, 2006). En effet, une réduction des inégalités n'est pas seulement souhaitable pour des raisons de justice sociale, mais aussi en raison de leur impact sur le développement humain. Comme le souligne le Rapport sur le Développement dans le Monde 2005[9] (PNUD, 2005), les inégalités en matière d'opportunités ont un rapport direct avec ce que les individus sont capables ou pas de réaliser, c'est-à-dire les *capabilités* au sens de Sen. En ce sens, même si aucun des Objectifs du Millénaire pour le Développement n'évoque explicitement la question des inégalités, cette dernière est sous-jacente car les progrès réalisés par le quintile le plus pauvre de la population sont inférieurs à ceux de la population générale alors même que ce sont ceux-là qui ont le plus de chemin à parcourir pour atteindre les objectifs définis.

Le deuxième paradoxe dans la distribution de l'épidémie du VIH/Sida dans le monde concerne la proportion des femmes infectées. Alors que l'épidémie s'est déclarée dans les populations homosexuelles masculines des pays riches, les femmes constituent aujourd'hui 60% des personnes infectées en Afrique sub-saharienne, alors

[7] Parmi les pays de l'OCDE, seuls le Mexique et la Turquie ont un coefficient de Gini plus élevé que les Etats-Unis (49,5 et 43,6 respectivement) et la valeur moyenne du coefficient pour l'ensemble des pays est de 32,53.
[8] Cuba et l'Etat de Kerala en Inde ont un revenu de moins de 6.000 dollars par habitant et une espérance de vie de 76,5 ans comparable à celle des Etats-Unis (76,9 ans).
[9] Rapport sur le Développement dans le Monde 2005, Chapitre 2: Inégalité et développement Humain.

qu'elles représentent juste la moitié de la population[10]. Plus important encore, elles représentent les trois quarts des nouvelles infections dans les régions en développement (UNAIDS, 2007). Face à ce nouveau visage féminin de l'épidémie, il faut clairement reconnaître que non seulement le développement est au centre de l'épidémie du VIH/Sida, mais que les inégalités de genre sont au cœur de la problématique du développement et du VIH/Sida. Il n'est donc pas surprenant que l'échec des politiques de développement (qui ont n'ont pas abordé les questions de genre) ait également contribué directement aux processus économique, social et culturel qui ont créé les conditions de prolifération du VIH (Cohen et Smith 2000, UNDP). L'effet de la pauvreté est exacerbé par la situation vulnérable des femmes dans beaucoup de sociétés africaines. La recherche de la survie à travers le commerce sexuel ou le mariage place de nombreuses femmes en situation de risque de contracter le virus. Même dans des circonstances où les femmes sont informées des risques et que les préservatifs sont disponibles, la seule volonté des hommes empêche l'utilisation des préservatifs lors des rapports sexuels à risques. Ceci est particulièrement vrai dans le contexte du mariage car l'utilisation systématique des préservatifs signifie ne pas avoir d'enfant. A cet effet, Bill Gates a insisté sur le fait qu' «aucune femme ne devrait demander à son partenaire la permission de protéger sa propre vie.»[11].

Nous définissons l'inégalité socio-économique à partir de deux dimensions : l'inégalité de revenu et l'inégalité de genre. L'inégalité socio-économique représente ainsi l'écart dans l'accès aux opportunités entre individus d'une même société, entre riches et pauvres, mais aussi entre hommes et femmes. Si les conséquences de l'épidémie du VIH/Sida sur la santé, l'économie et la société toute entière sont relativement bien documentées, paradoxalement il n'en est pas de même de ses déterminants, du moins en ce qui concerne la recherche en économie. La plupart des recherches faites sur le sujet appartiennent au domaine de la sociologie (Le Palec 1999, Becker et *al* 1999), de la médecine (Center for Diseases Control 1999, Do et *al* 2001) ou de l'épidémiologie (Auvert et *al* 2005, Caldwell et Caldwell 1996). De même, la plupart des travaux économiques réalisés à ce jour sur les déterminants de l'épidémie du VIH/Sida utilisent des données transversales. La spécificité de notre travail est

[10] Les femmes représentent 50,22 % des 806 millions de personnes vivant en Afrique Sub-Saharienne (United Nations Staistics Division on men and women, 2007)
[11] Discours à l'Ouverture du 16ème Congrès International sur le SIDA à Toronto, 14 Août 2006.

d'apporter une dimension temporelle à l'analyse. En utilisant des données de panel, nous pouvons analyser l'évolution de l'impact des différents facteurs, impact qui est susceptible de varier dans le temps.

Cette thèse est composée de quatre chapitres. Le premier étudie les déterminants de l'état de santé en général et s'attache plus particulièrement à l'examen des liens entre état de santé d'une part et inégalités de revenu et de genre d'autre part. On ne saurait aborder le problème du HIV sans parler du contexte général des maladies infectieuses. En effet, il ne faut pas oublier qu'avant de s'étendre à plusieurs niveaux pour former la redoutable pandémie à laquelle est confrontée l'humanité, l'impact du virus est d'abord ressenti au niveau personnel, sur la santé de l'individu. En ce sens, l'analyse des déterminants de la santé est cruciale pour commencer à comprendre celle de l'épidémie du VIH/Sida. De fait, le système de santé, les déterminants sociaux de la santé et les caractéristiques individuelles sont les principaux facteurs qui conditionnent l'état de santé d'une population. L'inégalité de revenu agirait sur la santé par des canaux qui ne font pas toujours l'unanimité. Il s'agit notamment de l'accès aux opportunités de la vie (Benzeval et *al* 2000, Deaton 2001), de la cohésion sociale (Kawachi et *al*, 1997) et des facteurs psychosociaux (Wilkinson 1996, Marmot 2001). L'inégalité de genre quant à elle agit sur l'état de santé à travers des canaux tout à fait différents. Tout d'abord, son impact sur l'état de santé est indirect puisqu'il transite par des déterminants-clés de l'état de santé que sont l'éducation et le revenu, mais également par la répartition du pouvoir. L'inégalité de genre est assez visible en matière d'état de santé car si les femmes vivent en moyenne plus longtemps que les hommes, elles se déclarent moins souvent en bonne santé (Waldron, 1986). Cette inégalité est également assez remarquable en ce qui concerne l'accès aux soins qui est plus restreint pour les femmes et les filles et qui dans de nombreux cas se résume aux soins de santé de la reproduction.

Le deuxième chapitre présente une description de l'épidémie du VIH/Sida, tant en ce qui concerne l'état des lieux actuel que par l'analyse des déterminants aussi bien au niveau théorique que par la revue des différents modèles économiques de la littérature. Nous faisons ici le point de notre connaissance sur l'épidémie du VIH/Sida. A la fin de 2006, environ quarante millions de personnes vivaient avec le virus dans le monde. Même si ce chiffre semble se stabiliser par rapport aux années antérieures, il faut le rapporter au nombre de nouvelles infections qui continuent d'augmenter, et ce

plus particulièrement dans les pays dits de la « nouvelle vague ». En tant qu'indicateur d'évolution d'une épidémie, le taux de prévalence du VIH présente des caractéristiques spécifiques qu'il convient de prendre en compte afin d'éviter des estimations biaisées. En effet, deux raisons rendent cette analyse différente de l'analyse des variables économiques classiques: (1) en tant que pourcentage, le taux de prévalence a une limite supérieure et (2) la différence de potentiel de nouvelles infections entre le début et suite de l'épidémie se traduit par une courbe en *S*. En dehors des facteurs purement personnels qui peuvent pousser deux personnes l'une vers l'autre, ce qui fait que la conséquence du contact entre ces personnes résultera en termes de transmission du virus dépend d'un ensemble de facteurs qu'il convient de déterminer afin de maîtriser l'évolution de l'épidémie. Ces facteurs tiennent autant de l'environnement dans lequel vivent les individus que de leur comportement. Plus généralement, on peut les regrouper en facteurs socio-économiques, socioculturels et épidémiologiques. L'examen de la littérature à travers les études de Over, Bonnel, Nattrass, Stillwaggon et Zanakis nous confirme la pertinence des déterminants évoqués plus haut. Si l'impact de certaines variables comme le niveau de revenu national ne font pas l'unanimité, d'autres comme les inégalités de revenu et de genre apparaissent comme des facteurs d'aggravation de l'épidémie dans toutes les études où elles sont introduites.

Dès lors, nous nous intéressons aux liens entre inégalités de revenu et de genre et épidémie du VIH/Sida. Dans notre troisième chapitre, nous étudions l'impact des inégalités socio-économiques sur le cours de l'épidémie, plus particulièrement les canaux par lesquels cet impact transiterait en Afrique sub-saharienne dans le cas d'une maladie infectieuse comme le VIH/Sida. Si l'on comprend à peu près que la privation matérielle et l'accès aux opportunités de la vie[12] véhiculent l'impact de l'inégalité de revenu sur la santé, on voit mal comment les facteurs psychosociaux seraient à l'œuvre dans la transmission du VIH/Sida en Afrique sub-saharienne. De fait, des spécificités liées au continent et à la transmission du virus se combinent aux canaux traditionnels de l'inégalité de revenu, amplifiant ainsi son effet sur le cours de l'épidémie dans cette partie du monde. Ainsi, le commerce sexuel et le travail des migrants, deux activités génératrices de revenu exposent les individus à l'infection. Elles sont pourtant parmi les seules à la portée de nombreuses femmes et plus généralement de pauvres qui ne

[12] Il s'agit là des deux premiers canaux de transmission de l'inégalité de revenu sur l'état de santé.

disposent pas d'autres sources de revenu. Enfin, la crise économique qu'ont connue la plupart de ces pays dans les années 80 et qui a été à l'origine des programmes d'ajustement structurel n'a pas du tout épargné le secteur de la santé. Dans certains d'entre eux à l'instar du Cameroun, c'est même le secteur qui a le plus souffert de la crise et de l'ajustement qui en a résulté (Samba, 2004). En ce qui concerne les inégalités de genre, les canaux de transmission sont tout à fait différents de ceux de l'inégalité de revenu. Tout d'abord, la problématique du VIH/Sida se trouve à l'intersection entre les concepts de genre, développement et santé. De plus, les inégalités de genre agissent à deux niveaux sur l'épidémie ; elles accroissent en premier lieu la susceptibilité des femmes à la transmission du virus. Ensuite une fois infectées, leur vulnérabilité est également plus importante que celle des hommes.

A partir d'une analyse en données de panel de 42 pays d'Afrique sub-saharienne au cours de cinq périodes, nous mettons en évidence dans le dernier chapitre les relations décrites dans les trois premiers. La variable dépendante qui nous permet de capter l'épidémie du VIH/Sida est le taux de prévalence. Nos deux variables d'intérêt seront les inégalités de revenu et de genre. Cependant, ces deux variables feront l'objet de traitements différents ; alors que l'inégalité de revenu est simplement représentée par le coefficient de Gini, l'inégalité de genre sera capturée à travers trois de ses manifestations. Il s'agit notamment de l'inégalité de genre en matière de revenu, d'éducation et de l'importance des bénéfices accordés lors des congés de maternité. Après avoir spécifié la forme de la variable dépendante qui tient compte de l'évolution propre aux épidémies, nous estimons le modèle en effets aléatoires pour tenir compte de l'impact de certaines variables qui ne varient pas dans le temps. Il apparaît ainsi que les inégalités socio-économiques sont des déterminants cruciaux de l'épidémie en Afrique Subsaharienne. Leur introduction dans le modèle permet d'obtenir près de 30% de pouvoir explicatif en plus. De plus, une analyse dynamique conforte l'importance des inégalités socio-économiques comme facteurs déterminants de l'épidémie. Nous constatons ainsi que l'impact de ces variables tend à augmenter au cours du temps. Enfin, nous essayons de quantifier l'effet d'une réduction des inégalités sur le cours de l'épidémie. Les prédictions sont surprenantes dans la mesure où une réduction raisonnable des inégalités[13], engendrerait une baisse du taux de prévalence qui va de 3,5% à 18%.

[13] Il s'agit de combinaisons de réductions de 5% de l'inégalité de revenu et de jusqu'à 20% de réduction des inégalités de genre.

CHAPTER 1

SOCIO-ECONOMIC INEQUALITIES AND HEALTH

> "The health of a population depends not just on the size of the economic pie, but on how the pie is shared." ***Daniels, Kennedy and Kawachi*** *in Justice is Good for Our Health (2000).*

Introduction

The study of health determinants is of great importance, as health constitutes the second part of human capital accumulation (along with education). Health is undoubtedly a very complex outcome, influenced by many determinants. Health determinants are elements that have been proved to have an influence on health outcomes in populations. Those are sanitary factors (health system, organization of care system, state of medical knowledge, qualification of health staff, access to quality health care), political factors (economic and social planification, health legislation, international aid), socio-economic factors (accommodation, urbanization, rural amenagement, lifestyles, employment, literacy rate, consumption modes and leisure), psycho-cultural factors (traditional laws and customs, beliefs in health matters, mentality), demographic factors (age and sex structure of population, family planning, urban concentration, internal migrations), geographic factors (natural wealth, climate). In terms of policy, it is

important to assess the impact of each determinant on health. For example, using US data, Genver (1976) found that health system contributed to 11% of morbidity, while it received up to 90 % of health expenditure. Meanwhile lifestyle, which is a combination of environment and behaviour accounts for 43 % of mortality, although it received a mere 1, 5 % of health expenditure. In the same way, Latouche (1995) established the contribution of genetic factors at 16 %. Health determinants are generally analyzed in the framework of models and in this chapter, we present two models of health determinants. These models were developed by Grossman (1972) and Mosley and Chen (1984).

The relationship between income inequality and health is not as obvious as the relationship between income and health. Income inequality impacts health through material deprivation, social cohesion and psychosocial factors. In addition to its impact on health, income inequality also gives birth to health inequalities in a society, translating into the presence of a socio-economic gradient and social inequalities in health.

As for gender inequality, its impact on health transits through the influence it has on some major determinants of health such as income, education and power relations. The gender gap in health is observable on both health status and access to health services.

In the next section of this chapter, we analyze health determinants in general, before describing the links between income inequality and health in the following section. The last section deals with the link between gender inequality and health.

Section I: Health determinants

The Alma Ata conference stated that "… health, which is a state of complete physical, mental and social wellbeing, and not merely the absence of disease or infirmity, is a fundamental human right and that the attainment of the highest possible level of health is a most important world-wide social goal whose realization requires the action of many other social and economic sectors in addition to the health sector" (Declaration of Alma-Ata, 1978). So, this event highlighted the importance of socio-economic factors as major determinants of health.

The Commission on Macroeconomics and Health created in 1999 by the World Health Organization (WHO) identified poverty, social exclusion, poor housing and

poor health systems as the main social causes of ill health. For a few years, the paradigm has shifted from the study of health determinants at macroeconomic level to what is now known as social determinants of health. Social determinants of health are the social conditions in which people live which determine their health. Social determinants, which are major determinants of health, greatly influence collective and personal well-being. These forces acting at a collective level shape individual biology, individual risk behaviours, environmental exposures, and access to resources that promote health (Daniels et *al*, 2000). McKeon (1976) showed that the extension of life expectancy observed during the 20th century is due more to the change in life conditions than to medical progress. Genetic factors have an influence on individuals' predisposition to diseases, but their impact on populations' health is much less obvious. In addition, the causes of health differentials between individual can differ from the causes of health differentials between populations (Rose, 1985).

Some authors tried to develop theoretical model to explain the evolution of health status. In this section, we present the models developed by Grossman (1972) and Mosley and Chen (1984) before studying major health determinants.

I- Some models of health determinants

We briefly present the Grossman model, which is a model of health production and the model of Mosley and Chen, which is a model of determinants of infant mortality.

A- Grossman (1972)

The model developed by Grossman (1972) is not speaking of a sole model of health determinants, but a model of health production. Since health cannot be bought directly, the demand for health is indirect and very difficult to measure. Because it is not possible to purchase units of health directly, Grossman used the 'Household production function model of consumer behaviour' to construct a model of health demand. Since the works of this author, the individual is no more considered only as a health subject, but also as an actor because he influences his health status through the investment he realises. For him, health can be viewed as a durable capital stock. So individuals inherit an initial stock of health that depreciates with time and can be increased with investment. In his model, health is demanded by consumers for two

reasons: (1) as a consumption good (because sick days are a source of disutility) and (2) as an investment good (because it determines the total amount of time available for other activities). So, the shadow price of health depends on many other variables in addition to the price of medical care.

Health production function can be written as:

$$H_{i,t+1} = H_i + I_i - \delta_i H_i \qquad (1.1)$$

Where $H_{i,t+1}$ is the health of individual *i* at period *t+1*

H_i is the health of individual *i* at period *t*

I_i is the investment individual *i* realises in health at period *t* and

δ_i is the natural depreciation rate of health.

I_i includes individual's behaviour and the environment in which he lives. Thus, it accounts for health determinants.

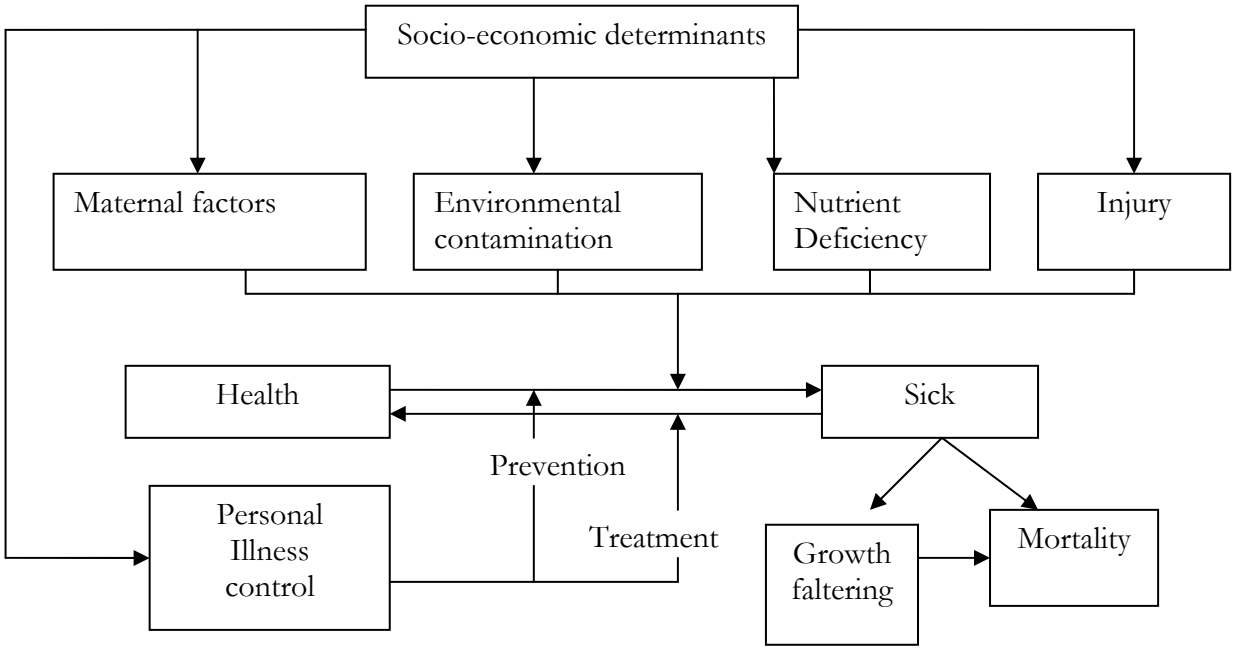
B- Mosley and Chen (1984)

In their model, Mosley and Chen (1984) try to correct the flaws in the explanation of infant mortality as proposed by social and medical sciences. The interest of their model lies precisely in the fact that it combines social sciences and medical sciences methodologies. According to the authors, on the one hand social sciences fail to determine the mechanisms through which socio-economic factors can influence infant mortality and just determine underlying relations, leaving unexplained the mechanisms from which stem these relations. On the other hand, medical sciences focus on biological and dietary factors that provoke diseases and deaths and ignore socio-economic determinants which are at the root of phenomenons. This approach considers that socio-economic factors influence infant mortality through intermediate biological determinants called proximate determinants.

Infant mortality is thus not only the result of the worsening of a diseases, but the final stage of a process that links variables interacting each on the others and influenced by socio-economic variables. Proximate determinants are regrouped in five categories: maternal factors (age, parity and birth interval), environmental contamination (air, food, skin and vectors), nutrient deficiency (calories, proteins and micronutrients), injury (accidental and intentional.) and personal illness control (personal preventive measures,

medical treatment). Figure 1.1 describes the links existing between the five groups of determinants.

Figure 1.1: Proximate determinants of child health



Source : Mosley and Chen, 1984

II- Health systems and related sectors

Health services, namely those which aim at improving health and reducing illness contributes to a better general health. But health infrastructure is not confined to health sector. Government policies and actions on other sectors can bring significant contribution to health.

A- Health systems

Recently, the major life-saving scientific innovations in medical procedures and new pharmaceuticals have had a major effect, particularly on reduced mortality from cardio-vascular disease. There have also been important health innovations whose effect has been mainly in poor countries. For example, the development of freeze-dried serums that can be transported without refrigeration, and of oral rehydration therapy for preventing children dying from diarrhoea.

The performance of health systems depends on the knowledge, skills, motivation and deployment of the people responsible for organizing and delivering services. Human resources—which include all clinical staff like doctors, dentists and nurses and health system staff such as managers, drivers and accountants—also represent the biggest single recurring cost for a health system. Skilled health workers are unable to deliver services effectively without appropriate physical capital – facilities and equipment – and consumables such as medicines. Thus health system budgets need balance among these three vital exigencies – human resources, physical capital and consumables–. In many countries, two-thirds or more of the regular health budget reflects labour costs. In the face of limited resources many countries are left with insufficient funds for equipment, drugs and supplies. The causes of these shortages are manifold. There is a limited production capacity in many developing countries as a result of years of underinvestment in health education institutions. Moreover, the training output is poorly aligned with the health needs of the population. There are also "push" and "pull" factors that encourage health workers to leave their workplaces, mainly related to unsatisfactory working conditions, low salaries, and poor career opportunities. The migration of health workers to developed countries has dire consequences for the health systems in developing countries, which are already suffering the effects of years of neglect, poorly managed health care reforms and economic stagnation. There is a clear correlation between the population density of health care providers and the levels of coverage attained with essential health interventions, such as immunization and skilled attendance at delivery. But many countries lack the human resources needed to deliver these interventions. (WHO 2006)

Access to preventive and other health services covers several factors, including access to health units and hospitals, trained staff providing quality services, cost-effective technologies, the best use of available technologies, and information to improve the community's knowledge base and best practices. One of the most striking features of mortality decline during the 20th century was its decreasing dependence on national income levels. As the century progressed, populations were able to attain ever-higher life expectancy at given levels of real income, gaining from 'shifting the curve' relating income to life expectancy, rather than from 'moving along the curve'. Fundamental to 'shifting the curve' has been developments in the production, use and dissemination of

public health infrastructures and their associated knowledge, which have acted synergistically to improve health.

B- Related sectors

Castro-Leal et *al* (2000) examined public spending on curative health care for seven African countries and found that it rather favours the well-off than the poor. Grossman (1981) stated that "actual and potential government policies with respect to a variety of non-medical inputs can have important impacts on health outcomes". Although it is more visible there, health infrastructure is not confined to health sector. Access to safe water and sanitation, information on hygiene and rural amenagement, prevention of international transmission of diseases are all components of health infrastructure, as are also infrastructure, transport, energy and agriculture.

III- Socio-economic determinants of health

Social determinants of health include not only poverty but also the circumstances in which people live and work. It is well known that among rich countries, there is little correlation between gross national product (GNP) per person and life expectancy. To borrow Geoffrey Rose's term, there is a need to examine "the causes of the causes", that are the social conditions that give rise to high risk of non communicable disease whether acting through unhealthy behaviours or through the effects of impossibly stressful lives. Careful analysis of the global burden of disease has pointed to the importance of risk factors, such as being overweight, smoking, alcohol, and poor diet.

A- Poverty/ Income

The link between poverty and health works in both directions, as poverty causes poor health and poor health exacerbates poverty.

1) Poverty and Health

As stated by Kofi Annan, the former Secretary General of United Nations in his allocution to the World Health Assembly (2001), poverty is the biggest enemy of health in the developing world. Even in the most affluent countries, people who are less well off have substantially shorter life expectancies and more illnesses than the rich.

The positive relation between income and health is known in the literature (Wagstaff and van Doorslaer 2000, Deaton 2003) as the Absolute Income Hypothesis, which

states that it is the absolute income of individuals that determines their health status. At national level, Gross National Product (GNP) is likely to influence mortality indirectly through its effects on food, sanitation, housing, medical care or education and this relationship is concave as demonstrated by Preston (1975). The chance of survival depends on national income because of the accessibility to a doctor, hospital, on availability, even before affordability. According to Summers and Pritchett (1996), the relationship between income and health at national level is so straightforward that it can be summarized in the phrase: 'Wealthier is healthier'[14].

At the individual level, there are solid grounds (Subramanian et *al* 2002) for asserting that the lack of income is causally linked to poorer health. Indeed, higher incomes provide greater command over many of the goods and services that promote health, including better nutrition, access to clean water, sanitation, housing, and good quality health services. Poor people suffer worse health and die younger. They have higher than average child and maternal mortality, higher levels of disease, and more limited access to healthcare and social protection. Income determines health status because it gives access to proper nutrition, adequate accommodation, health care as well as other inputs normally found in the health production function. A non-linear relation has also been found at individual level by Benzeval et *al* (2001).

For Sen (1999) however, the relationship between health and income is all but straightforward, as evidenced by numerous examples of poor countries which have been successful in achieving good health without economic growth (Sri Lanka, pre-reform China, the Indian State of Kerala), or on the contrary, which exhibit fair economic indicators, but poor health indicators[15] (Brazil, Gabon, Namibia and South Africa). A higher income surely constitutes a positive factor for health, but due to the influence of other factors, rich people can live shorter lives that poorer people. Sen reminds that it would be just as inexact to claim that higher income is not a contributory factor to better health and longer survival as it would be to assert that it is the only contributory factor. The usefulness of wealth lies in the things that it allows people to do, the substantive freedoms it helps us to achieve, including the freedom to

[14] This phrase is indeed the title of a paper on the link between income and health: Pritchett, L, Summers, L.H, 1996, *Wealthier is healthier*, *Journal of Human Resources*, 31(4), 841–68.

[15] Greece for example, with a GNP at purchasing power parities of just more than US$17 000, has a life expectancy of 78·1 years; Costa Rica and Cuba stand out as countries with GNPs less than $10 000 and yet life expectancies of 77·9 years and 76·5 years, while the USA, with a GNP of more than $34 000, have a life expectancy of 76·9 years.

live long and to live well. But this relation is neither exclusive, since there are significant other influences on our lives other than wealth nor uniform, since the impact of wealth on our lives varies with other influences. In fact, the statistical connection between the two is not always close. Anand and Ravallion (1993) showed that life expectancy does in fact have a significantly positive correlation with gross national product (GNP per capita), but that this relationship works mainly through the impact of GNP on the incomes specifically of the poor, and public expenditure particularly in health care. In fact, once poverty and public expenditure on health are included in the statistical exercise, little extra explanation can be obtained from including GNP per capita as an additional causal variable.

2) Health and poverty

However, the relation between income and health also works in the other way. Health is not only the absence of illness. It also determines people's ability to develop their full potential during the entire life. In that way, health is an asset individuals possess, which has intrinsic value (being healthy is a very important source of well-being) as well as instrumental value. In instrumental terms, health impacts economic growth in a number of ways. For example, it reduces production losses due to worker illness, it increases the productivity of adult as a result of better nutrition (Fogel, 2000), and it lowers absenteeism rates and improves learning among school children. Health also allows for the use of natural resources that used to be totally or partially inaccessible due to illnesses. Finally, it permits the different use of financial resources that might normally be destined for the treatment of ill health. Thus, health affects economic growth directly through labour productivity and the economic burden of illnesses. Health also indirectly impacts economic growth since aspects such as child health affect the future income of people through the impact health has on education. Healthy and well-nourished children will perform better in school and a better performance in school will positively impact their future income. In quantitative terms, poor health's impact on development can be considerable; Arcand (2001) estimated that malnutrition's effect amounted to a decrease in the annual GDP per capita growth worldwide of between 0.23 and 4.7 % and Barro (1997) estimated that a 10 % increase in life expectancy could raise economic growth by 0.4 % yearly.

B- Education

There are at least three reasons why higher education favours health. First, education improves the efficiency in resource utilization and increases the amount of these resources; second, more educated people tend to be more conscious of their own health and thus have better access to heath care. Finally, education is associated with low fertility for women and low fertility in turn is correlated with better mother and child's health.

Grossman (1975) has suggested that education can enhance one's ability and desire to exercise healthy habits and avoid unhealthy ones. Education is assumed to enhance health directly by, for example allowing wise use of medical care and indirectly through encouraging healthy habits and caution in the choice of occupation (Leigh 1983). Feinstein et *al* (2006) found considerable evidence that education is strongly linked to health and to determinants of health such as health behaviours, risky contexts and preventative service use. Moreover, a substantial element of this effect is causal. Education does not act on health in isolation from other factors. Those with more years of schooling tend to have better health, well-being and healthier behaviours. Education, as one of determinants in a social environment, is mentioned as an opportunity for better job, higher income, and better socio-economic status. Education teaches people how to apply basic hygiene rules and to avoid unhealthy practices, and more indirectly, it is a good predictor of future socioeconomic status. Moreover, education is the key to one's position in the stratification system. It shapes the likelihood of being unemployed, the occupational class, and the income level. It also gives a feeling of control over one's life circumstances that is associated with low stress.

Health quality and life expectancy are both positively associated with higher education level (Leigh 1983, Feinstein et *al* 2006). Average health performance also depends on how well health-related knowledge is rooted in society. For instance preventive behaviour results from knowledge of risks incurred with hazardous behaviour. For the individual this knowledge is determined by his/her education and by access to appropriate information. The availability and the diffusion of this information are determined by the distribution of education across society. Education can therefore play two different roles in the aggregate production function of health services. First, the level of education of the household's head enhances the longevity of its members.

It seems reasonable in fact that education affects crucial factors such as the understanding of treatments or feeding children healthily. Second, the average level of education in the economy improves its absorption capacity for health-related technology and ideas.

The health effect of education has been most documented about the link between mother's education and child mortality. The knowledge gained through education will not only enable mothers to have a greater awareness of sanitation and a more hygienic way of living, eating and providing nutritious food and to use health-care facilities and family planning more, but also to have improved skills and more self-confidence, to marry late, to take well-paid jobs and to be more exposed to the media and other information which may have a favourable impact on infant mortality. Caldwell (1979) found a strong and robust impact of mothers' education on child health in Nigeria. Glewwe (1999) establishes three mechanisms through which mother's education could raise child health: (1) direct acquisition of basic health knowledge in school may provide future mothers with information useful for diagnosing and treating child health problems; (2) literacy and numeracy skills learned in school may enhance mothers' abilities to treat child illnesses, conditional on health knowledge, and also should help mothers increase their stock of health knowledge after leaving school; and (3) exposure to modern society in general via schooling may change women's attitudes toward traditional methods of raising children and treating their health problems.

C- Social exclusion

It is not simply that poor material circumstances are harmful to health; the social meaning of being poor, unemployed, socially excluded, or otherwise stigmatized also matters. As social beings, individuals need not only good material conditions but, from early childhood onwards, they need to feel valued and appreciated. They need friends, need more sociable societies, we need to feel useful, and they need to exercise a significant degree of control over meaningful work (Marmot and Wilkinson, 2003). Relative poverty means being much poorer than most people in society and is often defined as living on less than 60% of the national median income. It denies people access to decent housing, education, transport and other factors vital to full participation in life. Being excluded from the life of society and treated as less than

equal leads to worse health and greater risks of premature death. As we have already mentioned, absolute poverty is a major determinant of ill-health. The poor are more likely to exist on the fringes of society because of their inability to afford decent housing, education and other things. According to the World Health Organization, the resultant social exclusion is 'socially and psychologically damaging', materially costly and harmful to health. Social stability, acknowledgement of diversity, security, interpersonal trust and social cohesion all form a set of favourable social condition that lowers many health hazards. Societies that exhibit higher levels of these factors are associated with lower rates of alcohol and tobacco use, more regular practice of a physical activity, lower obesity, hypercholesterolemia and arterial hypertension. A number of studies (Wilkinson 1996, Kawachi et *al* 1997, Kawachi and Kennedy 2002) show that limited affective support and restricted participation to social activities have a deleterious impact on health and well-being. Loneliness and loss of self-worth lead people to believe they are useless, and so they live with this sense of hopelessness, or far too often choose to end their lives. As well as the direct effects of being poor, health can also be compromised indirectly by living in neighbourhoods blighted by concentrations of deprivation, high unemployment, poor quality housing, limited access to services and a poor quality environment.

A telling example of health inequalities within countries is the 20-year gap in life expectancy between Australian Aboriginal and Torres Strait Island. Dirty water, lack of calories, and poor antenatal care cannot account for the 20-year deficit in life expectancy of Australian peoples—life expectancy is 56·3 years for men and 62·8 years for women—and the Australian average. This fact is not to deny that poverty is important. But the form that poverty takes and its health consequences are quite different when considering chronic disease and violent deaths in adults, compared to deaths from infectious disease in children.

Place does not only means geographic space. Indeed, spatial characteristics translate into people's physical and psychosocial conditions that are relevant to their health (Siegrist, 2000).

D- Culture

Culture is a product of both personal history and wider situational, social, political, geographic, and economic factors. Culture is important in shaping the way people interact with a health care system, their participation in programs of prevention and health promotion, access to health information, health related lifestyle choices, their understanding of health and illness and their priorities. Cultural factors that affect people's health include people's beliefs, their sense of having an identity, and their culturally based philosophies, practices, and values, including values that relate to rights and responsibilities within the family.

Caldwell (1993) showed two examples of settings where the difference in health achievement could only be attributable to cultural factors. The first case occurred in a Malaysia's plural society where it was discovered that controlling by a range of socioeconomic and biological factors failed to eliminate quite substantial differences in the survival chances of Chinese, Indian and Malay babies. In the second example, the United Nations study focused on 15 developing countries in Africa, Asia and Latin America. For eleven of these countries where ethnic contrasts and ethnicity was identifiable, the results of the study showed that, once controlled for other variables, ethnicity was the only factor other than parental education where large child mortality differentials persisted. Moreover, in seven of the eleven countries child mortality in one ethnic group remained at least 50% greater than in a contrasting ethnic group of the same country.

Religion also underlay differences in child survival but is in many cases difficult to distinguish from ethnicity. In any case the differentials were more easily reduced by controlling for other socioeconomic factors; although some contrasts between Muslim and non-Muslim populations remained, that can be attributed to different attitudes to priorities within the family.

In addition to these factors, for the particular case of developing countries we can cite nutrition, immunization coverage and urbanization.

IV- Individual characteristics

Individual characteristics add to the previous factors to influence health status. They include occupation and lifestyles, genetic factors and age.

A- Occupation and lifestyles

Blue/white collar: white collar live a healthier life than blue collar, from an objective measure such as stress indicators (Wilkinson 2002) as well as from their own perception of their health status. This is due to differences in health habits, life and job satisfaction, mental stress and fatigue. Having little control over one's work is particularly strongly related to an increased risk of low back pain, sickness absence and cardiovascular disease (Marmot and Wilkinson, 2003). These risks have been found to be independent of the psychological characteristics of the people studied. In short, they seem to be related to the work environment.

Vegetarian: Vegetarians who follow a well-balanced low-fat high-fibre vegetarian diet have been shown to have lower incidence of coronary artery disease, osteoporosis, hypertension, diabetes, obesity and some forms of cancer (Dwyer, 1988).

Sedentary: According to the WHO, sedentary lifestyle is increasingly becoming a global public health problem. It is a cause of disability, disease and death. Physical inactivity increases mortality, the risk of cardiovascular disease, type II diabetes and obesity.

B- Genetic factors

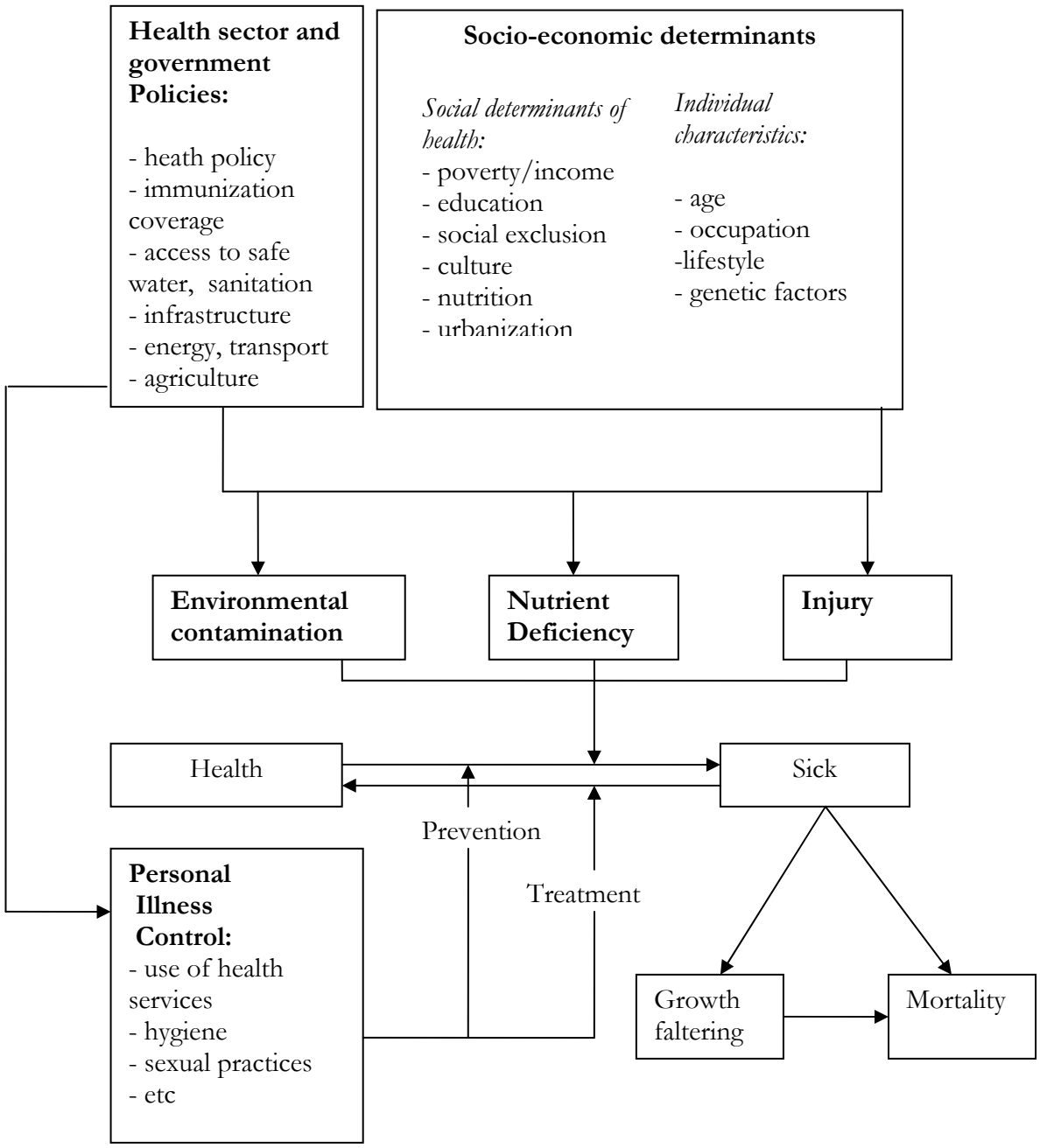
While socio-economic and environmental factors are significant determinants of health at community level, individual susceptibility to a disease under certain conditions is determined by genotype. Many diseases are indeed due to genetic disorders. For example, other factors being constant, people genetically predisposed are more at risk than others to contact affections such as heart disease, high blood pressure, Alzheimer's disease, arthritis, diabetes, cancer or obesity. Therefore, studies on genetic factors are as important as environmental ones. Without knowing the genetic predisposition one gets an incomplete picture of potential risks and blocks preventive measures and activities from being directed onto the most vulnerable ones. Data on genetic health determinants opened up dilemmas and created reasons for worry, but they also brought about unforeseen possibilities, the dimensions of which are unimaginable.

C- Age

During the first years of life, people are more fragile, then they become healthier and chronic diseases arise when people get old. Thus, the relationship between age and health follows an inverted-U shape, with a maximum during adulthood.

Figure 1.2 summarizes health determinants and the way they impact on health status and mortality. Maternal factors have been removed from this figure because Mosley and Chen's initial model represent the determinants of child health.

Figure 1.2: Health determinants



Source: Author, adapted from Mosley and Chen (1984)

Section II: Income inequality and health

Unlike poverty or national income, the link between income inequality and health is not obvious. It has been widely admitted that income level is one of the

determinant factors of health status, both at individual (Lynch et *al* 2000, Lynch and Smith 2004) and international level, and especially in poor countries (Preston 1975; Summers and Pritchett 1996). The broad link between poverty, inequality and health was first established by Rodgers (1979) using data from both rich and poor countries. He found that life expectancy was correlated with both national income (GNP per capita) and income distribution. Subsequently, the World Health Report (1995) established poverty as the main cause of death in the world; the situation was exacerbated by the inequality accompanying it: the gap in life expectancy between the healthiest and the least healthy country has widened passing from 35 to 49 years (the highest life expectancy at birth is for Japan and has increased from 78 to 82 years between 1995 and 2005 while the lowest was 43 for Uganda in 1995 and is 33 for Swaziland in 2005). States with high levels of income inequality tend to agree less on the nature of public expenditure (especially social expenditures such as health care) to engage in, leading to a decrease in these types of expenditures which are important determinants of health status. Ever since the works of Wilkinson (1996) who postulated that "*the quality of the social life of a society is one of the most powerful determinants of health, and in turn […] is very closely related to the degree of income inequality*"[16], the relationship between income inequality and health remains open to debate. Income inequality impacts health through material deprivation, social cohesion and psychosocial factors. Despite numerous papers documenting the link between income inequality and health, the nature and the channels through which this link could transit are still unclear and criticized. Income inequality impacts on average health, but it also widens disparities in health inequality, translating into the presence of a social gradient in health, as well as social inequalities in health.

I- Income inequality and health: a disputed relationship

About two centuries ago, in his book "The Wealth of Nations", Adam Smith described the concept of relative deprivation, referring to the "necessaries" of daily life: "*By necessaries I understand, not only the commodities which are indispensably necessary for the support of life, but whatever the custom of the country renders it indecent for creditable people, even of the lowest*

[16] Unhealthy societies, Chapter 1: The social economy of health, page 5.

order, to be without[17]*"*. Health status being an indisputable element of welfare, relative income has an impact on it through these "*necessaries* ". Lynch et *al* (1996) go as far as quantifying mortality attributable to income inequality as exceeding the combined deaths resulting from lung cancer, diabetes, motor accidents and HIV/AIDS 1995 in USA.

A- Income Inequality Hypothesis

Wagstaff and Van Doorslaer (2000) summarized the different theories which postulate that an individual's health is directly affected by income inequality under the income inequality hypothesis.

The equation relative to the income inequality hypothesis at individual level is the following:

$$h_i = f_i\ (y_i, I_p)\ \ (f_I^{'} \prec 0) \qquad (1.2)$$

where: y_i is individual *i*'s income

I_p is income inequality in population P.

Thus, if income inequality increases in population, individual *i*'s health will worsen, and if his income increases, his health status will improve.

According to Deaton (2003), the relationship between health, income and income inequality can take the form of the following equation:

$$h_{is} - \bar{h} = \alpha + \beta(y_{is} - \bar{y}) - \nu(y_{is} - \bar{y})^2 \qquad (1.3)$$

Where ν denotes the pure effect of income inequality on individual *i*'s health.

$h_{is} - \bar{h}$ is the differential between individual *i*'s health and average health in group *s*

$y_{is} - \bar{y}$ is the differential between individual *i*'s income and group average income $\bar{y}$.

Thus, income surely promotes health, but less for the rich. Also, if average income is high, there is still need that everyone's income is high enough, at least beyond the point at which income distribution has much effect on health.

At the aggregated level, the same relationship translates as:

[17] The Wealth of Nations, Book V, Chapter II, Part II

$$h_s - \bar{h} = \alpha + \beta(y_s - \bar{y}) - \gamma(y_s - \bar{y})^2 - \gamma v_s \quad (1.4)$$

The impact of average income on average population's health is thus given by:

$$(h_s - \bar{h})'_{y_s} = \beta - 2\gamma(y_s - \bar{y}) < 0. \quad (1.5)$$

The impact of income inequality on state's health is given by:

$$(h_s - \bar{h})_{v_s} = -\gamma, \quad (1.6)$$

which is constant.

The direct implication of (4) and (6) is that, as income increases, its impact on health decreases while income inequality's impact remains constant. That means the effect of income inequality becomes more important than the effect of income as countries become richer. Then, the absolute income and the income inequalities hypotheses coexist in Deaton's model. Both are not mutually exclusive. Indeed, among the poorest countries, average income is what matters for population health whereas income inequality is relatively less important. Among rich countries, average income is less important and income inequality is relatively more important.

Table 1.1 provides a review of the empirical studies on the link between income inequality and health. Among the eighteen studies summarized, six (Rogers, Kennedy et *al*, Kawachi and Kennedy, Lynch, Lobmayer and Wilkinson, Subramanian and Kawachi) conclude to a strong positive association between income inequality and mortality (or a negative association between income inequality and health). Meanwhile, five other studies (Lynch and Davey-Smith, Mellor and Milyo, Wildman et *al*, Deaton and Lubotsky) argue that there is no clear correlation between income inequality and health. Only two studies conclude to a positive correlation between income inequality and health (Kaplan and Poder (a)). For the remaining five studies (Marmot and Bobak, Wolfson, Judge et *al*, Fiscella and Franks, Poder (b)), the observed association between inequality and health is weak or not consistent.

B- Transmission channels

Marmot and Wilkinson (2001) argued that material deprivation and psychosocial pathways act in association to channel the impact of inequality on health. Lynch et *al* postulated three explanations: individual income, psychosocial factors, and neo-material interpretations. McKeon et *al* (1996) point in the same direction, attributing the

contemporary upsurge of mortality in former Soviet Union to three factors: impoverishment, increasing social inequality and the breakdown of political institutions. Broadly, there are three interpretations to the link between income inequality and health status that summarize the different transmission channels.

1) Access to life opportunities and material deprivation

Education, health expenditure and employment are also determined by relative (in addition to absolute income). In fact, when the gap between rich and poor widens, interests diverge translating into reduced social spending (Kawachi et *al* 1999). Kaplan et *al* (1996) found a positive and significant correlation between the level of inequality at state level and support to human services. Richer people allocate a greater share of their income to competitive spending and try to escape from the neighbourhood. They are also more reluctant about spending that benefit the whole community.

Additionally, there is a dynamic dimension in access to life opportunities. Differences in health status are caused by differences in the degree, the length and the frequency of the exposition to a health factor (favourable or not), as well as the number of factors people are exposed to. Thus the theoretical link between inequality and health transits through income's effect on investment in education and health, the two-way link between nutrition and low-wages and political capacity to deliver public goods in a context of inequality. High incomes help acquire goods necessary to produce health and to participate in social life, while low incomes limit the scope of choices individuals face and acts against favourable behaviour change for health (Benzeval et *al*, 1995). In that view, differences in health would primarily be due to differences in material well-being all life long. Deaton (2001) tested for the two hypotheses (absolute and relative income) and found that the only clear link between income inequality and health transits through the purchase of health care and third factors, such as education.

2) Social cohesion/ social capital

Social tensions due to lack of trust result in hostility, suspicion and social isolation. The consequence is a decrease of the importance given to material deprivation and the role played by social politics in promoting health. Kawachi and Kennedy (2002) found that when preferences are heterogeneous (low degree of social cohesion), the cost of consensus is high and it becomes more difficult to agree about the nature of expenditure to engage in. In addition, the low level of public expenditure, support and

social capital have a considerable impact on population health. Social capital is a concept which attempts to describe the quality and quantity of social interactions in a community. It has been defined as "those features of social organisations (such as networks of secondary associations, high levels of interpersonal trust and norms of mutual aid and reciprocity) which act as resources for individuals and facilitate collective action" (Kawachi et *al*, 1997). As the gap between rich and poor deepens, interests diverge, resulting in lower social expenditure. Unlike poverty that affects only the health of the poor, income inequality is detrimental to everyone's health because it weakens social infrastructure and destroys social cohesion.

Over the past several years, there has been a rapid emergence of research on social capital within the social epidemiological literature. The social capital theory of Bourdieu (1986) explains that social capital impacts health through positive externalities arising from individual's membership to networks. As for Putnam (1995), social capital can be contrasted with physical and human capital. It refers to "features of social organization, such as networks, norms, and social trust, that facilitate coordination and cooperation for mutual benefit", then social networks have value. The amount of social capital in a community, a collective characteristic generated via norms of reciprocity and trust among residents, has implications for a multitude of beneficial outcomes for that community.

Several causal mechanisms have been postulated for the linkages between social capital and health. Social capital may (a) influence healthy behaviours, in part, by establishing social norms supporting those behaviours; (b) lead to the development of, and foster accessibility to, healthcare services; (c) foster mutual trust and respect leading residents of an area to take more responsibility for each other; (d) foster egalitarian democratic political participation and thereby lead to the development of policies that protect all citizens; (e) would seem to build the social infrastructure for a community to prevent and respond to infectious disease outbreaks and; (f) social isolation may be linked to poor health and socially isolated individuals tend to live in areas low in social capital.

3) Psychosocial factors

Income inequality affects heath status through the perception individuals have of their relative position in society. The awareness of one's subordinated position in social or professional hierarchy causes chronic stress, hypercholesterolemia and an abnormally

high cortisol rate in blood, with all their negative effects on health. According to Wilkinson (1996), beyond a GNP per capita of $10.000, what matters for health is not more income, but income differentials between the richest and the poorest citizens. This relation transits through psychosocial factors; economic insecurity and lack of control over one's life and work result in chronic stress and anxiety that weaken the immune system. These situations also contribute to the lack of self-confidence, development of depression anger and destructive behaviours with a deleterious impact on health and that are well spread in inegalitarian societies. Anxiety and stress from severe social inequalities cause chronic pathology and illness. It could foster unhealthy behaviour (smoking or alcohol for example) or it could lead directly to neuro-endocrine perturbations that influence disease risk.

The impact of psychosocial factors on health occurs following the genesis of stress; it is assumed that the inability to respond to new challenges and the inability to relax after challenges may affect health in humans and animals (Murison and Overmier, 1993). This form of loss in dynamic capacity could be a cause for the higher susceptibility to disease that has been found more often among individuals with low Socio-economic status. This hypothesis is based on the assumption that sustained activation is more common in individuals with low socio-economic status. The indirect effects of socio-economic status relate to motivation for change, effort and choices of lifestyle. The high reactivity may be a condition for high performance. However, increased hostility, due to frustrations when coping is no longer possible may be a risk factor i.e. if these efforts do not lead to the expected results, the high reactivity may become sustained and dangerous (Wilkinson 1996). Cornia and Panicià (2000) provided evidence that stress is the main cause of increased mortality in transitional economies of Eastern Europe.

In addition to the above mentioned channels, the link between income inequality and health might simply transit trough income inequality's impact on economic growth. Thus, reduced income growth via low investment in human capital, social tensions and declining work incentives, decreasing returns to capital, policy distort and government failure are channels through which inequality affects economic growth.

II- Critics of the income inequality hypothesis

To date, the link between income inequality and health, though largely documented is far from being consensual. Critics about this link are based on theoretical grounds as well as on empirical demonstrations.

A- Theoretical critics

From a theoretical point of view, some authors criticized the income inequality hypothesis because of its conceptual vagueness.

- First, the mechanisms through which income inequality impacts health are poorly understood (Wagstaff and van Doorslaer, 2000); it is not obvious why an individual with a decent income, sufficient to afford his basic needs including all the inputs in a health production function, could have poor health outcomes because he compares himself to his richer peers. By the same way, it is difficult to admit that relative deprivation in a geographically marginalized population would impact the health outcomes of the population living in richer neighbourhood (even if they were located within the same town). Lastly, albeit the fact that the impact of income inequality can be decomposed into inequality in the access of commodities essential for the production of health on one hand and the relation between the consumption of these commodities and health outcomes on the other, the form of this relation itself remains ambiguous[18].

- Second, though it is adequately proved that inequality poses a hazard to health, it is not certain that it is precisely income inequality that is in cause, especially when it is comforted by other forms of inequalities or when there can be confounding with these other forms of inequalities. In that view, Deaton (2003) warns about a misinterpretation of income inequality coefficient in econometric regressions. The fact that the other inequalities cannot be captured raises the methodological problem of omitted variables. Moreover, if there is confounding with income, the interpretation of income inequality coefficient in the regressions is even more erroneous since it capture at the same time income effect, other inequalities effect and finally, income inequality effect itself. To focus only on income inequality carries the risk of neglecting other kinds of inequality,

[18] A typical example of this ambiguity described by Deaton (2003) is the relationship between alcohol consumption and health. Up to a certain dose (on average one glass), wine consumption deters the occurrence of cardiovascular diseases. Above this quantity however, it favours the occurrence of these diseases. The same can be said about medicines consumption, especially antibiotics that can develop resistance in patients, thus limiting the effectiveness of future treatments.

which also impact considerably on health, like gender inequality (which causes a high mortality gap at young ages, especially in settings where boys are better cared for than girls) or inequality of access to education and health care infrastructure. Thus, individual income as a measure of access to resources is more relevant that inequality to explain the relationship between income and mortality. Psychological or social factors related to income inequality may nevertheless have important health effects, as suggested by Wilkinson.

Indeed, in studies conducted in some rich and non-poor countries, namely USA, Brazil and South Africa the proportion of the population who live in poverty or at least in relative poverty is quite big; thus the measure utilized to quantify income inequality may actually reflect poverty. As a result, the income inequality's effect in these countries would in reality represent a poverty effect. In addition, it is because there are poor people even in rich countries that they are also poor health outcomes. By the same way, women, rural people and minorities are generally poorer. In this way, income inequality' effect captures the impact of these other forms of inequality. In order to be more credible, studies of the inequality hypothesis should control for income at the individual level and use direct measures of factors such as social cohesion and perceived socioeconomic deprivation to advance our understanding of the relative contribution of these factors to health. Moreover, according to Deaton (2003), health status depends not on income inequality itself but on variables that are correlated with income inequality such as education, wealth (which might be different from income) or social rank. Finally, income inequality could be important in the way that it brings out other inequalities and relative deprivations; thus it would have a negative impact on individuals' health. So, even though income inequality is not important per se, social environment is important. Along these lines, Sen (1999) notes that "the effectiveness of freedom as an instrument lies in the fact that different kinds of freedom interrelate with one another, and freedom of one type may greatly help in advancing freedom of other types", thus implying that the removal of any deprivation would contribute to the suppression of the others[19].

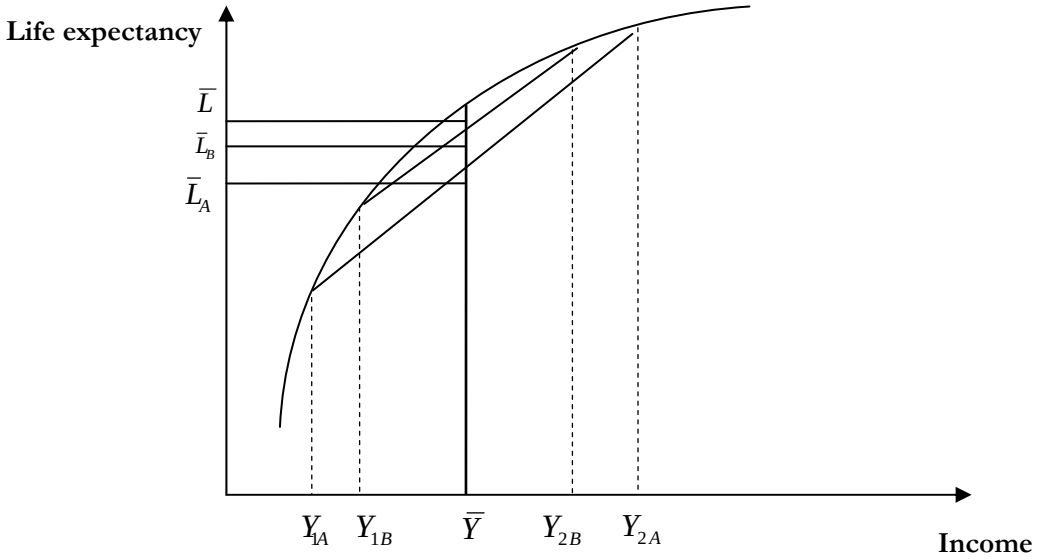
[19] (Development as Freedom, Chapter 2: The ends and means of development. page 37)

B- Empirical critics

In addition to these theoretical critics, there are also some econometric problems that have been raised in relation to the link between inequality and health.

- Some authors considered the relationship between income inequality and health presented in most paper as a pure statistical construction. Gravelle (1998) showed that the relationship found at aggregated level simply reflects the concavity of the function linking health and income level. Besides, the conclusions of the studies establishing a link between income inequality and health have a limited validity because results found with aggregated data are not always reproducible at individual level. In other words, the observed relation between income inequality and health could simply reflect the fact that inequality rates are inadequately measured at individual level and yield an "ecological fallacy" (Fiscella and Franks, 1997). At least part of the relation found between income inequality and health are in reality '*statistical artefact'* and then spurious. To explain the relationship between those two variables, one only needs the absolute income hypothesis and the concavity of the relation between income and health. One should then use individual-level data in order to have an unbiased and non-spurious correlation. Figure 2 below describes the "statistical artefact" in the relationship between income inequality and health as demonstrated by Gravelle (1998).

Figure 1.4: The 'statistical artefact' in the relation between income inequality and health



Country A has an income distribution more unequal than country B:

$$(Y_{2A} - Y_{1A}) > (Y_{2B} - Y_{1B}).$$

Average income is the same in both countries:

$$\overline{Y}_A = \overline{Y}_B = \overline{Y},$$

but life expectancies are different:

$$\overline{L} > \overline{L}_B > \overline{L}_A.$$

Thus, the virtual country with perfect income distribution and the same average income than countries A and B would have the highest life expectancy $\overline{L}$. Country A has a lower life expectancy than B due to its increased level of inequality. We do not really know the structure of income distribution in both countries, but just than country A is less egalitarian than country B. Yet, we are able to show a link between income inequality and health, simply because of the concavity of the relationship between income and health.

- Finally, it is not always acceptable to validate the link between income inequality and health because of certain aspects in the measurement of inequality. Even though there exist other forms of incomes such as food stamps, Gini coefficients for income inequality are computed solely based on monetary income of individuals[20]. Besides, in most rich countries, where most of the studies on the link between income inequality and health are done, the worst-off are sometimes exempt from paying for certain current expenditure or tax which corresponds in fact to an increase of their real income. Finally in poor countries, measures of income that account for monetary income or consumption only are not always relevant, as they do not account for home consumption.

[20] Gini coefficients can also be computed on other measures of wellbeing, such as households expenditure (Poder 2008). However, Gini coefficients for income inequality are computed only on actual income (in developed countries) or on the values individuals declare as their income (in developing countries).

Table 1.1: A review of the empirical studies on the link between income inequality and health

References of study (authors, year)	Health variable	Income inequality variable	Data level	Conclusion
Rodgers (79)	Life expectancy at age 0 and 5 Infant mortality rate	Gini coefficients and quintile distribution	56 developed and develop-ping countries	Strong positive and significant association between income inequality and all measures of mortality
Kaplan (96)	Age-standardized and sex-standardized mortality rates, low birth weight, number of homicides.	Share of income of poorest 50%	50 states (US)	Positive link between income inequality and health measures
Kennedy, Kawachi, Prothrow-Smith (96)	Age-standardized mortality rate , under-five mortality rate, mortality by cause	Robin Hood index, Gini Coefficient	50 states (U.S)	Positive link between income inequality and mortality, even when poverty and tobacco use are controlled for.
Fiscella, Franks (97)	Survival probability during follow-up	Share of income of 50% of PSU	14,407 individuals in 105 counties (U.S)	Small effect of income inequality on self-rate health, nut none on mortality
Judge, Mulligan Benzeval(1998)	Life expectancy Infant mortality	Gini coefficient, share of income of the richest 5%, of the 1^{st}, 2^{nd}, 6^{th} et 7^{th} percentile	16 industrialized countries	Correlation betwen income inequality and life expectancy not significantly different from 0, but significant between inequality and infant mortality
Kawachi, Kennedy (98)	Probability of reporting fair or poor health	Gini coefficient	State level (U.S)	Negative correlation between income inequality and health
Lynch (98)	Age-standardized mortality rate, age-adjusted mortality rate	Ratio of the share of 9th to 1st quintile Gini, Theil and Atkinson indexes	282 urban zones (US)	Positive correlation between income inequality and age-adjusted mortality rate.
Wolfson (99)	Survival during follow-up	Share of income of poorest 50% of the state	National level(U.S)	Weak association between expected mortality and income inequality
Lobmayer,	Potential life years lost,	Ratio of the share of	14	Positive correlation between income inequality

Wilkinson (2000)	age-adjusted mortality rate, sex-standardized mortality rate	income of the 5th to 1st percentile.	industrialized countries	measures and potential life years lost and also with under 30 age-specific mortality. No correlation with >65 mortality rate.
Marmot, Bobak (2000)	Change in life expectancy	Change in Gini coefficient	12 countries of western Europe	Negative correlation between change in income inequality and change in life expectancy. Unclear effect when per capita GDP is controlled for.
Lynch, Davey-Smith (2001)	Age-standardized mortality rate, low birth weight, life expectancy	Gini coefficient	16 industrialized countries	No correlation between income inequality and life expectancy. Inconsistent association between income inequality and all causes mortality, positive correlation between income inequality and low birth weight and negative correlation with > 65 mortality.
Mellor, Milyo (2001)	Mortality rate, all causes	Gini coefficient	48 states (U.S)	No effect of income inequality on health after controlling for income, except for mortality due to homicides
Mellor Milyo (2002)	Mortality rate	Gini coefficient based on family income	Panel data at State level	No link between income inequality and health after controlling for fixed effects
Wildman, Gravelle, Sutton (2003)	Infant mortality rate	Share of income of the 5% richest	71 countries, 16 of which with time-series data	No association between income inequality and health, neither in cross-section, nor in time-series.
Deaton, Lubotsky (2003)	Age and sex-adjusted mortality rates	Gini coefficient	50 states (U.S) et 276 urban zones	No direct effect of income inequality on health after controlling for race.
Subramanian, Kawachi (2003)	Self-rated health	Gini coefficient		Negative correlation between income inequality and health
Poder (a) (2008)	Infant mortality rate	Gini coefficient	84 developed and develop-ping countries	Positive correlation between income inequality and infant mortality rate
Poder (b) (2008)	Height for age Z-score (0-5)	Gini coefficient, Theil index	5339 Guate-malan children	Impact of income inequality on health depends on data aggregation level (statistical artefact)

III- Income inequality and Health inequality

Health inequality is a generic term used to describe disparities in health outcomes across individuals or groups. For example, if prevalence rate of a given infection is higher in group A than in group B, then there is health inequality according to this infection. The link between income inequality and health inequality depends on the way income inequality affects health. Then the material deprivation explication postulates a graded link between socio-economic position and access to tangible material goods that favour good health, like nutrition and accommodation. Meanwhile, psychosocial interpretation describes the existence of health inequalities as the direct consequence of stress, resulting from one's consciousness of an inferior position in social hierarchy and economic disadvantage. These two interpretations are different, but not mutually exclusive, as an individual with a low socioeconomic status can have poor health outcomes due to unhealthy housing and also to a smoking behaviour to cope with stress.
Health inequalities, a merely descriptive situation are different from health inequities that entail a moral judgement. Moreover, they translate into a socioeconomic gradient in health (even among the non-poor) and social inequalities in health.

A- Health inequalities and health inequities

Health inequalities are systematic differences in health outcomes among different groups of population[21]. These inequalities can be modified or not. Health inequities are health inequalities that are 'unnecessary, avoidable and unjust'. They are considered as unfair or arising of some form of injustice. Inequities broadly refer to socioeconomic position, gender or race. Thus, they reflect the presence of systematic disparities in indicators (or main determinants) of health between groups of different levels of wealth, power or prestige. Health inequities entail a moral judgement in the way that they place already disadvantaged or marginalized people (poor, women, homosexual, racial or religious minorities) at further disadvantage in regard to health status. The distinction between health inequalities and health inequities is important; in fact some health inequalities are attributable to biological factors (for example the difference in prevalence rates in cardiovascular diseases between young and old people) or the result of free choices (for example the difference in prevalence rates of lung cancer between smokers and non-smokers) while other are solely due to external environment and to circumstances out of the control of concerned individuals (for example the difference in life expectancy among racial groups of a population). In the first case, it is impossible or ethically unacceptable to change health

[21] This definition is borrowed from Wagstaff in the WHO Bulletin, vol 78 (2), 2000.

determinants and thus, health inequalities are unavoidable. While in the latter case, the unequal distribution of health determinants seems avoidable and unfair so that health inequalities resulting also translate into health inequities.

B- Socio-economic gradient in health

According to Deaton (2001), socioeconomic gradient in health refers to lower health status of groups located lower on the social ladder, defined by income, occupation or education. However, as suggested by Marmot (2005, 2001), the principal investigator of the Whitehall studies, these inequalities are not observable only between rich and poor, but persistent over the social hierarchy so that socioeconomic groups exhibit poorer health outcomes than the group above.
The two Whitehall studies on British civil servants revealed that each group had poorer health outcomes than the group immediately above on the social ladder. The first phase of the Whitehall studies, found there were higher mortality rates in men of lower employment grade specifically due to coronary heart disease, as well as increased mortality rates due to all causes for lower status men. Even after these standard risk factors were controlled for, the lowest grade still had a relative risk of 2.1 for cardiovascular disease mortality compared to the highest grade. By design, the Whitehall studies have been focused upon a single swath of related occupations, wherein relatively little heterogeneity exists within occupational grades, yet clear social distinctions between grades are inherent. The second phase, or Whitehall II, sought to examine the association between the psychosocial work environment and subsequent rates of absenteeism due to illness. Among the conclusions drawn from the second phase were that stress due to the psychosocial work environment factors predicts rates of sickness absenteeism, and that enhanced control of task management and support could have beneficial effects, such as increasing productivity and improving employee health and well-being.

C- Social Inequalities in Health

Despite generalized access to health services in rich countries (social security in France, National Health Service in Great-Britain and Medicaid in the USA), health inequalities persist across social groups, showing that their sources are not confined to medical sector. The differences also lie in behaviours and environments; the most disadvantaged people are more likely to engage in unhealthy lifestyles and are more exposed to health hazards such as pollution and unhealthy housing (Wagstaff, 2002). It is not always possible to understand the social rate of disease simply by studying individuals.

Social inequalities in health are inequalities in health outcomes related to the distribution of social determinants of health within a population. These social determinants are factors that make up the personal microenvironment such as family context during childhood, educational attainment, employment, working and living conditions, the quality of family and friendly relationships and participation to social life. The role that a determinant of health plays in health inequality depends on the one hand of its repartition across socio-economic groups and on the other hand on its impact on health. Thus most social inequalities in health are a mere reflection of inequality in social determinants of health. Nonetheless, these health inequalities can also be the coherent result of different behaviour choices, such as the practice of breastfeeding, alcohol and drug use and the practice of some dangerous hobbies. Sociologists increasingly believe that choices and trade-offs are underlined by inequalities and that for disadvantaged people, alternatives are not often possible. Furthermore, decisions of investments in health are biased since due to influences in early life, many adult cannot make well-lit decisions because of differences in education levels. Finally, survival probabilities are determined by contextual factors and other people's behaviour like those of passive smokers. In that view, even genetic differences, childhood living conditions and environmental exposition that are a priori random appear to be unjust. Benzeval et *al* (1995), decompose lifetime in three periods (childhood, adolescence and adulthood) and find that characteristics that appear in childhood influence health in adulthood through two mechanisms: first, the income potential, which is an accumulation of *capabilities* as described by Sen (1999), competences and education that determine future employment and ability to generate income and second health capital, which is the accumulation of health resources, be they physical, psychosocial, innate or acquired during earlier stages of life and that determine current and future health status.

Health inequality refers to a context of socioeconomic indicators, yet there are other important levels of inequality like gender and race. Moreover, it is increasingly admitted that some social groups with poor health outcomes are grouped in neighbourhoods and localities, thus creating a kind of health communautarism (Wagstaff, 2002).

In an attempt to eradicate social inequalities in health, Rose (1985) addresses the 'causes of the causes'; as the main determinants of health are socio-economic, remedies should also be found on socio-economic ground. On the international level, health inequalities are maintained by the speed of diffusion of the innovation between rich hand poor countries, which are determined by the administrative red tape that allows drugs importations, and the existence of patents for firms that produce these innovations. Interpretation of links between income inequality and health

must begin with the structural causes of inequalities, and not just focus on perceptions of that inequality.

Section III: Gender inequality, development and health

Gender inequality is probably the most 'equal inequality', as it cross-cuts the other dimensions of disadvantage in social relations (class, race, ethnicity). Like race, ethnicity, and class, gender is a social category that largely establishes one's life chances, shaping one's participation in society and in the economy.

According to Sen (2001), gender inequality is not one homogeneous phenomenon, but a collection of disparate and interlinked problems. He identifies seven dimensions of gender inequality: mortality (widely encountered in North Africa and in Asia, including China and South Asia), natality characterized by sex-selective abortion (in East Asia, China, South Korea, India and South Asia), basic facility (especially obvious in Afghanistan where girls were excluded from school, but also in Asia, Africa and Latin America), special opportunity (gender bias in higher education and professional training can be observed even in some of the richest countries in the world, in Europe and North America), professional (even in Japan progress to elevated levels of employment and occupation seems to be much more problematic for women than for men), ownership (homes and land property in many developing countries), household (division of labour within households, sharing the burden of housework and child care).

Before performing gender analysis, there is a need to avoid confusion in terms by making some concepts clear. Indeed, sex and gender, inequality and discrimination, are different notions, each carrying a specific meaning. They are however often used interchangeably, as if they were synonymous. In order to prevent confusion in these terms, we explain these notions in Box 1.2.

Box 1.2: A clarification of concepts

SEX/ GENDER

Sex refers to a biological distinction between men and women; a person's sex is biologically determined as female or male according to certain identifiable physical features which are fixed. However these biological differences cannot explain why women have less access to power and lower status than men. To understand and challenge the cultural value placed on someone's biological sex, and unequal power hierarchies, we need the relational concept of 'gender'.

Gender is a social construct that differentiates the power, roles, responsibilities, and obligations of women from that of men in society, it describes how a person's biology is culturally valued and interpreted into locally accepted ideas of what it is to be a woman or man. 'Gender' and the hierarchical power relations between women and men based on this are socially constructed, and not derived directly from biology. Gender identities and associated expectations of roles and responsibilities are therefore changeable between and within cultures.

Thus, people are born female or male but learn to be girls and boys who grow into women and men. This learned behaviour makes up gender identity and determines gender roles. To use the term gender, rather than sex, signals an awareness of the cultural and geographic specificity of gender identities, roles and relations. It also recognises gender inequality as the outcome of social processes, which can be challenged, rather than as a biological given. (*Baden and Reeves, 2000*)

DISCRIMINATION/ INEQUALITY

Gender discrimination is the systematic, unfavourable treatment or consideration of individuals which denies them rights, opportunities or resources on the basis of their sex. It means women's differential access to power and control of resources within households (in the sharing of resources), communities (sex selective abortion or female feticide), markets (unequal pay, occupational exclusion or segregation into low skill and low paid work for women), and states (lack of representation and voice in decision making bodies). Across the world, women are treated unequally and less value is placed on their lives because of their sex.

Gender inequality describes the situation in which males have greater choices, opportunities and resources than females. For example, enrolment rates are usually lower for girls than for boys in developing countries and females' share in labour force is lower than males' share in nearly every country. Gender inequality is then the result of sex differences combined with gender discrimination. Gender discrimination denotes the process and gender inequality the outcome

I- Gender inequality for specific health determinants

Throughout much of the world, families and societies treat girls and boys unequally. Girls face disproportionately privation, lack of opportunity and lower levels of investment in their health, nutrition and education. Women have fewer educational opportunities than men, receive unequal distribution of land and access to resources such as food and health care and take a usually have a lesser participation to public life. Thus, health disparities between men and women reflect a gradient in socioeconomic status and power (Amaratunga, 2000). Gender inequality and discrimination harm girls' and women's health directly and indirectly, throughout the life cycle; and neglect of their health needs prevents many women from taking a full part in society. Moreover, better access of women to these resources would allow to improve not only women' health, but also children's and the entire community's health.

A- Gender Inequality in education

Although considerable strides have been made over the past two decades in education, it is an area with significant disparities between men and women. Women still outnumber men by two to one among the world's illiterate people and girls constitute the majority of the more than 103.5 million children without access to primary school (UNDP, 2005). In some countries, there are only 60 young women in secondary school for every 100 young men. There are many factors that keep children out of school, but girls in particular face significant barriers. When money is scarce, parents tend to place greater value on educating their sons, who are assumed to be the future breadwinners of the family (Sen and Drèze, 1989). As the heads of the 189 states gathered at the UN Millennium summit acknowledged, education is one of the most efficient tools for health promotion. They also acknowledged that educated women are more likely to send their daughters to school, to care for their own health and that for their family and to have fewer children. Educated women will be better able to assume a more active role in social, economic and political decision-making throughout their lives. Besides being recipients of health care, women are also providers and promoters of health and their education impacts not only on their own health, but moreover on children and even on men's health.

Indeed as early as 1979, Caldwell showed that women's education is associated with a lower probability of child mortality, its impact transiting through various pathways: enhanced socio-economic status, greater health choice for children, immunization and proper nutrition, hygiene, emphasis on child quality in terms of fewer children, and capital investments. Moreover, this effect of women's education on children's health remained after controlling for a set of socioeconomic characteristics. Education can also influence health by delaying childbearing age.

Since educated mothers tend to begin childbearing later and to have smaller families, their children are less likely to suffer the excess-mortality risks associated with childbearing too early or too late (Hobcraft, 1993). More recently, Saleem and Bobak (2005) showed that education increases contraception use, that have been shown to be benefit both women's and children's health. Higher education levels among women are associated with better economic prospects, better reproductive health and higher awareness of the dangers posed by risky behaviours. By providing young women with greater economic options and autonomy, education also affords them the knowledge, skills, and opportunities they need to make informed choices about how to delay marriage and childbearing; have healthier babies; avoid commercial sex and other risky behaviours; and gain awareness of their rights.

B- Gender Inequality in income and participation to economic life

Despite recognition of women's subordinate situation in most aspects of public and private life and subsequent efforts to address the issue, gender inequality persists in most countries around the world. This inequality is most obvious in the distribution of income and wealth. Around the world, women now make up about 70 % of those who are poor (UNESCO, 2005). This 'feminization' of poverty is found in both rich countries[22] and in poor countries and reflects women's unequal situation in the labour market and their low status within the household. Women and men everywhere do not have equal access to the material and emotional resources needed to sustain health. In some parts of the world, food, income and medical care may not be distributed according to need, but to sex (Sen, 1988). In many societies, cultural norms dictate that males in the household have the principal share of income and wealth as well as higher status and greater decision-making power. And putting additional incomes in the hands of women within the household tends to have a larger positive impact than putting that income in the hands of men, as studies of Bangladesh, Brazil, and Côte d'Ivoire show (World Bank 2001). Unfortunately, rigid social norms about the appropriate gender division of labour and limited paid employment for women restrict women's ability to earn income. In nearly every country, women work longer hours than men, but are usually paid less and are more likely to live in poverty. Despite recent increases in women's educational attainment, women continue to earn less than men in the labour market, even when they have the same education and years of work experience as men. Women are often limited to certain occupations in developing countries and are largely excluded from management positions in the formal sector. In industrial countries women in the wage sector earn an average of 77 percent of what men earn; in developing countries, 73 percent but only about a fifth of the wage gap can be explained by gender

[22] For example, in 2000, 11% of all families in the United States lived in poverty, but 28% of families headed by single mothers did so (Dalaker 2001).

differences in education, work experience, or job characteristics (World Bank 2001). Due to these many forms of discrimination they confront, many women end up working in the informal sector, where work is unregulated, poorly paid, insecure and often unsafe.

In reference to women's economic activity, the measures commonly used (eg. gross domestic product, income per capita, labour force etc.) are very inadequate and inaccurate as reflections of reality. There is clearly an omission from standard economic measures of the vast amount of unpaid labour contributed by women in all economies. For example, in rural Africa, the unpaid work of women in gathering fuel wood, collecting water and processing food contributes immensely to the survival of households and to the well being of present and future workers, yet rarely are such activities accurately measured in the context of collecting standard national income data. Furthermore, the caring work that women undertake, the need for which has risen dramatically in the context of the HIV epidemic, is also generally omitted from economic data. By hindering the accumulation of human capital in the home and the labour market, and by systematically excluding women from access to resources, public services, or productive activities, gender discrimination diminishes an economy's capacity to grow and to raise living standards.

C- Gender Inequality in power relations and participation to public life

According to the State of World Population Report 2005 by the United Nations Population Fund (UNFPA), gender inequality hinders not only the wellbeing of affected individuals, but also the evolution of societies and the development of countries. According to the Report, greater equality in the power relations between women and men, combined with increased access to adequate reproductive health services, would save the lives of thousands of women. Women in most societies are expected to take the major responsibility for domestic tasks, and care of children, the elderly and the sick while men are allocated the responsibility for supporting the family. Gender disparities in rights constrain the sets of choices available to women in many aspects of life, often profoundly limiting their ability to participate in or benefit from development. Unequal rights and poor socio-economic status relative to men also limit their ability to influence decisions in their communities and at the national level. Women remain vastly underrepresented in national and local assemblies.

As well as material discrimination, women's lives are also affected by the cultural devaluation of femaleness that is a significant element of everyday thinking in many societies. Work which is done at home for instance, is deemed to be of less value than waged work and those who perform it are treated accordingly. Adolescents learn the social and gender norms that

prevail in their communities. These norms confront girls with special challenges, including restrictions on their independence and mobility, inequality in educational and employment opportunities, pressure to marry and start bearing children at an early age, and unequal power relations that limit their control over their sexual and reproductive lives

Men hold overwhelming power in decisions on sexual matters, including whether to use condoms. In many societies, women are expected to know little about such matters, and those who raise the issue of condom use risk charges of being unfaithful. Because of their lower social status and their economic dependence on men, women may be unable to negotiate the use of condoms as a STIs preventive measure. For many girls, marriage (and their sexual experience) starts when they are young, to husbands who are much older. These age differences between spouses reflect expectations about male earning capacity, female fecundity and a balance of power that favours men over women. These inequalities in influence and power within the household can also affect women's reproductive lives, constraining their ability to make fully informed choices about sexual practices or about fertility control.

II- Gender gap in health

There are concretely no societies in which women are treated as equals with men (World Bank 2001), and this inevitably affects both men's and women's health. Girls face disproportionately privation, lack of opportunity and lower levels of investment in their health, nutrition and education. Women have fewer educational opportunities than men and receive unequal distribution of land and access to resources such as food and health care. Women and men may also respond differently to treatment, have different access to health care and are treated differently by health providers. Thus, being male or being female has a major effect on an individual's health and well-being. Gender inequality and discrimination harm girls' and women's health directly and indirectly, throughout the life cycle; and neglect of their health needs prevents many women from taking a full part in society. Moreover, better access of women to these resources would allow to improve not only women' health, but also children's and the entire community's health. Gender characteristics, which are socially constructed, determine the capacity of both women and men to realise their potential for health.

A- Gender gap in health status

The health of both sexes is influenced by biological factors including, but not confined to their reproductive characteristics. Gender divisions shape the lives of both women and men in fundamental ways, health being one of these inequalities. Women receive inadequate medical care

in many societies, and they don't suffer the consequences alone. Healthy women are the foundation of healthy families, which foster healthy and prosperous societies.

Patterns of health and illness in women and men show marked differences. Most obviously, women tend to have longer life expectancy than men in the same socio-economic circumstances as themselves. Part of women's advantage in relation to life expectancy is biological in origin. Indeed far from being the weaker sex they seem to be more robust than men at all ages (Waldron, 1986). In all societies significantly more male foetuses are spontaneously aborted or stillborn and in most societies this pattern of excess male mortality continues to be marked during the first six months of life. In adult life too, women may have a biological advantage at least until menopause as endogenous hormones protect them from ischaemic heart disease. But even when women's potential for greater longevity is realised, this rarely results in them being healthier than men during their lifetime (Macintyre and *al*, 1996). We can identify at least three contributory factors why this is so. First, women's greater longevity is itself a cause of their higher rates of morbidity, as deteriorating health is a frequent part of the ageing process for both sexes and women make up the majority of elderly people in the world especially the octogenarians. Second, throughout their lifetime, both women and men are at risk from sex-specific diseases. For example, women are more likely to suffer from osteoporosis, diabetes, hypertension, arthritis and most immune disorders, and that biological factors are likely to play some part in this (US National Institutes of Health, 1992). Women will thus bear a heavier burden than men of reproductive health problems and this susceptibility is exacerbated during their childbearing years. Last, their capacity to conceive and bear children brings women into the arena of the health care system more often than men.

Although these difficulties take the form of biological disorders, social factors often play a major part in causing them, with gender discrimination in nutrition, heath care and social support[23]. Although 99 percent of maternal deaths during pregnancy and delivery are preventable, it has been estimated that 529,000 women died from complications of pregnancy and childbirth in 2000 and almost all occur in developing countries (WHO, 2002). In addition, for every woman who dies during pregnancy and delivery, approximately twenty more suffer serious harm. China, Korea, and South Asia have excessively high female mortality. Estimates indicated that there were at least 100 million (Sen, 1990) fewer women alive in 1990 than there would be in the absence of sex-selective abortion, one of the cruellest faces of gender discrimination. Violence against

[23] While women and girls, especially the poor, often bear the brunt of gender disparities, gender norms and stereotypes impose costs on males, too. As an example in the transition economies of Eastern Europe men have experienced absolute declines in life expectancies in recent years. These increases in male mortality rate -the largest registered in peacetime- are associated with growing stress and anxiety due to rapidly worsening unemployment.

women continues to be a curse worldwide, affecting all societies and all social strata. Its toll on women's health is greater than that of traffic accidents. Gender-based violence can take many forms and includes domestic violence, rape, female genital mutilation and 'honour' and dowry-related killings.

B- Gender gap in access to health services

Most medical research is based on the assumption that men and women are physiologically similar in all respects apart from their reproductive systems. Other biological differences are ignored as are the social differences which have a major impact on health. The consequence of this approach is the generation of biased knowledge. Also, common problems that cause considerable distress for women have received little attention if they are not central to their reproductive roles. Failure to provide a complete picture of women's health status stems in part from the fact that many developing countries lack a complete and accurate vital registration system. However, this is often compounded either by an official reluctance to recognise the importance of gender issues or by the complex social issues that surround so many women's health problems. Similar concerns can be raised about gender bias in access to medical care and in the quality of care received. The obstacles women face include lack of culturally appropriate care, inadequate resources, lack of transport, absence of alternative care for their families and sometimes the refusal of their husband to give permission. Of course limited public expenditure on health care will affect men as well as women but in conditions of scarcity it is often the females in the family whose needs are given the least priority. Households in certain regions of the world, spend less on health care for women and girls (Das Gupta 1987; Sen 1988). These financial constraints are reinforced in settings where customs and values deny women the right to travel alone or to be in the company of men outside their immediate family. In circumstances where female health workers are not available, treatment by a man may dishonour a woman and her family and she may need to go without care in order to avoid this. Gender differences in decision-making may also affect access to health facilities. While the price of treatment affects both sexes, women's unequal economic power as compared to men may make access to treatment particularly difficult for them. Low self esteem limits women's ability to make demands, and this may be reinforced by embarrassment if the problem is one that the community disapproves of.

If women do gain access to health care, there is also evidence that the quality of care women receive is inferior to that of men. These problems are reflected particularly in the context of reproductive services where dehumanising and insensitive treatment can affect women's willingness to return. Traditionally, women's health services have focused on their reproductive

needs, especially contraception and safe childbearing (Desjarlais et *al*, 1995). This has an obvious logic in the face of the huge toll of reproductive ill health that continues to affect some of the world's poorest women. However, it has also had serious limitations. First and most importantly, it means that millions of young women and those who are post-menopausal are denied access to any health care at all during periods of great need in their lives. Second, women of childbearing age find it not easy to obtain health care for non-reproductive problems (Paltiel, 1987).

Conclusion

This chapter focused on the analysis of the determinants of health. These determinants are so numerous that we focus on some that were studied as social determinants of health, individual characteristics and those related to health systems. Through their models, Grossman (1972) and Mosley and Chen (1984) proposed conceptual framework in which health determinants should be analyzed.

The relationship between income inequality and health is not as obvious as that linking income and health. Income inequality is likely to impact on health through access to material conditions, social cohesion and psychosocial factors. While the relationship between income and health is clearly established, the channels through which inequality affects health do not make the unanimity among the authors. In addition, they might seem irrelevant for poor countries, which still have to deal primarily with poverty as the most important factor of proliferation for infectious diseases, the major constituents of disease burden in these countries.

There are solid grounds for asserting that women's health is affected not only by the average income and education in the society, but also by the distribution of wealth and education across both sexes. Throughout the world and especially in Sub-Saharan Africa, socially constructed gender roles prevent women, even rich women from making the optimal decisions for their health. Thus, gender inequality and moreover gender discriminations have a detrimental effect on health, in addition to the impact of poverty. Concerning the impact of inequalities on health, the channels are completely different for income and gender inequalities. Gender's inequality detrimental impact on health transits through differentiated access to health services, gender gap in education and income and gender gap in power relations.

The following chapter is devoted to the study of the determinants of a specific affection, HIV/AIDS epidemic that is at the core of our analysis in this dissertation.

CHAPTER 2

THE DETERMINANTS OF HIV/AIDS EPIDEMIC

'The microbe is nothing, the terrain everything', ***Louis Pasteur*** *1822-1895.*

Introduction

The first reported cases of the disease subsequently named AIDS were discovered in Atlanta, Georgia (USA) in 1981, by Centre for Disease Control (CDC). Two years later, the virus responsible for that affection was detected by scientists at the Institut Pasteur, and named HIV. Since then, the virus has become one of the greatest killers of the planet and does not claim less than 33 millions of deaths in the world (UNAIDS 2006).

HIV is now a well-known disease. Rarely had a unique affection been devoted such an amount of information in newspapers, books, on the internet, dedicated conferences and received such an inflow of funds from international community. Nonetheless, we should admit that we know a lot and so few about the epidemic. It is true that a lot is known about the epidemic because of the amount of information that is released everyday through media. UNAIDS website provides a deep insights of HIV statistics pertaining to burden, new infections and deaths attributable to the virus. The

epidemiology of HIV/AIDS is also relatively well apprehended, since the main modes of transmission are well identified, as is also the epidemiological model. On the other hand, still little is known about the epidemic as comprehensive response fails to tackle the epidemic to date. Prevention programmes, which are mainly based on behaviour change, are still missing core groups by ignoring their specific needs. Despite tremendous progresses achieved, treatment provision is still short for 72 % of people who need it in developing countries and there is no announcement of a vaccine for the foreseeable future.

What sets HIV apart from many infectious diseases is its main mode of transmission, which is through sexual intercourse and also the reverse causality between the pandemic and its determinants. These determinants are numerous, complex, have many interactions among them. Socio-economic determinants shape the environment in which people live while socio-economic determinants influence their behaviour. Lastly, epidemiological determinants add to the picture by affecting the level of risk in sexual encounters. Although most studies devoted to the study of the determinants of HIV/AIDS epidemic belong to the fields of medicine, sociology or anthropology, five authors developed pertinent economic models, linking HIV/AIDS epidemic to its determinants at macro level.

The remainder of this chapter is organized as follows: in the first section, we briefly present the state of current knowledge of HIV/AIDS epidemic, in terms of burden, epidemiology and response addressed to the epidemic. The second section analyses the determinants of HIV/AIDS, classifying them into socio-economic, socio-cultural that add further to epidemiological determinants to make an unprecedented epidemic. The last section of this chapter deals with a presentation of the main studies that carried out an empirical analysis of the determinants of HIV/AIDS epidemic at macroeconomic level.

Section I: HIV/AIDS epidemic: What do we know?

HIV/AIDS epidemic is now a 'well-known' disease, as virtually everyone with access to broadcast or newspaper had heard about it. But what do we really know from this disease, in terms of what is the current level of the epidemic (new infections, deaths attributable to the virus, geographic distribution), its epidemiological model

transmission and the response that policy makers and international community oppose to the epidemic?

I- State of the art (AIDS epidemic update)

As of end of 2006, the total number of people living with HIV/AIDS virtually was 39.5 million, of which 37.2 million were adults and 2.3 million were children. Among these adults, 17.7 million, near half are women. The total of new infections occurred in 2006 is 4.3 million and 2.9 million people died in 2006 due to AIDS (UNAIDS 2007).

A- Burden

HIV pandemic is a combination of multiple epidemics that affects different populations at different levels in different regions. Thus, aggregated figures masks regional disparities, as the epidemic is now rampant in regions where it was only concentrated among high-risk groups. There is also evidence that it is slowing in some high-prevalence countries. The impact of HIV on women and girls is also growing; they represented 41% of infected population in 1997 and 48 % in 2006 (59 % for Sub-Saharan Africa).

Sub-Saharan Africa is the world's worst-affected region. Seventy percent of all people infected with HIV live in Sub-Saharan Africa, even though it is home to only 11% of the world's overall population. After Sub-Saharan Africa, the Caribbean is the next hardest-hit region as measured by HIV/AIDS prevalence. In Haiti, for example, the national adult HIV prevalence rate is estimated at around 5.5%. Eastern Europe is now experiencing the fastest-growing HIV/AIDS epidemic in the world, with 210,000 new infections in 2004 - bringing the total number of HIV-positive people in the region to approximately 1.4 million. In Asia and the Pacific, approximately 7 million people are living with HIV/AIDS; three countries in this region (Cambodia, Myanmar and Thailand) have HIV prevalence rates above 1% among 15 to 49 year old (UNAIDS, 2007).

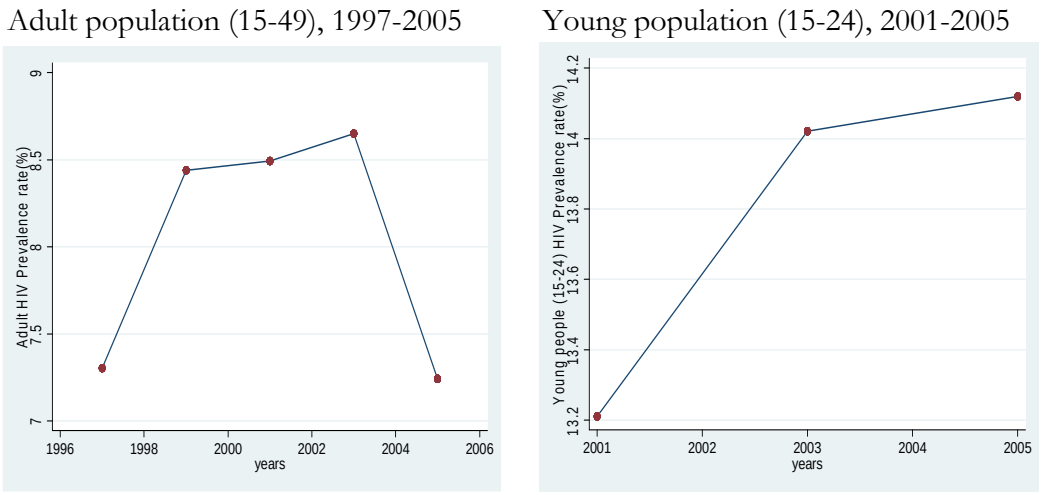
B- New infections

There were 4.3 million new HIV infections worldwide in 2006 with 2.8 million (65%) of these occurring in Sub-Saharan Africa. Significant increases are reported in Eastern Europe and Central Asia, where there are some indications that infection rates have risen by more than 50% since 2004. In North America and Western Europe, the

number of new infections has remained the same. In low and middle-income countries, there are only a few examples of countries that have actually reduced new infections. And some countries that had showed earlier successes in reducing new infections, such as Uganda, have either slowed or are now experiencing increasing infection rates (UNAIDS, 2007).

When looking at the prevalence since 2000, the epidemic seems to level off, since the number of infected people is constant around 40 million people. However, a look at the number of new infections tempers our enthusiasm, since the number of new infections is increasing and generalizing, especially in some parts of the world, where it was until now confined to high-risk groups. Figure 2.1 below demonstrates this paradox Sub-Saharan African countries for the period 1997- 2005.

Figure 2.1: Evolution of HIV prevalence rate in Sub-Saharan Africa



HIV/AIDS is a youth-driven epidemic, as most new infections occur among young people aged 15-24. The so-called 'next wave countries' are now experiencing generalized epidemic. Unfortunately, these countries are also the most populated of the planet (China, India, Russia, Nigeria, and Ethiopia). As a result, though prevalence rates in theses countries are still relatively low[24], there is a significant increase in the number of new infections.

[24] Except Nigeria and Ethiopia, with prevalence rates at 4% and 1.4 % respectively of adult population in 2005, prevalence rate in these 'new wave countries' does not currently exceed 1% of adult population. These figures are from UNAIDS for Nigeria and from DHS for Ethiopia.

C- Deaths

At the end of 2005, the amount of cumulative deaths attributable to HIV/AIDS amounted 25 million at the end of 2005, with 2.9 million deaths for 2005 alone. This figure can be decomposed as 2.6 million adults, and 380.000 children under 15 (UNAIDS, 2006).

AIDS-related mortality disproportionately affects Sub-Saharan Africa; in 2006, there were 24.7 million adults infected with the virus and 2.1 million deaths attributable to AIDS, which translates into almost one in twelve. In comparison, there were 65 000 AIDS-related deaths for 1.7 million infected adults (a factor of one in 26) in Latin America, 590 000 AIDS-related deaths for 7.8 million infected adults (a factor of one in 13) in South East Asia, and 18 000 AIDS-related deaths for 1.4 million infected adults (a factor of one in 77) in North America. But the gap in AIDS mortality between industrialized and developing regions is most striking for children, with 340 000 deaths for 2.1 million infected children infected with AIDS in Sub-Saharan Africa, a factor of one in six. This proportion is the same for Southeast Asia where 29 000 children died of AIDS-related complications while there are 180 000 other infected with HIV. In contrast, in Latin America, 2900 AIDS-related deaths occurred for 33 000 children infected with HIV (one in eleven) while there were 2300 deaths in central Asia and Eastern Europe for 10 000 children infected with HIV (one in four). This higher discrepancy in mortality due to AIDS among children highlights the importance of the issue about mother-to-child transmission of the virus, as well as pediatric antiretroviral treatment (ART).

Table 2.1: Regional HIV and AIDS statistics and features, end of 2007

	Adults & children living with HIV	**Adults & children newly infected with HIV**	**Adult prevalence (15-49) [%]**	**Adult & child deaths due to AIDS**
Sub-Saharan Africa	22.5 million [20.9 – 24.3 million]	1.7 million [1.4 – 2.4 million]	5.0% [4.6% – 5.5%]	1.6 million [1.5 – 2.0 million]
Middle East & North Africa	380 000 [270 000 – 500 000]	35 000 [16 000 – 65 000]	0.3% [0.2% – 0.4%]	25 000 [20 000 – 34 000]
South and South-East Asia	4 million [3.3 – 5.1 million]	340 000 [180 000 – 740 000]	0.3% [0.2% – 0.4%]	270 000 [230 000 – 380 000]
East Asia	800 000 [620 000 – 960 000]	92 000 [21 000 – 220 000]	0.1% [<0.2%]	32 000 [28 000 – 49 000]
Latin America	1.6 million [1.4 – 1.9 million	100 000 [47 000 – 220 000]	0.5% [0.4% – 0.6%]	58 000 [49 000 – 91 000]
Caribbean	230 000 [210 000 – 270 000]	17 000 [15 000 – 23 000]	1.0% [0.9% – 1.2%]	11 000 [9 800 – 18 000]
Eastern Europe & Central Asia	1.6 million [1.2 – 2.1 million]	150 000 [70 000 – 290 000]	0.9% [0.7% – 1.2%]	55 000 [42 000 – 88 000]
Western & Central Europe	760 000 [600 000 – 1.1 million]	31 000 [19 000 – 86 000]	0.3% [0.2% – 0.4%]	12 000 [<15 000]
North America	1.3 million [480 000 – 1.9 million]	46 000 [38 000 – 68 000]	0.6% [0.5% – 0.9%]	21 000 [18 000 – 31 000]
Oceania	75 000 [53 000 – 120 000]	14 000 [11 000 – 26 000]	0.4% [0.3% – 0.7%]	1200 [< 500– 2700]
TOTAL	33.2 million [30.6– 36.1million]	2.5 million [1.8 – 4.1 million]	0.8% [0.7% - 0.9%]	2.1 million [1.9 – 2.4 million]

Source: UNAIDS, 2007.

II- Epidemiology of HIV/AIDS epidemic

There are two types of HIV: HIV-1 and HIV-2. Both types are transmitted by sexual contact, through blood, and from mother to child, and appear to cause clinically indistinguishable AIDS. HIV transmission is determined by a number of factors including age of the epidemic, the state of immune system, male circumcision, prevalence of ulcerative sexually transmitted infections (STIs) and condom use.

A- Transmission modes

HIV can be transmitted through three main modes: infected blood (transfusion or needle sharing especially), unprotected sexual contact and from mother to child during pregnancy, delivery or breastfeeding[25]. Transmission through sexual contact accounts for 75 to 85 percent of the nearly total number of infections with HIV that have occurred so far, although the probability of infection through sexual contact appears to be lower than that of infection through other routes of exposure (Royce et *al*, 1997). The variability observed among and within routes of HIV exposure depends partly on the viral dose and also on whether the virus is transmitted directly into the blood or onto a mucous membrane. In addition, these differences are influenced by a variety of host factors. The greatest risk for transmission of HIV involves an HIV-contaminated blood transfusion, as seen in Table 2.2 below.

Table 2.2: Routes of Exposure and risk of infection with HIV

INFECTION ROUTE	RISK OF INFECTION
Sexual Transmission	
Female-to-male transmission	1/700 to 1/3,000
Male-to-female transmission	1/200 to 1/2,000
Male-to-male transmission	1/10 to 1/1,600
Fellatio	0 to 6%
Parenteral (With Infected Blood) Transmission	
Transfusion of infected blood	95/100
Needle sharing	1/150
Needle stick	1/200
Needle stick/AZT PEP	1/10,000
Transmission From Mother To Infant	
Without AZT treatment	1/4
With AZT treatment	Less than 1/10

Adapted from Royce, Sena, Cates and Cohen, NEJM 336, 1072 (1997)

[25] It is not possible to become infected with HIV through insect or animal bites, touching, hugging or shaking hands, eating food prepared by someone with HIV, toilet seats, coughing or sneezing, kissing, going to a public bath/pool, working or going to school with a person who is HIV infected, using telephones, sharing cups, glasses, plates, or other utensils (source: Center for Disease Control website).

B- Epidemiological factors

These factors are those that determine virus transmission during contacts between individuals. These are the age of the epidemic, the ability of the body to defend itself (through its immune system), the prevalence of sexually transmitted infections among population, the nutritional status, the practice of male circumcision and more importantly condom use.

There have been claims that HIV-2 might be less infectious than HIV-1[26] (Kanki et *al*, 1994), and that individuals infected with HIV-2 tend to survive for longer than those infected with HIV-1 (Whittle et *al*, 1994). Meanwhile, Gray et *al* (2001) conclude that the probability of HIV transmission per sex act in Uganda is comparable to that in other populations, suggesting that infectivity of HIV subtypes cannot explain the explosive epidemic in Africa[27]. Viral load, genital ulceration and age are the main determinants of the probability of transmission per coital act. Thus, there is no consistent evidence, of differences between these subtypes in terms of their infectiousness or pathogenic potential.

1) Age of the epidemic

An important feature of an epidemic with person-to-person transmission (exactly the case of HIV/AIDS epidemic among adult population) is that the probability that a contact will lead to a new infection is higher at its beginning (Chin and Lwanga 1991, Brookmeyer 1989). This is due to the fact that not many people are aware of the illness and do not use prevention measures; in addition as the epidemic spreads out, potential new infections are less because a big part of population is already infected.

2) Immune system

Greater attention to host factors helps to explain the extremely high rates of heterosexual HIV transmission in sub-Saharan Africa. Two factors that we find across sub-Saharan Africa that are known to undermine immune system response are malnutrition and parasite infection. Host susceptibility is considered a key to the emergence of the new and resurgent infectious diseases, of which HIV/AIDS is only the most notorious.

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[26] There are also differences between regions in terms of the strains of HIV that are common. The HIV-1 strain is most common, and is found in all parts of the world, while the HIV-2 strain is mainly confined to the countries of West Africa. Ten subtypes of HIV-1 group have also been identified, with substantially different geographical distributions.

[27] In their study, Gray et al found that HIV-1 was the most prevalent sub-type in Uganda.

Rates of perinatal transmission were found to vary greatly between rich and poor countries. In Europe, without intervention, 14 per cent of infants born to HIV-positive mothers are infected, and in the United States, 17 to 25 per cent are infected without intervention. In Africa, vertical transmission occurs in 25 to 40 per cent of births to mothers with HIV, indicating the importance of host factors (both maternal and child) for HIV transmission. In the United States, the average length of time from infection to illness is now more than a decade, and some people have lived with HIV for more than 15 years without becoming ill with AIDS-related diseases.

3) Nutritional status

There are interactions between HIV/AIDS and nutritional status at biological level as well as at the socio-economic level. The weakness of immune system is one of the direct consequences of malnutrition. In her comparison of pandemic's determinants in Sub-Saharan Africa and in Latin America, Stillwagon (2000) suggests that population's nutritional status could be the most important factor of the epidemic in Latin America. She considers that HIV/AIDS is an infectious disease before all and that its pandemic dimension should not make people forget that determinant variables for infectious diseases transmission are still relevant for HIV/AIDS transmission. Malnutrition and parasitical diseases are endemically spread in poor countries and it is well known that they weaken body's immune function. For instance, vitamin A is important for epithelial integrity, which plays a determinant role in STIs protection, especially those ulcerative that make virus transmission easier. Thus, they would then be determinant in the pandemic evolution. Nutritional status also acts as transmission canal between poverty and the infection; food insecurity leads to risky behaviours such as seasonal migrations or sex work, strategies people use to diversify their income. Louria (2003) considers the link between poverty and under-nutrition or malnutrition quite clear, as is also the nexus between under-nutrition and infectious diseases. Malnutrition influences incidence, prevalence, clinical appearance and clinical progression of a variety of infections. Thus deficient vitamin A among infants and small children results in a marked increase in prevalence of respiratory tract infections and diarrhoeal diseases.

4) Sexually Transmitted Infections (STIs) prevalence

Epidemiologists (Royce et al, 1997, Fleming and Wasserheit, 1999) establish STIs presence as the most important cofactor of the epidemic. Most STIs cause ulcers that in turn become real culture mediums for HIV. A study conducted by UNAIDS (2000)

in Mwanza (Tanzania) shows that the probability of virus transmission was higher when people were already infected with an ulcerative STI. In that study, the new infections rate decreased from 40% in the treatment-group and remained the same in the control group. On the contrary, a study conducted in Rakai, another Tanzanian city suggests that STIs' treatment does not affect the evolution of the epidemic. The explication of this paradox lies in the fact that the two cities had different infection rate before the intervention; it was 16 % in Rakai compared to 4 % in Mwanza. Thus, if the epidemic is just beginning, STIs presence can be an important factor of propagation of the infection while it depends more on behavioural factors when the epidemic is well established. Effective diagnosis and treatment of STIs has on the other side been shown to decrease HIV incidence by as much as 40 % (Philpott 2002).

Classical STDs likely play a central role in the HIV epidemic. Mucosal STIs (gonorrhea, Chlamydia, trichomonas) and genital ulcer pathogens affect both infectiousness and susceptibility. In this way, STIs might be particularly important in the early stages of a localized HIV epidemic, when people with risky sexual behaviour are most likely to become infected (Galvin and Cohen, 2004). STDs clearly facilitate HIV transmission because: i) STDs cause mucosal breaks and increase tissue inflammation and the number of receptive cells; ii) inflammation associated with STDs increase the concentration of HIV in genital secretions ; iii) STDs and HIV are frequently co-transmitted.

5) Male circumcision

Male circumcision, which is common to African religions and compulsory in Muslim religion, has a demonstrated impact on HIV transmission. This practice consists of the prepuce removal for young boys. There are two potential reasons justifying the negative relationship between circumcision and HIV/AIDS infection. First, the prepuce's presence exposes men to HIV/AIDS infection due to potential tearing during sexual intercourses; in addition it exposes them to STIs that are cofactors of the epidemic (Halperin et *al*, 1999). The ANRS 1265 randomized controlled trials in South Africa concluded to a reduction in the likelihood of female-to- male sexual HIV transmission by 60%, attributable only to male circumcision (Auvert *et al*, 2005). To put this in context, after many years of research there is still no sign of an HIV vaccine on the horizon with anything approaching the 50-60% efficacy rate of male circumcision. So the results are significant and raise important questions about how we best use

circumcision in the response to HIV. Further modelling suggests that increased male circumcision coverage in southern Africa could prevent as many as 2 million HIV infections over ten years (Williams et *al*, 2006) and a cost-effectiveness analysis by Kahn *et al* (2006) indicates that male circumcision could be cost-saving. However, the protection provided by male circumcision may be partially offset by increased HIV risk behaviour, or 'risk compensation[28]', especially reduction in condom use or increases in numbers of sex partners. Risk compensation may be especially important for MC because avoiding the sexual dissatisfactions of condom use and the desire to have more sex partners are likely to be significant motivations for men to seek circumcision (Kalichman *et al*, 2007). Indeed, circumcised men in the ANRS 1265 trial reported 18 % more sexual contacts at follow-up than did uncircumcised men, but no other sexual behaviour differences were obtained (Auvert *et al*, 2005). Risk compensation might be even higher in a non-research study, so it is necessary to adjust downward to reflect the increase in sexual risk behaviours among circumcised men when assessing the effectiveness of male circumcision. There is no evidence that circumcision increases or decreases the risk of HIV transmission by HIV-infected men. So, risk compensation by HIV-infected circumcised men could substantially increase the risk of transmission to their sex partners. This suggests that, in the short term at least, if not accompanied by appropriate prevention messages, circumcision would reduce the incidence of HIV among men, but increase the incidence among women, translating to greater risk to men. In addition, communicating the benefits of male circumcision entails to determine the acceptability of non-circumcising communities, to define the effect of male circumcision on women and to address the issues of safety and complications during interventions (Sawires *et al,* 2007*)*

6) Condom use

HIV/AIDS is transmitted through sperm, vaginal secretions and blood. Even in the absence of any ulcerative STI, a non-protected sexual intercourse can lead to virus transmission between individuals. Latex condoms provide protection against HIV transmission to both partners and reduce the impact of cofactors such as genital ulcers and other STDs. Thus, WHO recommends condom as the only contraceptive that also protects from HIV/AIDS when used efficiently. The Centre for Disease

[28] Risk compensation occurs when individuals adjust their behaviour in response to perceived changes in their vulnerability to a disease.

Control and Prevention (CDC, 1999) warns that only latex or polyurethane condoms provide a highly effective mechanical barrier to HIV. Moreover, for condoms to provide maximum protection, they must be used *consistently* (every time) and *correctly*. Pinkerton and Abramson (1997) go as far as assessing the effectiveness of inconsistent condom use at 79 % and that of consistent use at 90 to 95 %.

Despite the fact that people are well informed about the risks of HIV transmission and how to prevent it, many still engage in unprotected sex. Reasons for this are multiple and vary with the context. Campbell (2000), studying a cohort of Southern African mineworkers and sex workers concluded that the decision to use a condom during commercial sex encounters comes from men, who almost always prefer not using it. Sex workers lacked both economic and psychosocial power in relation to their male clients. In a study conducted in the township of Khutsong (South Africa), McPhail and Campbell (2000) identified six factors that hinder condom use among on adolescents; lack of perceived risk, peer norms, condom availability, adult attitudes to condoms and sex, gendered power relations and the economic context of adolescent sexuality. Cleland and Ali (2006), reviewing survey data, discovered that condom use is not always related to the severity of HIV epidemic. Most women in the survey report using condom primarily for pregnancy prevention.

C- Epidemiological model

HIV/AIDS is primarily an infectious disease. As such, its evolution depends on a reproductive rate. The reproductive rate is the number of secondary infections from each primary infection[29]. As the epidemic matures, the value of R should decline, as an important part of the population is already infected. The value of 1 is a threshold which defines an epidemic as becoming a pandemic or as disappearing. When R is greater than 1, the epidemic tend to spread because more secondary infections are occurring as results of the current primary infections. Conversely, when R is lower than 1, the epidemic tend to disappear and it remains stable when R equals to 1.

For an infectious disease, the reproductive rate, which determines the prevalence rate, is based on its epidemiological model. Over and Piot (1992) developed a model for STIs

[29] The underlying assumption behind the reproductive rate R is that the contact is between an infected person and an uninfected one.

that is also relevant for HIV/AIDS epidemic. Here, the reproductive rate is given by R[30], which value depends on three parameters.

$$R = a * Q * D \qquad (2.1)$$

a: the number of contacts per day

Q: the probability of infection for a single contact

D: the number of days a person remains infectious (duration of infectiosity).

In the ABC (Abstain, Being faithful and use Condoms) approach to prevent HIV, A and B are meant to influence the parameter a while C (and also circumcision) is directed towards the parameter Q. Education is of critical importance since it would influence C (and thus Q), (through behaviour change), but also D through rapid access to tests and treatment.

For Royce and Eron (1998), theoretically, the population reproductive rate of HIV is comprised of several reproductive rates representing each route of exposure to HIV (sexual, needle stick or use, mother-to-child, and transfusion) as well as population subgroups with distinct values for the parameters a, Q, and D, because different risk behaviours are associated with different degrees of risk.

Philipson (2000) and Kremer (1996) stress the role of behavioural changes on the course of the epidemic. For the former, the availability of a vaccine (or cure), coupled with a high prevalence, increase the demand for a vaccine, which lowers future prevalence that in turn lowers demand, resulting in an increase of susceptible persons, leading eventually to the regenerescence of the epidemic. As for Kremer (1996), whereas increased prevalence will lead low-activity people to reduce their sexual activity, it may lead high-activity people to become fatalistic, and thus either reduce their activity only slightly or actually increase it. In either case, the composition of the pool of available partners will worsen, creating a positive feedback. If this effect is strong enough, there may be multiple *equilibria* in sexual activity for given prevalence.

The epidemiological model described above links the future evolution of the epidemic to epidemiological factors (a, Q and D). In Box 2.1 below, we present the

[30] The parameter R is of different importance whether the stakeholder is an economist or a public health professional. For economists, reduce the value of R to less than 1 is a rationale for State intervention because when R >1, there are negative spillovers, leading to market failure, a straightforward case for externalities. For public health professionals, reduce R is the straightforward means to make the epidemic disappear.

relation linking the evolution of the epidemic to a broader set of determinants, which in turn determines the functional form relevant when analyzing prevalence rate.

Box 2.1: Functional form of HIV/AIDS prevalence rate

Let's assume p is the probability that an individual randomly drawn from the population is HIV positive (p is the prevalence rate), implying that 1- p is the proportion of the non-infected in the population.

Let $$p = \frac{1}{1+e^{-\mu}} \quad (1)$$

$$1-p = \frac{1}{e^{\mu}+1} \quad (2)$$

Assume that prevalence rate is linked to a vector of determinants X_i by the following function μ : $\mu = \alpha + \sum \beta_i (X_i)$ (3)

By multiplying p and p -1 by a fraction, which denominator and numerator are both equal to e^{μ}, thus leaving their value unchanged,

$$p = \frac{1}{1+e^{-\mu}} \cdot \frac{e^{\mu}}{e^{\mu}} = \frac{e^{\mu}}{e^{\mu}+1} \quad (4)$$

$$p-1 = \frac{e^{\mu}}{e^{\mu}+1} - \frac{e^{\mu}+1}{e^{\mu}+1} = \frac{-1}{e^{\mu}+1} \quad (5)$$

The odd-ratio is given by:

$$\frac{p}{1-p} = \frac{e^{\mu}/e^{\mu}+1}{1/e^{\mu}+1} = e^{\mu} \quad (6)$$

Defining $\log it(p)$ as:

$$\log it(p) = \log\left(\frac{p}{1-p}\right), \quad (7)$$

it follows that :

$$\log it(p) = \log(e^{\mu})$$

$$\log it(p) = \mu \quad (8)$$

$$\log it(p) = \alpha + \sum \beta_i (X_i) \ , \quad (9)$$

The functional form defined in equation (9) is the one we use for our econometric analysis in Chapter 4.

Source: Adapted from Mead Over's Lectures in the Masters of Health Economics in Developing and Transition Countries in CERDI, March 2007.

III- The comprehensive response to AIDS epidemic: Prevention, Treatment and Vaccine

Reaching the Millennium Development Goal on HIV/AIDS – to halt and reverse the spread of the epidemic by 2015– comprehensive response that includes greater access to HIV prevention services, treatment and vaccine than is currently available. The world (during the UNGASS) has committed to providing universal access to AIDS prevention, treatment, and care; but, unless the number of people who become infected is reduced, the costs of treatment and care will mount into tens of billions of dollars every year for the foreseeable future[31] (*IAVI 2005)*. Governments, organisations and individuals have promised large sums, and more funding than ever before is being directed at the fight against AIDS. As a matter of fact, the Global Fund to Fight AIDS, Tuberculosis & Malaria, conceived at the 2000 G8 summit in Genoa was established in January 2002 to dramatically increase global financing for interventions against the three pandemics, but mainly for HIV/AIDS. As of end of 2005, the Global Fund has committed US$ 7.7 billion in 136 countries to support aggressive interventions against all three diseases and disbursed 4 billion, with more than half (2.3 billion) devoted to HIV/AIDS alone[32].

Prevention and treatment are intertwined; provision of antiretroviral treatment (ART) is facilitated by prior prevention efforts. Thailand offers a telling example of how ART can prevent an exhaustion of health system because prevalence has been reduced earlier through systematic condom use among sex workers. It has good prospects to meeting its challenge to provide ART to all those in need and this ability rests on the success of its past prevention efforts. Indeed, in avoiding about 7.7 million HIV cases between 1991 and 1992, the country has realised substantial savings in terms of current ART needs (Over et al, 2006). On the other hand, ART is likely to influence HIV prevention through behaviour changes among infected as well as non-infected population. In Haiti, Farmer *et al* (2001) noticed that the demand for HIV testing and opportunity for counselling rose since antiretroviral therapy made available.

The overall impact on HIV incidence will further depend on the extent to which risk behaviours are affected by the availability of treatment. A pessimistic view will fear that access to treatment may produce a "dishinhibition effect" in both the seronegative and

[31] This annual cost does not even take into account the challenges associated with rising drug resistance and the associated need for ever-more expensive alternative drugs

[32] Source: Global Fund progress report, excel spreadsheets. Available at http://www.theglobalfund.org/

seropositive segments of the population and a consequent increase in HIV-related risky behaviours. Indeed, in developed countries, "disinhibition" effects have been reported by Del Romero et al (2001) in a cohort of gay men in Madrid. On the other hand, in order to facilitate their access to treatment, HIV-infected individuals may have more incentives to become aware of their serologic status and to share information about it with their partners and/or relatives. By giving a prospect of hope, longer survival and better quality of life, ART may also facilitate secondary prevention among infected individuals. Indeed, access to care has been associated with increased adoption of preventive behaviours among recently diagnosed HIV-infected patients in Tanzania (MacNeil et *al*, 1999). Also, Moatti et *al* (2003), comparing sexual behaviours of HIV-infected patients in Côte d'Ivoire whether or not they had access to antiretroviral treatment, concluded that access to ARV was not associated with an increase in HIV-related risky sexual behaviours .

Table 2.3 below highlights the possible effects of ART on prevention

Table 2.3: Possible Effects of Art on HIV prevention

Beneficial	Adverse
Encourages prevention, *especially* diagnostic testing. Therapy may increase participation in prevention activities, particularly Voluntary Counselling and Testing (VCT).	"*Disinhibits"* risk behaviours. People receiving ART and both HIV-positive and HIV-negative people may engage in more risky behaviours than they would if the therapy were not available.

Source: Adapted from Over *et al*, 2004.

A- Prevention

Across the world, a small number of countries have reduced HIV incidence (Uganda, Thailand, Botswana, Burundi, Côte d'Ivoire, Kenya, Malawi, Rwanda, Tanzania and Zimbabwe essentially) through sound prevention efforts. However, in 2005, there were still 4.2 million new HIV infections with over 40% of new adult infections occurring among young people aged 15-24. Although it reaches only one in ten of those most at risk - such as young people, women and girls, men who have sex with men, sex workers and their clients, injecting drug users, and ethnic and cultural minorities-, HIV

prevention works but needs to be focused and sustained (UNAIDS 2007). Components of successful prevention efforts include clear and accurate communication about HIV/AIDS and methods to prevent infection, HIV counselling and testing, and treatment of sexually transmitted infections. New data from UNAIDS[33] show that increased HIV prevention programmes that are focused and adapted to reach those most at risk of HIV infection are making inroads.

Positive trends in young people's sexual behaviours -increased use of condoms, delay of sexual debut, and fewer sexual partners- have taken place over the past decade in many countries with generalized epidemics. Declines in HIV prevalence among young people between 2000 and 2005 are evident in Botswana, Burundi, Côte d'Ivoire, Kenya, Malawi, Rwanda, Tanzania and Zimbabwe (WHO-UNAIDS 2006).

UNAIDS (2007) reports increasing evidence of HIV outbreaks among men who have sex with men in Cambodia, China, India, Nepal, Pakistan, Thailand and Viet Nam and Latin America whereas most AIDS programmes fail to address the specific needs of these people. Moreover, HIV prevention programmes are failing to address the overlap between injecting drug use and sex work within the epidemics of Latin America, Eastern Europe and particularly Asia. Furthermore, weak HIV surveillance in several regions including Latin America, the Caribbean, the Middle East, and North Africa often means that people at highest risk—men who have sex with men, sex workers, and injecting drug users—are not adequately reached through HIV prevention and treatment strategies because not enough is known about their particular situations and realities. In addition, levels of knowledge of safe sex and HIV remain low in many countries, as well as perception of personal risk; even in countries where the epidemic has a very high impact, such as Swaziland and South Africa, a large proportion of the population do not believe they are at risk of becoming infected.

"Know your epidemic" is now the new UNAIDS' paradigm in order to understand the drivers of the epidemic. Therefore, action must not only be increased dramatically, but must also be strategic, focused and sustainable to ensure that prevention services reach those who need it most.

[33] AIDS epidemic update 2006.

According to UNAIDS (2007), there were six new infections for each person on treatment in 2005[34]. Thus, the epidemic is still spreading and the straightforward implication of this fact is that governments will soon be overwhelmed if the provision of ART is not accompanied by an enhanced prevention. Over et *al* (2004) emphasize the importance of prevention in a context of ART provision, modelling the course of HIV/AIDS epidemic in India under different condom use scenarios. The difference between benefits and costs is always enormous, no matter the financing scenario for ART and the measure of benefits used[35].

B- Treatment

The first medicine use to treat HIV was Zidovudine discovered in 1987. In 1996, scientists discovered that its efficiency was limited, namely due the resistance reported in many patients after years of use. The combination of many drugs[36] proved to be more efficient in prolonging the life of HIV/AIDS patients and a significant breakthrough in the research was the implementation of the tri-therapy in 1998, which include three classes of molecules: 2 nucleoside analogues (NRTIs) and either a nonnucleoside reverse transcriptase inhibitor (NNRTI) or a protease inhibitor (PI). The tritherapies that associate two different classes of antiretrovirals (two inverse transcriptase nucleoside inhibitors and a non-nucleosidic inhibitor of this same viral enzyme), used as a basic treatment, are effective and well tolerated, as demonstrated by a clinical trial in Cameroon[37] (Laurent et *al*, 2004). While HAART does not offer a definitive solution, it represent real progress, giving hope to millions of AIDS victims across the world by extending their life expectancy and quality of life by several years[38] (Moatti et al, 2003).

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34 There were 4.2 million new infections and 700.000 new people on HIV treatment in 2005.

35 The two measures of benefits used in this study are discounted life-years saved relative to baseline and orphan-years saved relative to baseline. The three alternative antiretroviral therapy policies compared by the authors are: ADHERE (the National Antiretroviral Therapy Capacity Building) that yields a cost of $146 per life year saved, MTCT+ (the Government-Financed Antiretroviral Therapy for Mothers) with a cost-effectiveness ratio of $199 per life year saved and Below the Poverty Line (the Government-Financed Antiretroviral Therapy for the Very Poor) with a cost-effectiveness ratio of $280. These three policies are evaluated assuming that there is no change in condom use among population. When combining Below the Poverty Line policy with an increase in condom use (from 50 % baseline) to 70 and then 90 % use, the cost-effectiveness ratio plummets to $51 and $30 respectively.

36 Since 1996 at the 11th World Aids Conference in Vancouver, the multi-drug treatment regime known as HAART (Highly Active Anti-Retroviral Therapy) has proven extremely effective in allowing people with AIDS to battle opportunistic infections and lead close-to-normal lives.

37 The clinical trial was conducted in Cameroon by IRD (Institut de Recherche pour le Développment) researchers working with other organizations and supported by the ANRS.

38 Some problems with HAART have become apparent, including the failure of patients to comply with a very complex treatment regime and metabolic imbalances caused by the "protease inhibitors," a key element of the treatment.

Although the success of Highly Active Antiretroviral Therapies (HAART) in reducing HIV-related mortality and morbidity became evident as early as 1996, access to Anti-Retroviral Treatment (ART) was not considered a feasible technical and economic option for developing countries by most experts in the field. Pilot projects sponsored by the governments of Côte d'Ivoire, Senegal and Uganda with the support of UN organisations have proved the technical feasibility of Antiretroviral (ARV) delivery in sub-Saharan Africa. These experiences have established that similar virological and immunological outcomes, probability of an adverse event, and estimated survival, levels of patients' adherence, while maintaining limited viral resistance, have been obtained with patients enrolled in these African Drug Access Initiatives (DAIs) than with ART treated patients in the USA and Europe. Although it is obvious that inequality between rich and poor nations regarding access to HIV care and treatment constitutes a "moral scandal", many experts and policy-makers argued that improved access to ART was not a good investment choice in developing countries[39].

In 2000, treatment with HAART costed around US$15,000 a year and was not affordable in much of the developing world and therefore to the vast majority of people with AIDS. To improve access to care in the hardest-hit regions of the world, UNAIDS launched the Accelerating Access initiative, which consisted of assisting countries in implementing comprehensive packages of care for their citizens living with HIV/AIDS. The Accelerating Access initiative was followed by the "3 by 5" initiative, launched by UNAIDS and WHO in 2003. This initiative was a global target to provide three million people living with HIV/AIDS in low- and middle-income countries with ART by the end of 2005. As a result, the cost of ART in some developing countries has fallen from US$15,000 per year per patient to less than US$150 per year[40] and the prospects for treating people infected with HIV with antiretroviral drugs in low and middle-income countries have improved. This lower price brings it within reach of many middle-income countries, but ART continues to be a costly and complex challenge for low-income countries, where annual public health expenditures are often less than US$20 per person per year. In addition, the cost of medicines is only a part of

[39] Refer to Moatti et al (2003), pp 248-249 for a detailed discussion on why some economists considered ARV delivery option as a heresy in the South.

[40] Price reduction in the South was accelerated due to two effects: Brazilian and Indian generics production and the launch of the AAI programme.

the cost of the treatment; there are also costs associated with laboratory, especially CD4 count and viral load.

In 2003, fewer than 300,000 people in the developing world were receiving the anti-retroviral drugs that help treat the virus. ART coverage is 28% in the developing world and 2.2 million people in developing countries received the drugs in 2006. However, this aggregate number masks huge regional disparities: coverage is 75% in Latin America, 5% in the Middle-East and 28% in SSA. This is of course the result of tremendous efforts from international community, since the coverage was less than 1% in 2000 for SSA.

C- Vaccine

Although prevention and treatment programs are expanding rapidly in many countries, they are unlikely to reverse the rising number of new infections, and they will not end the pandemic on their own. For this, a vaccine is a critical tool in the effort to control the pandemic. The benefits to families and societies, measured in terms of suffering, economic loss and social disruption avoided, would be enormous. To date, there is still no sign of a cure for AIDS or for infection with HIV, and no expectation of an effective vaccine against AIDS becoming available in the immediate future, although the first human vaccine trials commenced in 1987. Nonetheless, many initiatives have come into being throughout the world, all devoted to an AIDS vaccine development. To name few, there are the International AIDS Vaccine Initiative (IAVI), South African AIDS Vaccine Initiative (SAAVI), Kenyan AIDS Vaccine Initiative (KAVI), WHO-UNAIDS HIV Vaccine Initiative and African AIDS Vaccine Programme. An annual conference, where researchers can share their advances, is even devoted to AIDS Vaccine.

Developing a vaccine against HIV is a very difficult challenge for scientists. Reasons for this include: *(i)* the unavailability of a natural mechanism to imitate, as nobody has ever recovered from HIV infection, *(ii)* the destruction of the immune system cells that are meant to fight against HIV, *(iii)* the insertion of genetic material into human cells by HIV soon after infection, where it remains hidden from the immune system, *(iv)* the existence of several subtypes, each of which is very different from the others, and *(v)* the absence good animal models to use in experiments (IAVI, 2006). Nonetheless, there are reasons to be optimistic about the search for an AIDS vaccine, despite the slow progress so far. Indeed, historically vaccines took many

decades to develop, whereas HIV was only discovered in the mid 1980s. It is therefore much too early to give up hope, especially given the current speed of scientific progress. One particular reason for remaining hopeful is that most people remain healthy for several years after becoming infected with HIV, and a small minority have survived as long as 20 years without developing AIDS, even though they never entirely rid themselves of the virus. Also it appears that a few people have some kind of natural resistance to HIV infection, meaning they never become infected despite repeated exposure to the virus. Presently, research on AIDS vaccines is ongoing, with over $600 million spent each year and more than 30 vaccine candidates currently being tested.

IAVI (2006) estimates that the potential positive impact[41] of an AIDS vaccine would be enormous. Even in a relatively conservative scenario, an effective preventive HIV vaccine could prevent almost 30 million of the 150 million new infections projected in the coming decades. A highly effective vaccine could even prevent over 70 million infections in fifteen years. The results suggest that even under very conservative assumptions, the impact of a vaccine would be considerable, lowering the number of people infected annually by more than 30%. However, it should be noted that complete eradication of the AIDS epidemic using vaccines alone is unlikely. Moreover, availability of a vaccine could lead to riskier behaviour, erasing some of the gains from vaccination. As a result, vaccines must be accompanied by other prevention efforts.

As might be expected, the largest impact would be in the developing country regions with the highest current burden of illness and deaths from AIDS: Sub-Saharan Africa, South and Southeast Asia. In the medium scenario, a vaccine could prevent as many as 28 million infections – two million each year – in sub-Saharan Africa. The impact of a

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[41] The three scenarios were combined with long-term projections from UNAIDS on the expected trajectory of the AIDS pandemic over the next twenty-five years, assuming that existing prevention and treatment programs continue to grow until 2012, then plateau at 80% coverage. The simulation also assumes that an AIDS vaccine is introduced in 2015. The *Base Scenario* is the situation with no vaccine. The number of people infected would rise to 10.2 million a year by 2030. Between 2015 and 2030, 150 million people would be infected. In the *Low Scenario,* a 40% effective AIDS vaccine provided to 20% of the population would reduce the number of people infected annually by 32%, to seven million by 2030. It would prevent 29 million new infections or 19% of people who would otherwise be infected from 2015 to 2030. In the *Medium Scenario,* an AIDS vaccine with 60% efficacy provided to 30% of the population would reduce the annual number of new infections by 54% to 4.7 million by 2030. It would prevent 47 million new infections or 31% of those people who would otherwise be infected from 2015 to 2030. In the *High Scenario,* an AIDS vaccine with 95% efficacy provided to 40% of the population could substantially affect the course of the pandemic. It would reduce the annual number of new infections by 82% to 1.8 million by 2030. It would prevent 71 million new infections, 47%, over 15 years

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vaccine in South and Southeast Asia would also be substantial – 7 to 19 million HIV infections avoided, depending on the scenario.

Section II: Macroeconomic determinants of HIV/AIDS epidemic

To date, HIV/AIDS has reached the proportions of a pandemic. While factors determining the transmission of virus from one person to another are purely individual, what makes this disease an epidemic is due to factors much more complex and global. Determinants of HIV/AIDS transmission are those factors (biological, behavioural, cultural, political and socio-economic) that put people at risk of infection with the virus. Factors such as unsafe sex, injecting drug use and transfusion with contaminated blood pose a direct threat of infection to the individuals concerned. Others such as poverty, lack of knowledge, gender inequality and conflict create an environment that is conducive to the spread of HIV and renders groups and even whole societies vulnerable to the virus. Thus, a number of biological co-factors interact with social and political variables to increase the transmission of HIV. While each of these factors increases individual susceptibility to HIV infection, together they also describe epidemic patterns within populations, since these biological co-factors are at least in part a result of locally dominant cultural, social, and political relations. A number of social conditions have been identified that affect a population's vulnerability to HIV. These include poverty, social inequality, state capacity, migration, and the nature of sexuality and gender relations. According to Louria (2003), emerging and remerging infections are caused by massive urbanization; wars; poverty; malnutrition; forced migration; human behaviour (particularly sexual behaviour); massive irrigation projects and dam construction; extensive travel; and planetary population aging. This author stresses that these determinants which are closely inter-related, are virtually all modifiable by individual actions, by small but important changes in our educational system, and by political actions.

Sexual transmission of the virus, which its main mode of transmission, implies a critical role of behaviour in the spread of the epidemic. Coupled with a specific environment, risky behaviour is likely to generate a pandemic, which actually occurred in Sub-Saharan Africa. The most common classification of determinants distinguishes what is attributable to socio-economic environment, to behaviour induced by socio-economic

variables and to the nearest context of sexual encounters, made off epidemiological variables.

I- Behaviour *versus* environment

Many studies have been conducted to explain the difference of in the spread of HIV/AIDS pandemic in different parts of the world. They can be classified into those in favour of behaviour and those giving importance to environment.

A- The importance of behaviour

The critical role of behaviour is the main lesson drawn from the success of Uganda in containing its HIV/AIDS pandemic (UNAIDS 1998, 1999). Many qualitative and quantitative studies were done at a small scale on the evolution of behaviour in Uganda. Although they do not allow making direct comparisons in time, they led to the conclusion that attitudes and behaviours had changed notably in Uganda. More than three hundred documents were examined by UNAIDS and allow to note a link between a decrease in the number of new infections and changes observed in condom use, age at the onset of sexual activity and sexual intercourses with multiple and non regular partners.

In the developed world, the pandemic had first appeared and was confined to homosexual and injection drugs users' populations and this is precisely why prevention policies were exclusively based on a safe behaviour. This orientation was maintained in HIV/AIDS prevention policies in sub-Saharan Africa, which until recently consisted of the 'ABC' approach consisting in altering behaviour by practicing Abstinence; Being faithful and use Condoms.

Since the success recorded by the antiretroviral drugs combinations in terms of increased life expectancy and quality, incidence rates are rising in some segments of population in developed countries, showing the occurrence of a riskier behaviour. There is some disturbing evidence of an increased incidence of STDs and high-risk behaviours as ART has become widely available (Do et *al* 2001, Dodds et *al* 2000). This lack of prudence is due to the fact that people interpret the amelioration in infected people's health as a definite cure. In contrast, an optimistic view refers to the evidence from cohort studies in the North indicating that individuals receiving HAART tend to

adopt protective behaviours more frequently than those who are not on treatment (Bouhnik *et al*, 2002).

In developing countries, Jha *et al* (2001) illustrates the effects of announcements of "AIDS cures" (Kemron and Pearl Omega) on condom use among sex workers; in Kenya where it occurred, it took at least one year after each announcement to recover initial levels of condom use. In the contrary, a survey conducted in Abidjan (Côte d'Ivoire) in a sample of HIV-infected patients seeking care in the medical centres revealed that those who had access to ART were more likely to maintain sexual activity, in association with the improvement of their health status, but declared significantly more frequent condom use than non ART-treated individuals (Moatti and Kazatchkine, 2001). Similar studies conducted in Rio de Janeiro and Chile found that adherence to condom use also increased in the ART-treated population (Barroso et *al* 2003, Sgombich et *al* 2002).

B- The importance of environment

UNAIDS (1999) tried to explain the difference in the speed of spread of the pandemic in Central and Occidental Africa (where prevalence rates were relatively low) and in Austral and Oriental Africa (where they were quite high). It comes out that differences in behaviour alone do not explain the difference in prevalence rates. Behaviour differences in this case are more than compensated by differences in risk of transmission of the virus. So, even though median age to first sexual intercourse was lower in austral and Oriental Africa and age difference between sexual partners is higher there, partner change and contacts with sex workers were more frequent in Central and Occidental Africa. Condom use during occasional sex was low on each site. Except for chlamydia, STI's infections were more frequent on Kisumu (Kenya) and Ndola (Zambia) sites than on Cotonou (Benin) and Yaounde (Cameroon) sites. Moreover, in Cotonou and Yaounde, almost all men declared to be circumcised against 10% only in Ndola and 30% in Kisumu[42].

Stillwagon (2000) warns about the "exceptionalization" of HIV/AIDS pandemic in Africa. According to her, priority must be given to sanitary and economic conditions when studying the determinants of the pandemic. So poverty, malnutrition, lack of

[42] Male circumcision in Sub-Saharan Africa is more a cultural that a religious practice. In that way, circumcision is widely practised in countries like Cameroon and Benin, and the percent of male population which is circumcised is much higher that the percent of the population which is Muslim. In general, male circumcision is less usual in Southern and Eastern Africa than it is in central and Western Africa (Caldwell and Caldwell, 1996).

hygiene and care for STIs are of highest importance, especially since these characteristics are also present in other regions in the world that are facing an emergent HIV/AIDS pandemic. As she put it: "AIDS flourishes where people are dying of other diseases"… That is not a mere coincidence…. Conditions were ripe for an AIDS epidemic in SSA"[43].

Bonnel (2000) points out the politico-legal climate as one of the catalysts of the pandemic; which is also a variable on which HIV has a negative impact.

In virtually all countries with a generalized epidemic, the spread of HIV/AIDS is associated with (i) high rates of poverty and consequently poor living conditions, (ii) gender inequality, (iii) incomplete epidemiological transition with high prevalence of communicable diseases, (iv) low levels of knowledge as a result of which population will be less than fully aware about methods of reducing risk of HIV infection and (v) migrant and displaced people (UNDP, 2003).

The most common classification of determinants distinguish what is attributable to socio-economic environment, to behaviour induced by socio-economic variables and to the nearest context of sexual encounters, which represent epidemiological variables.

II- Socio-economic determinants

The most recurrent in literature (Bonnel 2000, Nattrass 2006, Over 1997) are income and gender inequalities[44]. Other socio-economic determinants are poverty, national income, development, internal and external mobility, education level, governance, access to health services and access to information.

A- Poverty

There is a clear relationship between poverty and the spread of communicable diseases and HIV/AIDS is not an exception. Poor households typically have few financial or other assets and are often politically and socially marginalised. These conditions of social exclusion increase the problems of reaching these populations through programmes aimed at changing sexual and other behaviours (Cohen 1998). Thus they tend to lack adequate information. Poverty also impacts on HIV transmission through less access to health services. Moreover, as noted by Stillwaggon (2002), people are also

[43] AIDS and the ecology of poverty, 2005 (October 6th) page 10.
[44] Income and gender inequalities will not be studied in this section. Instead, they will be at the core of the analysis in the next chapter.

biologically more vulnerable to HIV infection when they are malnourished and/or experience parasitic infections (that weakens immune system), two features that are more prevalent among poor people. Finally, when poor people are aware of the infection, they are not able to protect themselves and are more likely than others to engage in risky behaviours such as migrant work and commercial sex work. In Cambodia, the country among those with the most advanced epidemics in Asia, Bloom et *al* (2001) demonstrated that the poorest segments of society have much less knowledge of how AIDS is transmitted and prevented; are more likely to have sex at a younger age; use condoms less frequently; and, in the case of young women, are more likely to turn to sex work as a means of supporting themselves and their families.

However, when people are quite destitute, their social isolation has also tended to protect them from aspects of development that favour HIV epidemic. These are transport infrastructure that increase mobility, access to leisure activities, some of which increase risky behaviour.

A noteworthy fact is that the relationship between poverty and HIV infection is growing stronger over time, both between and within continents, as evidenced by the Commission on Macroeconomics and Health (2002).

B- National income

Fist HIV/AIDS cases have been discovered in developed countries, but the epidemic spread the fastest in poor countries. The main reason for this discrepancy is that developed countries could implement prevention measures and care for sick people, thus decreasing their infectivity and the number of new infections. When examined continent, therefore, HIV appears to be either affecting the rich more than the poor (as in Africa) or is income neutral as found everywhere else (Commission on Macroeconomics and Health, 2002). Between continents, however, the picture looks different. Ninety-five per cent of those infected with HIV live in less-developed countries, home to 80 % of the world's population. This negative association between income and HIV prevalence at national level reflects the role that better infrastructure, access to health services and nutrition seem to play in the spread of the disease. In addition, in a resource-constrained setting, there could be certain reluctance of governments to confront AIDS among other priorities (Ainsworth and Teokul, 1997).

C- Development

Although poverty reduction might be thought to reduce HIV/AIDS rates, in some cases the development process may itself strengthen epidemics. Development is associated with infrastructure development, urbanization, increases in disposable income, the growing importance of cash in agriculture, and growing mobility. Furthermore, inequality often grows in the early stages of development (Deaton and Lubotsky, 2001), creating increased internal migration. Workers migrate to centres of wealth and employment, which is a significant risk factor: men travel away to work, but occasionally return to their families in their village of origin. Development is thus likely to bring greater opportunities for multiple partnering and a growth in the commercial sex industry. Finally, inequality can create changes in gender relations that may facilitate the spread of sexually transmitted diseases (Farmer, 1999).

D- Internal and external mobility

Mobility significantly increases HIV-related risk as migrant workers move from generally low-prevalence rural regions to urban centres, where HIV prevalence is much higher and risk behaviours are more frequent. As urbanization increases, infrastructures in cities need to welcome a much more important population. Unhealthy environment (accommodation, lack of access to safe water and poor hygiene) coupled with an overwhelmed health system can do little to prevent the spread of infectious diseases. Furthermore, rural exodus induces the same behaviour for young people going to big cities as migrant workers. In addition, they can engage in commercial sex work as a survival strategy if they are unemployed for long time.

Migrant workers form an important part of labour force in poor countries. Industrial and mining projects are almost located in remote areas where workers cannot see their regular partners for long time. Commercial sex develops in these areas for boredom, loneliness and economical reasons. Generally, neither sex workers, nor their clients are aware of their serologic status. Then both incoming population (workers will infect their regular partners once back at home) and welcoming population (sex workers will infect their occasional and regular partners when migrant workers will leave the area) are affected by the epidemic (Cohen, 1998).

HIV and migration do not have a linear, cause and effect relationship, but are laterally linked. HIV is a manifestation of the inequalities and deprivation faced by migrants.

Hostile and lonely environments, separation from families, lack of access to information and services and social support systems can lead to social and sexual practices that make them more susceptible to HIV exposure. However, it may be noted that migration in itself is not a vulnerability factor for HIV, but it is the unsafe process of migration that creates conditions of vulnerability (UNDP, 2006)

E- Education

One key determinant of the infectious diseases is education. At the onset of epidemic in Sub-Saharan Africa, the virus affected educated and non-educated people in an indifferent manner. People's education can be a factor that exposes them more to virus infection, as it goes hand in hand with higher socio-professional category. Indeed, those people from high socio-professional categories (especially men) travel more than others for work and are more likely to support a 'second office[45]', given their higher income. Indeed, Over (1997) found a positive socioeconomic gradient[46] in the HIV/AIDS epidemic, as also did Lachaud (2007), using data from Burkina-Faso. However, as the epidemic spread out educated people are expected to be less affected than the rest of population. This can be due to many reasons; first, educated people are better informed about prevention measures. Like access to health care, access to education is a mean of being informed on prevention methods, especially if educative programs include sexual education. Public awareness is widely done on the radio and through posters. The fact that a large part of population can read and speak official language(s) makes it sheltered from misinterpretations due to translation errors. Next, since infected educated people are no more productive at the onset of sickness and will not be able to perceive an income, education increases the opportunity cost of being infected. Last**,** education impacts on HIV/AIDS prevalence by delaying age at the onset of sexual activity, especially in regions where girls are sent into marriage as soon as they abandon schooling.

De Walque (2004) showed that the sign of the correlation between socio-economic status and HIV infection changes over time. Parker (1997) concluded that three-quarters of people newly diagnosed with HIV in the early 1980s in Brazil had a

[45] The 'second office' is a term used in Central Africa in reference to extra-marital affairs because unfaithful men usually pretend that they were working late when confronted by their wife.

[46] The socioeconomic gradient as defined by Over is the bias in the distribution of the epidemic among individuals according to their socioeconomic status. Thus, the pandemic is said to be upward if the epidemic affect more people in higher social strata and downward biased inversely. It is equally distributed if the population is affected uniformly.

secondary or university education, but by the early 1990s this share had fallen to one-third.

F- Governance

The influence of this variable on the epidemic necessary goes through the priority government gives to HIV/AIDS as a real public health issue. For most African countries, disastrous economic situation is caused more by bad governance than by international adverse conditions. In a corrupted political environment, decision-makers can be not incited to invest enough in HIV/AIDS prevention activities. In that way, good governance appears as a factor of the epidemic's slowing down. For Bonnel (2000), the politico-legal climate, which determines health expenditure, is among the catalysts of the epidemic.

Governance can also influence HIV/AIDS epidemic through the implementation of national programmes for the fight against HIV/AIDS (which plays a major role in countries like Cameroon) or the role of institutions (like in Uganda).

G- Access to health services

The main route through which access health services impacts on HIV/AIDS epidemic is the non-treatment of STIs. According to Lurie (1995), structural adjustment programs are to be blamed for creating conditions favourable to the epidemic by deepening poverty and cutting back health services at a time when the continent was already vulnerable to other infectious diseases. Hence, these policies dictated on African governments, have decreased access to curative health services. Fragile health services were in turn not able to implement HIV/AIDS preventive measures (especially prevention of mother-to-child-transmission), adequate treatment for STIs and treatment for opportunistic infections that could reduce infectivity in HIV positive people.

Today, a great deal of money is devoted to the fight against HIV/AIDS epidemic. Most of these funds are not even integrated in ministries of health's budgets and their amount usually exceeds the amount devoted to the fight of any other single disease. However, this situation is quite recent. This has not always been the case and health sector was among the vehicles through which the epidemic passed to reach vulnerable populations. This situation is explained is Box 2.2 below.

Box 2.2: Health sector crisis and HIV/AIDS epidemic in Sub-Saharan Africa

Today, a great deal of money is devoted to the fight against HIV/AIDS epidemic. Most of these funds are not even integrated in ministries of health's budgets and their amount usually exceeds the amount devoted to the fight of any other single disease. However, this situation is quite recent. This has not always been the case and health sector was among the vehicles through which the epidemic passed to reach vulnerable populations.

From the mid-80s, health sectors in most African countries experienced a long and deep crisis, translating into the degradation of health outcomes which where improving from their independence level. The causes of this crisis are multiple; they can be attributed to Bamako initiative as well as to structural adjustment or more simply to overall economic crisis prevailing in the continent at that moment and which was the justification of structural adjustment. Authors are not unanimous about the effects of structural adjustment on health indicators in Africa. Jones (1998) found no difference between adjusting and non-adjusting countries, neither in the change of government expenditure, nor in the share of health sector in government expenditure in these two sets of countries. For Sahn (1992), the effects of reforms are confused with effects of economic crisis which were at the origin of structural adjustment. Finally, Samba (2004) and Lurie (1995) believe that measures imposed by Bretton Woods on African economies are the cause of health sector crisis in Africa. Whatever the actual causes of the crisis, debt burdens favours HIV/AIDS epidemic in two ways. First, governments had to observe strict restrictions on their spending, including social expenditure such as health and education expenditure. Second, those export revenues used to reimburse debt were no more available for imports such as drugs and medical equipment, critical ingredients for the functioning of health system. Thus, efforts African countries made to honour their engagements resulted in reduced health expenditure, countries spending on average three times for debt service what they spent on basic services for the population. This has led to an increase in the contribution of an already impoverished population. Yet the most tragic consequence of health sector crisis in Sub-Saharan Africa was felt on the quality of health services. Audibert and Mathonnat (2000) pointed out that the quality anticipated by the users of health care is a key element, because it has an influence on attendance rates at health establishments. For Olivier de

Sardan (2004), "Africans… want it (modern medicine) to be dispensed efficiently and with kindness". The worsening of living conditions as a result of budgetary constraints and the fall of wages under efficiency wages led to lack of motivation, preventing demoralised doctors often cannot provide their patients with a decent service. In addition, it became usual for health personnel to claim money from the patients to perform the work they were supposed to be paid for.

Health sector crisis in Sub-Saharan African countries did also favor the epidemic in a more direct manner: (1) mother-to-child-transmission could not be reduced in Sub-Saharan Africa as successfully as it was in other parts of the world; while some combinations of medicines (for example a combination of zidovudine and lamivudine) or single medicines (nevirapine) which are proved to be efficient in reducing vertical transmission were available and subsidized in these countries, infrastructures problems and poor quality of staff training rendered the implementation of these interventions impossible,(2) health workers are directly exposed to the risk of contamination due to direct contact with blood and patients secretions. Post-exposure prophylaxis measures that can reduce the risk of infection in medical field were not implemented early enough at the onset of the epidemic, (3) health services did not automatically treat patients for STIs and opportunistic infections, thus favouring the dissemination of the virus. These two kinds of infections are associated with higher rates of transmission, the former as a catalyst and the latter as an enemy of immune system that reinforces infectivity of sick people and accelerates the onset of full-blown AIDS, thus increasing fear and stigma associated with the disease and (4) Voluntary Counseling and Testing services, an important component of the fight against HIV/AIDS were not always available and when they did, the conditions required for them to be efficient were not fully fulfilled (confidentiality, tests kits, skilled workers).

H- Access to information

Many infections still occur because they are not informed appropriately about transmission modes and preventive measures. While awareness of the epidemic is on the rise, specific knowledge about HIV is still inadequate. Although efforts are increasingly made to sensitize populations and especially young people about the

pandemic, there are persistent misconceptions about how HIV is transmitted; meanwhile, these people constitute the age group most exposed to the infection. AIDS awareness is still far from universal. From this view, information is of great importance to stop or slow down the pandemic. Information and education have a synergic action; information campaigns are almost of posters display, advertising messages on the radio and television, generally in the official language that can be different from local ones. In the 2001 Demographic and Health Survey from Cameroon, the percentage of respondents aged 15-19 who knew that a healthy-looking person can have HIV varies from 74% of girls (68% of boys) still in school to 55% of girls (48% of boys) no longer in school. The figure is of course worse among young people who have never been in school (17% of girls and 9% of boys). In fact, none of the 18 countries in which young people were surveyed by the Demographic Health Survey/AIDS Indicator Survey between 2001 and 2005 had knowledge levels exceeding 50% (UNAIDS 2006).

III- Socio-cultural determinants

These determinants are the least subject to change in a society. These are some religious and cultural practices, ethnic diversity, the average age at the onset of sexual activity and conflicts.

A- Religious and traditional practices

Traditions can favour the epidemic through age of the onset of sexual activity. Girls are generally sent into marriage during adolescence and age difference between partners is generally high, another cofactor of virus transmission. Christian and Muslim religions on the contrary would have a negative impact on the course of the epidemic through delayed age at the onset of sexual activity due to religious education. However, the religious and traditional practice that has been proved to have a greater impact on HIV transmission is male circumcision (Auvert et *al*, 2005).

B- Ethnic diversity

The degree of ethnic fragmentation is typically an environment variable. It would affect HIV/AIDS prevalence rate essentially through growth. Numerous economic works point out the negative relation between ethnic diversity and growth, namely those of Easterly and Levine (1998). According to them, ethnic diversity tends to make

cooperative solutions more difficult to obtain in case of disagreement. That would also be the case for a consensus on the portion of public spending allocated to the fight against HIV/AIDS. A big number of ethnic groups lowers confidence between economic agents and raises transaction costs. Collier (1998) points out that impact of ethnic diversity on growth passes through economic policy choices and then notes the importance of political institutions. Arcand, Guillaumont and Guillaumont-Jeanneney (2000) find that impact of ethnic diversity on growth is direct because it affects public and private resources allocation. They indicate communication between different ethnics as solution to this problem. Audibert et *al* (2002) raised the concern that ethnic polarization can influence public spending on health.

C- Age at the onset of sexual activity

This variable is thought to favour the epidemic mainly through girls' age at the first sexual intercourse. The younger they begin sexual activity, the more they are exposed to tearing, since their partners are almost always older and their body is still immature. More importantly, many people aged less than fifteen have not received enough formal sexual education to use contraception. This sexual education is rather learned in the street, with all what it embodies as misinterpretations and generally accepted ideas. People who engage early in sexual activity are thus less likely to protect themselves or to do so adequately.

Socio-economic variables interact among them; for instance, a low education level leads to an over-valuation of qualified labour force, which in turn increases income inequalities. There are also interactions between socio-economic and socio-cultural variables; the important proportion of Muslim population into total population tends to result in a lower education rate and in the same way, bad governance can be the result of ethnic diversity. These two types of variables directly influence HIV/AIDS prevalence rate. Poor countries cannot nor allocate many funds to vaccine research neither offer antiretroviral therapy to sick people. Last, they influence epidemiological variables; for example, income level influence nutritional status and circumcision is associated with some cultural and religious practices.

D- Armed conflicts

Wars impoverish populations and displace them, placing them at increased risk of infection by disrupting social structures. Louria (2003) states that wars create the milieu

for infection in many ways: massive injuries that invite microbial infection; forced migration of non-immunized persons into areas inhabited by disease-carrying vectors; crowding in refugee camps with inadequate sanitation facilities; migration of disease-carrying individuals into uninfected areas; exposure to disease-carrying rodents; malnutrition, starvation, destruction of public health infrastructures and lack safe water supply. Displaced populations also lack access to prevention services as well as STI diagnosis and treatment.

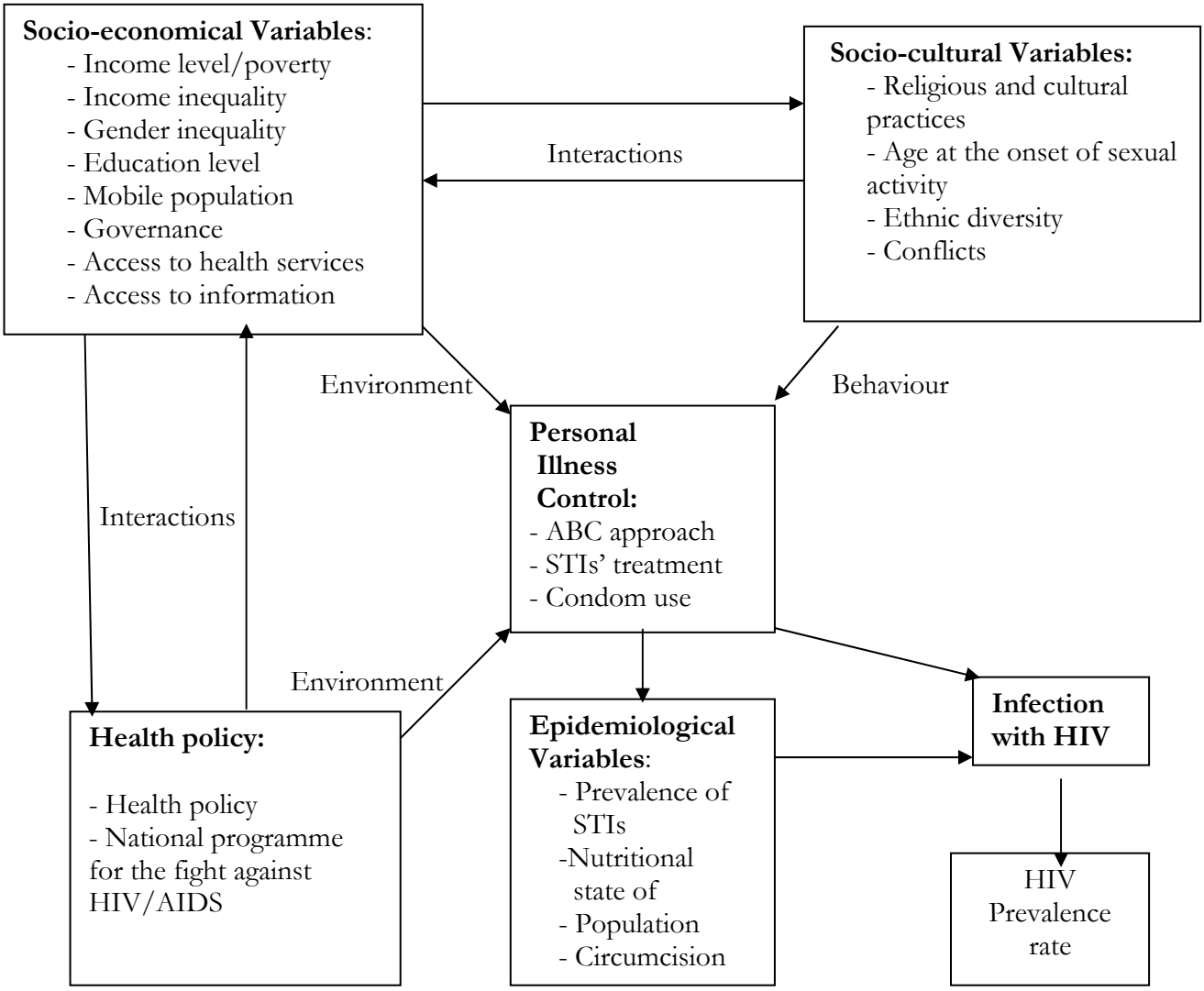
Most armed conflicts in Sub-Saharan Africa were sparked by internal and external conflicts which contributed to famines, producing massive flows of refugees with consequent food shortages and crowding in unsanitary camps (Stillwaggon 2002). Moreover, warfare presents major opportunity costs for poor countries. Resources flow to arms and equipment purchases, military salaries, replacement costs, and hundreds of other large and small expenditures. Yet, these resources in the mid and late 1980s could have been going for improved access to health care, especially STD treatment and other forms of HIV prevention (Collins and Rau, 2000).

Additionally, mass rape can become a vehicle for spread of sexually transmitted diseases, when it is used as an intentional military and governmental technique of intimidation and cruelty.

Evidence in Sub-Saharan Africa, especially in Uganda however fails to confirm that internally displaced persons and refugees are more likely to be HIV-infected than people in more stable settings (UNAIDS, 2006). This might be due to the reduced mobility and increased access to health and prevention services of women in some of the camps (Fabiani et *al*, 2006). A recent review of HIV literature on displaced persons in eight countries (including Uganda) also failed to find evidence that conflict increases HIV transmission (Spiegel and Harroff-Tavel, 2006).

Figure 2.2 summarizes the action of each group of determinants on the prevalence rate.

Figure 2.2: HIV/AIDS determinants and action channels



Source: Author

Section III: Empirical evidence on HIV/AIDS determinants

If the study of the determinants of HIV/AIDS epidemic is the subject of many articles and scholarly papers, most of them do however belong to the fields of medicine, sociology or anthropology. Five authors developed pertinent economic models, linking HIV/AIDS epidemic to its determinants at macro level. These authors are Over (1998, 2004), Bonnel (2000), Stillwaggon (2000, 2002), Nattrass (2006) and Zanakis (2005).

I- Over

Mead Over (1998) assessed the impact of societal variables on HIV prevalence rate in urban settings, using cross-sectional data for urban areas of eighty-three developing countries. In his study, he reminds the three major causes that increase the infectivity of the virus: the presence of ulcerative STIs, the absence of male circumcision and the sub-type of the virus[47].

At the time the paper was written, the most complete dataset on HIV/AIDS prevalence rate was US Bureau of the Census' (BUCEN)[48]. A non-negligible advantage of BUCEN dataset is that it decomposes the population according to behaviour, so that prevalence rates are expressed for low-risk groups (members of the general population or as pregnant women attending antenatal clinics) and high-risk groups (prostitutes or STI patients). The dataset is quite heterogeneous in terms of HIV prevalence; of the 83 countries composing the sample, from 0 to 8.3 per cent of urban groups practising high-risk behaviour are infected. Fourteen countries have less than 1 per cent infection rates in these groups and at the other extreme another 14 have more than 45 per cent infection rates. Among low-risk groups in 96 countries, infection rates remains at 0 per cent in 29 of the countries, but ranges up to 33 per cent in others

After a *logit*[49], transformation of the HIV prevalence rate, he designed a linear model with the following explanatory variables:

- country GNP per capita (to assess the possibility of alternative to commercial sex),
- age of the epidemic (that indicates the potential for new infections in population),
- the percent of Muslim (to capture cultural norms that shape a society's sexual practices)
- the percentage of foreigners to account for migration
- the percent of military forces in population (both in order to assess the demand for commercial sex),
- the male/female gender ratio,

[47] These three elements can easily be linked to the parameter Q in the formula of the reproductive rate (see Section I).

[48] UNAIDS began to release prevalence rates at the end of 1997 for all countries in the world

[49] The *logit* transformation of HIV prevalence rate is linked to the non-linear evolution of the infection. Refer to Chapter 3, section II for an in depth insight on the functional form.

- the male/female literacy gap (both indicators of gender inequality that would increase the potential for commercial sex work) and
- Gini coefficient for income inequality.

The author performed a first estimation using ordinary least squares and his model's explicative power (as indicated by the value of 0.650 for R-*squared*) is remarkably high, as the variables can explain at least half of the cross-country variation in infection rates. Income inequality and gender inequality (measured as the male/female literacy gap and male/female ratio in urban population) appear to favour HIV transmission and GNP per capita is linked negatively to the infection. In the same way, age of the epidemic, the percent of foreigners and military forces among total population are correlated positively with national HIV prevalence rate while the percentage of Muslim in total population is negatively associated with the infection rate. All the coefficients carry their expected sign and most of them are statistically significant. These results are also robust to the inclusion of regional dummies and of the prevalence rate among high-risk groups. Moreover, the addition of twenty-two countries to the sample does not significantly change the results.

Because the similarity in the value of coefficients appeals for further investigation, the author estimates its equation on the pooled sample. Interestingly enough, pooling the data on low- and high-risk groups into a single regression improves the precision of the coefficient estimates substantially, even after taking into account the within-country correlation between the residuals from the low- and high-risk groups

This work is noteworthy as it is practically the first empirical study of AIDS economic determinants. In addition, the interesting dataset allows explaining the synergy between high-risk groups' behaviour and socio-economic variables on low-risk groups, which in turn determines prevalence rate at national level. Last, but not least, this study is the only one that explicitly accounts for the particular evolution of an epidemic. Then, it allows a relevant econometric analysis of an epidemiological phenomenon.

II- Bonnel

Bonnel (2000) assessed the impact of HIV/AIDS epidemic on growth in Africa, with the hypothesis that the impact of the epidemic on per capita growth is indeterminate ex ante due to two opposite factors; on the one hand lower labour force growth due to AIDS-related mortality would increase per capita income while reduction in the stock

of capital due to increased health expenditure would lower per capita income. Basically, AIDS epidemic is likely to affect African economy due to the following factors: *(i)* the scale and the speed of the epidemic have been worse than projected, *(ii)* the epidemic reduces the stock of physical and human capital, *(iii)* AIDS destroys social capital by tearing away at (swamping) existing institutions and *(iv)* feedback effects further amplify the impact of AIDS on growth, leading to a vicious circle of AIDS and poverty.

Classifying its determinants into three headings[50], Bonnel also examine the reverse issue, which is the impact of growth and economic variables on HIV/AIDS epidemic[51]. The variables included in the analysis are access to information, growth, gender inequality, ethnic fragmentation, education, migration, age of the epidemic and the share of the population that is Muslim.

Using cross-sectional data for 59 developing countries in 1997 and ordinary least squares, he estimate the equation below:

$$Log(HIV_i) = \beta_0 + \beta_1 Log(Phone) + \beta_2(growth) + \beta_3(Gender) + \beta_4(Mus\lim) + \beta_5(Ethnic) + \beta_6(Time) + \beta_7(migration) + \beta_8(Education) + \varepsilon \quad (2.2)$$

Where: HIV is HIV/AIDS prevalence rate

Phone is the number of phones per person (en indicator for development)

Growth is the growth rate of GDP per capita between 1980 and 1990

Gender is the share of female labour in industry

Muslim is the percentage of the population which is Muslim

Ethnic is the ethnic fractionalisation

Time is the time since fist HIV case was reported

Migration is labour migration (proxied by the share of factor receipts in exports)

Education is secondary school enrolment rate.

Quite surprisingly, the growth rate of GDP per capita and secondary school enrolment rate were not significant. Moreover, the two variables most strongly correlated with HIV prevalence rate (value of the coefficient and of *t*-statistic) are not economic ones[52].

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[50] Bonnel classifies AIDS determinants as socio-economic, socio-cultural and epidemiological. The classification we used in the previous section is adopted from this paper, although epidemiological determinants are analyzed in Section I.

[51] This second issue is the only one we shall analyze in the rest of the sub-section, since our purpose is mainly on AIDS determinants.

[52] The two variables most strongly correlated with HIV infection are indeed the percent of Muslim in total population and the time since first HIV case was reported in the country. These variables are epidemiological ones, since Muslim religion is associated with a more regular practice of male circumcision. It is thus not really surprising

In this model, access to information, gender inequality, ethnic fractionalization and labour migration were also strongly correlated with HIV/AIDS infection. Bonnel's model fits well, with an *R-squared* value of 0.69.

In order to account for the interactions among HIV/AIDS, policy and institutional variables and growth, Bonnel then used a system of three equations. In the first equation, economic growth is a function of macroeconomic rating, institutional variables and traditional determinants of growth.

$$Growth_i = \alpha_0 + \alpha_1(Y_i) + \alpha_2 Ln(Phone_i) + \alpha_3(Macro_i) + \alpha_4(Law_i) + \alpha_5(HC_i) + \alpha_6 Z_i + U_i \quad (2.3)$$

Where: Macro is the rating of macroeconomic outcome by country in 1998

Law is the rating of the legal framework

HC is an indicator of human capital

The second equation in turn links macroeconomic policy ratings and institutional variables to HIV prevalence rate.

$$Macro_i = \gamma_0 + \gamma_1 Ln(Y_i) + \gamma_2 Ln(Phone_i) + \gamma_3 HC_i + \gamma_4 ICRG_i + \gamma_5 Growth_i + \gamma_6 Ln(HIV_i) + \gamma_7 Ln(HIV_i)^2 + \gamma_8 Dummy_i + V \quad (2.4)$$

Where: ICRG is the rating of macroeconomic outcome by country in 1990.

The third equation is equation (1) previously described, linking HIV/AIDS epidemic to its determinants. There are only negligible changes in the results when correcting for these potential feedback effects.

Bonnel's model is remarkable and sophisticated in a number of points. First, it accounts for the lag between infection and the apparition of AIDS cases by using lagged values for all determinants[53]. Second, the analysis takes into account feedback effects between AIDS epidemic and macro-economic environment. However, it fails to address some important determinants of the epidemic; the author mentions ulcerative STIs as the most important co-factor of the epidemic, but this variable is not included

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that these two variables are the most significant, since they are theoretically the variables acting the most directly during HIV transmission (see figure 2 of this chapter).

[53] Though the intention is noteworthy, it is not really meaningful here since prevalence rates are estimations of the number of people infected with HIV and those developing full-blown AIDS. Thus there is no indication on the average time infection took place.

in the analysis. In addition, poverty and income inequality and access to health care, which are of critical importance for the spread of the epidemic, are excluded from the estimations.

III- Stillwaggon

In her comparison of African and Latin American HIV epidemics, Stillwaggon (2000) focussed on the economic and biomedical environment in which HIV transmission occurs. She insists that HIV/AIDS infection is primarily an infectious disease and as such, it is fuelled by poverty, malnutrition, urbanization and international migration. A better understanding of the difference of the spread of the HIV/AIDS epidemic between countries would require looking at the factors that usually distinguish developing countries from developed ones, rather than attribute this difference to behaviour only. Indeed, malnutrition and parasitosis are widely recognized as depressing immune function, destroying immune system and impeding the production of B-cells and T-cells. Moreover, protein-energy malnutrition, iron-deficiency anaemia and vitamin-A deficiency weaken physical barriers and immunity. Malnutrition and parasite infection increase HIV susceptibility, not only to opportunist infection after HIV infection, but also to HIV transmission, just as they increase susceptibility to other infectious diseases. Yet, Sub-Saharan Africa shares most of these features with many countries in Latin America. Indeed, most Latin America countries are ranked as middle-income and of medium human development. That ranking does not represent an intermediate stage between the developing and rich nations, instead it simply results from averaging the affluence of modern sector and the extreme poverty of the rest of the population. In that way, African epidemic is not an exception, it could easily spread with the same speed and to the same extent in other parts of the world that are also *fertile*.

Focusing exclusively on what makes Sub-Saharan Africa a *fertile* terrain for the epidemic, Stillwaggon (2002) reminds that it is the continent with the greatest area in the tropics and therefore suffers more than other continents from parasitic diseases and poor tropical soil. Low and falling food production and consumption, low food quality because of the infertility of tropical soil, and endemic parasitic diseases that drain the population of essential nutrients are all conditions more descriptive of sub-Saharan Africa than of any other part of the world.

Thus, regardless of behaviour (multiple partners or condom use), these factors widespread in Sub-Saharan Africa, increase the risk of transmission of HIV there. Income inequality and ulcerative STIs make up the context of HIV transmission. Income distribution is important because it tells us more about the living conditions of the poor than does average income. Thus, it is not GDP per capita that is relevant, but the rapidly worsening situation of the poor. Though South Africa, Botswana, and Namibia are relatively wealthy compared to the rest of the continent, they have highly skewed income distributions. One-fourth of South Africa's population and one-third of Botswana's population live on less than one dollar a day. While STDs are common in the industrialised countries and can act as co-factors in the transmission of HIV, they play a more significant role in Africa. Not only are the kinds of STDs in Africa more likely to be cofactors for HIV transmission, but the STDs go undetected and untreated, increasing the likelihood of transmission of an ulcerative STD and HIV. Cumulative cases of STIs the United States for 1989 to 1996 were just over 17,000, compared to 4.1 million cases of gonorrhoea, 2.6 million cases of Chlamydia, and 1.2 million cases of syphilis. Genital ulcer diseases are most common in areas where water is difficult to acquire and personal hygiene suffers. While there is convincing evidence that all STDs can increase the transmission of HIV, genital ulcers increase the risk five- to tenfold (World Bank, 1993).

The results of her multivariate ordinary least squares regression showed that the four variables (real GDP per capita, urbanization rate, international migration and calorie supply) were all significantly[54] associated with HIV prevalence rates in Latin America's countries. The explicative power of the model (R-*squared* = 0.828) should however be interpreted with caution given the small size of the sample (*N*= 20). This analysis also displays on Latin American data the paradox mentioned by Nattrass, which prevails on African continent concerning the positive association between the epidemic and national income[55]. It also confronts with Over's result who found a negative correlation between GDP and HIV prevalence rate. Stillwaggon herself points out that this preliminary analysis appeals more tests, time-series studies and also interactions among

[54] All the regression coefficients are significant at the 95 percent level of confidence and most are even significant at the 99 percent level of confidence.

[55] Unlike in Africa where there is not a clear explanation to the paradox, in Latin America however, the positive association between national income and HIV prevalence rates lies in the fact that the initial infections were among international travellers, who brought them back from USA. Yet, the number of international migrants in a country is positively linked with GDP per capita.

variables to further explore the relations between the four variables (poverty, malnutrition, urbanization and international migration) and HIV prevalence rate.
The model is slightly different for the sample of African countries, as explanatory variables are now change in calorie intake, Gini coefficient, change in urban population, real GDP per capita and change in real GDP per capita. All variables, except real GDP per capita are significantly correlated with the epidemic (Table 2.4). This model can explain at least half of the cross-country variation in prevalence rates. This is not really surprising, given that the analysis ignore some important determinants of the epidemic; the author forcefully states the importance of STIs as co-factor of the epidemic, but this variable does not appear at all in the econometric model. By focusing on the context of transmission, Stillwagon *de facto* exclude from her analysis factors like access to information, education or circumcision, which are strong predictors of the epidemic in other studies.

IV- Nattrass

The first of the three objectives[56] of this paper is to explore for the year 2003 the relationship between HIV prevalence on the one hand and inequality within countries and differences in per capita income between countries on the other hand. Nattrass (2006), assessing the impact of national income and income inequality on eighty-four developing countries (which account for 90% of total infections in the world) demonstrated a paradox in HIV/AIDS distribution. Although it is undoubtedly correlated negatively with national income worldwide, HIV/AIDS epidemic exhibits a different pattern over the sub-sample of African countries; as the epidemic is more prevalent in African countries with higher GNI per capita. In order to mitigate the impact of 'outliers' countries of Southern Africa, HIV prevalence and per capita income are expressed in log terms, as well as a dummy variable to account for Southern Africa countries.

Despite a fairly good explicative power (R-*squared* = 0.55), the results of his study are to be treated with cautious, as his aim is not to provide a full description of the determinants of HIV prevalence, but simply to examine the afore-mentioned relation. Furthermore, attention, should be paid to the fact that the direction of the

[56] The two other issues are the change in life expectancy that has occurred in these countries as a consequence of the AIDS epidemic as well as the provision of highly active antiretroviral therapy (HAART).

relationship between the variables is not clearly stated: just as lower per capita income and inequality is likely to create conditions conducive to the spread of AIDS, high rates of HIV prevalence may well undermine economic growth, reduce economic security and possibly result in greater inequality.

Although the model does not explicitly incorporate poverty, Nattrass establishes it as the link between per capita income and inequality. Given the relationship between poverty and HIV transmission, countries with relatively low levels of per capita income are likely to experience greater levels of poverty, and thus higher rates of HIV prevalence, than higher income countries. Middle-income countries with high levels of inequality may also manifest a substantial degree of poverty because inequality in such countries could be a proxy for the presence of poverty, though not necessarily so.

V- Zanakis et *al*

Unlike most of the previous studies, this analysis's ultimate aim is not to identify HIV/AIDS epidemic determinants, but to measure countries' efficiency (defined as a low number of infections) and classify them accordingly. So, the authors assessed countries' efficiency in avoiding the epidemic by the Data Envelopment Analysis (DEA) method, using cross-country data on hundred and fifty-one countries, with five dependent variables and fifty explanatory variables. They identified the following as critical HIV/AIDS determinants: nation's wealth, migration and access to health services.

The five dependent variables used by these authors are HIV/AIDS Cases per 100,000 people, the absolute number of adults aged 15–49 living with HIV/AIDS virus, the percentage of adults aged 15–49 living with HIV/AIDS, the estimated number of AIDS-related deaths for adults and children and the percentage of male sexual transmitted diseases patients diagnosed with HIV/AID[57]. Influential socio-economic factors of the epidemic (interest variables here) are national income, migration and access to health services. Covariates are education, unemployment rate, energy use, environment, demographics, access to information and sex-related

[57] All data for dependent variables are for year 2000.

behaviour. For each of these determinants, the authors use a plethora of indicators in each equation[58].

Outliers and variables with many missing data are excluded from this analysis. All the dependant variables are subject to log-transformation, but this is not the case for most of the explanatory variables.

Zanakis et *al* (2007) came out with the following estimations:

Ln (HIV/AIDS Cases per 100.000 population) = *1,93* + *0,22* (Commercial energy GDP output) + *0,36* (Dependency ratio of population economically dependent as active age group ages 15–59) – *3,78* (Population fertility rate) – *1,66* Ln (Doctors per 100;000 people) – *0,045* (Health public expenditure per capita in international $) – *0,73* Ln(Labor force % in agriculture). (2.5)

Ln (Adults 15-49 living with HIV/AIDS virus) = *10,58* + *0,88* Ln (GNP) – *8,77* (Health System Performance Index) + *0,91* (Population Natural Rate of Increase) + *0,54* Ln(Population Density) + *1,065* Ln(Out-of-pocket expenditure per capita in international $) – *0,60* Ln(Radios per 1000 people) – *0,46* Ln(Nurses per 100; 000 people). (2.6)

Ln (Percentage of total adults living with HIV/AIDS) = *4,21* – *7,66* (Overall health system performance index) +*1,76* Ln(Out-of-pocket expenditure per capita in international $) + *1,11* (Population natural rate of increase) + *0,39* Ln(Population density) – *0,47* Ln (Radios per 1000 people). (2.7)

Ln (AIDS-related deaths for adults and children) = *13,86* – *9,82* (Health system performance) + *0,83* Ln(GNP) – *0,84* Ln(Radios per 1000 people) + *0,94* (Population natural rate of increase) + *0,65* Ln(Population density) – *0,10* (Hospital beds per 1000 people) – *0,01* (Health public expenditure per capita in international dollars). (2.8)

[58] Migration is an exception here; for this variable, only the net migration rate (migrants/1000 population) was used as a measure of international migration and no urban–rural migration data were found to account for internal migration.

Ln (Percentage of male sexual transmitted disease patients diagnosed with HIV/AIDS) = *7,78*+ *0,08* (Dependency ratio) + *1,66* Ln(Health out-of-pocket expenditure per capita in international $) - *5,63* (Overall health system performance index) + *0,005* (Commercial energy net imports). (2.9)

Data published by Zanakis et *al* reveals the strong positive correlation between national income and HIV prevalence previously found by Stillwaggon. However, this correlation is not homogenous across the sample; although all five HIV/AIDS indicators exhibit overall negative correlations with GNP per capita, the picture changes if segmented by region: the HIV/AIDS case rate correlation with GNP per capita remains negative for OECD and high income countries but reverses to a significant positive correlation for Latin America, Caribbean, East Europe, Central and South Asia regions. Nations net migration rate correlation to any of the AIDS measures is practically nil and there is a direct linear relationship of higher AIDS indicators in countries with lower health system performance. Among the covariates, only access to information and the structure of the labour force are significant and carry the expected sign.

The important finding of this research is that countries with lower population density that manage to provide better health system performance, per capita support (doctors, nurses and hospital beds) with better media information (radio, phone and TV access), and not necessarily higher GNP are more likely to exhibit lower HIV/AIDS indicators.

Zanakis *et al's* paper is innovatory in three points: first, it is a multi-country explanatory assessment of HIV/AIDS indicators; all other studies use data for developing countries exclusively, while national prevalence rates are higher in poor countries worldwide, the epidemic is also well spread among ethnic minority and poorer populations in wealthy nations. Second, the paper addresses the countries efficiencies in battling the pandemic. Last, the authors use many measures of the prevalence rate, so that the robustness of their analysis could *de facto* be assessed.

Table 2.4: A review of the empirical studies on HIV/AIDS determinants[59]

	Over		**Bonnel**		**Stillwaggon[60]**		**Nattrass**	
Dependant variable	Prevalence rate in urban population		Log of HIV prevalence rate in adult population		AIDS cases per 100,000 people **National prevalence (UNAIDS data)**		Estimated HIV prevalence (adults 15- 49)	
Period of study	1997		1997		**1998**, 1997		2003	
Countries location	SSA, LAC, Asia, Middle East		Africa		Latin America SSA		SSA, LAC, SEA, EA, SA, EE, CA[61]	
Explanatory variables	coefficient	*t*-stat	coefficient	*t*-stat	coefficient	*t*-stat	Coefficient	*p*-value
Age of the epidemic	0.47	3.6	0.379	2.9				
GNP per capita (log)	-1.0	-2.9			0.059	8.15	-0.937	0.000
					0.0003	**0.406**		
Growth rate of GDP			4.58	0.5	**0.763**	**1.475**		
Foreign-born per cent (log)	0.28	2.0						
Per cent Muslim	-0.018	3.3						
Gini index of inequality	10.6	4.0			**22.45**	**2.58**	0.048	0.022
Male-female literacy gap	0.069	2.4						
Urban M/F gender ratio	2.9	2.6						
Military forces (% of Urban population	0.056	3.1						
Urbanization rate					86.83	2.92		
					1.011	**2.41**		
International migration					18.41	3.62		
Calorie supply					-6.04	2.20		
					- 18.96	**3.36**		
Number of phones fixed lines (log)				2.2				
Share of female labour			-0.0035	1.7				
Labour migration			0.003	3.2				
Ethnic fractionalization			0.027	3.5				
Enrolment rate			-0.016	1.2				
Southern Africa dummy							2.925	0.000
R2 or adjusted R2	0.621		0.69		0.828		0.5534	
					0.517			
Observations	72		59		20 **44**		57	

[59] Zanakis et *al* used more than one dependant variable and much more explanatory variables than the other authors; his analysis is well described in the devoted paragraph and not summarized in this table.
[60] Figures in bold relate to Stillwaggon's second model on Sub-Saharan Africa, while the normal font indicates Latin America's model.
[61] LAC: Latin America and the Caribbean; SEA: South East Asia; EA: East Asia; SA:South Asia, EE: Eastern Europe; CA: Central Asia.

Conclusion

HIV/AIDS epidemic is one of the most mediatised affections in human history. Its burden, toll and evolution are well documented and scientists working on the disease can regularly share their advances during numerous conferences. Like most infectious diseases, the evolution of the disease is due to a conducive environment, which allows HIV to spread easily among individuals. In the same way, behavioural factors contribute to the propagation of the virus. Therefore, the epidemiological model of HIV transmission looks like any infectious disease's, with a reproductive rate that depends on the number of contacts per day, the probability of infection for a single contact and the duration of infectiosity for the infected person. However, unlike most infectious pathogens agents, HIV is a complex virus with multiple subtypes and strains and the aptitude to destroy the immune system cells that are meant to fight against HIV and to insert genetic material into human cells. For this reason, the response addressed to the epidemic still fails to provide a definite cure and a vaccine, despite tremendous progress achieved in the recent years.

The comprehension of variables that influence the course of HIV/AIDS pandemic constitutes an important stake both at the economic and humanitarian levels for Sub-Saharan Africa because it is the first step in eradicating the disease. Socioeconomic, socio-cultural and epidemiological variables interact among them and influence the HIV/AIDS prevalence rate, thus creating an unprecedented epidemic. Health determinants cited in the previous chapter, to which add variables influencing behaviour, constitute the framework of HIV/AIDS transmission. Thus ethnic diversity, age at the onset of sexual activity and some cultural or religious practices also favour HIV transmission. Due to the nature of the infection, some epidemiological variables such as condom use and the prevalence of ulcerative STIs among population complete the picture to shape the frame into which HIV transmission occurs.

Most of the above-mentioned determinants have been submitted to empirical analysis and seem to confirm the relations we described; both Over and Bonnel found that the course of the epidemic is influenced by its age. Worldwide, HIV/AIDS is a disease of poor countries, as found by Over and Nattrass, notwithstanding this finding, it can be more prevalent in richer countries at regional level, as evidenced by Stillwaggon. Migration and urbanization are also strong drivers of the epidemic. Finally income inequality as measured by Gini coefficient and gender inequality, measured by literacy gap between male and female and the share of female labour are shown to fuel the epidemic.

Sub-Saharan Africa is the poorest region of the world, but it is the region that exhibits the highest disparities in income, and in social and economic outcomes when data are disaggregated by sex. In the next chapter, we focus our analysis on income and gender inequalities, two socio-economic variables that have not been studied in this chapter.

CHAPTER 3

SOCIO-ECONOMIC INEQUALITIES AND HIV/AIDS EPIDEMIC: THEORETICAL LINKS AND METHODOLOGICAL ISSUES[62]

"The only well-demonstrated cofactors are social inequalities, which have structured not only the contours of the AIDS epidemic, but also the course of the disease once a patient is infected", ***Paul Farmer*** *in Infections and Inequalities (1999).*

"A range of structural inequalities intersect and combine to shape the character of the HIV/AIDS epidemic everywhere", ***Richard Parker*** in *The Global HIV/AIDS Pandemic, Structural Inequalities, and the Politics of International Health (2002).*

Introduction

Income inequality is regularly cited as one of the most important determinants of the HIV/AIDS epidemic. At the international level, it is generally admitted that HIV/AIDS epidemic is fuelled by poverty; countries with the highest prevalence rate are also the poorest of the world. However, in Sub-Saharan Africa, the region most heavily affected by HIV/AIDS in the world, countries with the highest rate of prevalence are not necessarily the poorest but those with the least egalitarian income distribution, which also happen to be the wealthiest. For example, in 2005 Botswana which had the second highest prevalence rate (just behind Lesotho) and South Africa which had the highest absolute number of infected persons are in fact the two richest countries of the continent and belong to the group of the upper-middle income countries. Yet, these countries display a high level of income inequality as measured by Gini index of income

[62] The article "Gender Discrimination and HIV/AIDS Epidemic: Evidence from Sub-Saharan Africa", which is taken from Section 3 of this chapter (combined with the econometric analysis of chapter 4) has received the first prize of the 2007 Global Development Awards and Medals Competition in the category: *Women's Rights, Security and Development: Challenges and Opportunities.*

distribution. Unlike poverty, the link between income inequality and HIV/AIDS epidemic is not obvious. States with high levels of income inequality tend to agree less on the nature of public expenditure (especially social expenditures such as health care) to engage in, leading to a decrease in these types of expenditures which are important determinants of health status. Differences in income also give room for the opportunity for high-risk activities such as commercial sex work and migrant work.

In the early stages of the pandemic, HIV infection was predominantly among men in many developed and most developing countries. As of the end of 2005, however, approximately half (seventeen million and half) of the thirty-eight million adults living with HIV/AIDS globally are women compared to forty-one percent in 1997. This trend is even more pronounced in Sub-Saharan Africa, where women account for close to sixty percent of people living with HIV. Seventy-seven percent of all HIV-positive women in the world are African (UNFPA, 2005). Young women represent the group most susceptible to be infected in the world: they account for sixty-seven percent of all new cases of HIV among people aged 15 to 24 in developing countries and this figure is three quarters for Sub-Saharan Africa (UNAIDS 2005). Currently, according to UNAIDS (2007), some seven thousands girls and women become infected with HIV every day. Thus, there is clearly a need to address the role of gender inequalities on women's susceptibility and vulnerability to HIV/AIDS.

Indeed, there are important differences between women and men in the underlying mechanisms of HIV infection and in the social and economic consequences of HIV/AIDS (Moss, 2002). These stem from biology, sexual behaviour and socially constructed *gender* differences between women and men in roles and responsibilities, access to resources and decision-making. Most traditional strategies to prevent the spread of HIV have focused on behavioural changes such as abstinence, faithfulness and the promotion of condom use. While this 'ABC' approach has certainly prevented large numbers of people from becoming infected, many of the world's women are simply not in a position to abstain from sexual activity, rely on their partners' fidelity, or negotiate condom use. Women's low socioeconomic and socio-cultural statuses appear mainly through commercial and transactional sex, violence, lack of negotiation of safe sex and lack of adequate information.

In analyzing the impact of socio-economic inequalities on HIV/AIDS epidemic, we first present some usual problems associated with the empirical analysis of inequality, then we present the effects of income inequality on HIV/IDS epidemic. In a last section, we address the links between gender inequality and HIV/AIDS epidemic in Sub-Saharan Africa.

Section I- Analyzing inequality: conceptual and empirical issues

The analysis of inequality comes along with some issues that can be conceptual or empirical. In the case of income inequality, there are many links, more or less obvious between inequality, poverty and growth that need to be clarified. In addition, there exist many kinds of inequality and other confounding and this can lead to a misinterpretation of the results, since an observed effect could be mistakenly attributed to income inequality. Last, inequalities measures are numerous, complex and should be chosen according to the characteristics one is interested in. The main problem associated with the analysis of gender inequality is the absence of a single measure that could encompass all the dimensions of the concept.

I- Measures of inequality

There is no best measure of income inequality. Some measures are more "bottom-sensitive" (more strongly correlated with the extent of poverty) than others. There are one-dimensional and composite measures of inequality. The measures perform differently under various types of income transfers. One should then select the measures based on the hypothesis to be addressed. Inequality measures should carry at least one of the following properties: fix range between 0 and 1, scale invariance, transfer principle and decomposability.

A- Percentiles and percentiles' distributions.

Deciles, quintiles and quartiles represent the share of income held by a certain percentage of population. The less this percentage is than the percentage of the population, the high the inequality in the considered population. Another way to use percentile is to compute a ratio, for instance the share of income of the lowest decile as a percent of the share of income of the highest decile. A low value of this ratio indicates an inegalitarian distribution. Finally, one could simply compare the share of these percentiles in the overall distribution, assessing for example that the highest quartile receives half of the income whereas the lowest receives only 5% of the total income and a higher gap between these two shares indicates a less egalitarian distribution.

B- Indexes

Among them, the most common used is the Gini index, Robin Hood index, Theil's entropy index and Atkinson index. The former two are derived from the Lorenz curve.

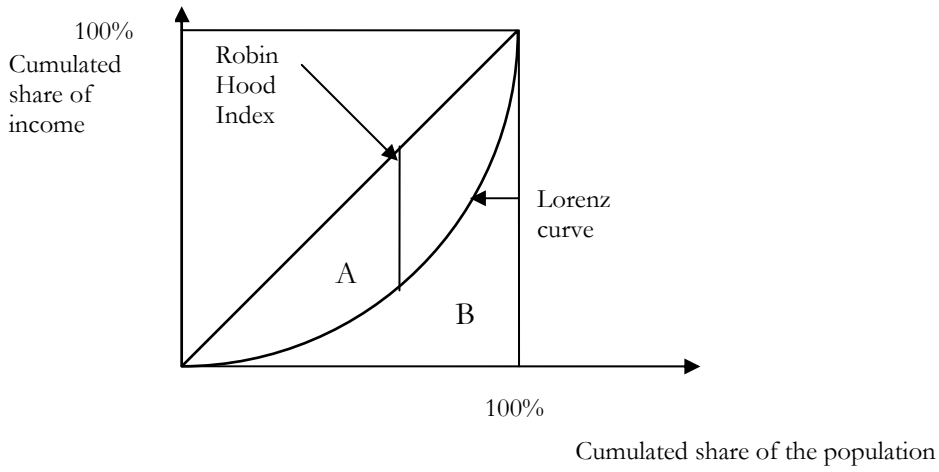
1) Lorenz Curve

The Lorenz Curve is a graphic used to display the relative inequality in a distribution of income values. The curve is obtained by connecting points whose abscissa is the cumulated frequency of

the population and ordinate is the cumulated frequency of income received. A perfectly equal income distribution in a society would be one in which every person has the same income and then, the curve would be a straight line, actually first bisector linking all the points representing n % of the population receiving exactly n % of the income. In practice however, the curve is always located under this line, but also above the curve with the right angle, the one which passes through the point (0, 100) representing the perfect inequality where one person has all the income and everyone else has none.

Figure 3.1 provides a graphical representation of the links between Lorenz Curve, Gini index and Robin Hood index.

Figure 3.1: Lorenz curve, Gini index and Robin Hood index



2) Gini index

The Gini index is the most commonly used measure of inequality. It is derived from the Lorenz curve and its value is comprised between 0 and 1, where 0 corresponds with perfect equality (where everyone has the same income) and 1 corresponds with perfect inequality (where one person has all the income and everyone else has no income). The Gini index is the double of the value of the ratio (A/B) on Figure 3.1. It is given by the following formula:

$$G = \left| 1 - \sum_{k=1}^{n} (X_k - X_{k-1})(Y_k + Y_{k-1}) \right| \qquad (3.1)$$

Where: G is Gini index

X_k is cumulated proportion of the population, for k = 0,...,n, with $X_0 = 0$, $X_n = 1$,

Y_k is cumulated proportion of the variable under study, for k = 0,...,n, with $Y_0 = 0$, $Y_n = 1$.

While Gini index carries a set of advantages that makes it the most commonly used measure of inequality. Thus, it is considered as a standard tool for economic works, so its interpretation is easy. It also accounts for all the observations in the distribution and does not depend on the size of the population considered. Finally, it allows to make comparisons between distributions of different sizes.

However, it also soiled by some shortcomings. For example, it entails elaborate calculations. The interpretation of the coefficient is not concise, as the same value of the coefficient could be associated with two different distributions[63]. Furthermore, it is scale-sensitive so that the value of the coefficient measured for a large geographically diverse country will generally result in a much higher coefficient than each of its regions has individually. And it is claimed that the Gini index is more sensitive to the income of the middle classes than to that of the extremes. Conversely, it is less sensitive to transfers between values near the average than between the two tails of the distribution.

3) Robin Hood index or relative gap to the mean

It measures the portion of the total income that would have to be redistributed in order for there to be perfect equality (hence its name in reference to the hero of Sherwood forest). It can be graphically represented as the longest vertical distance between the Lorenz curve, or the cumulative portion of the total income held below a certain income percentile, and the 45 degree line representing perfect equality. Robin Hood index is non sensitive to transfers between individuals in the same side of the distribution according to the mean.

4) Theil's Entropy index

It is a decomposable inequality measure, defined as a measure such that the total inequality of a population can be broken down into a weighted average of the inequality existing within subgroups of the population and the inequality existing between them.

[63] For example, a value of 0,5 for the Gini coefficient could describe a distribution where half of the population has no income and all the income is equally distributed among the other half , as well as a situation where an individual holds half of the total income and the other half of the income is equally distributed to all the others.

Theil index is more elaborate than Gini index, and as other advantages, it can be computed with individual as well as with group data, it is a decomposable index (thus it allows to assess the share of within-group and between-group inequalities), it is more sensitive to low incomes, so it is quite relevant for works on poverty. Despite these indisputable advantages, Theil index raises two principal critics: it entails very sophisticated calculations and it does not allow the comparison of populations of different sizes and structures.

5) Atkinson index

The Atkinson Index is one of the few inequality measures that explicitly incorporate normative judgments about social welfare (Atkinson 1970). The index is derived by calculating the so-called equity-sensitive average income (y_e), which is defined as that level of per capita income which if enjoyed by everybody would make total welfare exactly equal to the total welfare generated by the actual income distribution. The equity-sensitive average income is given by:

$$y_\varepsilon = (\sum_{i=1}^{n} (y_i) - y_i^{1-\varepsilon})^{1/(1-\varepsilon)} \qquad (3.2)$$

Where y_i is the proportion of total income earned by the *i*th group, and ε is the so-called inequality aversion parameter. The parameter ε reflects the strength of society's preference for equality, and can take values ranging from zero to infinity. When ε > 0, there is a social preference for equality (or an aversion to inequality). As ε rises, society attaches more weight to income transfers at the lower end of the distribution and less weight to transfers at the top. The Atkinson Index (*I*) is then given by: $I = 1 - y_\varepsilon / \mu$ where μ is the actual mean income. The more equal income distribution, the closer y_ε will be to μ, and the lower the value of the Atkinson Index. For any income distribution, the value of *I* lies between 0 and 1.

Table 3.1 summarizes the properties of the inequality measures described earlier.

Table 3.1: Axiomatic properties of various inequality measures

	Gini index	**Robin Hood index**	**Theil index**	**Atkinson index**
Range between 0 and 1	Yes	Yes	No	Yes
Scale independence	Yes	No	No	No
Transfer principle	Yes, but tails-sensitive	Yes, but transfers only from one side of the mean to the other	Yes	Yes, but depends on the value of ε
Decomposability	No	No	Yes	Yes
Suitability for comparisons	Yes	Yes	No	Yes, but with the same value of ε
Ease of calculation	Yes	Yes	No	No

II- Growth, Poverty and Inequality

No study on the effects of inequality on health can ignore the impact of poverty. If a majority of the population gets poorer, there will be an increase in inequality even though the rest of the population does not get richer. By the same token, there can be an increase in poverty without any increase in inequality if all income classes get poorer in the same proportion. Finally, societies that are economically unequal usually have higher level of poverty. Since the works of Kuznets (1955), economic development, which is often measured by per capita income, is cited as a determinant of income inequality. Indeed, inequality is low when national income is low because nearly everyone is living at or near the subsistence level. In the initial stages of the growth process, rapid population growth, urbanization, and industrialization lead to increased income inequality, but as the process continues, social and political factors emerge which then act to reduce income inequality; this evolution is known as the Kuznets curve. It is now well understood that there is no set law governing the relationship between inequality and growth as once proposed by the Kuznets curve. Instead, some macroeconomic variables related to development could be significant, along with others, in explaining country differences in income distribution, the most important being poverty. While these three notions seem clear, a clarification of each concept is necessary in order to fully understand the connections linking them. Thus, in what follows, we define poverty as the proportion of the population below a

particular poverty line (poverty gap), inequality as disparities in relative income across the whole population and growth as the percentage change in mean welfare level, following Bourguignon (2002). There are obvious, but also less clear links between these three variables, and they can be assessed by answering to the following questions: Is growth always good for the poor? Does growth contribute to lessen inequality? Does inequality play a critical role in the effect of growth on the poor? The answers to these questions lie in the theoretical links between poverty and growth, poverty and inequality, and lastly growth and inequality.

A- Poverty and growth

There is little controversy among economists that growth is essential for poverty reduction under the assumption that the distribution of income remains more or less constant. However, since a couple of years, evidences show that the link between these two variables may not be as direct as one might think. In fact, much evidence points in the opposite direction (Deininger and Squire 1996, Dollar and Kraay 2001, Bourguignon 2004). Is growth always good for the Poor? The answer to this question depends on growth distribution effects on income. Thus, income inequality is the most obvious channel through which growth impacts on poverty (Mehanna, 2004). Lopez (2004), after reviewing a plethora of paper concluder that growth is fundamental for poverty reduction and in principle growth as such does not seem to affect inequality. As for Ravallion (2004), a strong correlation between economic growth and poverty reduction means that either all that matters is economic growth and only policies that are targeted on growth can generate poverty reduction or only policies that are successful in reducing poverty can yield higher growth in aggregate.

B- Poverty and inequality

Income inequality is different than poverty. The former represents the distribution of income among the different classes of people while the latter describes the percent of the poorest category of the population. Nonetheless, both are tied with narrow links. For example, holding inequality constant, an increase in per capita income (in other words, growth) reduces poverty. Second, holding per capita income constant, an increase in inequality raises the level of poverty.

Poverty rates are measured by the percent of the population living below the national poverty line (the international poverty line is below $2 a day, and below $1 a day for extreme poverty). Income inequality can be measured in different ways (Dollar, 2004; Deaton 2001). The most visible link between income inequality and poverty is the mathematical one. Thus if we consider the formula of Gini index in paragraph I of this section, we can see that the greater the

extent of poverty, the higher the value of Gini index in this population for a given average income.

For Feldstein (1998), what matters is poverty and not inequality, as both can move in opposite ways. A reduction of poverty can occur when there is growth enough, so that both rich and poor experience an increase in their income. However, if the increase in rich people' income in more than that of poor people, there will also be an increase in inequality. There would thus be a simultaneous increase in poverty and inequality.

C- Growth and inequality: limits of the Kuznets curve

The inverted U-shaped relationship between inequality and growth established by Kuznets has been subject to critics because the inverted U-shape in the curve comes from historical differences between countries, rather than from progression in the development of individual countries. In the dataset used by Kuznets, many of the middle income countries were in Latin America, a region with historically high levels of inequality. When controlling for this variable, the U-shape of the curve tends to disappear. So, in the 1990s debates on growth, inequality and poverty were revolutionised as the World Development Report opened the decade making reference to 'broad-based growth'. Sen's *capabilities* framework and the related UNDP's work on human development further challenged the mechanistic assumption that growth automatically led to poverty reduction.

Indeed, growth is hardly distribution neutral, as the process of economic growth modifies income distribution depending on the forces behind growth. But these effects may be attenuated or compounded by growth and distribution policies. Moreover, economic models developed by Alesina and Rodrik (1994), Persson and Tabellini (1994) showed that inequality can negatively affect economic growth through political constraints, limited investment decisions, hindered development of capabilities and social strife.

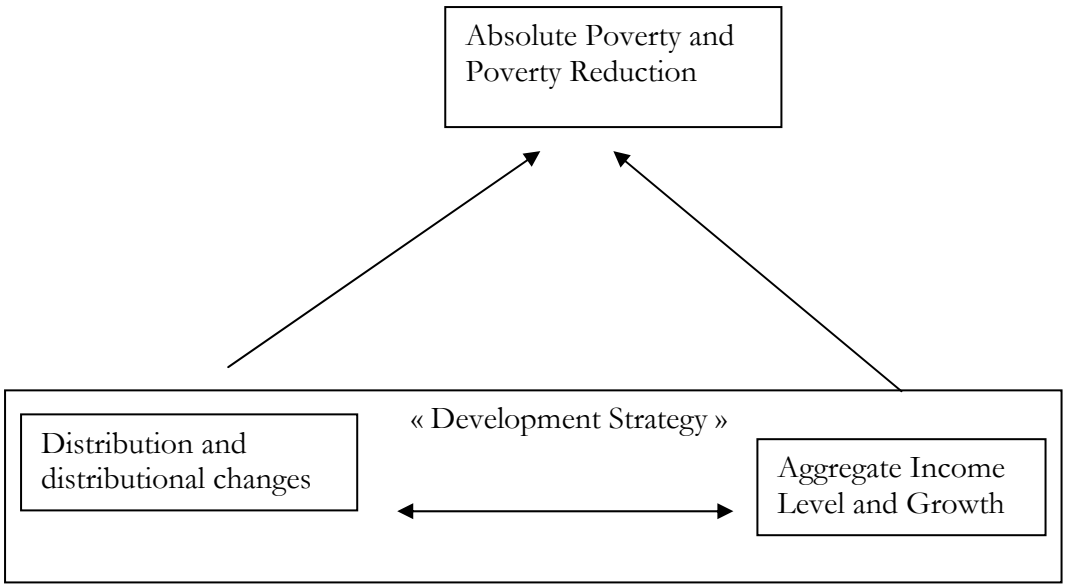
According to Bourguignon, there is an arithmetic identity between the growth of the mean income in a given population, with the change in distribution and the reduction of absolute poverty (See Box 3.1).

The rate of economic growth also affects income inequality; with accelerated growth, the absorption of labour into the higher growth sectors occurs at faster rates. Unless a country is at a very low level of development, one would expect income inequality to be lower in those countries which are growing the fastest. Growth can also affect income distribution through trade, making it will likely be lower.

In conclusion, rising inequality threatens growth and poverty reduction targets. In order to meet the global targets for reducing poverty, it will be essential to make pro-growth policies more favourable to equal distributional and to this end, structural reforms should be considered.

Box 3.1: The Poverty-Growth-Inequality triangle

The poverty-Growth-Inequality triangle



Source: Bourguignon, 2004.

The simple arithmetic of poverty, inequality and growth yields:
Change in Poverty= F (average income, distribution, growth, change in distribution)

It can also be written as: $\Delta Poverty = \Delta Growth - \Delta Gdp + \Delta gini$

An arithmetic *identity* links the growth of the mean income in a given population, with the change in distribution and the reduction of absolute poverty. A change in the distribution of income can be decomposed into two effects. First, there is the effect of a proportional change in all incomes that leaves the distribution of relative income unchanged called the *growth* effect. Second, there is the effect of a change in the distribution of relative incomes which, by definition, is independent of the mean called the *distributional* effect.
As illustrated in the figure above with the "Poverty-Growth-Inequality (PGI) Triangle", a development strategy is thus fully determined by the rate of growth and distributional changes in the population.

Bourguignon also emphasizes the importance of the growth elasticity of poverty and how it is affected by distributional changes, as well as by initial inequality and a country's level of

development. Specifically, he pinpointed two channels as to how redistribution affects growth: a permanent redistribution of income reduces poverty instantaneously through what was identified as the distribution effect; but also redistribution contributes to a permanent increase in the growth elasticity of poverty reduction - therefore accelerating the rate of poverty reduction for a given rate of growth.

Thus the "Poverty-Growth-Inequality (PGI) Triangle indicates that a development strategy is entirely determined by the rate of growth and distributional changes in the population. The final word is that there is no need to trade off between growth and inequality, both are important for poverty reduction.

Source: Bourguignon, 2004.

III- Gender inequality: How to measure a multi-faceted concept?

When it comes to gender, the right concept is gender discrimination rather than gender inequality. Unlike income, gender is not an objective indicator. As 'gender' refers to socially constructed roles, which could vary from one society to another, it is likely to change significantly following the evolution of societies. Moreover, it is a multifaceted notion. Therefore, measuring and assessing the many dimensions of gender inequality are tricky and difficult, and the lack of gender-differentiated data is a real obstacle in gender analysis. Thus, due to this lack of data, gender inequality is usually defined in terms of outcomes. But, this view is simplistic for two reasons. First, different cultures and societies can follow different paths in their pursuit of gender equality. Second, equality implies that women and men are free to choose different (or similar) roles and different (or similar) outcomes in accordance with their preferences and goals. Unfortunately, there is at present no gender equivalent of the Gini index, the summative measure of income inequality in a community (Phillips and al, 2005). In that way, one can only observe some characteristics, some overt manifestations of gender discriminations that translate into relative deprivation in education, inequality in income and participation to economic life and maternal leave benefits. Besides these measures, there are also two indexes developed by UNDP, the Gender-related Development Index (GDI) and the Gender Empowerment measure (GEM).

A- Inequality in education

Education and an independent income are well demonstrated as means to give women more voice and agency within the family (Drèze and Sen, 1989). However, throughout the world, women are less educated than men and the discrepancy is even more striking for Sub-Saharan Africa. As shown by our data, female literacy rates are always less than men and overall literacy

rates. It is expected that educated women are more able to ear about the disease, how it is transmitted and how to prevent infection. Then a high value of the variable would negatively affect the epidemic.

B- Inequality in income and participation to economic life

All over the world, women account for less than men in official labour force participation. Furthermore, jobs occupied by women often show a tendency towards lower pay, difficult career opportunities and lower reputation. Working in the agricultural sector, rural women in many African countries produce about 80 % of total food consumption, but receive only 1% of all credits given to agriculture (World Bank, 2001). While increased feminization of the workforce appears to be a global phenomenon, it has been pointed out that the increase in female work participation has been taking place in low-paying and unprotected jobs, leading to a rise in the precariousness of the female workforce (Ghosh, 1996). When women take part to economic life, they are supposed to rely less on risky activities like commercial or transactional sex, and also to have greater access to adequate sanitation and health services, which can prevent them from malnutrition and treat STIS, both cofactors of the epidemic.

B- Maternal leave benefits

The length of maternal leave and the percentage of wage women receive during maternal leave could be viewed as the value the whole society grants women for their non-productive work. Thus, a high value of these indicators would indicate that society has a great consciousness of women's activities that are not taken account into national accounts, though crucial: carry and raise children.

Although these indicators apply to the formal sector only and a great proportion of women in Sub-Saharan Africa work in the informal sector, they are still relevant in indicating the importance a society attaches to women's activities that are not assessed by the market. What these indicators are meant to represent are not the benefits of women working in the formal sector relative to other women in the country, but the value society would grant to the work of the average woman in the country, which cannot be assessed by the market.

D- UNDP indexes : the GDI and GEM

After several unsuccessful attempts to measure female discrimination across countries, the UNDP introduced two indicators to quantify the degree of gender inequality in 1995: The Gender-related Development Index (GDI) and the Gender Empowerment Measure (GEM). The GDI is based on three variables, namely life expectancy at birth, educational attainment, which is

measured by literacy rate and school enrolment, and access to resources in terms of GDP per capita converted at purchasing power parity exchange rates. These variables are also used to calculate the Human Development Index (HDI); however, the GDI adjusts the values for gender equality. The GEM combines income shares, professional opportunities and participation in economic decision making and parliamentary participation as shares of parliamentary seats for both males and females. A common feature for both indicators is that they combine absolute values for the considered indicators with a penalty for inequality. A common critic of these indicators is the overweighted income variable, as GDI is strongly correlated with the absolute level of income. GDI and GEM, therefore, may underestimate gender inequality in richer countries.

Section II: Income inequalities and HIV/AIDS epidemic

The relationship between poverty and HIV/AIDS transmission is more complex that it first appears. Although it is generally admitted that it is poverty that causes AIDS, it is also noteworthy that there are rich countries that are badly affected by the epidemic and within a particular country, the most affected people are not always the poorest. Even among infected people, there is inequality between rich and poor; infected poor people are susceptible to develop AIDS earlier than the rich ones due to malnutrition and poor access to treatment for opportunistic infections and even to antiretroviral therapy. HIV/AIDS infection is not randomly distributed in the population. Indeed, though it seems to affect more people from higher socio-professional categories at the onset of the epidemic, it is thereafter fuelled by poverty like any other infectious disease. Income inequality favours high-risk behaviour, thus creating conditions that encourage the spread of HIV/AIDS. In addition to the broad links between poverty, inequality and health, HIV/AIDS epidemic carries a set of specificities that magnify these links. Furthermore, Sub-Saharan Africa exhibits some peculiarities that intensify the relation between HIV/AIDS, poverty and income inequality. In this way commercial sex work and migrant work, two income generating activities to cope with poverty acted as catalysts of the epidemic.

The risky behavior adopted by commercial sex and migrants workers do not appear justified *a priori* but became pertinent if one bears in mind that it can be rational for people who are uncertain about their future to adopt a risky behavior that yields an immediate utility[64]. Indeed, sex workers in precarious situation maximize a welfare function, whose argument depends on the expected revenues from sex work and future health status. If one further considers that even for

[64] Refer to Appendix A.1 for a model of inter-temporal choice, which explains risky behaviour from people facing financial pressure. This model is suitable for the understanding of commercial sex and migrant work, even in a setting where HIV/AIDS prevalence is high.

the most skeptical the existence of the virus is not certain or that they perceive the risk of infections as low, then the adoption of a risky behavior seems to be an optimal solution. Moreover, the long delay between the infection with HIV/AIDS and the onset of full-blown AIDS reduce the belief in its real and actual hazard.

I- Commercial sex work

Poverty and wealth inequality between men and women can fuel HIV transmission as women engage in unsafe sex in exchange for money, housing, food or education. Heterosexual intercourse is the main mode of transmission of HIV/AIDS in Africa. Meanwhile commercial sex work, a survival strategy directly linked to poverty and income inequality contributes to its spread. Commercial sex work is usually the only income generating activity available to women in a setting where unemployment rates especially among women are high. In almost all countries with a generalized epidemic, sex-workers are among the main high-risk groups identified. Sex work has a big dissemination power in total population because the clients do not usually use condom during sexual intercourse with their life partner, regardless whether they use it or not in their commercial relationships. Thus prevention efforts among that group would be the most efficient, as they would prevent the infection of a big part of population (Ainsworth and Over, 1997).

Two basic requirements for the onset of commercial sex work are fulfilled in Sub-Saharan Africa: a concentration of a sexually active population and, more importantly, the socioeconomic disparities that make sex work affordable by the client and an economic opportunity for the worker. Poverty experienced by women and men in developing countries has been deepened by increasing global economic inequalities. But unequal gender relations and unequal access to economic resources have made women poorer than men. Sex work appears to be fostered when a demand for sexual services and a favourable setting coexist. Sex workers are also more subject to sexual violence which increases the risk of virus transmission.

Transactional sex, which is exchange of sexual services for money, gifts and protection, is even more common than sex work and people engaged in this kind of transaction are not always aware of the situation. Thus, most young girls attached to "sugar daddies" qualify themselves as "resourceful" rather than sex workers. Although many women do not view themselves as sex workers, most of them have at one moment in their life resort to transactional sex, that is using sex as a commodity in exchange for goods, services, money, accommodation, or other basic necessities, too often with older men (Halperin and Epstein, 2004). A high proportion of unmarried adolescents aged 15 to 19 years engage daily in sex for money or goods in almost all

parts of sub-Saharan Africa. Intergenerational sex and transactional sex are frequently intertwined. Research in parts of Africa, for example, has found that older men often help girls' families meet essential needs such as school fees, transport costs and groceries (Buve and al 2002; Luke and Kurz 2002). The dependencies built into these relationships limit women's abilities to protect themselves from HIV infection, especially when the perception of younger women as 'pure' encourages men to avoid using condoms. Indeed, older men often seek younger girls because they are thought to be free of the infection.

The culprit with both sex work and transactional sex is unprotected sex, as women (and increasingly men) engaged in these activities are not often in position to negotiate safe sex. In this way, sex work is likely to put women at more risk of HIV infection because clients are ready to pay more for unprotected sex than for protected sex (Gertler et al, 2005).

Following are some relevant facts about unsafe sex and commercial sex work in Africa: (1) in many countries, sex workers are not routinely treated for STIs. While STIs' presence is known to be a cofactor of the infection, their prevalence is higher among sex workers in Sub-Saharan Africa than in the whole population. Senegal, the only country which succeeded in containing its prevalence rate at less than 1% of adult population has targeted its intervention among sex workers only. They were offered condoms and prevention campaigns and treated for STIs, (2) it has been assessed that only 36 % of sex workers in Sub-Saharan Africa have access to prevention services against HIV/AIDS (United nations, 2006), (3) in a setting where young girls offer sexual services in exchange of financial protection, they are not able to impose the use of condom to their partners, even when they are confident in its efficacy in protecting them against the infection.

II- Migrant work

Because there are some countries or regions within countries that are more developed, richer than other, people, especially young working age people move to these regions in their search for employment. In general, accelerated development of a region relative to its neighbors will lead to an increase income inequality between them and contribute to regional migration. And within a country, income inequality between rural and urban areas will generate rural exodus especially of young and unmarried people in search for a better future to the cities. This displacement of rural dwellers is partly responsible for higher infection rates in the cities relatively to the villages as these rural newcomers are exposed to the same risks as migrant workers. Indeed, economic migration is driven by opportunity gaps between richer and poorer regions and lower transportation costs in travelling to a closer region. The epidemiological relationship between

migration and HIV is well established. Kane et al, (1993) found that 27 percent of the men who had previously travelled in other African countries and 11.3 percent of spouses of men who had migrated were infected with HIV in Senegal. In neighbouring villages where men had not migrated less than one percent of the people were HIV positive. High HIV prevalence rates in areas of high out-migration have also been documented in Mexico (Santarriaga et *al.*, 1996), Senegal (Pison et *al.*, 1993), but also in Ecuador and in southeast Ghana (Decosas, 1996). Brockerhoff and Biddlecom (1999) found that the incidence of HIV in Kenya is higher near roads, and amongst people who either had a personal migration experience or have sexual partners who are migrants. In Southern and West Africa, Anarfi (1993) found that migrant workers (and their sexual partners) have higher levels of infection than the general population. Itinerant traders and long-distance truck drivers are also at increased risk to contract HIV infection, as evidenced by Wilson et al (1993). Border towns have high rates of HIV prevalence, being places where transient populations such as truck drivers encounter a more stable local population, and which are often not reached by national HIV and AIDS intervention programmes.

The current distribution of the HIV epidemic across the continent is also the clue to its link with mobility. The highest incidence is not in Africa's poorest countries, but in Southern African countries such as South Africa and Botswana which have a good transport infrastructure, relatively high levels of economic development, and considerable internal and cross-border migration. The contribution of migrant work to the spread of HIV/AIDS epidemic is the result of both recent trends in migration and specific risks associated with some kinds of migrant works.

A- Recent trends in African migration

The report "HIV/AIDS, Population Mobility and Migration in Southern Africa Defining a Research and Policy Agenda" points that it is difficult to define clear trends in African migration. It nevertheless identifies the following as general migration trends for the past two or three decades:

1) Rapid urbanization:

In 1970, Africa's urban population was around 82 million people, or 23 per cent of the continent's population; by 2000, 295 million Africans, or 37 per cent of the population, lived in urban areas.

2) Changing geography of migration

To long-established sources, routes and destinations, new ones have been added. For example South Africa has seen a significant increase in international migration since the end of Apartheid, with people coming into the country from all over the African continent.

3) Increased access to transportation

Increased transportation has improved personal mobility and enabled movement across longer distances and to a wider range of destinations. It has also enabled migrant workers to return home more frequently. As a result, on the one hand this could lessen the pressures for migrants and their spouses to engage in extra-marital relationships. On the other, it means that the rural areas have become far less "insulated" from HIV than before.

4) Political instability and environmental catastrophes

At different times in different countries, political turmoil has led to internal as well as international displacement of people. Environmental catastrophes and famines have also led to major episodic human migrations.

5) Migration of skilled workers

Through the brain drain, Africa faces a significant loss of its skills base as skilled professionals take advantage of opportunities arising from globalization and immigration policies of industrial countries. The brain drain is depleting the skills base of African countries. Its impact is particularly acute in the public health sector where migration is undermining the ability of health systems to cope with the burden of HIV/AIDS.

6) International migration

Some movements of people take place within national borders, but there is also significant and increasing international migration, both across borders within Africa and from Africa to countries outside the continent, especially in Europe.

7) Feminization of migration

According to the International Organisation for Migration (IOM), women make up almost half of the world's 185 million migrants. While women move in order to escape economic deprivation, lack of education often restricts them to unskilled jobs such as commercial sex work, informal trading, agricultural labour, or domestic work. Indeed, as women can expect to be compensated for sex outside their primary relationships, they are more likely to engage in transactional sex and sex work. They are also more likely to becoming victims of trafficking into the sex industry. A field study in southern Africa conducted jointly by Care International and the

International Office of Migrations (IOM) suggests that migrant women who work as traders or agricultural labourers supplement their incomes with transactional sex. Migrant women are vulnerable to sexual harassment, coercion, and violence. For example, Jewkes and Abrahams (2002) found that informal traders have been exposed to sexual harassment and rape by officials when crossing borders, and by truckers or taxi drivers while travelling to and from markets and other sales sites. In fact, migrant women regularly use sex as a tool to obtain food, transport, or leniency.

B- Risks associated with migrant work

Migrant populations are more vulnerable than native populations as they are subject to discrimination, xenophobia, exploitation and harassment. In addition, they have no or little access to legal resort or social protection; they are also more difficult to reach, whether for prevention education, condom provision, HIV testing, or post-infection treatment and care. Besides, as they usually don't speak the national language of their host country, they have less access to information on HIV/AIDS, health services and STIs. Finally, migrant populations play a central role in the transmission of the virus from the host country to their own country when they return home.

It is not so much movement per se, but the social and economic conditions that characterize migration processes that put people at risk for HIV. With their wages, usually ready availability of liquor and peer support, migrant workers can easily induce women into either short or longer-term sexual partnerships (Collins and Rau, 2000). Migration can also be a risk factor not simply because men return home to infect their rural partners, but also because their rural females partners – both those who are partners of migrants and those who are partners of nonmigrants – are likely to become infected in the rural areas from outside their primary relationships, as evidenced by Crush (2004).

While the risks highlighted above account for any work migrants do, there are specific risks associated with the occupation in which they are involved. Indeed, mine, commercial farms, transport and domestic work carry a specific risk. So do also informal trade and military work. Southern African migration makes the bulk of the literature on the links between migrant work and HIV/AIDS epidemic.

1) Mine workers

The South African gold mining industry employs migrant mine workers from South Africa, Botswana, Mozambique, Lesotho and Swaziland. Because most of these migrant men live almost exclusively in single-sex hostels, without their wives or families, they are seen as being at high risk

of contracting STIs, including HIV (Crush et *al*, 2002). Campbell (1997) found that for mine workers, working away from their families in South Africa, drinking and sex were two of the few diversionary activities easily available on a day-to-day basis. One informant in her study commented that the risk of HIV/AIDS appeared minimal compared to the risks of death underground, and suggested that this was the reason why many mine workers did not bother with condoms. In addition, the prevalence of commercial sex and alcohol-related business supported by miners create conditions that render other groups in the surrounding communities more susceptible to infection as well, as showed by Jochelson et *al* (1998) and Steen et *al* (2000).

2) Commercial farm workers

South African commercial farms employ significant numbers of migrant workers from other Southern African countries, mainly from Mozambique, Zimbabwe and Lesotho. Siziya et al (1999) revealed that workers are confronted with difficult basic conditions: poor pay and exploitative working conditions, but also overcrowded accommodations, poor sanitation, long absences away from home, boredom, limited recreational opportunities, and a meager hand-to-mouth existence with little hope for the future. Within this context, HIV is seen as a distant threat. The study also revealed a lack of access to information, high levels of misconceptions about HIV and AIDS and high levels of reported risky sexual behaviour. A significant number of workers reported having two or more concurrent sexual partners, and there were indications that during the harvesting season, transactional sex increases dramatically at the farm compounds.

3) Transport Workers

The road freight industry within Southern Africa has grown exponentially and has played a significant role in the spread of HIV in Southern Africa. In Mozambique, for example, provinces with major transportation arteries have higher HIV prevalence rates than those that do not (IOM and PHAMSA, 2005). More important than the movement itself is the opportunity to find sexual partners along major transportation corridors. Truck drivers frequently have partners at stops along their route which provide a social network for them, and which they help to support financially (Laukamn-Josten et al, 2000). The wives that are left behind by the truck drivers may also have sex with other partners in their husbands' absence. Ramjee et al studied sex workers and their truck driver customers along a South African trucking route and found that about 56 per cent of both the truck drivers and sex workers were infected with HIV. Some 66 per cent of the truck drivers reported having an STI in the past six months.

4) Domestic Workers

Domestic work includes cleaning, cooking, and caring for the young and elderly. Domestic workers represent a vulnerable group because they often work in social isolation, have low levels of education, and are exposed to poor working conditions, which include lack of privacy and low wages. Few domestic workers have any kind of written contract, paid leave, benefits or medical aid, as highlighted by Peberdy and Dinat, their vulnerability also lies in their social isolation and vulnerability in the workplace. Domestic workers are likely to be at increased risk of HIV infection because of a number of factors including gender inequality, social isolation, poverty, low levels of education, lack of access to health care services and medical aid. The living conditions of domestic workers and their status as migrants could have a serious affect on their health, in particular their reproductive and sexual health. On the other hand, the socially isolated lives of many domestic workers may reduce their potential vulnerability.

5) Informal traders

Informal trade provides an income earning opportunity for entrepreneurs around Southern Africa, and appears to comprise a significant proportion of intraregional trade. In general the majority of informal cross border traders are female, relatively young and single. As an example, a study carried out by Nyatanga et *al* (2000) indicated that eighty percent of traders going to South Africa from Zimbabwe were women. Women may are particularly vulnerable to sexual assault and extortion, including demands for sexual favours from officials at border crossings or truck drivers, whereas male informal traders face many of the same circumstances that render truck drivers vulnerable to HIV infection, including long periods away from home, lack of access to health care, and extended waits at border crossings, where the only affordable accommodation is often in the homes of sex workers (IOM and PHAMSA, 2005).

6) Military personnel

Most armed forces personnel are young men and women in their 20s and 30s and, as such, they represent one of the professional groups most affected by HIV and AIDS. Uniformed services personnel generally have an ethos of risk taking that can place them at higher risk of HIV infection (IOM and PHAMSA, 2005). Soldiers and peacekeepers are often posted away from their families and communities for long periods of time, removing them from the social discipline that would normally prevail in their home communities. During conflict, both consensual and non-consensual sexual encounters tend to increase, and adherence to prevention measures declines (UNAIDS, 2004).

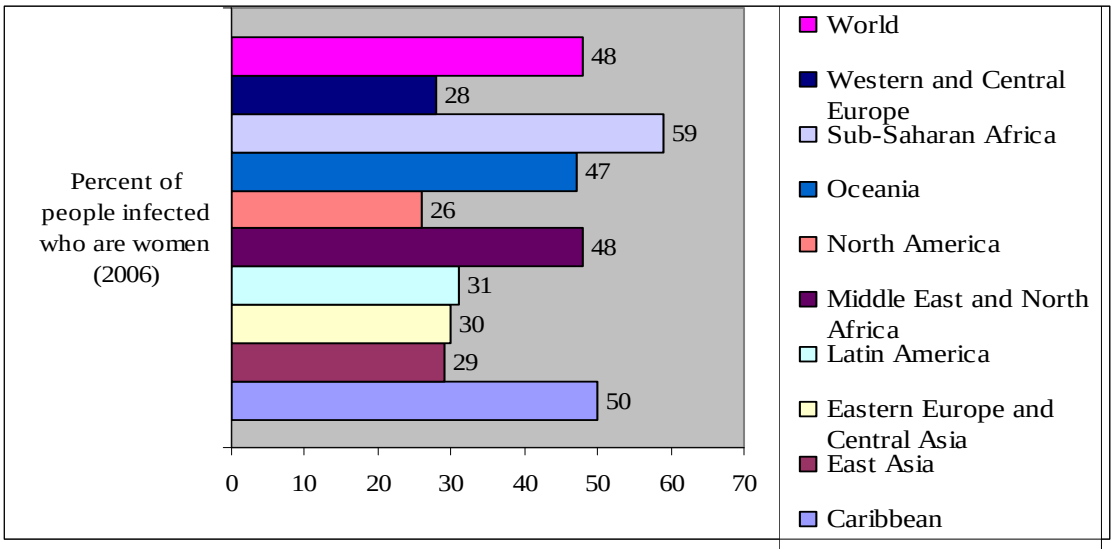
Section III: Gender inequality and HIV/AIDS epidemic

Men and women are differentiated by social -or gender- characteristics on the one hand and by biological -or sex- characteristics on the other. The distinct roles and behaviours of men and women in a given culture, dictated by that culture's gender norms and values, give rise to gender differences, most of which are in fact gender discrimination, that are differences between men and women which systematically empower one group to the detriment of the other. Gender roles are learned and can be affected by factors such as education, culture or economics. They vary widely within and among cultures, are slow to change, but they are not static. In no region of the world are women treated as equal as men. Although women's economic situation has been improving, there are still significant inequalities between women and men, and there is evidence that these inequalities play a role in increasing both exposure risk and vulnerability to HIV/AIDS.

Though the epidemic in the world initially affected mostly men, today approximately half of the 40 million people living with HIV are women. This is due to the fact that it spread rapidly in Sub-Saharan Africa where transmission is primarily heterosexual. In addition, there is a discrepancy in the distribution of the epidemic across both sexes between Sub-Saharan Africa and the world average; women now make sixty percent of infected people in Africa. More importantly, young girls constitute two thirds of new infections (UNAIDS, 2006). This distribution of the epidemic across the sexes is the result of direct exposure to the virus (due mainly to biology), but also of the lower socio-economic and socio-cultural statuses experienced by women on the continent.

The economic vulnerability of women makes it more likely that they will exchange sex for money or favours, less likely that they will succeed in negotiating protection, and less likely that they will leave a relationship that they perceive to be risky. Women have greater susceptibility than men to infection due to biological, social, cultural and physiological reasons. The pattern of women's and men's roles and relationships in Africa puts women at greater risk. In this way, polygamy, sexual coercion and violence against women all contribute to the distressing gender gap in HIV/AIDS infection. Figure 3.2 illustrates the gender gap in HIV prevalence in selected regions while Table 3.2 depicts how the difference between Sub-Saharan Africa and the world average has evolved over time.

Figure 3.2: Percent of Adults (15-49) Living With HIV Who Are Female, 2006



Source: UNAIDS data (AIDS epidemic Update 2006).

Table 3.2: Percentage of women among infected people

	1997	2000	2002	2003	2005
Sub-Saharan Africa	50	55	58	59,8	60
World	43	46	47,2	49,7	50

N

Note: Author's calculations from UNAIDS data (AIDS epidemic Updates 2000, 2002, 2003, 2005 and 2006).

I- Gender and development

Institutions, households, and the economy are the factors standing in the way of transforming gender relations and eliminating gender inequalities. Societal institutions (social norms, customs and rights), laws as well as economic institutions, such as markets, shape roles and relationships between men and women and influence what resources women and men have access to, what activities they can or cannot undertake, and in what forms they can participate in the economy and in society. Like institutions, households play a fundamental role in shaping gender relations from early in life and in transmitting these from one generation to the next. But families make decisions in the context of communities and in ways that reflect the influence of incentives established by the larger institutional and policy environment. Finally, because the

economy determines many of the opportunities people have to improve their standard of living, economic policy and development critically affect gender inequality.

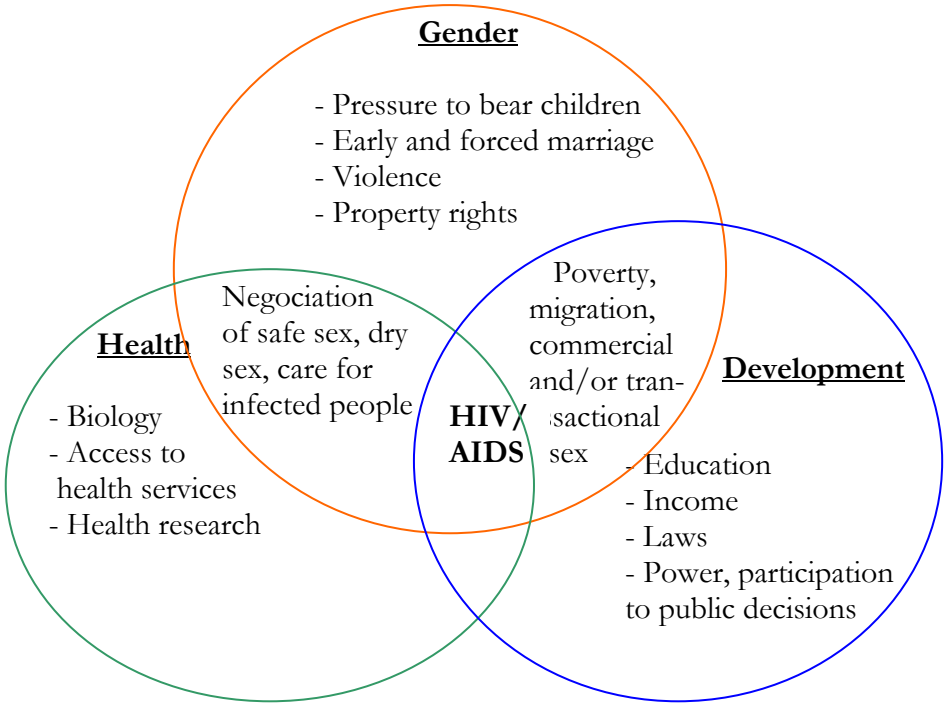
Gender gaps are widespread in access to and control of resources, in economic opportunities, in power, and political voice. While disparities in basic rights; in schooling, credit, and jobs; or in the ability to participate in public life take their most direct toll on women and girls, the full costs of gender inequality ultimately harm everyone. For these reasons, gender equality is a core development issue, a development objective in its own right. It strengthens countries' abilities to grow, to reduce poverty and to govern effectively. In many societies, women have fewer educational opportunities than men and receive unequal access to resources such as food and income (Sen 1988), all of which are strong predictors of health status. Finally, even in settings where women have access to the resources they need to improve their health, power relations in the household as well as social norms often prevent them to make good use of what is available to them. Most women have access to fewer resources than males in the same social situation than themselves and these inequalities clearly have a major impact on their health. Essentially, abstinence is often not an option for poor women and girls who have no choice but to marry at an early age. Being faithful will not protect a woman whose partner is unfaithful and using condoms is not a decision most woman can make independently of her partner.

The discourse of development is being fundamentally transformed by the introduction of gender analysis. Given the fundamentally different social and economic roles of men and women, their different economic behaviours, including different patterns of consumption and expenditure, and their differential access to income and resources, it is no longer effective to construct an analysis of an economy or a society, at micro or macro levels which is not gender-sensitive. Even in societies where there is *de jure* equality (for example, of pay or employment or property rights), these differences nevertheless remain as significant features of economic and social life. Gender disparities are inextricably linked to poverty. On the one hand, poverty exacerbates gender disparities; inequalities between girls and boys in access to schooling or adequate health care are more acute among the poor than among those with higher incomes (World Bank 2001). On the other hand, gender inequalities hinder development. Gender inequalities harm well-being of men, women, and children, and affect their ability to improve their lives, thus hindering development. In addition to these personal costs, gender inequalities reduce productivity in farms and enterprises and thus lower prospects for reducing poverty and ensuring economic progress. Gender inequalities also weaken a country's governance and thus the effectiveness of its development policies. Moreover, gender biases and distortions are increasingly seen as not simply inequitable, but also inefficient, in that they may hamper effective

adjustment and women's ability to escape from poverty by limiting labour market opportunities. The latter are limited because of lesser labour endowments (education, skills etc) and/or socio-cultural biases limiting their labour market participation. Finally, gender inequality undermines the effectiveness of development policies in fundamental ways, acting through three powerful tools for development: education, income and participation to public life.

HIV/AIDS epidemic lies at the intersection of gender, development and health issues, as represented in Figure 3.3.

Figure 3.3: HIV/AIDS in gender, development and health nexus



Source: Author

II- Direct exposure to the virus

Women are more exposed to virus transmission than men due to physiological reasons and cultural.

A- Biology

Biologically, the risk of HIV infection during unprotected vaginal intercourse is two to four times higher for women than men. This is because women have a bigger surface area of mucosa exposed to their partner's sexual secretions during intercourse. Semen also contains a higher concentration of HIV than vaginal secretions. Last, semen can stay in the vagina for hours after intercourse. In addition, women are also more likely than men to have other STIs, which can

increase the risk of HIV infection by three to four times, but STIs are more frequently asymptomatic in women than in men, and when symptoms do occur in women, they are more subtle. Young women are even at greater risk as their physiologically immature cervix and scant vaginal secretions put up less of a barrier to HIV.

B- Practice of dry sex

Second, there are some traditional sexual practices that are especially dangerous to a woman's health. The practice of dry sex, common in Southern Africa, is harmful to women and put them at greater risk of infection. Dry, abrasive vaginas are seen as desirable in sexual intercourse in the vast majority of southern African cultures. Dry vaginas that are swollen with friction are perceived to enhance men's pleasure. Furthermore, many believe that naturally lubricated vaginas are evidence of infidelity (Nyirenda, 1992). Yet, dry sex promulgates HIV/AIDS in three ways: the lack of lubricant results in lacerations in the delicate membrane tissue, making it easier for the lethal virus to enter. In addition, the natural antiseptic lactobacilli that vaginal moisture contains aren't available to combat sexually transmitted diseases. Third, condoms break more easily due to the increased friction.

C- Care for an infected relative

Finally, while this route is very rare, there is little evidence of contamination during daily life with an infected person (CDC 1999); indeed the vast majority of women and girls who care for infected family members are at risk, doing so with very little material support and this might result in their own infection with the virus (Ogden and Esim, 2003). They receive no training, no formal materials such as gloves, medication. Home care programmes are critically short of kits containing gloves, soap, disinfectants and other basic necessities.

III- Women's lower socio-economic status

The challenge of gender interacts with the problem of poverty. Women are generally poorer than men, in part because they are less educated on average, but also because there are gender discriminations in the way wages are set (Baliamoune, 2005). They are relatively powerless when confronting husbands, boyfriends, employers and other men. The combination of poverty and inequality compounds the fundamental problem of gender relations. The economic vulnerability of women makes it more likely that they will exchange sex for money or favours[65]. Violations of women's rights heighten their vulnerability to AIDS. Women's subordinate position in society, discriminatory property and legal rights, are just some of the factors that sustain the escalation of

[65] The links between commercial sex work and HIV/AIDS epidemic have already been examined in the previous section on income inequality and HIV/AIDS epidemic.

the pandemic. Also, health services, especially HIV/AIDS prevention services do not always properly address the issue, which is still considered taboo in many countries.

A- Denial of women's property rights and inheritance laws

Women's vulnerability to HIV is further exacerbated by unequal property and inheritance rights. Women face additional obstacles due to the pervasiveness of discriminatory legal frameworks which fail to guarantee equal rights or equal protection before the law. In many cases, inequitable divorce and property laws make it difficult for women to leave abusive relationships. In much of sub-Saharan Africa, property is usually owned by men, with women occasionally acquiring rights mainly by virtue of marriage. Multiple legal regimes overlap in many African countries, incorporating old colonial laws, more recent constitutional law, and ongoing customary law. Shifts in customary law during colonial rule in southern Africa, for example, have meant that the law is often interpreted to the detriment of women. Colonial administrations superimposed concepts of private property and a rigidly patriarchal system on traditional property dispensations. This helped transform the principle that men administer and inherit property to the benefit of the clan into claims of individual property ownership without corresponding.

The payment of bride-price upon marriage tightens men's control over women and property; in some countries women remain legal minors even after marriage (Human Rights Watch, 2003). The outcome is a status quo that often fails to recognize or uphold women's property rights, that reduces women's economic security and can lead to women having to endure abusive relationships or resort to sex for economic survival. In some countries, women whose male partners die of AIDS (many of whom are already infected with HIV) are subjected to property stripping by their spouses' relatives, they may have no legal rights to land and property (due to customary inheritance laws or the difficulties of enforcing existing remedial legislation) after their husbands' death. Impoverishment may force them to send some of their children away, engage in occasional sex for money or earn a living as commercial sex workers.

B- Lack of information: failure of HIV/AIDS prevention services

Because sex is a taboo topic in many countries, large numbers of young people do not get sufficient information or the skills to refuse sex or negotiate safer sex practices. That lack of knowledge magnifies their risk of HIV infection. While most young people have heard about HIV/AIDS, few know enough to protect themselves against infection. Surveys from 40 countries indicate that more than half of the young people have misconceptions about how HIV is transmitted. In Botswana, where one in three people is living with HIV/AIDS, virtually all young people have heard of AIDS and more than 75 per cent know the three

primary means of protection. Still, 62 per cent of girls had at least one major misconception about how HIV is spread. Surveys in Cameroon, Lesotho, Mali and Senegal indicated that two thirds or more of young women (aged 15 to 24 years) did not know three HIV prevention methods (UNAIDS 2005).

In many countries HIV/AIDS information and services are provided primarily through family planning, prenatal and child health clinics, which are typically not designed to reach men or meet men's needs. As a result, men may be less likely than women to receive HIV/AIDS information, counselling and treatment services. Moreover, girls and women may be reluctant to seek advice, for fear of stigmatization. They may also be deterred by the unhelpful and discriminatory attitude of staff in some centres.

IV- Women's lower socio-cultural status

The pattern of gender roles and relationships in Africa puts women at greater risk. In this way, violence and sexual coercion against women contribute to the distressing gender gap in HIV/AIDS infection. Gender role also make it less likely that they will succeed in negotiating protection, and that they will leave a relationship that they perceive to be risky. In addition, patterns of marital life in Sub-Saharan Africa and some practices associated with widowhood further increase women's vulnerability to the epidemic.

A- Violence and stigma

Many women experience violence in their daily life, and this violence increase their risk of becoming infected with HIV. In addition, stigma surrounding HIV/AIDS makes it difficult, if not impossible to discuss about the issue, whether to ask for prevention or to disclose one's status.

1) Violence

As well as economic and social insecurity, many women also have to face the threat of physical violence if they are not sufficiently responsive to a partner's desires. Under these circumstances, many will prefer to risk unsafe sex in the face of more immediate threats to their well-being. Violence in the form of coerced sex or rape may also result in the acquisition of HIV, especially as coerced sex may lead to the tearing of sensitive tissues and increase the risk of contracting the HIV virus. Studies in adolescent girls from several countries have found that an important proportion of them report that their first intercourse was forced. Young women and girls are at greater risk of rape and sexual coercion because they are perceived to be more likely to be free from infection, or because of the erroneous but widespread belief in some regions that sex with a virgin can cleanse a man from infection. Young women, too, may view sexual violence or sex that

is obtained through force, fear or intimidation as normal, reflecting perverse gender norms in some communities or societies. One South African study found that sexual violence and coercion against young girls was so widespread it was referred to as 'everyday love'. It is not only a coincidence that the country was the one with the highest number of infected people; it is also notorious as the country with the highest rate of rape in the world.

Around the world, as many as one in every three women has been beaten, coerced into sex, or abused in some other way — most often by someone she knows, including by her husband or another male family member. Gender-based violence both reflects and reinforces inequities between men and women and compromises the health, dignity, security and autonomy of its victims. For many women worldwide the threat of violence that permeates their everyday lives exacerbates their vulnerability to HIV. Fear of violence prevents women from accessing HIV/AIDS information, being tested, disclosing their HIV status, accessing services for the prevention of HIV transmission to infants, and receiving treatment and counselling, even when they know they have been infected. This is particularly true in settings where HIV-related stigma remains high. Both HIV negative and HIV positive women face violence in day-to-day life, but HIV positive women are at greater risk because of their HIV-positive status. Gugu Dlamini, the first women to disclose her HIV positive status in South Africa was beaten to death in December 1998. Yet she lived in Kwa-Zulu Natal, a region where HIV prevalence rate at that time was estimated at 20-30% of adult population.

2) Stigma and fear of abandonment

Both women and men living with HIV/AIDS experience discrimination and stigma. However, there are gender differences in the way stigma affects women and men. Women may be more affected by stigma and discrimination than men because of social norms concerning acceptable sexual behaviour in women, and because women are often more economically vulnerable than men. The shame associated with AIDS is a major obstacle to its prevention, and the stigma that surrounds people living with HIV is compounded by discrimination against women. Hundreds of thousands of HIV-positive women avoid testing and treatment services for fear of abandonment and other repercussions from husbands, families, communities and health providers. In a 2001 survey conducted in Kenya (WHO 2003), more than half of the women surveyed who knew that they were HIV positive had not disclosed their HIV status to their partners. Indeed, disclosure of their status would expose them to violence or abandonment. Lack of confidentiality in testing services is a well-grounded concern, so that women sometimes discover their HIV status last, long after their husbands or in-laws. Only 5 per cent of HIV-positive people are aware of their

status, and testing during pregnancy is often the only way that a family learns of HIV. Even if they contracted HIV from their husbands, women are sometimes blamed for bringing AIDS home[66] and may face violence or ostracism as a result. Health care providers sometimes deny HIV-positive women proper care during and following delivery.

Studies conducted in Southern Africa found that many young women knew little about their bodies, contraception and STIS. Many reported a fear of seeking information on sex or condoms, as this would label them as sexually active, regardless of the true extent of their sexual experience. Stigma associated with HIV/AIDS is a major factor preventing many women and men from accessing services (WHO, 2003). VCT and HIV/AIDS-related services may deter women and young people from accessing care since, in such cases, use of services may be seen as tantamount to an admission of having a sexual infection, and thus lead to stigmatization.

B- Negotiation of safe sex

When women and girls have sexual relations, it tends to be with older men, increasing the likelihood that their partners are already infected. Once in these relationships, girls have little power to negotiate the use of condoms. Social norms frequently hold that it is the male's responsibility to acquire condoms and for a young woman, to carry condoms suggests that she intends to have sex (Childhope, 1997). At the same time, the prevailing norms in many settings dictate that since reproductive and sexual health are 'female' concerns, women must be the ones to suggest contraceptive use. Physical violence, the threat of violence, and the fear of abandonment act as significant barriers for women who have to negotiate the use of a condom, discuss fidelity with their partners, or leave relationships that they perceive to be risky. Some women experience the threat of, or actual, physical violence when attempting to negotiate safer sex through the use of condoms. Much of the resistance to condom use encountered by condom promotion programmes is gender-related. While it is estimated that perfect use of the female condom may reduce the annual risk of acquiring HIV by more than 90 % among women who have intercourse twice weekly with an infected male, the price of the female condom (4–10 times that of male condoms) renders it inaccessible to most women.

Within marriage it is particularly difficult for women to negotiate condom use, especially if wives are much younger than their husbands. It is especially difficult, if not impossible to negotiate condom use in the following settings: adolescent girls married with older men, women who experience physical or sexual violence, women who are economically dependant on their partner, newly wed women pressured to produce a child in order to be accepted by husband and in-laws,

[66] On that subject, see Le Palec. 1999. Le Sida, une maladie de femmes.

sex worker with clients paying more the usual rate for unprotected sex. Finally, abstinence messages have reinforced the perception that to use a condom is a 'sin,' implying that the girl has actively agreed to have sex and is therefore a prostitute.

C- Patterns of marital life

The first pattern of marital life that puts women at greater risk of infection in Sub-Saharan Africa is polygamy. Polygamy, the marriage of a man to more than one wife, is inherently discriminatory, and, because the man may be having unprotected sex with multiple partners, exacerbates the risk of HIV transmission. Furthermore, the payment of a "bride price or dowry by a man to a woman's family, which is more frequent in case of polygamy makes the woman her husband's property and denies her any authority in marriage, including over sexual practices.

Another characteristic of marital life in African countries is early marriage for girls. In many countries, including several with high rates of HIV infection, girls are married in their teens, often as a poverty reduction strategy at household level. Early forced marriages for girls create high risks of HIV infection. Husbands and families also apply considerable pressure on young wives to have a child soon after marriage, increasing their risk of maternal death or injury and hampering efforts to prevent STIs and HIV through regular condom use. At this relatively early stage in their physical development, girls are particularly susceptible to HIV infection. Adolescent girls are likely to be married to older men with more sexual experience who are more likely than single men to be HIV-positive and who are also less predisposed than younger males to use condoms. In Kisumu, Kenya, 30% of male partners of married adolescent girls were infected with HIV. Unmarried but sexually active girls are more likely to have relationships with younger men, who are more inclined to favour condom use. There is evidence that the age gap between partners affects the chances that young women will become infected (Kelly et al. 2003, Luke 2003). Thus, in southern Africa women and girls often get infected with HIV almost at their sexual debut. In a study in Zambia, 18% of women who said they had been virgins a year before being tested for HIV were found to be HIV-positive, while in South Africa, 21% of sexually active girls 16–18 years of age tested HIV-positive. HIV prevalence was approximately 16% among teenage girls (15–19 years) in rural Zimbabwe whose last partner was less than five years older than themselves, but among girls with partners 10 or more years older, HIV prevalence was twice as high (Gregson et al, 2002). In Kisumu, Kenya, among women three years or less the junior of their husbands, none was found to be infected with HIV, but half the women with husbands 10 years or more their senior were HIV-positive (UNFPA, 2005).

Third, gender norms increase susceptibility to HIV infection, especially the views about manhood and womanhood. Simply by fulfilling their expected gender roles, men and women are likely to increase their risk of HIV infection. In almost all cultures masculinity is associated with virility. On the other hand, feminity is associated with softness and pureness. Women infidelity is highly condemned, often resulting in the woman being ostracized while men having multiple sex partners are viewed as virile, bold and daring. A report based on research conducted in seven countries (Cambodia, Cameroon, Chile, Costa Rica, Papua New Guinea, the Philippines and Zimbabwe) found that notions of masculinity encourage young men to view sex as a form of conquest (WHO, 2003). Besides, since ignorance is construed as a sign of weakness, men are often reluctant to seek out correct information on HIV/AIDS prevention. Also, they may be under pressure to keep their HIV infection status secret for fear of dismissal from work, and of being unable to play their traditional gender roles as breadwinners. In this context, simply being married is a major risk factor for women who have little or no control over abstinence and condom use at home or husband's sexual activity outside. Married women are more likely to contracting HIV/AIDS from their homes, than sex workers who are normally considered to be at high risk. Cultural beliefs and religious convictions oblige wives to be respectful of and obedient to their husbands even when there is enough ground to suspect infidelity. As an illustration, in two districts of Uganda, only 26 per cent of women said it was acceptable for a married woman to ask a husband to use a condom (UNFPA, 2005).

D- Window inheritance and ritual cleansing

Wife inheritance and cleansing rituals are customary practices common in some communities in sub-Saharan Africa. In wife inheritance, a male relative of the dead husband typically marries the widow to ensure that the children remain within the late husband's clan and that the widow and her children are provided for. The deeply rooted tradition of widow inheritance is widely practiced by Luo groups in Uganda, Tanzania, Zaire, and Sudan. A study conducted by Luke (2002) in the Luo community of Kenya points out that this practice of sexual networking whereby men who inherit widows have multiple sex partners, high frequency of exchange between widows, and low levels of condom use, however, encourages the spread of HIV.

In some forms of ritual cleansing, a widow has to have sex with a social outcast who is paid by the dead husband's family[67]. Wife inheritance and ritual cleansing are closely related to women's property rights in that many widows are not allowed to stay in their homes or on their lands unless they succumb to these practices and in both practices, condoms are seldom used. These

[67]This practice is supposed to cleanse the woman of her dead husband's evil spirits. It is widely believed that if this is not observed the spirit of the dead man will be visiting those living to make demands (Luke 2002).

practices are allegedly protective for women since they gain the legitimacy and security of being in a male-headed household. The other side of the story is that these practices are predatory and exploitive (in that the 'protector' gains the widow's labour and her deceased husband's property) and contribute to the spread of HIV/AIDS.

Conclusion

This chapter aimed at shedding light on how income and gender inequalities could favour HIV/AIDS epidemic. In spite of the relatively evident theoretical links between income inequality and health, the analysis of these links is not an easy task. First, inequalities measures are numerous and do not carry the same content, there is a choice to make according to the sense one wants to give to his analysis. Second, income inequality cannot be analyzed independently from poverty and growth, as these three notions are inextricably linked. Finally, gender inequality is not a homogeneous phenomenon and it can only be studied through some of its overt manifestations, such as difference in education and in income and participation to labour market between males and females.

The channels through which inequality affects health are access to material conditions, social cohesion and psychosocial factors. Moreover, in Sub-Saharan Africa, there are aggravating factors such as commercial sex work and migrant work, which magnify the links between income inequality and health, thus creating an unprecedented epidemic in the continent.

Our second aim was to show how gender inequalities could favour HIV/AIDS epidemic. The relationship between gender inequality and HIV/AIDS epidemic is embedded into the nexus made of by gender, health and development. Although income inequality between men and women constitutes a significant component of gender inequality, the phenomenon is much more complex. Due to the main mode of transmission of the virus, this debate is even more sensible in the case of HIV/AIDS epidemic. Biological differences, social norms, women's low socioeconomic and socio-cultural status and stigma raise African women's exposure to HIV infection, making them now outnumbering men in infected people.

Now that we are familiar with the links existing between inequality (both income and gender) and HIV/AIDS epidemic, the ways the former can affect the latter, we have to go further with the empirical assessment of the impact of these variables. The next chapter is then dedicated to the empirical analysis of the impact of income and gender inequalities, using data from Sub-Saharan Africa.

CHAPTER 4

INCOME AND GENDER INEQUALITIES AND HIV/AIDS EPIDEMIC: MACRO-ECONOMETRIC EVIDENCE FROM SUB-SAHARAN AFRICA

Introduction

The second chapter of this dissertation set the framework in which HIV/AIDS epidemic should be analyzed and the third chapter made us familiar with the mechanisms through which income and gender inequalities is likely to affect HIV/AIDS epidemic. They also highlighted some of the most common methodological issues that we have to come through in analyzing income and gender inequalities. In the present chapter, we attempt to provide empirical insights into the connections mentioned in the previous chapters. The central issue we address in this chapter is then the relevance of income and gender inequalities as determinants of HIV/AIDS epidemic. Along with this central issue, we also address some related issues, such as the components of gender inequality that really affect the epidemic, the interactions between the two inequalities and the transmission channels through which income inequality actually impacts on HIV/AIDS epidemic in Sub-Saharan Africa.

Although the links between inequalities and HIV/AIDS epidemic have been mentioned earlier in economic literature (Over 1998, Bonnel 2000, Nattrass, 2006), they have not been studied as core determinants of the epidemic. Meanwhile, researchers in other fields have placed socio-economic inequalities at the centre of their analysis (Farmer 1999, Rao Gupta 2002 and 2006). Moreover, the immense majority of the studies completed so far on HIV/AIDS epidemic determinants used cross-section data. They could thus seize the importance of these determinants, but not how their impact evolves over time. Our contribution to the research in this field is to use panel data, so that we can study the evolution of the determinants over time as well as their impact on the epidemic. Indeed, many of our control variables are time-invariant and the most relevant model to account for this kind of variables is the random effects.

The remainder of this chapter is organized as follows. In a first section, we describe the data that will serve us to perform our empirical analysis. The second section is a description of our basic model. In the third section of this chapter, we try to address the flaws and weaknesses of our basic model, namely by using alternative specifications of the dependent variable, data that measure incidence rate and a dynamic model to account for the impact of the lagged value of the dependent variable. Since the *logit* transformation of the dependent variable does not allow a straightforward interpretation of the coefficients, we attempt to represent the impact of inequality on the epidemic, using simulations based on our model. The fourth section assesses the quality of our predictions and the results of simulations. In the last section, we provide some concluding remarks to this chapter.

Section I: Data and descriptive statistics

Before addressing the main issue of this chapter, which is to assess the impact of socio-economic inequalities on HIV/AIDS epidemic, we present a thorough description of our data and justify the choice of some indicators. Our analysis is performed using panel data for two samples of Sub-Saharan African countries. These data come from various sources that we briefly present before taking a close look to data characteristics.

I- Data

Our main sample is made of 42 Sub-Saharan African countries (see Appendix 4.1), for which we used panel data over five periods (1997, 1999, 2000, 2003 and 2005). We used data from the World Institute for Development Economics Research (WIDER), World Development Indicators (World Bank), Human Development Reports (UNDP), UNAIDS' AIDS epidemic updates[68], and United Nations' Women's Indicators and Statistics Database (WISTAT). Since there could be many indicators available for one variable, we want to expose the reasons why we chose ours among other.

A- Income inequality

Among all measures cited in chapter 3, we restricted our analysis Gini index because it is the only indicator for which data are available. The most relevant database for Gini coefficient is the one from WIDER II (original dataset of Deininger and Squire (1996), actualized with new data and released in May 2005). Although this dataset is the most complete on inequality, it does not provide us with the coefficients for all our countries and for all our periods. Thus, we used alternative sources, essentially national sources and Human Development Reports (HDR) data in order to fill this lack. Finally, we have data for only 35 countries on all years. Moreover, the value of the coefficient is the same for 1997 and 1999[69].

B- Gender inequality

There is no equivalent of Gini coefficient for gender inequality and this determinant is still excluded from most economic studies or, at best represented only through one indicator. In order to capture the phenomenon in the fullest possible extent, without confining it to only one dimension, we focused on three dimensions of gender inequality: deprivation in education, inequality in income and participation to economic life and maternal leave benefits[70].

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[68] HIV/AIDS prevalence rates are obtained from UNAIDS. These in turn are a product of demographic modelling using raw data from sentinel sites and ante-natal surveys (see www.unaids.org for data and a discussion of the modelling).

[69] In our regressions on the big sample, we used only actual values of Gini coefficients and due to missing data, our observations in most estimations amounted 115 for 29 countries. Because of the smaller size of the second sample however, we tried to overcome the missing data problem by imputing values of Gini coefficients whenever possible. So, when we had data for two periods, we made extrapolations from these two values to generate the missing value and when we had no value, we predicted the value of Gini coefficient from the other variables.

[70] We first thought of building a gender inequality index, combining all the three dimensions of income inequality. From preliminary estimations however, it appeared that this multidimensional phenomenon was not appropriately captured through an index and there are at two main reasons for that. First, the different dimensions of gender

C- National Income

The indicator for national income is the gross domestic income. GNI is GDP (the sum of values added by all resident producers) plus any product taxes (less subsidies) not included in the valuation of output plus net receipts of primary income (compensation of employees and property income) from abroad. It is usually preferred over the GDP as an estimation of country's income because it gives the indication of the actual wealth received by the country (De Janvry, 2007). While indicating the average level of development a country has achieved, the GNI is not a good indicator for the extent of poverty people experience in the country.

D- Poverty

The most commonly used poverty indicators are income-based. The poverty headcount ratio indicates the incidence of poverty by calculating the percentage of the population whose incomes fall below a certain threshold. The poverty gap ratio measures the degree to which the mean income of the poor differs from the established poverty line (depth of poverty). Income poverty is sometimes measured in per capita GNP terms. However, this indicator is a very crude measure and can often be misleading, as poverty is not related to income only. Since 1997, the UNPD computes a multidimensional measure of poverty, the Human Poverty Index (HPI). The HPI informs in synthesized form about longevity (percentage of the population expected to die before age 40), adult illiteracy, access to health services and to safe water, and under five malnutrition rates. While the HPI depicts certain key and easily measurable elements of human poverty, many other elements of poverty, as perceived from a human capability perspective, are not included in the HPI. Due to the lack of data, we could use neither the poverty gap ratio, nor the poverty headcount ratio. The HPI data, which is actually available, would not perform well in this analysis. Indeed, life expectancy, a component of the index is directly influenced by mortality, which in turn is strongly affected by HIV/AIDS epidemic, what would make it an endogenous

discriminations do not always point in the same direction, so that there can be no or only little variation in the value of the index, while its constituents (especially women's education and economic activity/income, since length of maternal leave and percent of income perceived are time-invariant) have varied greatly in the same time. Second, there is no reason *a priori* that gender inequality's components affect the epidemic the same manner. In particular, within the same society, there can be a low level of women formal employment, but fairly good compensation for women during maternal leave, or even a long duration of maternal leave, but without preservation of wage. There can also be a case where women's education (both enrolment and literacy rates) are high, but do not necessarily mean that women have greater access to labour market. Thus, for this reason, we renounced to our first temptation that was to use the gender index in our regressions and decided to address the dimensions of gender inequality separately.

variable. In order to correct this obvious flaw, we created a poverty index, which is a combination of lack of access to safe water and malnutrition. These two indicators can give an indication of poverty experienced by population; they are both included in the calculation of the UNDP's HPI. Our poverty index is given by:

$$\text{Poverty} = \frac{[100 - (\text{access to safe water}) + \text{malnutrition}]}{2} \qquad (4.1)$$

E- Access to health care

The best indicator of access to health care is the contact rate of individuals with health services. Other common indicators, which are biased because they do not account for unequal geographical repartition, include the number of physicians or nurses for 1000 people or the number of births attended by trained health staff. Data for these three indicators are not available for our sample on the whole period of study. To represent this variable, we resorted to National Health Accounts published by the World Health Organization (WHO). Among all the proposed indicators, we chose health care expenditure per capita because it expresses the amount of money that was spent for the average citizen for health purposes. This indicator does not differentiate whether the expenditure is public or private, or whether it is expressed in total or as percentage of the GDP. Besides, we shall later use the ratio of private to public health care expenditure and immunization rate as instruments for health care expenditure per capita.

F- Male circumcision

In order to capture this variable, we used the percentage of the population which is Muslim. While male circumcision is compulsory in Muslim religion, the use of this indicator is not very accurate for Africa, since male circumcision is also a traditional practice in many countries and is used either as a form of initiation or routinely at birth. Nonetheless, male circumcision is not a common cultural practice in many high prevalence countries[71], so that it is confined to Muslim population only in these countries.

[71] In their categorisation of male circumcision prevalence for 118 developing countries, Drain et *al* (2006) only eight countries of our sample (Botswana, Burundi, Malawi, Namibia, Rwanda, Swaziland, Zambia and Zimbabwe) were classified as low prevalence (< 20 %) of male circumcision countries.

Table 4.1 below provides a detailed list of indicators for our variables, as well as their source, with the first column reporting the signs coefficients are expected to take in our regressions.

II- Descriptive statistics

In Tables 4.2 and 4.3, we present the summary statistics of the variables used in the econometric analysis. These variables are classified into dependent variables, variables of interest and control variables.

A- Dependent variables

The most relevant variable expressing the evolution the epidemic is the incidence rate[72]. However, incidence rates are not easy to observe. UNAIDS, which release the most extensive database on AIDS data, provides routine data on prevalence only. As a proxy for incidence rates, prevalence rates among young people (15-24) are available for a limited number of countries (20) for three years (2001, 2003 and 2005).

Adult (15-49) HIV Prevalence rate: The average level of HIV prevalence rate among the adult population (15- 24) is 8.07%, a very high prevalence level considering that UNAIDS defines a generalized epidemic when the national prevalence rate is estimated at 1% of the adult population. In many countries of this region, especially in Southern Africa, more than 1 in 5 adult are estimated to be living with the virus. For example, South Africa had the highest number of infected people in the world (5.7 to 6.2 million), translating into a prevalence rate of 21.5% of adult population in 2003. Botswana and Swaziland are the countries with the highest prevalence rate (38.8%) for 2001 and 2003 respectively. Meanwhile, there are some countries with a relatively low prevalence rate (Angola has a mean prevalence rate of 3.12% over the period of study, Mauritius, Mauritania, Mali, Senegal, and Madagascar have all prevalence rates less than 1% of adult population). In addition to this intra regional disparity in prevalence rates (Gini coefficient of the distribution of the epidemic in our sample is estimated at 51%), the epidemiological situation has worsened over our period of study; the mean prevalence rate increased steadily from 7.41 % in 1997 to 8.5 % in 1999, 8.5 % in 2001 and 8.64 % in 2003; afterwards however, there is a sensitive amelioration since

[72] The incidence rate is the number of new infections while the prevalence is the total number of infections. Prevalence rate is not as good as incidence rate in indicating the evolution of the epidemic, as it accounts not only for new infections but also for mortality due to the infection.

Table 4.1: Data and source

Variable	Sign	Indicator	Source
Adult (15-49) HIV prevalence rate		HIV prevalence rate in adult population (15-49)	AIDS Epidemic Update UNAIDS (1998-2006)
Young people (15-24) HIV prevalence rate		HIV prevalence rate in young population (15-24)	
Income inequality	+	Gini coefficient for income inequality	WIDER II, UNDP
Poverty	+	Malnutrition prevalence rate, percent of population with access to safe water	Human Development Report (HDR) data (UNDP) 1995-2006
National income	-	Gross National Income, GNI (in Purchasing Power Parity US), fixed telephone lines per 1000 people	World Development Indicators database (World Bank) 2006
Adult literacy rate	-	Adult literacy rate (>15)	HDR (UNDP)1998-2006
Combined enrolment rate	-	Combined enrolment rate (primary, secondary, tertiary)	HDR (UNDP)1998-2006
Access to information	-	Number of television sets for 1000 people	WDI (World Bank) 2006
Access to health care	-	Health expenditure per capita ($), Private health expenditure (% of total), Public health expenditure (% of total)	HDR (UNDP)1998-2006
Contraceptive use	-	Contraceptive prevalence (any modern method)	HDR (UNDP)1998-2006
Religious and traditional practices (circumcision)	-	Percentage of Muslim in total population	ACT (AIDS Campaign Team), World Bank
Ethnic fragmentation	?	Ethnolinguistic fractionalization index	World Bank (Eastely and Levine, 1998)
Female income	-	Female estimated earned income (as % of male)	HDR (UNDP) 1995-2006
Female participation in economy	-	Percentage of women in total labor force, Female economic activity rate	HDR (UNDP) 1995-2006
Female literacy rate	-	Female literacy rate (> 15 years)	HDR (UNDP) 1995-2006
Maternal leave	-	Length of maternal leave (weeks), percent of wage perceived	Women's Indicators and Statistics Data base (Wistat), UN
Conflict	+	Dummy variable that takes the value 1 if the country did experience an occurrence of armed conflict	Centre for the Study of Civil War (CSCW)
Urbanization	+	Percent of population which lives in urban areas	HDR (UNDP) 1995-2006
Voice	-	Voice and Accountability	The Worldwide Governance Indicators (WGI) project, World Bank
Southern and Eastern Africa regional dummy	?	Dummy variable that takes the value 1 for countries located in southern and eastern Africa	Author

Source: Author

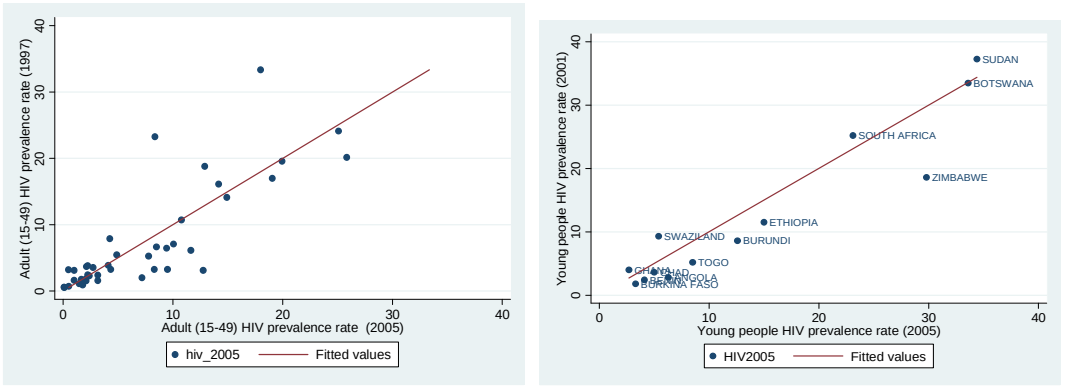
prevalence rate comes back to its initial level of 7.24% in 2005.

Young people (15-24) HIV prevalence rate: The picture looks quite different when we look at the prevalence rate among young people. First, the period of study is so short (2001 to 2005) that there is only a slight change in the mean value (13.7 % to 15.8 %). Moreover, unlike the growth rate of adult prevalence rate, the growth rate of young people prevalence rate is actually increasing (about 5% between 2001 and 2003 and 10% between 2003 and 2005). Although our sample for incidence rate is small, a direct implication of this is that we need to moderate our enthusiasm about the decrease observed in the distribution of adult prevalence rate. This evolution seems to be more the result of increasing mortality due to AIDS than that of decreasing number of new infections.

Figure 4.1 below describes the evolution of the epidemic during our period of study. Countries lying under the 45° line are countries which have experienced a worsening of their epidemic (prevalence rate is higher at the end than at the debut of the period of study).

Figure 4.1: Evolution of HIV prevalence rate (debut and end of the period)

Adult population (15- 49), 1997-2005 Young population (15-24), 2001-2005

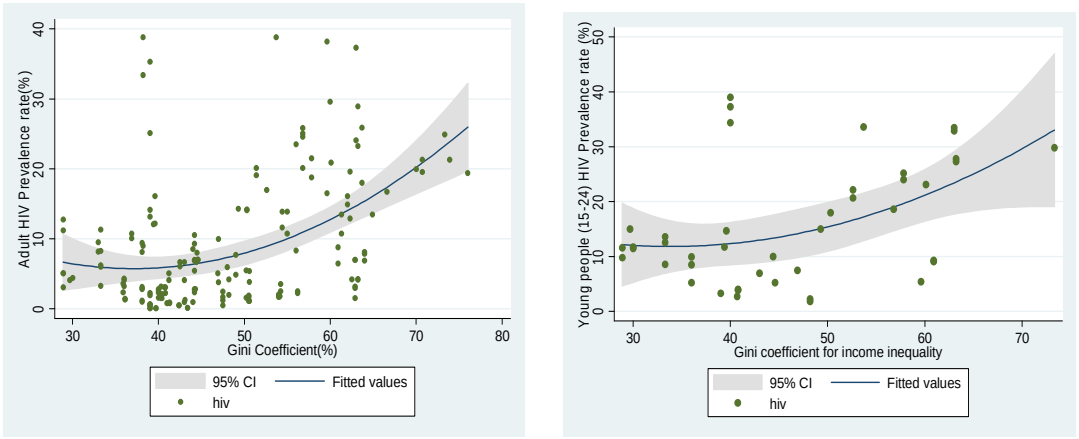


B- Core variables

Income inequality (Gini index): (mean is 47.79%) shows the magnitude of unequal distribution of income in Sub-Saharan Africa. Sub-Sahara has surpassed Latin America as the region with the less egalitarian income distribution in the World. Six of the ten countries with the highest Gini indexes lie in the Southern Africa sub-region. Tables 4.13 and 4.14 in Appendix of this chapter show the strong positive correlation (coefficients are 0.54 and 0.42 on the big and small samples respectively) between income inequality and HIV/AIDS prevalence rate. The simple reading of this statistic is that countries with higher income inequality would experience higher HIV prevalence rates. Figure 4.2 clearly demonstrates this relationship on our samples.

Figure 4.2: Income inequality and HIV/AIDS epidemic

Adult population (15- 49), 1997-2005 Young population (15-24), 2001-2005



Female enrolment rate (as percent of male): This variable is very unequally distributed across the sample, its mean value of 43% hiding values ranging from 11% for Niger to 96% for South Africa. Within countries, the variation in female enrolment rate can be an increase, which occurred in countries like Benin, Chad, Sierra Leone and Uganda or a decrease observed in Congo, in Swaziland, in South Africa or in Namibia.

Female participation to economic life: This variable is the product of female estimated earned income and the share of female labour, both as a percentage of male values. The means values for both indicators are 58.56% and 73.15% respectively. In no country do women earn the same income on average than men, except in Togo (1997) where this equality is probably due to the performance of the 'Nana Benz'[73]. The only example where the share of female labour exceeds that of male is found in Mozambique (2005).

Maternal leave benefits: Women rights about maternal leave benefits do not differ greatly across the sample. In most countries, women are on leave for twelve weeks and perceive the totality of their wage during that time. There are however some notable exceptions where the share of wage perceived by women during maternal leave is much less, a mere 25 % in Botswana, only 10 % in Uganda and none in Lesotho and Swaziland. The maximum length of maternal leave is found in South Africa, where women can be on leave for sixteen weeks, but they perceive only 60 % of their wage. The countries where women are the worst off when they are pregnant is Malawi where can perceive their whole wage, but for only 8 weeks (every three years). Maternal leave benefits' indicator is the product of length of maternal leave and percentage of wage perceived by women during this period. Our indicator is relevant for the formal sector only and there is a significant part of women who work in the informal sector in all countries of our sample[74]. Nonetheless, since the law is supposed to represent a society's values, our indicator is intended to capture the importance society grants all women for their non-productive (in the sense of market) work.

[73] Nana Benz's is the term used in Togo to indicate a wax trader. These women were so successful in their business that they were the only people who could afford the Mercedes Benz cars. They still constitute a powerful economic lobby in the country.

[74] Chen (2001) assessed the share of labour force working in informal sector in Sub-Saharan Africa at 78 % of non-agricultural employment, 61 % of urban employment and 93 % of new jobs. This percentage is likely to be higher for female labour force.

C- Other determinants

Our set of other HIV/AIDS determinants is made of variables presented in Figure 1.3 in Chapter 1. Unfortunately, there are some relevant determinants of the epidemic that could not be included in our regressions due to lack of data. These are average number of sexual partners, average age at the onset of sexual activity and treatment of STIs. Moreover, the percentage of Muslim population captures the effect of some religious and traditional beliefs, but also of male circumcision representing more than one variable. Our index of poverty is composed of malnutrition prevalence and access to safe water. And yet, malnutrition prevalence is an indicator of nutritional status, a proxy of immune system.

1) Socio-economic variables

GNI per capita (in Purchasing Power Parity dollars)): Average GNI per capita is 1860$, with a minimum of 430$ for Sierra Leone in 1999 and a maximum of 12451$ for Mauritius in 2005. Almost every country of the sample experienced a steady growth throughout the period of study, except Zimbabwe (with a decrease from 2590 in 1997 to 1940 in 2005) and Eritrea (with a decrease from 1070$ in 1997 to 1010$ in 2005) which followed the inverse path.

Poverty: The mean value of poverty index is 31,8 and the observations range from 3 (for Mauritius in 2005) to 68,5 for Eritrea in 1997. Poverty has decreased in countries like Central African Republic (52 in 1997 to 34 in 2005), Tanzania (39 to 29,5 in 2005), Gambia (38 to 22,5), Madagascar (57,5 to 45,5) or Eritrea (68,5 to 56,5). Meanwhile, it has sharply increased in other countries like Zimbabwe (19 to 31,5), Zambia (30 to 45,5), Congo (33 to 45,5) or Congo, Democratic Republic (44,5 to 56,5). It has been steady in a third group of countries including Ethiopia (60,5 to 62), Kenya (38,5 to 36) or Mauritania (29,5 to 28,5).

Health care expenditure per capita (in constant dollars): The value of this indicator varies widely; ranging from 3$ to 669$. Moreover, the value of this variable doubles on average in the sample the sample, with an upsurge in 2005. The two countries where the value of this variable decreases are Togo, with a reduction from 91$ in 1997 to 62$ in 2005 and Congo, democratic republic where the expenditure decreased from 19$ in 1997 to 14$ in 2005. Meanwhile, the increase was spectacular in countries such as

Equatorial Guinea, where it nearly quadrupled (44$ in 1997 to 179$ in 2005) and Ghana where the increase was also four fold (23$ to 98$ between 1997 and 2005).

Adult literacy rate: The mean value (57.13) masks wide disparities in this indicator, with a minimum of 12.84 for Niger and a maximum of 90 for Zimbabwe. There is also variation within countries during the period of study, with decreases in countries like Zambia (75 to 67%), Swaziland (77 to 69%) and Ghana (67 to 58%) and increases in other countries like Angola (40 to 67%), Burundi (44 to 59%) and Madagascar (40 to 64%).

Television sets (for 1000 people): The indicator for access to information varies across the sample and over the period of study. For example in Togo, the number of television sets per 1000 people was 16 in 1997 and 85 in 2005 while the figures are 12 in 1997 and 53 in 2005; 25 in 1997 and 75 in 2005 respectively for Angola and Botswana. This number is steady in Benin (11) and in Cameroon (71), while it has decreased in Uganda (from 26 to 11) and Mauritania (from 93 to 43).

Governance: We used the indicator *Voice and Accountability* from The Worldwide Governance Indicators (WGI) project, World Bank. This indicator measures the extent to which a country's citizens are able to participate in selecting their government, as well as freedom of expression, freedom of association, and a free media. Its value ranges from -2.5 to 2.5. A higher value indicates greater voice and accountability (Kaufman et *al*, 2003).

Armed Conflicts: We used a dummy variable created from the data from the Centre for the Study of Civil War (CSCW). However, the categorization of an event is very simplistic in this database. Most of the conflicts are classified as "minor" in the dataset, but all kinds of conflicts are assigned the dummy value 1. Thus, conflicts between Cameroon and Nigeria are considered as armed conflicts in the same way as conflicts in Eritrea and Ethiopia. In the same way, internal conflicts, like the one opposing Casamance to Senegal and Darfur to Sudan are considered as similar to external conflicts, as was the case between Ethiopia and Somalia.

2) Socio-cultural variables

Percentage of total population which is Muslim: In regard to this variable, countries are quite alike, since Islam is not the main religion in most of the countries in our sample. Muslim population is indeed quasi inexistent in some countries (Angola, Swaziland, Namibia and Zimbabwe) while it makes up the bulk of the population in others (Mauritania, Senegal and Mali).

Ethnolinguistic fragmentation index: It is a proxy for social cohesion. It expresses the likelihood that two persons selected randomly in the population are not from the same ethnic group. In our sample, it ranges from 4 in Burundi to 93 in Tanzania[75]. There are very homogenous countries like Madagascar (index is 6) and Rwanda (index is 14), but also countries where the population is more heterogeneous in regard to ethnic origin like Togo, Cameroon, South Africa or Nigeria with an ethnolinguistic fragmentation index of 90, 89, 88 and 87 respectively.

Regional dummy "southern and eastern Africa": We created this dummy to account for regional differences in the impact of determinants on the epidemic across the sample. There are eighteen countries belonging to the Southern and Eastern Africa sub-region.

3) Epidemiological variables

Contraceptive prevalence: Only married women aged 15-49 are surveyed for this indicator. The mean value across the sample is 19.14, with a minimum of 2 for Guinea and a maximum of 75 for Mauritius. The data do not vary during the period of study, since UNDP's data are expressed as a mean for the period 1996-2004. Moreover, the indicator considers any modern method of contraception and not only male condoms, which are the only effective protection against HIV transmission (see Chapter 2, Section I).

The percent Muslim among total population, which is primarily a socio-cultural variable, also gives an indication (though imperfect) of the practice of male circumcision (an epidemiological variable).

[75] A value of 4 for this index means that the odds that two people chosen at random in the population are not from the same ethnic group is 4, translating into 96 percent of probability that these two people are from the same ethnic group. A low value of this coefficient indicates an homogenous population, where there are few ethnic groups while a higher value indicates a population where people belong to too many ethnic groups.

Detailed descriptive statistics of all the variables are presented in Table 4.2 and Table 4.3. Moreover, Appendix 3 shows the correlation of key variables with HIV prevalence rate, the dependent variable while Appendix 4 depicts their distribution. The trend that emerges from the simple reading of these correlations is contra intuitive, as countries with more educated women are more likely to experience high HIV/AIDS prevalence rates. Countries with higher women's estimated income tend to have lower HIV/AIDS prevalence rates, while no clear pattern emerges about the link between the share of female labour on the one hand and HIV prevalence rate on the other hand.

Table 4.2: Summary statistics on the first sample (prevalence rates)

Variables	Mean	Min	Max	Standard Total	Deviaton Within Country
HIV prevalence rate (average)	8.07	0.07	38.8	8.47	2.51
1997 (N= 42)	7.41	0.08	25.84	6.83	
1999 (N= 42)	8.54	0.07	35.3	8.48	
2001 (N= 42)	8.49	0.07	38.8	9.62	
2003 (N= 42)	8.64	0.07	38.8	9.46	
2005 (N= 41)	7.24	0.07	33.38	7.95	
Gini Coefficient	47.58	28.9	76	10.72	3.63
1997 (N= 36)	47.36	28.9	70	10.61	
1999 (N= 36)	47.66	28.9	76	10.99	
2001 (N= 36)	47.97	28.9	73.9	11.74	
2003 (N= 36)	47.45	28.9	70.7	10.41	
2005 (N= 36)	47.45	28.9	70.7	10.41	
Female income	58.56	23.7	100	14.13	6.18
1997 (N= 32)	62.66	31.2	100	15.40	
1999 (N= 34)	56.23	30.31	89.61	12.87	
2001 (N= 33)	56.01	31.25	90.20	13.06	
2003 (N= 41)	56.17	31.76	92.85	12.63	
2005 (N= 42)	61.65	23.7	91.8	15.53	
Share of female labour	73.15	33	102	14.78	3.34
1997 (N= 41)	73.34	40	98	14.77	
1999 (N= 41)	73.34	40	98	14.77	
2001 (N= 41)	73.24	41	98	14.45	
2003 (N= 41)	73.31	42	98	14.27	
2005 (N= 42)	72.54	33	102	16.22	
Female enrolment rate (as percent of male)	42.94	11	96	18.68	6.44
1997 (N= 40)	39.37	11	94	20.41	
1999 (N= 42)	41.4	12	96	20.89	
2001 (N= 42)	43.5	14	81	18.93	
2003 (N= 41)	45	17	78	16.65	
2005 (N= 42)	45.30	18	77	16.35	
Wage during mat leave (%)	80.35	0	100	30.45	0
Maternal leave (weeks)	12.25	2.66	16	2.50	0
GNI per capita ($ PPP)	2207.07	430	12451	2453.81	485.05
1997 (N= 42)	1927.85	460	8870	2036.43	
1999 (N= 42)	2041.19	430	9080	2178.75	
2001 (N= 42)	2179.04	470	10320	2381.51	
2003 (N= 41)	2250.97	530	11280	2561.25	
2005 (N= 42)	2637.35	640	12451	3046.38	
GNI per capita (lagged value)	1859.85	420	10320	1985.86	403.26
1997 (N= 42)	1632.61	420	7760	1680.36	
1999 (N= 42)	1681.66	430	8120	1795.06	
2001 (N= 42)	1832.85	450	8690	1945.62	
2003 (N= 42)	1973.09	470	8820	2096.76	
2005 (N= 42)	2179.04	470	10320	2381.51	

Telephone lines per 1000 people	20.444	0.18	287	42.37	6.41
1997 (N= 42)	16.75	0.18	195.22	34.82	
1999 (N= 42)	19.19	0.18	218.91	39.33	
2001 (N= 42)	20.82	0.19	255.60	43.17	
2003 (N= 42)	22.53	0.20	285.20	47.36	
2005 (N= 42)	22.89	1	287	47.53	
Malnutrition prevalence	28.44	6	73	11.37	6.12
1997 (N=39)	27.87	9	50	9.60	
1999 (N=39)	27.87	9	50	9.60	
2001 (N=39)	27.82	9	50	9.61	
2003 (N=39)	27.82	9	50	9.61	
2005 (N=38)	30.92	6	73	16.97	
Access to safe water	61.75	7	100	19.21	9.79
1997 (N= 42)	53.59	7	100	21.80	
1999 (N= 42)	62.92	24	100	21.59	
2001 (N= 42)	63.38	22	100	17.26	
2003 (N= 42)	64.47	22	100	16.60	
2005 (N= 42)	64.38	22	100	16.69	
Health Care expenditure per capita	51.29	3	669	79.45	45.82
1997 (N= 42)	37.5	4	266	51.39	
1999 (N= 42)	37.5	4	266	51.39	
2001 (N= 42)	37.35	3	224	48.00	
2003 (N= 42)	37.35	3	224	48.00	
2005 (N= 42)	106.73	14	669	135.46	
Immunization rate	63.53	18	98	20.80	10.84
1997 (N= 42)	60.48	18	96	21.66	
1999 (N= 42)	55.92	21	97	24.07	
2001 (N= 42)	60.95	24	97	20.38	
2003 (N= 42)	69.26	25	97	19.18	
2005 (N= 42)	70.97	35	98	14.44	
Ratio PUB/PRI	1.47	0.16	9	1.46	0.91
1997 (N= 42)	1.57	0.21	9	1.59	
1999 (N= 42)	1.57	0.21	9	1.59	
2001 (N= 42)	1.21	0.2	9	1.42	
2003 (N= 42)	1.58	0.16	8.54	1.57	
2005 (N= 42)	1.43	0.16	5	1.10	
Adult Literacy rate	57.13	12.84	89.99	20.00	8.10
1997 (N= 42)	54.98	14.49	86.27	20.25	
1999 (N= 41)	54.04	13	85	19.82	
2001 (N= 42)	59.41	16.5	89.3	19.63	
2003 (N= 42)	58.20	12.84	90	20.67	
2005 (N= 42)	58.94	14.37	90	20.00	
Television sets per 1000 people	51.30	0.10	385.58	63.20	20.76
1997 (N= 39)	38.83	0.10	226.11	44.05	
1999 (N= 39)	45.94	0.3	242.67	50.03	
2001 (N= 42)	58.55	1	385.58	75.54	
2003 (N= 39)	57	1	300	70.92	
2005 (N=39)	57	1	300	70.92	
Contraceptive prevalence	19.14	2	75	14.96	4.00

Muslim population (% of total)	29.75	0	100	31.33	0
Ethnic fractionalization	65.96	4	93	23.01	0
Urbanization rate	36.05	16.80	85.6	16.80	3.82
1997 (N=41)	34.15	5.85	83.30	16.52	
1999 (N=41)	35.35	6.05	83.74	16.78	
2001 (N=41)	36.56	6.28	84.17	17.00	
2003 (N=41)	37.78	6.55	84.58	17.17	
2005 (N=41)	36.4	9.7	85.6	17.13	
Voice	-0.603	-2.05	1.21	0.72	0.25
1997 (N=42)	-0.57	-1.66	0.87	0.69	
1999 (N=42)	-0.58	-1.72	0.98	0.73	
2001 (N=42)	-0.62	-1.91	1.21	0.76	
2003 (N=42)	-0.63	-2.05	0.8	0.71	
2005 (N=42)	-0.60	-1.84	0.92	0.76	

Table 4.3: Summary statistics on the second sample (incidence rates)

Variables	Mean	Min	Max	Standard Total	Deviaton Within Country
HIV prevalence rate among young people (15-24)	13.76	1.8	39	10.75	3.94
2001 (N=18)	13.21	2.7	34.4	10.26	
2003 (N=19)	14.02	2.3	39	10.67	
2005 (N=15)	14.12	1.8	37.3	12.08	
Gini Coefficient	46.64	28.9	73.3	11.73	2.99
2001(N=17)	47.11	28.9	73.3	13.17	
2003 (N=17)	46.41	28.9	63.2	11.32	
2005 (N=17)	46.41	28.9	63.2	11.32	
Female income	58.46	23.7	91.8	15.67	5.99
2001 (N=18)	55.74	31.25	74.60	13.75	
2003 (N=20)	56.99	31.76	74.60	13.12	
2005 (N=20)	62.37	23.7	91.8	19.24	
Share of female labour	74.66	33	102	17.37	4.46
2001 (N=20)	74.85	41	98	16.34	
2003 (N=20)	74.8	42	98	16.23	
2005 (N=20)	74.35	33	102	20.16	
Female enrolment rate (as percent of male)	46.88	18	81	17.77	5.13
2001 (N=19)	71.36	24.5	98.6	21.77	
2003 (N=18)	64.06	14	96.2	23.75	
2005 (N=16)	64.80	23.2	95.6	22.09	
Wage during mat leave (%)	74.31	0	100	37.59	8.67
Mat leave (wks)	11.82	2.66	16	2.87	0.23
GNI per capita	2420.93	560	12121	2655.53	949.70
2001 (N=20)	2200.5	560	9500	2365.09	
2003 (N=20)	2378.5	590	10130	2562.01	

2005 (N=20)	2683.8	640	12121	3100.72	
GNI per capita (lagged value)	2041.66	530	9500	2154.52	667.83
2001(N=20)	1908	530	8690	2057.28	
2003 (N=20)	2016.5	560	8820	2131.29	
2005 (N=20)	2200.5	560	9500	2365.09	
Telephone lines	19.65	1	120	28.61	8.37
2001 (N=20)	18.22	1.39	110.51	28.46	
2003 (N=20)	20.05	2	120	29.30	
2005 (N=20)	20.68	1	120	29.48	
Malnutrition prevalence	28.13	9	68	12.04	6.77
2001 (N=20)	26.65	9	48	10.22	
2003 (N=20)	26.65	9	48	10.22	
2005 (N=19)	31.26	12	68	15.23	
Access to safe water	65.15	22	95	17.92	3.66
2001 (N=20)	63.65	22	95	19.11	
2003 (N=20)	65.9	22	95	17.72	
2005 (N=20)	65.9	22	95	17.72	
Health Care expenditure per capita	67.58	3	669	107.52	66.74
2001 (N=20)	42.25	3	224	55.83	
2003 (N=20)	42.25	3	224	55.83	
2005 (N=20)	118.25	15	669	160.05	
Immunization rate	72.91	27	97	15.18	6.76
2001 (N=20)	68.85	27	97	16.81	
2003 (N=20)	75.05	46	97	16.74	
2005 (N=20)	74.85	49	91	11.26	
Ratio PUB/PRI	1.46	0.16	8.54	1.30	0.90
2001 (N=20)	1.03	0.21	2.45	0.65	
2003 (N=20)	1.87	0.16	8.54	1.91	
2005 (N=20)	1.47	0.16	3.42	0.88	
Combined Enrolment rate	58.76	12.84	89.99	19.56	9.07
2001 (N=20)	50.9	22	80	18.61	
2003 (N=20)	51.55	24	78	15.89	
2005 (N=20)	49.8	26	77	14.58	
Television sets	53.15	1	385.58	76.85	11.80
2001 (N=20)	55.00	1	385.58	86.16	
2003 (N=18)	52.13	1	300	73.49	
2005 (N=18)	52.13	1	300	73.49	
Contraceptive prevalence	18.9	4	56	13.02	1.24
Muslim pop (%)	18	0	70	18.56	1.12
Ethnic fractionalization	62.05	4	90	23.79	0.69
Urbanization rate	31.13	6.28	59.19	14.18	3.22
2001 (N=20)	30.15	6.28	57.64	13.86	
2003 (N=20)	31.20	6.55	59.19	14.12	
2005 (N=20)	32.04	9.7	58.8	15.20	
Voice and Accountability	-0.652	-1.84	1.05	0.76	0.13
2001 (N=20)	-0.62	-1.75	1.05	0.80	
2003 (N=20)	-0.69	-1.71	0.73	0.71	
2005 (N=20)	-0.63	-1.84	0.82	0.82	

Section II: The basic model

In this section, we describe the model we intend to use to study the effect of inequalities on HIV/AIDS epidemic. In doing so, we first precise the specification of the dependent variable, and the functional form we give to our model. In order to assess the additional impact of inequality on the understanding of HIV epidemic, we regress our model on the traditional variables, which are used here as control variables, then we add inequality indicators, our variables of interest among the determinants of the epidemic. The pandemic nature of HIV/AIDS infection is dealt at regional level, as we include average prevalence rate in border countries and a regional dummy in our equation. Finally, we estimate our equation using the two-sample least squares (TSLS) estimator in order to correct for the endogeneity of national income and access to health care.

I- Specification and functional form

The specification of a dependent variable depends on various parameters, including its natural evolution. Prevalence rates are percentages and as such, they are bounded to one or 100%. In addition, as prevalence represents the number of people infected with a disease at a given moment, it follows the typical evolution depicted by the epidemiological curve. Such a variable can be adequately analyzed only if the appropriate transformation is done. In the particular case of panel data, the additional problem of the model specification itself arises, leading to the choice between fixed and random effects models.

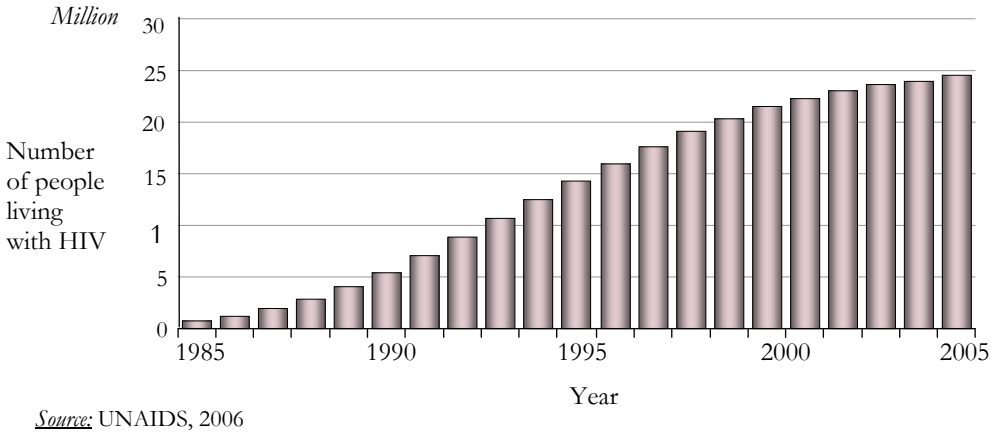
A- Specification of the dependent variable

HIV/AIDS infection is an infectious disease and as such, it typically follows an S-shaped curve (Over 1998). This curve describes an epidemic which prevalence is low at the start, then it increases sharply and finally it levels off at a plateau. Theoretically, there are two main reasons why HIV prevalence rate's growth declines over time. First, at the beginning of the epidemic, there are only very few people aware of the disease, who know how it is transmitted and how to avoid it, thus many people keep getting infected. Second, as the epidemic moves in time, the potential for new infections is low, since many people are already infected. Moreover, people have become aware of the epidemic and now try to avoid it.

Unlike the estimation of a typical economic variable (for example, growth rate) where the assumption can be made that all determinants have a constant elasticity (so that a log-log equation is a relevant model), the estimation of HIV prevalence rate should consider the fact that the impact of determinants can be different according to the rate of spread. The fact that the growth rate of the epidemic declines over time suggests precisely that the impact of its determinants is not constant.

Figure 4.3 represents the number of people living with HIV in sub-Saharan Africa, estimated by UNAIDS for the 1985-2005 period.

Figure 4.3: Estimated number of people living with HIV in sub-Saharan Africa, 1985–2005



Source: UNAIDS, 2006

To be able to apply any linear regression analysis to our nonlinear variable, need to transform the dependent variable, following Over (1998). The first step is to assume a maximum value for the dependent variable. The *logit*(*y*) is then defined as:

$$Logit(y) = \ln\left(\frac{y}{C-y}\right) \tag{4.2}$$

Where C is the assumed maximum value for the dependent variable.

Since there is no zero-value of the HIV prevalence rate in our sample, all observations can be transformed as *'logits'*. These *'logits'* of prevalence rates are the variables we explain in our statistical analysis and this transformation renders it possible to use a linear functional form.

B- Specification of the model

The random effects and the fixed effects models carry their own specificities and are relevant for different data. When performed on our data, the Hausman test does not favour the fixed effect model. Due to the specificities of our data, we prefer using random effects for two reasons; first, the time dimension of our panel is weak with only five periods and second, there are critical time-invariant variables in our series. The importance of cultural factors (ethnic fragmentation, traditional and religious practices) has been assessed in previous studies (Bonnel, 2000). These cultural factors are the slowest to change over time and for our period of study (1997-2005), they are not likely to have changed.

In analyzing the impact of inequality on HIV/AIDS epidemic, our basic specification is given by:

$$logit(hiv_{i,t}) = \alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \varepsilon_{i,t} \qquad (4.3)$$

Or:

$$hiv = \frac{1}{1 + \exp\{\alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \varepsilon_{i,t}\}}$$

Where $i = 1, ..., N$ denotes countries,

$t = 1, ..., T$ denotes time periods;

hiv denotes HIV/AIDS prevalence rate,

I_{it} is the matrix of inequality variables,

X_{it} is the matrix of traditional determinants covariates that always include period dummies in order to account for common shocks that affect all observations in a given period and

ε_{it} is a disturbance term *iid.*

Here, the coefficients $\beta_{.i,t}$ represent the constant increase in *logit*(*hiv*) for every unit increase in the associated variable, all other variables held constant. A constant increase in *logit(hiv)* does not mean a constant increase in *y,* as the *logi*t transformation is non-linear. In particular, the increase in *hiv* associated with a unit change in the determinants varies with the value of x_1 one begins with.

We first regress HIV prevalence rate on traditional determinants and then we analyse specific impact of inequality, adding it to these determinants. Among the variables making the matrix of covariates $X_{i,t}$ up, GNI per capita and television fixed lines per 1000 people are included in the equation after log transformation in order to reduce the spread between observations.

II- Basic results

Table 4.4 presents the results of equation (4.3). The first column does not include our determinants. Among the strong predictors of the epidemic in the covariates, we can cite access to information, contraceptive prevalence, the proportion of the population which is Muslim and ethnic fragmentation in population. On the other hand, poverty, access to health care, adult literacy rate and armed conflicts are also predictors of the epidemic, but carry a sign contrary to the one expected. Surprisingly enough, three variables that are expected to influence the epidemic do not appear determinant in our model. These are national income (which is negatively correlated with HIV/AIDS epidemic at international level[76]), urbanization rate and the governance indicator.

Introducing inequality variables in the model increases its explicative power by 34% (R-squared was 0.50 and is now 0.67); they are thus critical determinants of the epidemic. Income inequality is significant and appears to favour the epidemic, as postulated. Only women's participation to economic life among gender inequality indicators is not relevant in explaining the epidemic.

Except armed conflicts[77], all the determinants conserve their impact on HIV prevalence rate when inequality is added to the model, suggesting that the increase in explicative power is a pure extra effect.

[76] Worldwide, the most affected countries are developing countries.

[77] As mentioned in Chapter 3, previous studies also found no impact of armed conflicts on HIV/AIDS epidemic or even a negative impact. This can be due to the ambiguous categorization of conflicts. Also, the deleterious impact of armed conflicts on health is felt on displaced population only and does not diffuse to the whole population. Moreover, prevalence estimates are based on surveillance sites at antenatal clinics, while displaced people seldom access this kind of services.

Table 4.4: Inequalities and HIV/AIDS epidemic: basic results

Dependent Variable Estimator	**Logit of HIV prevalence rate** Generalized Least Squares	
	(1)	(2)
Income Inequality (Gini)		0.014 (2.00)**
Female participation to economic life		0.000 (1.61)
Female enrolment rate (as percent of male)		-0.009 (1.88)*
Maternal leave benefits		-0.001 (2.14)**
Log of GNI per capita (PPP)	-0.228 (0.84)	-0.314 (1.06)
Poverty	-0.024 (3.31)***	-0.021 (2.52)**
Access to health care	0.005 (3.66)***	0.005 (2.92)***
Adult literacy rate	0.009 (1.89)*	0.018 (3.13)***
Percentage of total population which is Muslim	-0.019 (4.37)***	-0.020 (4.78)***
Ethnic fractionalization	0.018 (2.02)**	0.017 (2.47)**
Log of Television sets (for 1000 people)	-0.202 (2.44)**	-0.218 (2.53)**
Contraceptive prevalence	-0.020 (2.24)**	-0.028 (2.50)**
Urbanization rate	-0.007 (1.22)	0.008 (0.97)
Conflict	-0.356 (2.61)***	-0.182 (1.19)
Voice and Accountability	-0.045 (0.33)	-0.047 (0.29)
Constant	-0.406 (0.19)	-0.266 (0.11)
R-squared (Overall)	0.48	0.63
R-squared (Between)	0.50	0.67
Hausman test *p*-value	0.97	0.31
Rho (Fraction of the variance due to *u_i*	0 .81	0.71
Observations	158 (36)	115 (29)

*Significant at 10% confidence level **Significant at 5% confidence level
***Significant at 1% confidence level
Figures in statistics are t-statistics.
(1) Generalized Least Squares, without inequalities variables.
(2) Generalized Least Squares, with inequalities variables.

III- Spatial correlation

Our basic results provide some help for the understanding of the spread of HIV epidemic in Sub-Saharan Africa. However, at this stage of the analysis, we believe additional understanding can be extracted from this model if we bear in mind that HIV has become a pandemic, and as such it affects all parts of the continent. To be present everywhere, the virus certainly crosses borders. In addition, a close look at our data shows us that countries belonging to southern and eastern Africa sub-region have on average higher prevalence rates than others. We explore these hypotheses by adding successively average prevalence rate in border countries, and a regional dummy.

A- Introduction of a regional dummy

When looking at the raw data, we noticed that countries belonging to the Southern and Eastern Africa have higher prevalence rates on average than others. The positive correlation between HIV/AIDS epidemic and the membership to the aforementioned sub-region persists even when the other determinants are controlled for. Moreover, this new variables provides a better understanding of the epidemic, as there is a noticeable increase in the value of our R-squared, the explicative power of the model. The importance of inequality as determinants of the epidemic is ascertained. However, among the components of gender inequality, only women's education remains significant. Control variables also remain significant and urbanization rate now has an impact on the epidemic.

The strong statistical significance of the regional dummy implies that the other variables cannot fully explain the epidemic in this part of Africa. We believe that this arises from traditional practices and customs[78] in this part of Africa, which place populations there at greater risk of infection. In fact, the value of *rho* for our estimation is about 0.56, translating into more than half of the variance being due to individual country random effects. Yet, these random effects are the very expression of cultural factors.

[78] Practices such as women inheritance, ritual cleansing and dry sex are more common in Southern and Eastern Africa and are shown to favour the transmission of the virus (Luke, 2002).

B- Border countries' prevalence rate

HIV/AIDS is an infectious, contagious disease. As such it ignores boundaries and can easily cross borders. In particular, commercial exchanges and migrant work are frequent along borders, with commercial sex work usually accompanying these activities. Migrants and sex workers, once back at the origin are likely to spread the virus to their regular partners as explained in Chapter 3, Section II. The introduction of border countries' prevalence rate among the determinants of the epidemic nevertheless contradicts our theory, as no extra explicative power is added to the model.

In what follows, we include only the regional dummy in our model, as it is the variable that accounts the best for spatial correlation.

IV- Endogeneity issues

The three sources of endogeneity are omitted variables, reverse causality and simultaneity bias. In our case, there are some good reasons to suspect national income and access to health care of endogeneity in our model.

A- National Income

First, the relationship between income and health is very often one of reverse causation (Fuchs, 2004). National income is basically defined as the overall wealth produced by all factors in the country. Yet, those factors, be it labour force or capital are affected by HIV/AIDS epidemic itself. In addition national income is also determined by some of covariates in our model such as education, ethnolinguistic fragmentation and the quality of governance. Unless we correct for the potential endogeneity by using instrumental variables method, we can consider all the previous results biased. The latest column of Table 4.5 takes this problem into account. We used logarithm of lagged (5 years) values of GNI and the number of fixed telephone lines as instruments for the logarithm of the GNI. The GNI levels five years ago are strongly corrected with the current GNI value (through growth rate), but not with the current HIV prevalence rate. The second instrument is the log of fixed telephones lines for 1000 persons. This variable is not likely to be correlated with the dependent variable, while it is obviously the case with country's GNI[79] .

[79] Unlike cell phones lines that depend on personal income, fixed telephone lines is part of a country's infrastructure and a country's ability to deliver adequate infrastructure directly influenced by its national income.

B- Access to health care

Because AIDS-infected people are likely to experience more illness episodes, we assume that they will be in contact with health services more often. Meanwhile, access to health services increases the likelihood that sexually active people could seek care for STIs or in order to get tested. A valid instrument for this variable would represent access to health care at best, without being correlated with HIV prevalence rate. We used immunization rate as a first instrument, since immunization occurs only at health facilities, but on babies so that it has no correlation with adult prevalence rate. A second instrument we used was the ratio of private to public health expenditure. The sum of the denominator and the numerator of this ratio is the value of our endogenous variable (health expenditure per capita), so they are strongly correlated. However, the structure of health expenditure in countries composing our sample is unlikely to have changed due to the epidemic. Indeed, there is no evidence of different fixing of prices for AIDS-related diseases and other affections at health facilities in the different countries. National and individual health budgets are likely to increase, but there is no reason *a priori* that their increase follows the same path. The Global Fund creation in 2002 would have influenced the value of this ratio, but the first grants were awarded not earlier than in 2003, so they are likely to have affected the prevalence rates for at most one period.

The values of the Sargan-Hansen tests associated with our regressions can be read at the bottom of Table 4.5 and confirm the instruments utilized valid for the endogenous variables, national income and access to health care indicators.

Broadly, our main conclusion is that HIV epidemic is strongly favoured by income and gender inequalities. This conclusion holds even when endogeneity of national income is considered in our analysis, the effect on inequality on HIV/AIDS epidemic is always positive and significant statistically. Moreover, many of the covariates that were significant under the Generalized Least Squares (GLS) estimation remain good predictors of the epidemic under the TSLS specification. These are contraceptive prevalence, ethnic fragmentation, the percentage of total population which is Muslim and the regional dummy.

Table 4.5: Inequalities and HIV/AIDS epidemic: Spatial correlation and instrumental variables

Dependent Variable	**Logit of HIV prevalence rate**		
Estimator	Generalized Least Squares		Two Sample Least Squares
	(1)	(2)	(3)
Income Inequality (Gini)	0.017	0.009	0.027
	(2.44)**	(1.33)	(2.39)**
Female participation to economic life	-0.000	-0.000	-0.000
	(0.09)	(1.43)	(0.11)
Female enrolment rate (as percent of male)	-0.013	-0.007	-0.018
	(2.51)**	(1.59)	(1.94)*
Maternal leave benefits	0.000	-0.001	-0.000
	(0.54)	(2.11)**	(0.67)
Log of GNI per capita (PPP)	-0.097	-0.239	-0.952
	(0.38)	(0.68)	(0.86)
Poverty	-0.023	-0.018	-0.031
	(3.15)***	(2.25)**	(2.47)**
Access to health care	0.003	0.003	0.015
	(1.74)*	(1.64)	(1.05)
Adult literacy rate	0.012	0.015	0.014
	(2.47)**	(3.02)***	(1.55)
Percentage of total population which is Muslim	-0.017	-0.019	-0.015
	(4.92)***	(4.31)***	(2.29)**
Ethnic fractionalization	0.019	0.019	0.014
	(3.45)***	(2.62)***	(1.78)*
Log of Television sets (for 1000 people)	-0.211	-0.191	-0.177
	(2.87)***	(2.07)**	(1.40)
Contraceptive prevalence	-0.038	-0.027	-0.039
	(3.51)***	(2.00)**	(3.65)***
Urbanization rate	0.014	0.008	0.005
	(1.88)*	(0.97)	(0.35)
Conflict	-0.338	-0.124	-0.413
	(2.04)**	(0.87)	(1.65)
Voice and Accountability	-0.139	-0.037	-0.271
	(0.97)	(0.23)	(1.50)
Southern and Eastern Africa (regional dummy)	1.612		1.303
	(5.26)***		(2.22)**
Prevalence rate in border countries		0.027	
		(1.58)	
Constant	-2.458	-1.020	3.697
	(1.26)	(0.38)	(0.46)
R-squared (Overall)	0.82	0.66	0.75
R-squared (Between)	0.87	0.70	0.84
Rho (Fraction of the variance due to *u_i*	0.56	0.73	0.31
Sargan-Hansen test p-value[80]			0.05
Observations (countries)	115 (29)	115 (29)	115 (29)

[80] The Sargan-Hansen test is not programmed on *stata* for the random effects model, so we had to perform it step by step. We first regressed the TSLS equation with the instruments and predict the residuals. Then, these residuals are regressed on the explicative variables, including the instruments. The R^2 of this late regression is then multiplied with the number of observations. The value obtained is the test statistics that follows a $\chi^2(F)$, with ***F,*** the degrees of freedom being the number of explicative variables. Then, the reading of the test for the pooled sample differs from that of the split sample. The test statistic (6.624) follows a chi-square with 2 degrees of freedom. So the probability to reject the null hypothesis (validity of the instruments) is the value in parentheses, which is 0.05.

(1) Generalized Least Squares estimation with "Southern and Eastern" dummy.
(2) Generalized Least Squares estimation with prevalence rate in neighbour countries.
(3) Estimation on the pooled sample using the Two Sample Least Squares.
Instruments used are the logs of lagged values of GNI (5 years ago) and the logs of the number of fixed telephones lines (for log of the GNI per capita) on the one hand and immunization rate and the ratio of private to public health care expenditure for health care expenditure per capita on the other hand.

Section III: Robustness

The basic results of our model have established that inequalities are strong determinants of HIV/AIDS epidemic. But before moving further to assert the impact of socio-economic inequalities on HIV epidemic, we need to ascertain the robustness of our previous result. To that end, we consider a series of econometric procedures that will allow us to assess the validity of the basic result. Thus, we first estimate our model, using the Hausman-Taylor fixed effects estimator, and then we change the specification of the dependent variable. The second step in this robustness exercise is to look for interactions between our variables of interest. Then we want to use a more accurate indicator of the evolution of a disease, incidence rate which is approximated here by prevalence rate among young population (people aged 15-24). Finally, we explore the possibility that the dynamics of our variable may not follow the same pattern over the whole period of study, even though its determinants remain the same. Moreover, the lagged value of the dependent variable itself is added into the model, to explore the hypotheses of potential inertia or convergence in the model.

I- Alternative specifications

In analyzing how our basic results might change according to the different specifications, we first use the fixed effects Hausman-Taylor estimator, keeping using the continuous *logit* as the specification of our dependent variable.

We chose the *logit* specification for our dependant variable because it is the most appropriate with its intrinsic evolution. However, our results might also be biased precisely because of the specification. To make sure that this is not the case, we estimate our model, using a different specification of HIV prevalence rate, which is still compatible with its evolution. In that end, we transform HIV prevalence rate, making it a binary variable. We chose the thresholds of 8% and 5% for prevalence rate (because the average prevalence rate in our sample is 8.14% and the median is 4.14) as marker for a severe epidemic. These binary variables are then estimated using a *probit*.

A- Fixed effects estimation with Hausman-Taylor estimator

The Hausman test performed on our equation (4.3) does not really help us to choose between the fixed and the random effects model. For the reasons we explained in Section II, we tilted for the random effects model. Basically, the fixed effects estimator cannot estimate the impact of time-invariant variables; however this limit can be overcome by the Hausman-Taylor estimator.

As our results suggest (Table 4.6, column 1), there are some differences between the random effects estimation and the fixed effect model using Hausman-Taylor estimator. In particular, income inequality and contraceptive prevalence are no longer determinants of the epidemic. Gender inequality in education remains an important variable for the spread of HIV/AIDS epidemic, as do also most of the variables significant under the random effects model, which conserve their sign and their statistical power.

B- *Probit* model

The models estimated are given by:

$$Hiv_{it} = \alpha_{it} + \beta_{1it} I_{it} + \beta_{2it} X_{it} + \varepsilon_{it}, \varepsilon_{it} \square N(0,1) \qquad (4.4)$$

Where Hiv_{it} =1 if Hiv_{it} > 8 % and Hiv_{it} = 0 otherwise.

Then, Hiv_{it} =1 if Hiv_{it} > 5 % and Hiv_{it} = 0 otherwise.

The explicative variables I_{it} and X_{it} are the same as in Section 2.

The coefficients $\beta_{.it}$ are estimated by maximum likelihood (ML).

The results pertaining to the *probit* specification are presented in the last two columns. As we can see from these results, the impact of gender inequality on HIV prevalence rate is not sensitive to alternative specifications of the dependent variable. Income inequality appears as a determinant of the epidemic only in the *probit* model, when 8 % is used as the breakpoint.

In addition among the covariates, the percentage of population which is Muslim, ethnic fragmentation, access to information and the regional dummy are strong predictors of the prevalence rate in the alternative specifications. On the other hand contraceptive prevalence is no longer significant under the *probit* specification.

Table 4.6: Inequalities and HIV/AIDS epidemic: alternative specifications

Dependent Variable	**Logit of HIV prevalence rate**		
Estimator	Hausman-Taylor	*Probit*	
	(1)	(2)	(3)
Income Inequality (Gini)	0.011	0.083	0.084
	(1.17)	(1.72)*	(1.19)
Female participation to economic life	0.000	-0.000	0.000
	(0.37)	(2.50)**	(1.11)
Female enrolment rate (as percent of male)	-0.013	-0. 128	-0. 07
	(2.08)**	(2.74)**	(1.95)*
Maternal leave benefits	-0.000	-0.000	0.000
	(0.59)	(0.45)	(0.34)
Log of GNI per capita (PPP)	-0.337	(0.30)	-3.104
	(0.98)	(0.29)	(2.14)**
Poverty	-0.020	-0.035	-0.085
	(2.48)**	(0.87)	(2.15)**
Access to health care	0.003	0.015	0.017
	(2.26)**	(1.48)	(1.32)
Adult literacy rate	0.012	-0.001	0. 064
	(2.32)**	(0.05)	(1.69)*
Percentage of total population which is Muslim	-0.012	-0.036	-0.054
	(1.78)*	(1.61)	(2.39)**
Ethnic fractionalization	0.018	0.046	0.052
	(2.46)**	(1.88)**	(2.58)**
Log of Television sets (for 1000 people)	-0.395	-1.242	-1.023
	(2.94)***	(2.73)**	(2.13)**
Contraceptive prevalence	-0.006	0.016	-0.005
	(0.70)	(0.41)	(0.08)
Urbanization rate	0.018	0.012	0.089
	(1.77)*	(0.27)	(1.59)
Conflict	-0.262	-3.302	-1.081
	(1.24)	(2.32)**	(0.94)
Voice and Accountability	-0.053	-1.243	0.496
	(0.32)	(1.11)	(0.56)
Southern and Eastern Africa (regional dummy)	1.355	5.795	3.967
	(2.89)***	(3.28)***	(3.15)***
Constant	-0.725	0.97	21.074
	(0.27)	(0.12)	(1.86)*
Observations	115 (29)	116(29)	116(29)

* Significant at 10% confidence level ** Significant at 5% confidence level
*** Significant at 1% confidence level. Absolute value of *t*-statistics in parentheses
All regressions used year-dummies. Time-invariant variables are the percentage of wage perceived during maternal leave*length, percentage of total population which is Muslim, ethnic fractionalization and the regional dummy. Endogenous[81] variables are: Log of GNI per capita (PPP), Health care expenditure per capita, contraceptive prevalence and female enrolment rate. The other variables are considered exogenous in this model.

[81] The Hausman-Taylor estimator considers as endogenous those variables that are correlated with country-specific effect α_i, but not with the error term υ_{it}.

II- Interactions between the variables

Income inequality might interact with gender equality or poverty, thus creating a negative synergic effect on the epidemic. In addition, political economy considers that armed conflicts are more likely to occur in countries where natural resources endowment is abundant. Yet these countries also experience higher levels of income inequality.

A- Introduction of interactive terms in the model

In the literature, claims have been made that the impact of income inequality should be more significant in richer countries because relative deprivation of poor people is higher there (Wilkinson 19996, Deaton 2003). The possibility of confounding with poverty or gender inequality (Sen, 1999) is not to exclude either. In order to explore these hypotheses on our sample, we introduce four interactive variables in the model. These variables are defined as follows:

*Income inequality * female participation to economic life, income inequality * female enrolment rate*: in order to account for possible interactions between income inequality and gender inequality.

*Income inequality * log of GNI per capita:* in order to account for possible interactions between income inequality and poverty (at national level).

*Income inequality * poverty*: in order to account for possible interactions between income inequality and poverty (at individual level).

Surprisingly enough, no extra explanation emerges from our model when theses variables are introduced and our model fails to detect any interaction between income inequality and the other variables. Because of the correlation coefficient of 0.49 between income inequality and female enrolment rate (Table A.1 in Appendix), one would logically expect income inequality's impact to be more significant in presence of higher female enrolment rates.

Table 4.7 presents the results of our estimations when interactive terms are added to the model.

Table 4.7: Inequalities and HIV/AIDS epidemic: interactions between variables

Dependent variable	**Logit of HIV prevalence rate**			
Estimator	Two Sample Least Squares			
	(1)	(2)	(3)	(4)
Income Inequality (Gini)	0.059	0.006	-0.124	0.023
	(1.94)*	(0.14)	(0.61)	(1.70)*
Female participation to economic life	0.000	-0.000	-0.000	0.000
	(0.76)	(0.06)	(0.34)	(0.49)
Female enrolment rate (as percent of boys)	-0.019	-0.03	-0.017	-0.017
	(1.96)*	(0.94)	(1.80)*	(1.80)*
Maternal leave benefits	-0.000	-0.000	-0.000	-0.000
	(0.51)	(0.59)	(0.59)	(0.74)
Log of GNI per capita (PPP)	-0.931	-0.670	-1.903	-1.038
	(0.87)	(0.61)	(1.16)	(0.77)
Poverty	-0.033	-0.029	-0.032	-0.025
	(2.63)***	(2.65)***	(2.84)***	(1.96)**
Access to health care	0.015	0.011	0.012	0.017
	(1.02)	(0.68)	(0.85)	(0.95)
Adult literacy rate	0.013	0.013	0.014	0.014
	(1.34)	(1.68)*	(1.62)	(1.71)*
Percentage of total population which is Muslim	-0.012	-0.016	-0.014	-0.014
	(1.78))*	(1.61)	(2.42)**	(1.79)*
Ethnic fractionalization	0.014	0.016	0.015	0.013
	(1.95)*	(1.93)*	(2.11)**	(1.47)
Log of Television sets (for 1000 people)	-0.209	-0.210	-0.215	-0.145
	(1.90)*	(1.57)	(1.91)*	(0.89)
Contraceptive prevalence	-0.039	-0.037	-0.034	-0.036
	(3.88)***	(3.94)***	(2.60)***	(2.92)***
Urbanization rate	0.004	0.008	0.002	0.003
	(0.28)	(0.56)	(0.18)	(0.19)
Conflict	-0.514	-0.399	-0.463	-0.303
	(2.21)**	(1.85)*	(2.07)**	(1.05)
Voice and Accountability	-0.378	-0.241	-0.307	-0.171
	(2.15)**	(1.46)	(1.81)*	(0.86)
Southern and Eastern Africa (regional dummy)	1.314	1.343	1.140	1.185
	(2.42)**	(2.91)***	(1.94)*	(1.63)
Income inequality* Female participation to economic life	-0.000			
	(0.87)			
Income inequality* Female enrolment rate		0.000		
		(0.50)		
Income inequality*Log of GNI per capita (PPP)			0.021	
			(0.77)	
Income inequality*Poverty				0.000
				(0.46)
Constant	2.481	2.659	10.597	4.154
	(0.31)	(0.41)	(0.89)	(0.44)
R-squared (Overall)	0.78	0.81	0. 80	0.71
R-squared (Between)	0.88	0.87	0. 87	0.79
Rho (Fraction of the variance due to *u_i*	0.18	0.32	0.23	0.46
Observations	115 (29)	115 (29)	115 (29)	115 (29)

*Significant at 10% confidence level **Significant at 5% confidence level ***Significant at 1% confidence level

B- Income inequality, natural resources and armed conflicts

Collier and Hoefler (1998) identified greed and grievance as the main causes of civil conflicts. This view is also shared by Anyanwu (2002) who found social fractionalization and the amount of natural resources (proxied by primary commodity exports-GDP ratio) as strong predictors of civil war occurrence in Sub-Saharan Africa.

Table 4.8 shows the relation between income inequality, HIV/prevalence rate, armed conflicts and the presence of mineral resources on our sample.

As we can notice, there is a clear correlation between income inequality and HIV prevalence rate. Surprisingly enough, no connection appears between income inequality, the presence of natural mineral resources and civil conflicts. Among the fourteen countries with average value of Gini coefficient higher than, only two (Sierra Leone and Central African Republic) experienced armed conflicts during our period of study[82]. On the other hand, some countries with relatively low values of Gini coefficients (Ethiopia, Eritrea, Sudan, Cote d'Ivoire, Rwanda and Burundi) experienced civil conflicts, most during the whole period of study. However, among the six countries with no values of Gini coefficients, three are often cited as countries with a high level of inequality. These are Angola, Congo and Congo, Democratic Republic. Yet, these countries also experienced armed conflicts during the period of study.

We cannot examine the relation between natural resources and armed conflicts (described as *greed* by Collier and Hoeffler) on our sample because we do not have an assessment of the value of natural resources[83]. Thus, petroleum is listed as natural resource for both Equatorial Guinea and Benin, but it is certainly more abundant in the former than in the latter. Moreover, a country can have only one or few listed natural resources, but these resources are worth more than many other listed in another country. This is the case with Botswana which has only diamonds, whereas Lesotho, Guinea-Bissau and Burkina-Faso have many natural resources, but whose value does not certainly equal that of Botswana's diamonds.

[82] Indeed, we look for the occurrence of armed conflict during our period of study of course, but also two years prior the beginning of this period, in order to account for the delay in the potential effect of conflict on the epidemic.

[83] In order to have natural resources for each country, we used a map from Michigan State University, available on the internet at: http://exploringafrica.matrix.msu.edu

Table 4.8: Natural resources, income inequality, armed conflicts and HIV/AIDS epidemic in Sub-Saharan Africa

Country	Average Gini coefficient	Occurrence Of Conflit	Average HIV prevalence rate	Natural Mineral Resource
Namibia	73.30	0	20.30	diamonds, copper, uranium, gold
Gabon	63.68	0	6.26	petroleum, manganese
Sierra Leone	62.90	1	4.34	diamonds, bauxite, iron ore
Swaziland	62.33	0	30.86	asbestos, coal, clay
Zimbabwe	62.30	0	24.10	coal, chromium ore, asbestos
South Africa	61.57	0	18.74	gold, diamonds, uranium, chromium
Malawi	57.77	0	14.72	limestone
Central African Republic	58.30	1	12.48	diamonds
Lesotho	57.33	0	22.72	water (hydro)
Zambia	56.47	0	17.87	copper, cobalt, zinc, lead
Botswana	54.03	0	32.12	diamonds
Mali	54.00	0	1.85	gold, phosphates
Guinea-Bissau	52.23	0	4.27	bauxite, phosphates
Kenya	51.10	0	9.27	limestone, soda ash, rubies
Senegal	49.73	1	1.21	phosphates, iron ore
Uganda	46.30	1	6.07	copper, cobalt
Cameroon	46.12	0	6.39	petroleum, aluminum
Eritrea	44.20	1	2.78	potash, zinc
Madagascar	43.30	0	0.76	graphite, chromite, coal, bauxite
Nigeria	44.20	1	4.79	petroleum, tin, columbite, iron ore
Gambia, The	42.73	0	1.89	
Sudan	41.40	1	1.69	petroleum, iron ore, copper
Mauritania	41.27	0	1.85	iron ore, gypsum, copper
Niger	40.93	0	1.24	uranium, coal, iron ore
Guinea	40.27	0	2.23	bauxite, iron ore, uranium
Cote d'Ivoire	39.40	1	8.32	petroleum, diamonds, manganese
Ethiopia	39.37	1	7.08	
Mauritius	39.28	0	0.17	
Mozambique	39.13	0	13.55	coal, titanium
Burkina Faso	39.00	0	4.80	manganese, limestone
Tanzania	38.16	0	8.36	tin, phosphates, iron

				ore, diamonds
Djibouti	38.10	0	2.20	
Ghana	37.50	0	2.89	gold, bauxite, manganese
Togo	34.80	0	5.87	phosphates, limestone
Burundi	33.3	1	7.02	gold
Rwanda	28.90	1	7.45	gold, tin ore
Congo, Dem. Rep. (Zaire)		1	5.30	copper, diamonds, cobalt, gold, zinc
Congo		1	4.85	petroleum, diamonds
Chad		1	3.43	petroleum, uranium
Angola		1	3.24	petroleum, diamonds
Equatorial Guinea		0	2.52	petroleum
Benin		0	2.03	petroleum

III- Estimation with incidence rate

As mentioned in section I, the indicator relevant for the evolution of an epidemic is incidence rate. However, since the number of new infections is very difficult to observe (if not impossible at all in the particular case of HIV/AIDS), the prevalence rate among young people is used as a proxy for the incidence rate.

Table 4.9 summarizes the results of the estimation of incidence rates on the same determinants used earlier for prevalence rates. In addition to the Generalized Least Squares estimator, we also estimate our equation using the Two Sample Least Squares in order to correct for potential endogeneity. Due to the small size of our incidence dataset, (20 countries and 3 periods and unbalanced panel yielding to 37 observations), we perform a normality test on our standard-errors. The test probabilities are 0.66 (for OLS estimation) and 0.83 (for TSLS estimation), so we cannot reject hypothesis H_0 of errors' normality.

There is a notable difference with our previous results when we use incidence rates, as gender inequality appears as the only determinant of the epidemic. More importantly, the component of gender inequality that is relevant in explaining the epidemic differs for prevalence rate and incidence rate. For the prevalence rate, this component is women's education as showed by the coefficient and significant sign of female enrolment rate, whereas the pertinent variable with incidence rate is women's economic power, which is the only inequality variable which remains robust when the equation is estimated with Two Samples Least Squares.

Table 4.9: Inequalities and HIV/AIDS epidemic: estimation with incidence rates

Dependent Variable	**Logit of HIV incidence rate**	
Estimator	Generalized Least Squares	Two Sample Least Squares
	(1)	(3)
Income Inequality (Gini)	0.019	0.019
	(2.46)**	(1.56)
Female participation to economic life	-0.005	0.004
	(2.06)**	(1.90)*
Female enrolment rate (as percent of boys))	0.046	0.061
	(0.10)	(061)
Maternal leave benefits	0.000	0.000
	(0.53)	(0.39)
Log of GNI per capita (PPP)	-0.180	-0.173
	(0.64)	(0.26)
Poverty	-0.001	-0.000
	(0.08)	(0.05)
Access to health care	0.003	0.002
	(1.03)	(0.33)
Combined enrolment rate	-0.006	0.005
	(0.37)	(0.17)
Percentage of total population which is Muslim	0.025	0.025
	(3.76)***	(3.75)**
Ethnic fractionalization	-0.017	-0.018
	(2.15)**	(1.70)*
Log of Television sets (for 1000 people)	0.158	0.160
	(2.38)**	(1.70)*
Contraceptive prevalence	-0.021	-0.001
	(0.24)	(0.18)
Urbanization rate	-0.005	-0.004
	(0.33)	(0.23)
Conflict	-0.713	-0.722
	(2.21)**	(1.28)
Voice and Accountability	-0.383	-0.38
	(1.63)	(1.74)*
Southern and Eastern Africa (regional dummy)	1.002	1.009
	(3.75)***	(2.26)**
Constant	-1.323	-1.35
	(0.50)	(0.26)
R-squared (Overall)	0.95	0.95
R-squared (Between)	0.96	0.96
Skewness/Kurtosis tests for Normality	0.66	0.83
Observations	37(17)	37(17)

*Significant at 10% confidence level **Significant at 5% confidence level
***Significant at 1% confidence level
(1) Generalized Least Squares
(2) Two sample Least Squares
In column (2), instruments used are the logs of lagged values of GNI (5 years ago) and the logs of the number of fixed telephones lines (for log of the GNI per capita) on the one hand and immunization rate and the ratio of private to public health care expenditure for health care expenditure per capita on the other hand.

IV- Dynamic analysis of the epidemic

Our period of study (eight years) is not a long period in regard of macroeconomic analysis. Nevertheless, it is a long period in the history of HIV/AIDS epidemic (26 years) and indeed, it covers the whole period for which routine data on the epidemic has been collected. Given the evolution of the epidemic, it is also a period during which significant changes are likely to have occurred in the impact of determinants or prevalence rate's acceleration. The epidemic was not certainly sensible to its determinants in 1997 in the same way it was in 2005. Moreover, among determinants, there can be a shift in the importance, so that determinants important at the beginning of the period of study are not the same at the end. To explore the potential difference in the way the epidemic is determined throughout the period of study, we split this period into two shorter sub-periods, following the recommendation of the Chow test performed earlier. Another way to perform a time analysis of our model is to allow past value of the dependent variable to be a determinant of the epidemic, a hypothesis we explore in a model of dynamic panel.

A- Chow test

Our period of study (1997-2008) is not really long, but there are significant differences in prevalence rates between countries and in the same countries over the period[84]. Furthermore, the *logit* specification implies that the elasticity of determinants is not constant over time. We consider these hypotheses in the Chow test[85] for the stability of our coefficients (Chow, 1960) by dividing the period of study into a sub-period from 1997 to 1999 and another from 2001 to 2005.

Since we cannot consider our coefficients as stable over time (as shown by the results of the test in Table 4.10), the next step in our quest of robustness is to split our sample into two sub-samples, which would allow us to study the potential differences in the manner the epidemic is determined during the two periods.

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[84] As shown in descriptive statistics, prevalence rates range from 0.077 in Madagascar and Mauritania to 38.8 in Lesotho. Concerning intra-country difference over years, prevalence rate plummeted from 8.5 in 1997 to 4.1 in 2003 in Uganda, as it did in Ethiopia (from 10.5 in 1999 to 4.4 in 2003) and in Congo (from 6.5 in 1999 to 3.5 in 2005). Meanwhile, the rates followed the inverse path in countries such as Lesotho were they rose sharply from 8.4 in 1997 to 28.8 in 2003, South Africa where the increase was a jump from 12,9 in 1997 to 21.5 in 2003 and Swaziland where it rose from 18.0 in 1997 to 38.8 in 2003.

[85] In order to correct for potential heteroskedasticity in our data, the estimations are corrected with White procedure whenever applicable. As for unit root, panel and time-series data are often flawed with stationarity, but this is likely not the case with ours, since our time dimension (5 periods) is very small compared to the individual one (42 countries).

Table 4.10: Inequalities and HIV/AIDS epidemic: Chow test

Dependent Variable	**Logit of HIV prevalence rate**
Estimator	TSLS
Income Inequality (Gini)	0.027
	(2.39)**
Female participation to economic life	-0.000
	(0.11)
Female enrolment rate (as percent of boys)	-0.018
	(1.94)*
Maternal leave benefits	-0.000
	(0.67)
Constant	3.697
	(0.46)
R-squared (Overall)	0.75
R-squared (Between)	0.84
Chow test	51.430
	(0.00)
Observations	115 (29)

B- Evolution of the epidemic over the two sub-periods

The curve of the epidemic, which is not linear, implies that the impact of different variables is not constant over time. In fact, our period of study corresponds to the part of the curve from when the epidemic accelerates through hopefully the beginning of its plateau. The Chow test validates this intuition, being unable to reject the null hypothesis of difference in coefficients when our sample is broken into two sub-samples.

The breaking point for our sample is arbitrary, since the only valid reason we have to choose another date leads to two very disproportionate sub-samples[86] (a sample of one year and the other made off the other four years), which is not suitable for econometric analysis.

When the sample is split into two sub-samples, the TSLS estimation's results show a picture different of the one which emerges from the estimation on the whole sample. Income inequality is important at the onset of the epidemic (first sub-sample),

[86] We first thought of the date of creation of the Global Fund as breaking point for our sample, but the first grants were awarded in 2003 and are likely to influence prevalence rates for 2005 only.

then it is or relayed by structural factors such as the percentage of population which is Muslim, ethnic fragmentation and the membership to southern and eastern Africa sub-region. All over the period of study, contraceptive prevalence is a strong determinant of the epidemic. Surprisingly enough, no gender inequality indicator appears having an impact on the epidemic when estimations are performed on two sub-samples.

C- Dynamic evolution of the dependent variable

The curve of the epidemic, as depicted in Figure 4.1 follows an S-shaped curve and is not totally randomly determined. In particular, the growth rate of the number of new infections is positive, though decreasing. That means countries with already high prevalence levels are likely to reach their plateau sooner, but that also means that these countries could have a higher number of new infections, since their initial prevalence rate is high. The sign of the lagged value of the dependent variable in the regression cannot be anticipated and depends on the magnitude of those two effects; the potential for new infections, which is positive and the decreasing growth rate of prevalence, which is negative.

The lagged value of the dependent variable is by construction an endogenous variable. If estimated with the GLS estimator, the subsequent estimation would yield correct, but not robust coefficients. The model that allows estimating lagged value of the dependent variable while correcting for this intrinsic endogeneity is the Generalized Method of Moments. There exist two variants of the GMM estimator in dynamic panel: the first difference estimator and the system estimator.

The first difference estimator developed by Arellano and Bond (1991) consists of taking for each period the first difference in order to eliminate specific country effects, and then instrumenting explicative variables of the first difference equation by their lagged values. As for the estimator GMM-system estimator, developed by Blundell and Bond (1998a), it combines first difference equations with level equations where variables are instrumented with their first differences. Using Monte-Carlo simulations, Blundell and Bond (1998b) showed that the estimator of GMM system performs better that the estimator in difference developed by Arellano and Bond (1991), the latter yielding biased results on finite samples when instruments are weak.

We use the GMM-system estimator for our dynamic estimation, where current values are used as instruments for exogenous variables; lagged values for one period are

used as instruments for weakly exogenous variables[87] and lagged values for at least two periods are used as instruments for endogenous variables.

Leaving from our initial equation:

$$logit(hiv_{i,t}) = \alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \varepsilon_{i,t}, \quad (4.3)$$

We now estimate the model:

$$\log it(hiv_{it}) - \log it(hiv_{i,t-1}) = \alpha_i^{'} + \beta_1^{'}(I_{it}) + \beta_2^{'}(X_{it}) + (\beta_{3,it} - 1)\log it(hiv_{i,t-1}) + \varepsilon^{'}_{i,t} \quad (4.5)$$

Where *t-1* denotes the previous period.

This dynamic equation can also be written as:

$$logit(hiv_{i,t}) = \alpha_{i,t} + \beta_{1i,t} I_{it} + \beta_{2i,t} X_{i,t} + \beta_{3i,t} logit(hiv_{i,t-1}) X + \varepsilon_{i,t} \quad (4.6)$$

The Sargan-test associated with our estimation validates our choice for instruments in this model; there is also no second order autocorrelation in this model, as showed by the p-value (0.192) of Arellano and Bond test for autocorrelation.

The value of coefficient $\beta_{3i,t}$ is 0.462 and represents the impact of previous prevalence rate on current prevalence rate. But this is different from the value of previous prevalence rate on the evolution of prevalence rate, which is given by $\beta_{3i,t} - 1$. The command *lincom* programmed under *stata* allows us to have the value of this coefficient, which is -0.538, significant at the 1% confidence level[88]. This negative sign translates into a convergence in prevalence rates, where high prevalence countries tend to reach their plateau before low prevalence countries.

Table 4.11 summarizes the results of the dynamic analysis of HIV/AIDS epidemic over the period of study. The first two columns present the results of equation (4.3) performed on the two sub-samples. Again, income inequality appears to significantly favour HIV/AIDS epidemic. However we can notice that income inequality is very important at the onset of the epidemic, which appears to be fuelled

[87] Weakly exogenous variables are defined here as variables that are predetermined or that can be influenced by past values of dependant variables, but which are not correlated with future realizations of error term.

[88] The *t*-statistic associated with this coefficient is 3.47.

thereafter by structural determinants such as the percentage of the population which is Muslim, ethnolinguistic fragmentation and the regional dummy. The contraceptive prevalence is also an important determinant of the epidemic all over our period of study. The variables other are no longer determinants of the epidemic.

The last column presents the results of equation (4.5) on the pooled sample, including the lagged value of the prevalence rate among the determinants of the model. The results of this model present minor differences with the model described by equation (4.3) on the pooled sample (Table 4.5, Column 1). Thus income inequality and gender inequality in education are significant under this specification, as are also access to information and the structural determinants of the epidemic. Armed conflicts also appear as determinants of the epidemic, but do not carry the sign expected, as already showed in our previous results.

Table 4.11: Inequalities and HIV/AIDS epidemic: Dynamic Analysis

Dependent Variable	**Logit of HIV prevalence rate**		
Estimator	Two Sample Least Squares		Generalized Method of Moments
	(1)	(2)	(3)
Income Inequality (Gini)	0.070	0.018	0.015
	(2.69)***	(0.74)	(1.79)*
Female participation to economic life	-0.000	-0.000	-0.000
	(1.10)	(0.58)	(1.38)
Female enrolment rate (as percent of boys)	-0.006	0.012	-0.011
	(0.34)	(0.30)	(1.87)*
Maternal leave benefits	0.000	0.000	-0.000
	(0.02)	(0.65)	(1.20)
Log of GNI per capita (PPP)	-1.688	2.133	0.098
	(1.54)	(0.98)	(0.38)
Poverty	-0.033	-0.014	-0.009
	(1.34)	(0.58)	(1.05)
Access to health care	0.018	-0.029	
	(1.46)	(0.70)	
Adult literacy rate	0.002	-0.034	-0.005
	(0.17)	(0.65)	(0.73)
Percentage of total population which is Muslim	-0.008	-0.034	-0.010
	(1.00)	(1.96)**	(3.92)***
Ethnic fractionalization	0.005	0.039	0.007
	(0.60)	(1.68)*	(2.40)**
Log of Television sets (for 1000 people)	-0.010	-0.191	-0.100
	(0.05)	(0.81)	(1.71)*
Contraceptive prevalence	-0.032	-0.060	-0.012
	(2.03)**	(2.99)***	(1.30)
Urbanization rate	-0.007	0.034	0.013
	(0.33)	(0.96)	(1.49)
Conflict	-0.313	-0.658	-0.419
	(0.59)	(1.42)	(2.27)**
Voice and Accountability	-0.727	-0.234	-0.168
	(1.97)**	(0.79)	(1.35)
Southern and Eastern Africa (regional dummy)	1.685	3.399	1.174
	(3.03)***	(1.98)**	(2.82)***
Logit of lagged HIV prevalence rate			0.462
			(2.98)***
Constant	-3.642	-17.904	-1.782
	(1.41)	(1.10)	(0.61)
R-squared Overall (Between)	0.77 (0.87)	0.66 (0.73)	
A-R (1) *p*-value			-2.94(0.003)
A-R (2) *p*-value			-1.08 (0.280)
Sargan-Hansen test p-value[89]	0.1	0.001	26.20 (0.159)
Observations (countries)	45(25)	70(27)	95(29)

[89] The Sargan-Hansen test is not programmed on *stata* for the random effects model, so we had to perform it step by step. We first regressed the TSLS equation with the instruments and predict the residuals. Then, these residuals are regressed on the explicative variables, including the instruments. The R^2 of this late regression is the multiplied with the number of observations. The value obtained is the test statistics that follows a $\chi^2(F)$, with ***F,*** the degrees of freedom being the number of explicative variables. Then, the reading of the test for the sub-samples differs from that of the pooled sample (GMM estimation). The test statistics (14.357 and 5.0175) follow a chi-square with 2 degrees of freedom. So the probability to reject the null hypothesis (validity of the instruments) is the value in parentheses, which are 0.1 (for the period 1997-2001) and 0.01 (for the period (2001-2005).

(1) Two Sample Least Squares Estimation for the period 1997-2001
(2) Two Sample Least Squares Estimation for the period 2001-2005
Instruments used are the logs of lagged values of GNI (5 years ago) and the logs of the number of fixed telephones lines (for log of the GNI per capita) on the one hand and immunization rate and the ratio of private to public health care expenditure for health care expenditure per capita on the other hand.
(3) Estimation on the pooled sample using the Generalized Method of Moments.
In Column (3) the log of the lagged prevalence rate, the log of GNI and health care expenditure per capita are our endogenous variables. Weakly exogenous variables are female enrolment rate and female participation to economic life. The other variables are considered exogenous.

Section IV: Simulation of the effect of a reduction of inequality on the epidemic

In the previous sections of this chapter, we attempted to assert the importance of inequalities as determinants of HIV/AIDS epidemic. This impact is significant and robust to the alternative specifications of the model, of the dependent variables, to the correction for endogeneity and to the inclusion of lagged prevalence rate as a new variable in the model. The interpretation of the coefficients in the *logit* specification is not straightforward, so to be able to visualize the impact of inequality on the epidemic, we perform simulations based on our estimations, trying to assess the effect of the reduction of inequality on the course of the epidemic.

The rationale behind our simulations is to estimate the model, with new values of inequality variables, but leaving all other variables unchanged. So the predictions are performed using the coefficients yielded by the initial regression. Since the simulations are based on the predictions of our dependent variables, we first estimate the ability of these predictions to match the actual data, and then we assess the effect of a reduction in inequality on the dependent variables, HIV/AIDS prevalence rates.

I- Quality of the predictions

The quality of our predictions is not uniform across the whole sample. For some countries, the predictions are a close match of the actual data. In the most cases where the predictions are not accurate, there is an overestimation of the value of the dependent variable, the extreme cases being the predicted value of adult prevalence in Botswana in 2005 (79.44 % instead of 24.10) and Mauritius in 2005 (6.06% instead of 0.55%). Zimbabwe is the most obvious case in which the value of the dependent variable is patently underestimated by our predictions. Examples of good predictions include Burkina-Faso, Burundi, Cote d'Ivoire, Ethiopia, The Gambia, Ghana, Mauritania and Rwanda. The value of the dependent variable is overestimated in countries like Kenya, Madagascar, Mauritius, Malawi, Niger and Tanzania.

Again, the spread of the epidemic in countries belonging to Southern and Eastern Africa is not well explained by our model. Only on the second sample in Burundi (2003), South Africa (2001

and 2003) and in Zimbabwe (2001 and 2005) are the predictions accurately matching with the reality of the data in the sub-region.

Surprisingly enough, the quality of predictions is better with incidence data; the gap between actual and predicted data is on average 1.35%, with a minimum of 0.03% (Zimbabwe, 2001) and a maximum of 18.6% (Lesotho, 2003). Uganda, Lesotho, Rwanda and Botswana also perform well in this model.

The detail of the predictions for the dependent variable can be found in Tables A.3 and A.4 in the appendix of this chapter.

II- Results and comments

When making simulations, it is important to make realistic hypotheses. Since income and gender inequality are shown to favour a tragic epidemic, our natural tendency is to sharply reduce them in order to maximize their impact on the epidemic's evolution. However, reducing income inequality is hard to implement in practice. This arduous task requires the implementation of many types of policy (redistributive, fight against poverty). In this section, we assign a reduction of 5% in income inequality[90], an objective which is ambitious, but not impossible in our context if political will is present.

The task is much easier with the reduction of our variables of gender inequality. Although increasing women's income or enrolment rates will not immediately translate into women being treated better in the societies in which they leave, these actions would nevertheless reduce HIV/AIDS prevalence rate. Moreover, increasing women's income or enrolment rates is more feasible than reducing income inequality. The reduction of gender inequality in education is one of the MDGs and countries are allocated the means they need in order to meet the target assigned by the UN. Concerning women's income and participation to economic life, the microfinance institutions are already doing a great job in helping them to acquire a form of economic independence. We simulate an increase of girls' enrolment rate of successively 5%, 10% and 20%. For simulations on the first sample, we first simulate the reduction in gender inequality in education, and then we combine it to the reduction of 5% in income inequality.

Since the only inequality variable robust on incidence data after bootstrap estimations is females' income relative to males, only the change in this variable will be simulated on our second sample.

[90] A reduction of income inequality can be done through an increase of the poor's income or a decrease of the rich's income. The latter modality can feasible through taxation. Unfortunately, tax systems in most of the countries composing our sample are still not effective, so that reducing income inequality translates into increasing the poor's income (or alleviating poverty).

The effect of the different *scenarii* about the reduction of socio-economic inequalities on average HIV/AIDS prevalence rate is reported on Table 4.11. The range of this impact is a reduction of prevalence rate varying from of 3.5 % to 18.1 % of the baseline value. The reduction of income inequality alone causes the least impact on prevalence rate, and this impact is at least increased by 60 % when combined with any postulated reduction in gender inequality in education. The most noticeable effect if obtained when increasing girls' enrolment rates by 20%, whether this measure is accompanied by a reduction in income inequality or not. Reducing income inequality alone lowers average prevalence rate by 5.4 %, but when this measure is combined with the increase in girls' enrolment rates, its impact tends to disappear, since the impact is identical to that of increase in girls' enrolment rates alone.

The reduction of gender inequality through an increase in women's income strongly affects the evolution of the epidemic among young people, as suggested by the results of our simulations. The proportionality factor here is about seventy percent, with 5% of increase in women's income translating into a reduction of average prevalence rate by 3.5%, as an increase of 10%, then of 20% translates into a reduction of 6.7 % and 13.25 % respectively in the average prevalence rate.

Table 4.12 reports the effects of a reduction in inequality on the average prevalence rates in and Table 4.13 represents the impact on average incidence rate.

This average effect, though important, masks significant differences across the sample. Indeed, as what could be logically expected, the impact is more striking among country with high initial inequality levels and high prevalence rates. The effect of the increase in women's relative income is a reduction on average prevalence rate among young population that ranges from 0.5 % for Sudan in 2005 to 26.5 % in Burundi in 2005. In Zimbabwe where our prediction almost matches the actual figure in 2001, an increase in women's income would have reduce prevalence rate among young population by 3,59 % to 13,76%, according to the amount of this increase. Meanwhile, the same reduction of gender inequality in income would have translated into a decrease of only 1.56 % to 5.37 % in Lesotho. When looking at prevalence rate among adult population, in Mauritania where the prediction is accurate for 2005, a reduction in inequality would have led to a decrease in prevalence rate by 5.15 to 18.85 %, according to the *scenarii* used to reduce inequality, whereas the reduction would range from 4.06 % to 10.12% in Burundi for 1997, a year when the model is also fit to predict the prevalence rate.

Table 4.12: Effect of the reduction of inequalities on HIV/AIDS epidemic (average prevalence rates)

Change in income inequality(%)	Change in gender inequality (%)	Effect on average adult HIV prevalence rate (%)
ΔGini = 0	ΔGirls' enrolment rate = 0	Baseline
ΔGini = 0	ΔGirls' enrolment rate = +5	8.9
ΔGini = 0	ΔGirls' enrolment rate = +10	12
ΔGini = 0	ΔGirls' enrolment rate =+20	18.1
ΔGini = -5	ΔGirls' enrolment rate = 0	5.4
ΔGini = -5	ΔGirls' enrolment rate = +5	8.9
ΔGini = -5	ΔGirls' enrolment rate = +10	12
ΔGini = -5	ΔGirls' enrolment rate = +20	18.1

Source: Simulations based on author's calculations

Table 4.13: Effect of the reduction of inequalities on HIV/AIDS epidemic (average incidence rates)

Change in gender inequality (%)	Effect on young people prevalence rate (%)
ΔWomen's income = 0	Baseline
ΔWomen's income = +5	3.50
ΔWomen's income = +10	6.7
ΔWomen's income = +20	13.25

Source: Simulations based on author's calculations

The interpretation of these results should be done with caution, as there are large benefits in prevalence rates that do not necessarily translate into large benefits in terms of infected population, and similarly small benefits in terms of prevalence rates that correspond to large benefits in terms of infected population. This is precisely the case with the decrease in prevalence rate in Mauritius that ranges from 4.89 to 26.37 % (Table A.3). Since population and prevalence rate in this country are low, the benefit in terms of infected people is not significant. On the

other hand, Ethiopia[91] offers an example of much larger benefit in terms of infected population with a decrease in prevalence rate that ranges from 3.83 to 13.5 %.

It is worth noting that the effect we computed considers the reduction in inequalities, holding everything else given, but precisely in this case, everything else is unlikely to remain unchanged. A reduction of income and gender inequalities is likely to have an impact on other determinants. As an example, throughout our analysis, contraceptive prevalence appears always significant. Enhancing girl's education and raising women's income would increase their awareness about the infection, preventive measures and make contraception more affordable. In this way, the effect predicted here is a minimum effect.

Table 4.14 presents a detailed picture of the effect of a reduction in inequality on the epidemic (average prevalence rates) in selected countries. Table 4.15 shows the impact on incidence rates.

Table 4.14: Effect of the reduction of inequalities on HIV/AIDS average prevalence rate in selected countries

Country (year)	Prevalence rate (actual value)	Prevalence rate (predicted value)	New value of prevalence rate* (predicted value)	Change in preva -lence rate** (%) (predicted value)
Burkina Faso (1999)	6,44	6,43	[6,12 - 5,76]	[4,87 - 10,44]
Burundi (1997)	8,30	9,07	[8,70 - 8,15]	[4,06 - 10,12]
Cote D'ivoire (2001)	6,70	6,22	[5,88 - 5,29]	[5,54 - 14,90]
Ethiopia (2003)	4,40	4,42	[4,25 - 3,85]	[3,84 - 12,93]
Gambia, The (1999)	1,96	1,97	[1,87- 1,64]	[5,09 - 16,63]
Ghana (1997)	2,38	2,15	[2,05 - 1,80]	[4,68 - 16,26]
Mauritania (2005)	0,68	0,70	[0,66 -0,57]	[5,15 - 18,85]
Rwanda (1999)	11,21	11,92	[11,51 - 10,17]	[3,42 - 14,69]
Senegal (2001)	0,80	0,74	[0,70 - 0,62]	[5,41 - 16,14]

Source: Simulations based on author's calculations

* Interval in which lies the new value of prevalence rate when inequality is reduced, calculations take the predicted value as baseline.

** Interval in which lies the amount of the change in prevalence rate when inequality is reduced.

The changes reported here are negative, thus values in parentheses are absolute values.

[91] Ethiopia's population is estimated at 77 million in 2006 and Mauritius' population is 1.3 million for the same year.

Table 4.15: Effect of the reduction of inequalities on HIV/AIDS average incidence rate in selected countries

Country (year)	Incidence rate (actual value)	Incidence rate (predicted value)	New value of incidence rate* (predicted value)	Change in incidence rate** (%) (predicted value)
Botswana (2005)	33,5	35,62	[34,99 - 33,13]	[1,7 - 6,99]
Burundi (2003)	13,6	13,41	[12,76 - 10,95]	[4,89 - 6,99]
Cote D'ivoire (2001)	10,0	9,39	[9,25 - 8,84]	[1,52 - 5,94]
Ethiopia (2001)	15,0	14,88	[14,49 - 13,38]	[2,60 - 10,07]
Lesotho(2005)	27,3	26,06	[25,55 - 24,06]	[1,96 - 7,69]
Rwanda (2001)	9,8	9,67	[9,25 - 8,09]	[4,32 - 16,31]
South Africa (2001)	23,1	22,46	[22,04 - 20,84]	[1,84 - 7,20]
South Africa (2003)	24,0	23,84	[23,40 - 22,13]	[1,83 -7,17]
Sudan (2001)	34,4	34,62	[34,36 - 33,58]	[0,75 -3,00]
Uganda (2001)	7,5	7,74	[7,38 - 6,38]	[4,71 - 17,65]
Uganda (2003)	7,0	7,55	[7,19 - 6,20]	[4,78 - 17,90]
Zimbabwe (2001)	29,8	29,81	[28,97 - 26,55]	[2,81 - 10,94]
Zimbabwe (2005)	18,6	18,18	[17,53 - 15,68]	[3,59 - 13,76]

Source: Simulations based on author's calculations

* Interval in which lies the new value of prevalence rate when inequality is reduced, calculations take the predicted value as baseline.

** Interval in which lies the amount of the change in prevalence rate when inequality is reduced.

The changes reported here are negative, thus values in parentheses are absolute values.

Concluding remarks

Using panel data on Sub-Saharan African countries for the period 1997-2005, we tried to assess the importance of inequality as determinant of HIV/AIDS epidemic. We focussed on socio-economic inequalities that we defined as income and gender inequalities. Analyzing inequality comes along with methodological and conceptual issues we explained in the two previous chapters. In particular, the choice of a correct indicator for gender inequality is a critical challenge, since there is no Gini coefficient equivalent for this kind of inequality. The first step towards our aim was to find the correct specification of our dependent variable; unlike most economic variables, HIV/AIDS prevalence is expressed as a rate and as such, it is naturally bounded to 100. In order to estimate this variable using a linear model, we had to transform it accordingly, with a *logit* in order to accounts for the ceiling.

Our basic results suggest that inequality fuels HIV/AIDS epidemic in Sub-Saharan Africa. When added to traditional determinants of the epidemic, it increases the explicative power of the model by 30 %, which is further enhanced when we account for spatial correlation. Our model carries some weaknesses we subsequently corrected for, using instruments for endogenous variables, alternative specifications of the model, and dynamic analysis of the model.

Our main finding is that inequalities are important predictors HIV/AIDS epidemic in Sub-Saharan Africa. The impact of income inequality on HIV prevalence rate is almost always significant in our analysis, across different specifications of the dependent variables, when endogeneity is corrected for and when the model is specified in dynamic form. Moreover, this impact of income inequality does not transit through social cohesion, since ethnic fragmentation (which could be viewed as a proxy of social cohesion) also has an impact of the epidemic. Armed conflicts and internal migrations are also accounted for in our model, so that the impact of income inequality that we observe can be considered a pure effect. Because of the low value of the coefficients of variables representing inequalities, one might underestimate the impact of our interest variables on the epidemic. Indeed, the *logit* transformation of the dependent variable alters the value of the coefficient which becomes much smaller that what it would be otherwise. The reduction of the average prevalence rate induced by a reduction of the level of socio-economic inequalities denotes precisely the impact of these variables on the epidemic, despite the weak value of the coefficient in the regressions. The complexity of gender inequality is an impediment to this analysis, as it allows assessing only the specific impact of some outcomes, but not the processes that give birth to this inequality (cultural norms, traditions and male chauvinism). In addition, we are unable to assess the channels through which gender inequality affects HIV/AIDS epidemic, mainly due to lack of data on violence, commercial sex work and traditional practices. Despite this methodological issue, gender inequality in education appears as a critical determinant of the epidemic, which is robust throughout our analysis. On a smaller sample, gender inequality in economic power appears to be one of the most robust determinants of the epidemic among young population.

While trying to answer the main question addressed in this study —is inequality a major determinant of HIV/AIDS epidemic in Sub-Saharan Africa? —, we have been led to assess the impact of the other determinants of the epidemic. In this way, some of our findings confirm those of previous studies. In particular, the percentage of Muslim among total population appears to curb the epidemic, a result already found by Over 1998). The positive coefficient of a regional dummy for southern and eastern Africa was also assessed by Nattrass (2006). Notwithstanding these similarities, our results contradict the impact of national income found by the authors.

Thus, this variable's impact is positive, as assessed by Zanakis (2005), Nattrass (2006) and Stillwagon (2000) or negative, as Over's results suggest. Our own model fails to provide evidence of any impact of national income on the epidemic. We are also at odds with Zanakis concerning the impact of access to health care and with Bonnel (2000) about the effect of female participation to economic life on adult HIV prevalence rate. None of the studies reviewed in Chapter 2 include poverty among the determinants of the epidemic. In our own analysis, we came out with mixed results concerning the impact of poverty on the epidemic in; poverty appears to be either protective or to have no impact on HIV/AIDS prevalence rate. This result can be justified in two ways. First, it could be due to our index, which is not an indicator of income poverty and might not accurately express the real poverty level in population. Second, poverty might well be negatively correlated with HIV/AIDS epidemic, not only in terms of national income (GNI, GDP, and GNP) but also in terms of individual income (poverty headcount ratio and poverty gap). Indeed, this result has been found previously in the literature, using microeconomic date (Lachaud, 2007).

Our second important finding is that the determinants important for adult prevalence rate differ from those of the incidence rate. Namely, the epidemic among young people (15-24) is more sensible to poverty, especially in terms of women's economic deprivation relative to men.

The last finding concerns the channels through which income inequality affects HIV/AIDS epidemic in our sample. Thus we can say that this impact does not transit through social cohesion, as this channel represented by ethnic fragmentation is robust when introduced in the equation among control variables. Besides, there is no measure of stigma, which could represent a psychosocial factor that would act as a channel for income inequality. So we favour the material deprivation pathway as the channel through which income inequality affects the epidemic on our sample.

Nonetheless, there are other important issues that remain unresolved after this analysis, in particular the interactions between the two kinds of inequality. We did not find evidence supporting the hypothesis that gender inequality's impact could be enhanced in presence of income inequality, or *vice-versa,* what one could logically expect. Also, we cannot either refute, or confirm that the effect of income inequality reported in our study results from confounding by income at the individual level. Indeed, since we did not use an income-measure of poverty, we cannot challenge the common critic that the impact of inequality captures the impact of poverty. Finally, we do not have prevalence rate by sex, so that we could compare the determinants of the epidemic for each sex. Such a comparison would have allowed us to confront the assumptions of Chapter 3 to the reality of data. At best, what we can say from the results of our analysis is that

gender inequality drives HIV/AIDS epidemic in adult and young population. However, since women make up more than half of infections (more than three quarters for young people) throughout the continent, they are disproportionately affected.

CONCLUSION GÉNÉRALE

Malgré l'absence d'un vaccin ou d'un traitement curatif VIH/Sida jusqu'à présent, le combat contre l'épidémie n'est pas encore perdu. De fait, de plus en plus de moyens financiers sont mis à disposition pour lutter contre l'épidémie, mais leur efficacité ne pourra être garantie que s'ils sont alloués dans les actions dont l'efficacité est maximale. L'étude des facteurs qui affectent la transmission du VIH/Sida est importante en ce sens qu'elle permet justement de savoir dans quelles activités allouer le plus de ressources humaines et financières dans la prévention pour obtenir un impact optimal.

Au cours de cette thèse, nous nous sommes intéressés à l'étude de l'impact des inégalités de revenu et de genre sur le cours de l'épidémie du VIH/Sida en Afrique Subsaharienne. Nous nous sommes attachés à montrer que l'étude des déterminants de l'épidémie était indissociable de celle des déterminants de la santé en général. En particulier, les inégalités de revenu affectent l'état de santé à travers trois principaux canaux : l'accès aux opportunités de la vie, la privation matérielle et les facteurs psychosociaux. Les deux premiers canaux qui sont les moins contestés dans la littérature sont également à l'œuvre dans la transmission du VIH en Afrique Subsaharienne. A ces deux canaux, s'ajoutent des spécificités liées à la région concernée comme le travail des migrants et la crise du secteur sanitaire des années 80 et 90, mais également à la transmission sexuelle du VIH comme le commerce sexuel qui est une stratégie de survie pour de nombreuses femmes dans le contexte de pauvreté du continent. Quant aux inégalités de genre, leur effet sur la santé passe nécessairement par l'impact qu'elles ont sur des déterminants clés de la santé comme le revenu et l'éducation. Pour donner à l'épidémie du VIH/Sida le visage féminin qu'on lui connaît désormais, aux facteurs précédemment cités viennent se greffer l'exposition plus importante des femmes au virus (du fait de leur constitution physique, de certaines pratiques traditionnelles et des soins prodigués aux personnes infectées), les spécificités de la vie maritale en Afrique (caractérisée par la polygamie, le mariage précoce des filles et la pression sociale pour avoir des enfants) et le faible statut socio-économique et culturel des femmes. Outre ces deux déterminants qui sont au cœur de notre problématique, les autres facteurs qui conditionnent l'état de santé tels que la pauvreté, le niveau d'éducation, les facteurs culturels, la nutrition ou l'accès

aux soins de santé déterminent également la transmission du VIH au sein d'une population. Nous soulignons également l'importance des facteurs structurels comme le degré de fragmentation ethnique, le pourcentage de la population qui est de religion musulmane et la localisation des pays à l'Est et au Sud du continent.

La première précaution à prendre avant l'analyse de l'épidémie du VIH/Sida concerne la spécification de la variable dépendante. Le traitement des variables purement économiques n'est pas approprié pour l'étude de l'évolution de l'épidémie. Par conséquent, nous privilégions la transformation logistique du taux de prévalence du VIH/Sida qui rend mieux compte de l'évolution d'une épidémie. Dès lors, nous avons tenté de déterminer par un modèle économétrique l'impact des inégalités de revenu et de genre sur le taux de prévalence du VIH/Sida. Nous soulignons alors un impact négatif des inégalités sur la santé des populations africaines, notamment parce qu'elles créent un environnement propice à la propagation de la pandémie. Parce que nous utilisons des données macroéconomiques qui ne donnent aucune indication sur le revenu des ménages, il nous est impossible ici de tester si l'effet de l'inégalité de revenus que nous observons capte celui de la pauvreté monétaire, comme le suggère Deaton (2003). Par contre, nous pouvons affirmer que cet effet observé de l'inégalité de revenu ne transite pas uniquement par la cohésion sociale approchée par l'indice de fragmentation ethnolinguistique[92]. De plus, nous n'avons pas pu déceler la présence d'interactions entre les deux types d'inégalités ou entre les inégalités et les autres variables par l'introduction de termes multiplicatifs dans le modèle.

Approfondissant cette relation, nos résultats soulignent que l'effet identifié est d'autant plus fort que l'on tient compte des spécificités régionales, l'impact des inégalités de revenus apparaissant logiquement plus significatif dans les pays où elles sont le plus élevées et où les taux de prévalence sont également les plus importants. D'autre part, la seule variable de genre qui reste significative une fois cette spécificité régionale prise en compte est l'inégalité de genre en matière d'éducation. Ceci pourrait bien traduire l'interaction entre les deux types d'inégalités que nous n'avons pas pu déceler par l'introduction des variables muettes, du moins au niveau des pays d'Afrique australe et orientale. Une analyse dynamique de l'impact des inégalités sur l'épidémie du VIH révèle que leur importance s'accentue au cours du temps. De plus, la valeur de la variable dépendante elle-même influence le cours de l'épidémie, les pays avec des taux élevés ayant tendance à connaître un ralentissement dans l'évolution du taux de prévalence. Ce résultat est

[92] Dans la mesure où l'inégalité de revenu et l'indice de fragmentation ethnolinguistique introduites dans les régressions sont toutes les deux significatives.

cohérent avec la spécification logistique du taux de prévalence dans la mesure où plus on se rapproche du plafond et plus les « gains » sont difficiles à obtenir. Ainsi, on assisterait à un phénomène de convergence au sein de l'échantillon, les pays à faible taux de prévalence ayant tendance à rattraper ceux aux épidémies bien avancées.

La deuxième application de l'impact des inégalités de revenu et de genre sur l'épidémie du VIH/Sida en Afrique Subsaharienne est réalisée sur un échantillon plus petit de pays pour lesquels les données sur le taux de prévalence pour la tranche d'âge des 15-24 ans sont disponibles. En fait, le taux de prévalence des jeunes est un bon indicateur du taux d'incidence qui mesure le taux de nouvelles infections dans une population. L'étude de l'effet des inégalités sur le taux de prévalence au sein de la population jeune a permis de mettre en évidence deux différences avec les résultats de l'épidémie dans la population adulte. Ainsi, les inégalités de genre constituent le déterminant le plus important de l'épidémie au sein de cette population. En outre, c'est l'inégalité de genre en termes de revenu et de participation à la vie économique qui est déterminante pour la transmission du virus chez les jeunes.

Plusieurs questions restent inabordées dans cette thèse, par manque de données essentiellement. Ainsi, il aurait été intéressant d'étudier l'impact de certains comportements sur le cours de l'épidémie, tels que l'âge moyen au premier rapport sexuel ou le nombre moyen de partenaires dans la population. De plus, nous n'avons pas étudié la relation entre inégalités socio-économiques et épidémie du VIH/Sida dans le sens inverse c'est-à-dire l'impact du VIH/Sida sur l'évolution des inégalités de revenu et de genre dans les pays d'Afrique sub-Saharienne.

Malgré les faiblesses citées plus haut, nos résultats permettent de dégager un certain nombre d'éléments à prendre en compte pour améliorer l'efficacité de la lutte contre le VIH/Sida. Premièrement, l'objectif de réduction de la pauvreté qui semble pourtant galvaudé est toujours d'actualité en ce qui concerne le VIH/Sida. Toutefois, son impact sur le cours de l'épidémie ne serait pas seulement direct, mais il passerait également par une baisse des inégalités de revenu, réduisant ainsi l'opportunité des comportements à risque tels que le travail des migrants ou le commerce sexuel chez les femmes les plus pauvres. Cependant, la réduction des inégalités de revenu toute seules est le scénario le moins efficace (puisqu'elle permet de réduire le taux de prévalence moyen de seulement 5,4%), mais aussi sûrement le plus difficile à mettre en œuvre. En effet, ce scénario ne peut se réaliser que soit par une réduction de la pauvreté (qui est l'objectif affiché des politiques de développement depuis que l'économie du développement existe, mais avec les résultats que l'on sait), soit par une réduction du revenu des plus riches, qui est un objectif non réaliste, d'autant plus que la majorité des pays de notre échantillon ont des systèmes de taxation assez flous.

Deuxièmement, en ce qui concerne la réduction des inégalités de genre pour freiner l'épidémie, un message important de cette thèse est qu'il faut appliquer des politiques différentes dans la population jeune et la population adulte ; il serait ainsi plus efficace d'éduquer et de sensibiliser les femmes adultes à l'épidémie tout en fournissant aux plus jeunes des alternatives pour être financièrement indépendantes. En effet, il est tout à fait logique de considérer que le différentiel d'éducation entre garçons et filles n'est pas une variable significative pour la propagation de l'épidémie chez les jeunes, dans la mesure où cette génération a grandi avec l'épidémie et est beaucoup plus informés des méthodes de prévention. Par contre, le différentiel de revenu entre hommes et femmes est une variable déterminante pour l'épidémie chez les jeunes en ce sens où de plus en plus de jeunes filles, notamment au lycée ou à l'université échangent des services sexuels contre la prise en charge financière de leurs études (phénomènes de type « sugar daddy ») ou épousent des hommes beaucoup plus âgés et riches. Toute politique qui fournirait aux filles et jeunes femmes des sources ou même des perspectives de revenu les mettrait à l'abri des comportements risqués qui conduisent à la transmission du VIH.

Troisièmement, une action sur les inégalités de revenu et de genre est susceptible de se répercuter sur les autres déterminants de l'épidémie. Ainsi, réduire les inégalités de revenu se traduirait presque automatiquement par une baisse de la pauvreté, qui à son tour accroîtrait l'accès à l'éducation et aux soins de santé et partant, l'accès aux activités de prévention[93]. De même, l'utilisation d'une méthode moderne de contraception (variable qui apparaît significative dans la plupart de nos estimations) serait fortement renforcée par l'augmentation du revenu des femmes[94], leur meilleur accès aux campagnes de prévention (notamment par l'accès à l'information) et un niveau d'éducation plus élevé des femmes.

Quatrièmement, cette thèse apporte un message d'optimisme dans la lutte contre l'épidémie. En effet, la plupart des ingrédients de la lutte contre le VIH/Sida existent déjà et ont fait leurs preuves ailleurs, pour venir à bout d'autres problèmes de développement. Il s'agit par exemple des programmes de micro-crédit qui accroissent le revenu des femmes les plus pauvres, des projets de développement locaux qui permettraient de réduire le travail des migrants et de l'alphabétisation de masse. De plus, comme l'a souligné Stillwaggon (2006), les institutions qui proposent ces services aux pauvres existent déjà et ont déjà fait leurs preuves. D'autres éléments

[93] Même si l'accès aux soins de santé, le niveau d'éducation et la pauvreté n'apparaissant pas significatifs dans notre modèle, il ne faut pas oublier que nous utilisons des données agrégées et qu'au niveau individuel, ces trois variables (notamment l'accès aux soins de santé) pourraient d'agir sur la susceptibilité à l'infection.

[94] L'utilisation des préservatifs masculins dépend généralement des hommes, mais le préservatif féminin correctement utilisé est également efficace contre la transmission du VIH. Malheureusement, l'utilisation de cette contraception qui est totalement contrôlée par les femmes est rendue prohibitive du fait de son coût élevé ; en effet il coûte en moyenne 10 à 20 fois le prix du préservatif masculin (le prix de vente dans les pays en développement négocié par l'ONUSIDA et Female Health Company, l'entreprise leader productrice de préservatifs féminins dans le monde varie entre 30 et 61 centimes de dollars contre 3 centimes pour le préservatif masculin, Mazina et Martens, 2002).

de lutte contre l'épidémie, notamment le développement des microbicides totalement contrôlables par les femmes sont actuellement à l'étude[95]. En outre, une des solutions les plus efficaces, qui consiste en l'augmentation des taux de scolarisation des filles est également la plus facile à mettre en oeuvre. De fait, la réduction des inégalités de genre à travers la hausse de la scolarisation des filles en vue de réduire l'écart avec celle des garçons conduit à une baisse du taux de prévalence moyen de 9 % à 18%. Or, il s'avère qu'augmenter la scolarisation des filles est nettement plus facile à mettre en œuvre que réduire les inégalités de revenu, du moins avec une volonté politique bien affichée.

La communauté internationale a enfin compris que le VIH/Sida n'est pas une urgence de court terme, mais une épidémie de longue vague qui nécessite une réponse auto-entretenue et durable sur plusieurs décennies et ce à la fois sur les causes immédiates et structurelles. La conclusion de la 7ème conférence AIDSIMPACT sur les aspects socio-économiques du VIH/Sida est claire se résume en 2 messages. Le premier est un vieux message qui a déjà été énoncé par McKeon (1976), à savoir que les progrès bio-médicaux sont une condition nécessaire mais non suffisante pour améliorer l'état de santé et partant, lutter contre l'épidémie. Il faut en plus prendre en compte les aspects sociaux, culturels, techniques et psychologiques. A ce vieux message, s'ajoute un deuxième message plus nouveau: il est impératif de faire sauter les blocages à l'accès universel aux traitements du VIH/Sida. En effet, malgré l'efficacité maintenant reconnue des antirétroviraux et la chute spectaculaire de leurs prix ou même leur gratuité dans certains pays, ces médicaments sont encore inaccessibles pour les couches moyennes et les plus défavorisés dans les pays pauvres. De ce fait, l'immense majorité des personnes qui ont besoin d'antirétroviraux n'y a pas accès. Pourquoi ? Quels sont les déterminants du prix et de la distribution d'antirétroviraux dans les pays pauvres ? Pourquoi existe-t-il encore une inégalité dans l'accès aux antirétroviraux dans les pays en développement ? C'est à ces deux dernières questions qu'il faut d'apporter des éléments de réponse pour faire le tour de la question des liens entre inégalités et épidémie du VIH/Sida.

[95] Lors de son discours d'ouverture lors du16ème Congrès International sur le VIH/Sida prononcé le 13 Août 2006 à Toronto, Bill Gates a souligné que le développement des microbicides permettrait de «mettre le pouvoir d'éviter la transmission du VIH entre les mains des femmes».

APPENDIX

Appendix 1: A model of inter-temporal choice to understand risky behaviour

Preference for present, riscophobia and investment choice in health: Explaining why a risky behaviour can be optimal for the poor and for vulnerable women?

Let us first consider the case of a determinist model, and then we move on to the case where the agent can choose the risk level.

I- A determinist model

Consider the inter-temporal utility function given by:

$$U(c_t, c_{t+1}) = u(c_t) + \beta u(c_{t+1}). \quad (1)$$

The budgetary constraints are given by:

$$c_t = w - s_t \quad (2)$$

$$c_{t+1} = y(s_t), y'(s_t) > 0, y''(s_t) \leq 0. \quad (3)$$

Here, s_t represents health investment in first period, while $y(s_t)$ is the return of this investment (wage) which is realized only in second period. The hypotheses on $y(s_t)$ hold that payment in second period is an increasing and concave function of first period investment in health. The optimization problem is given by:

$\max_{\{s_t, s_{t+1}\}} U(c_t, c_{t+1})$ Under the constraints (2) and (3).

Assuming non satiety ($U'(.) \succ 0$), the budgetary constraints are bound and the problem can be non constraint as follows:

$$\max_{\{s_t\}} u(w - s_t) + \beta u(y(s_t)) \quad (4)$$

The FOC is given by:

$$-u'(w-s^*_t)+\beta y'(s^*)u'(y(s_t)) \;=0, \qquad (5)$$

Whereas the SOC is:

$$u''(w-s^*_t)+\beta\left[(y''(s^*_t))+y'(s^*_t))^2 u''(y(s^*_t)\right]\prec 0 \qquad (6)$$

Given the initial hypotheses, the SOC is always satisfied and the FOC is not only necessary, but also sufficient. By implicit differentiation of equation (6):

$$\frac{ds^*_t}{d\beta}=-\frac{\frac{\partial}{\partial\beta}(-u'(w-s^*_t)+\beta y'(s^*_t)u'(y(s_t))}{\frac{\partial}{\partial s^*_t}(-u'(w-s^*_t)+\beta y'(s^*_t)u'(y(s_t))}$$

$$=-\frac{y'(s^*_t)u'(s^*_t)}{CSO}\succ 0$$

So, if β decreases (the individual weights more the present), his investment in health will decrease.

As for comparative static concerning aversion to risk, let us consider a simple example with a utility function parametric CARA: $u(x)=-\theta\{-\exp-\theta x\}$ where θ represents Arrow-Pratt's absolute risk aversion coefficient. The FOC is then given by:

$$-\theta\exp\{-\theta(w-s^*_t)\}+\beta y'(s^*_t)\theta\exp\{-\theta(s^*_t)\}=0$$

Taking the logs, we get:

$$\theta(w-s^*_t)+\ln\beta+\ln y'(s^*_t)-\theta y(s^*_t)=0 \qquad (7)$$

By implicit differentiation, we get:

$$\frac{ds^*_t}{d\beta}=-\frac{\frac{\partial}{\partial\theta}(\theta(w-s^*_t)+\ln\beta y'(s^*_t)-\theta y(s^*_t))}{\frac{\partial}{\partial s^*_t}(\theta(w-s^*_t)+\ln\beta y'(s^*_t)-\theta y(s^*_t))}$$

$$=\frac{(w-s^*_t)-y(s^*_t)}{\underbrace{-\theta-\theta y'(s^*_t)}_{CSO\prec 0}}$$

$$=\frac{w-s^*_t-y(s^*_t)}{\theta\left[1+y'(s^*_t)\right]} \qquad (8)$$

The sign of the derived function is given by the sign of the numerator $w-s^*_t-y(s^*_t)$. In addition, a simple differentiation of (8) yields:

$$\frac{d^2 s_t^*}{d\theta^2} = \frac{w - s_t^* - y(s_t^*)}{\theta^2\left[1 + y'(s_t^*)\right]} = -\frac{1}{\theta}\frac{ds_t^*}{d\theta}.$$

If we take the limit case where second period return for investment in health is linear $(y(s_t) = ys_t)$, equation (7) becomes:

$$s_t^* = \frac{\theta w + \ln\beta + \ln y}{\theta\ (1+y)}$$

$$\frac{ds_t^*}{d\beta} = \frac{w\theta(1+y) - (1+y)(\theta w + \ln\beta + \ln y)}{\theta^2(1+y)^2} = \frac{\ln\beta + \ln y}{\theta^2(1+y)} < 0$$

So in the case of linear return, investment in health is a decreasing function of risk aversion.

II- The case where risk level is chosen by agent

Let us now consider a model where investment in health does not affect payment in second period but, when the later is stochastic, it reduces its risk. Let *y*, second period's income, which is supposed to be a random variable following the density $f(y,\rho)$ on the interval $[y, y]$ and where ρ represents Rothschild and Stiglitz's increasing parameter defined by the usual integral conditions:

$$(i) \quad \int_{\underline{y}}^{\bar{y}} F_p(y,p)dy = 0$$

$$(ii) \quad \int_{\underline{z}}^{\bar{z}} F_p(y,p)dy \geq 0, \forall z \in \left[\underline{y}, \bar{y}\right],$$

where $F(y,p)$ is the cumulative density associated with $f(y,p)$,

$$F(y,p) = \int f_p(y,p)dy \text{ and } F_p(y,p) = \frac{\partial}{\partial p}F(y,p)$$

In this case, investment in health in first period aims at reducing income's risk in second period, which we pose by writing the budgetary constraint in first period as:

$$c_t = w - k(p), k'(p) \prec 0, k''(p) \geq 0, \qquad (9)$$

Whereas the budgetary constraint in second period is:

$$c_{t+1} = y.$$

The hypotheses in (9) imply that it is costly in terms of first period health investment, to reduce the second period risk. The optimization problem is given by the optimal risk level choice or:

$$\max_{\{p\}} u(w-k(\rho))+\beta E\left[u(y)\right],$$

Which is:

$$\max_{\{p\}} u(w-k(\rho))+\beta\int_{\underline{y}}^{\bar{y}} u(y)\, f(y,\rho)dy.$$

By the Leibnitz's rule, the FOC associated with this problem is:

$$-k'(\rho^*)u'(w-k(\rho^*))+\beta\int_{\underline{y}}^{\bar{y}} u(y)\, f_\rho(y,\rho^*)dy=0, \tag{10}$$

Whereas the SOC is given by:

$$0\prec k''(\rho^*)u'(w-k(\rho^*))+\left[k'(\rho^*)\right]^2 u''(w-c(\rho^*))+\beta\int_{\underline{y}}^{\bar{y}} u(y)\, f_{\rho\rho}(y,\rho^*)dy$$

Where the inequality will hold (and the FOC will then be not only necessary, but also sufficient) if we further impose the condition that $F_{\rho\rho}(y,\rho^*)\succ 0$.

By implicit differentiation of (10), it is immediate that:

$$\frac{d\rho^*}{d\beta}=\frac{\int_{\underline{y}}^{\bar{y}} u(y)\, f_\rho(y,\rho^*)dy}{CSO\prec 0}\prec 0$$

Where we know that the numerator is negative from a trivial application of the fundamental risk theorem. **It follows that if β decreases (the individual weights more present), he will make riskier health choices** $(\beta\downarrow\Rightarrow\rho^*\uparrow)$.

Concerning risk aversion, let us the consider a simple parametric example where utility function is CARA $(u(x)=-\exp\{-\theta x\})$ and y follows a normal distribution N (0, σ_y^2). The Rothschild and Stiglitz increasing parameter ϱ can in this case be assimilated to the distribution varaiance σ_y^2.

In this simple case, the optimization problem is given by:

$$\max_{\{\sigma_y^2\}} -\exp\{-\theta(w-k(\sigma_y^2))\} - \beta\int_{\underline{y}}^{\bar{y}} \exp\{-\theta y\}\frac{1}{\sqrt{2\pi\sigma_y}}\exp\left\{-\frac{y^2}{2\sigma_y^2}\right\}dy.$$

From the usual property of the moment's generative function of a lognormal density, we know that:

$$\int_{\underline{y}}^{\bar{y}} \exp\{-\theta y\}\frac{1}{\sqrt{2\pi\sigma_y}}\exp\left\{-\frac{y^2}{2\sigma_y^2}\right\}dy = \exp\left\{\theta^2\frac{\sigma_y^2}{2}\right\}$$

And we can rewrite the optimization problem as follows:

$$\max_{\{\sigma_y^2\}} -\exp\{-\theta(w-k(\sigma_y^2))\} - \beta\exp\left\{\theta^2\frac{\sigma_y^2}{2}\right\}$$

The FOC is given by:

$$-\theta k'(\sigma_y^{2*})\exp\{-\theta(w-k(\sigma_y^2))\} - \beta\frac{\theta^2}{2}\exp\left\{\theta^2\frac{\sigma_y^2}{2}\right\} = 0$$

By implicit differentiation of the FOC :

$$\frac{d\sigma_y^{2*}}{d\theta} = -\frac{\begin{bmatrix} -\left[\theta k'(\sigma_y^{2*})\right]^2\exp\{-\theta(w-k(\sigma_y^2))\} \\ -\theta k''(\sigma_y^{2*})\exp\{-\theta(w-k(\sigma_y^2))\} \\ -\beta\theta\ \exp\left\{\theta^2\frac{\sigma_y^2}{2}\right\} - \beta\frac{\theta^2}{2}\left(\theta^2\frac{\sigma_y^{2*}}{2}\right)\exp\left\{\theta^2\frac{\sigma_y^2}{2}\right\} \end{bmatrix}}{CSO \prec 0} \prec 0$$

It logically follows that the increase in agent's riscophobia will reduce the optimal risk in his health choices.

Appendix 2: Composition of the samples

Figure A.1: Composition of the first sample (adult prevalence rate)

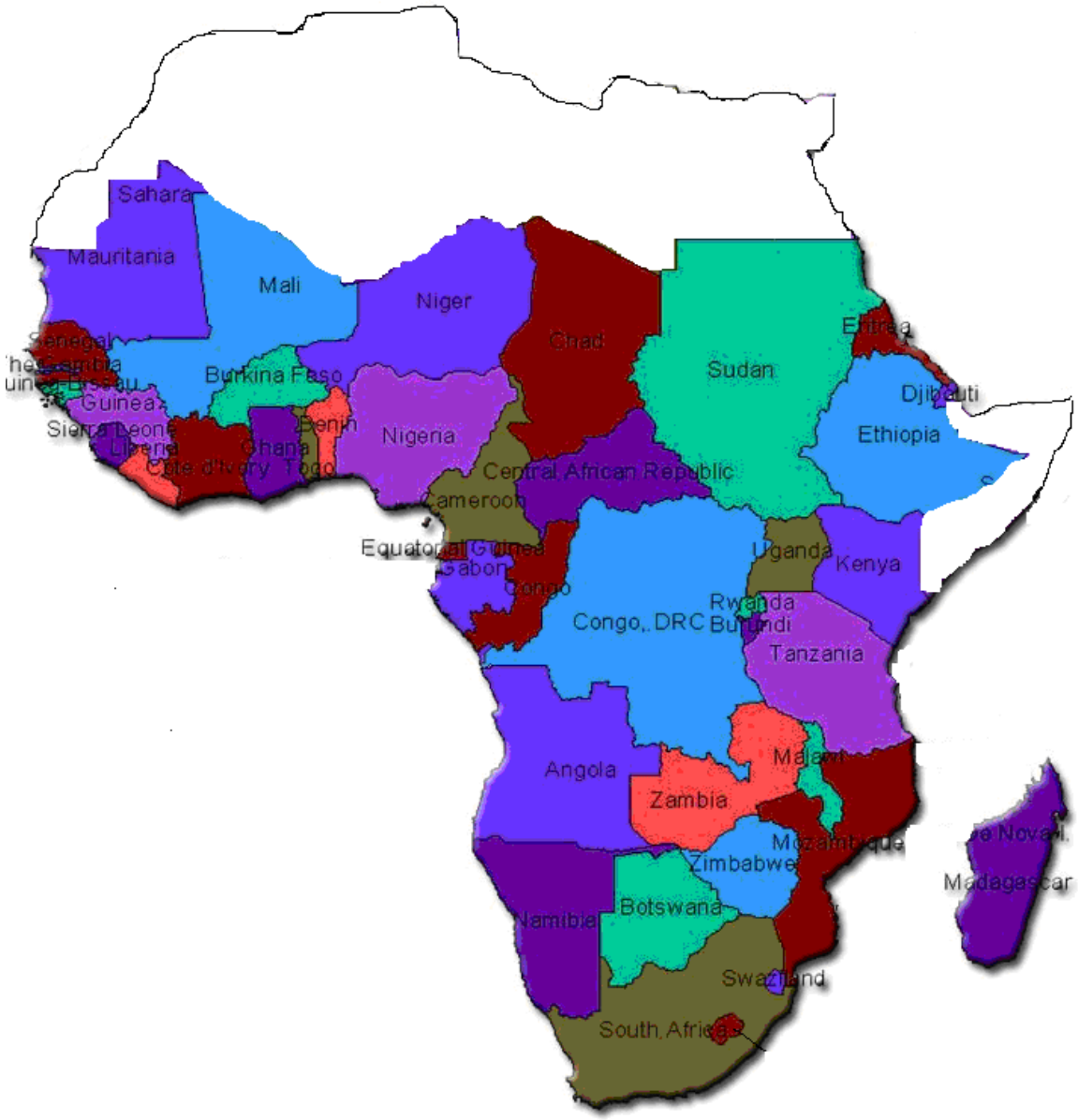
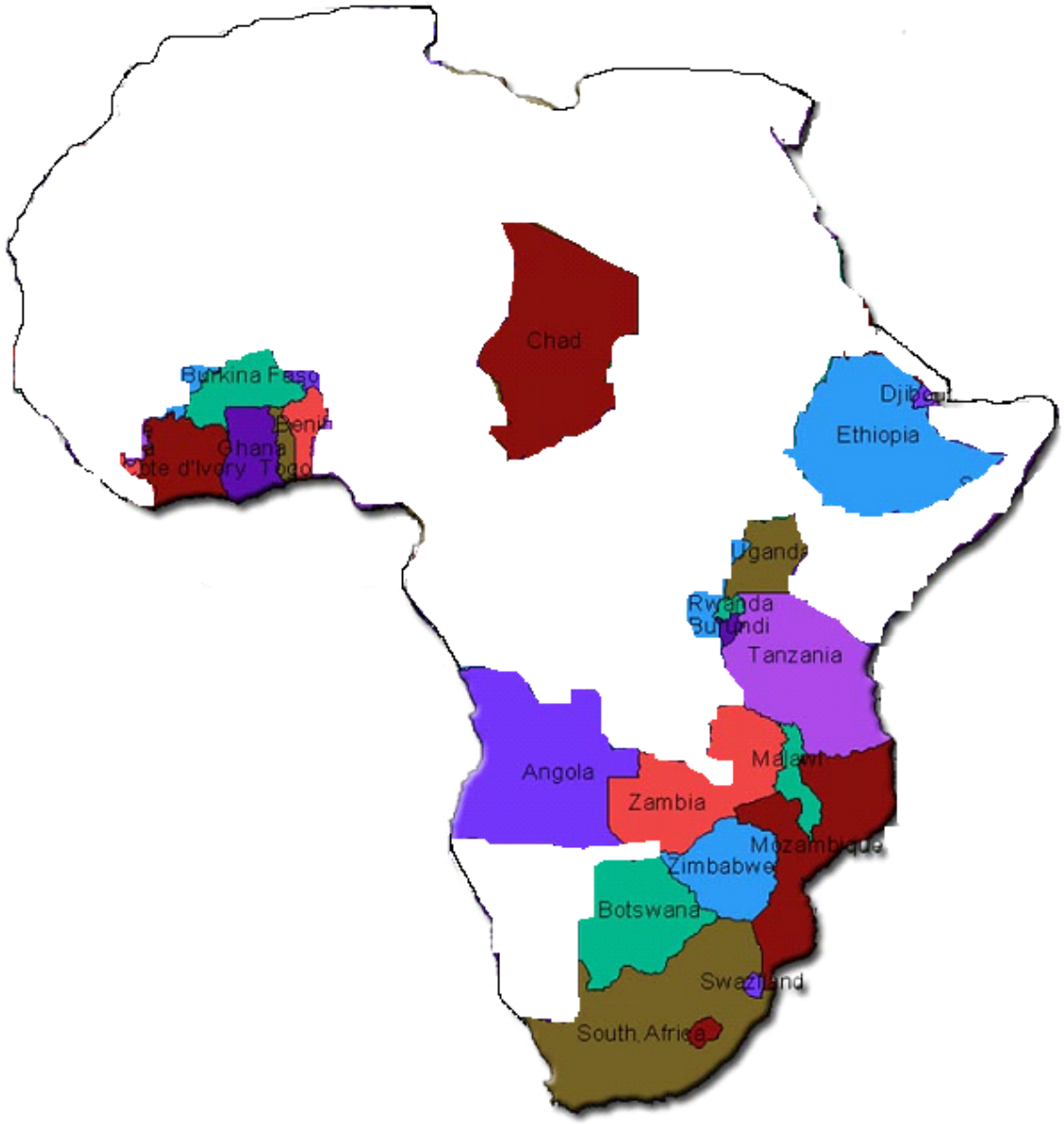


Figure A.2: Composition of the second sample (incidence rate)



Appendix 3: Correlations

Table A.1: Correlations between the variables on the first sample

	HIV Prev rate	Gini Coef	Fem-ale inco me	Shar of Fem labor	Fem enrol rate	Mat leave wage (%)	Mat leave wks	GNI	GNI (lag val)	Tel Main lines	Mal nut prev	Acce safe wa ter	HC Expe nd pc	Imm un rate	Ratio PUB /PRI	Adlt Lit rate (%)	Te Levis ions	Con trace ptive prev	Mus lim (%)	ELF	Urba nizati on rate	Conf lict	Voi ce
HIV prev rate	1.00																						
Gini Coef	0.54	1.00																					
Female income	-0.13	-0.23	1.00																				
Sh of fem labor	-0.07	-0.24	0.85	1.00																			
Female enrol rate	0.51	0.49	-0.21	-0.23	1.00																		
Wage during mat leave (%)	-0.48	-0.31	0.22	0.18	-0.24	1.00																	
Mat leave (wks)	-0.10	-0.02	-0.11	-0.11	-0.12	0.00	1.00																
GNI per capita	0.37	0.36	-0.36	-0.36	0.66	-0.22	0.16	1.0															
GNI per capita (lagged value)	0.40	0.39	-0.36	-0.34	0.67	-0.23	0.19	**0.98**	1.0														
Telephone lines	0.15	0.13	-0.34	-0.34	0.50	-0.03	0.07	**0.85**	0.83	1.0													
Malnut preval	-0.37	-0.29	0.23	0.25	-0.56	0.15	-0.08	-0.47	-0.48	-0.38	1.0												
Access to safe water	0.32	0.22	-0.18	-0.14	0.45	-0.13	0.01	0.53	0.55	0.50	-0.39	1.0											
Health Care expen per capita	0.35	0.34	-0.25	-0.27	0.56	-0.19	0.17	0.83	0.83	0.61	-0.39	0.42	1.0										
Immun rate	0.37	0.05	0.15	0.17	0.39	-0.02	-0.31	0.30	0.29	0.34	-0.30	0.38	**0.23**	1.0									
Ratio PUB/PRI	-0.16	-0.23	-0.01	-0.08	-0.18	0.08	0.04	-0.19	-0.19	-0.14	0.15	-0.11	**-0.10**	-0.23	1.00								
Adult Lit rate	-0.09	0.28	-0.23	-0.28	0.66	-0.18	-0.14	0.47	0.47	0.37	-0.41	0.32	0.35	0.27	-0.01	1.0							
Television sets	0.42	-0.01	-0.52	-0.57	0.31	0.11	-0.01	0.61	0.58	0.66	-0.27	0.37	0.42	0.02	0.00	0.42	1.0						
Contraceptive prevalence	0.33	0.21	-0.09	-0.15	0.58	-0.04	-0.01	0.64	0.67	0.72	-0.47	0.50	0.50	0.38	-0.08	0.53	0.44	1.00					
Muslim pop (%)	-0.41	-0.26	-0.07	-0.03	-0.38	0.12	0.11	-0.19	-0.19	-0.10	0.19	-0.09	-0.17	-0.17	-0.03	-0.45	0.08	-0.36	1.00				
Ethnic fractionalization	-0.01	0.21	-0.11	-0.22	-0.04	0.05	0.03	0.04	0.05	0.02	-0.02	-0.10	0.14	-0.18	0.17	-0.00	0.10	-0.01	0.20	1.0			
Urb rate	-0.04	0.23	-0.30	-0.34	0.18	0.04	0.41	0.35	0.37	0.21	-0.29	0.28	0.33	-0.22	0.07	0.25	0.38	0.18	0.17	0.38	1.0		
Conflict	-0.29	-0.22	-0.12	-0.06	-0.36	-0.09	-0.03	-0.33	-0.32	-0.25	0.32	-0.23	-0.29	-0.36	0.24	-0.14	-0.11	-0.38	0.06	-0.02	-0.13	1.0	
Voice	0.23	0.32	0.05	0.09	0.38	-0.05	0.15	0.48	0.47	0.50	-0.33	0.34	0.36	0.34	-0.35	0.05	0.13	0.44	-0.02	-0.02	0.13	-0.47	1.0

Table A.2: Correlations between the variables on the second sample

	HIV Prev rate	Gini Coef	Fem-ale inco me	Shar of Fem labor	Fem enrol rate	Mat leave wage (%)	Mat leave wks	GNI	GNI (lag val)	Tel Main lines	Mal nut prev	Acce safe wa ter	HC Expe nd pc	Imm un rate	Ratio PUB /PRI	Adlt Lit rate (%)	Te Levis ions	Con trace ptive prev	Mus lim (%)	ELF	Urba nizati on rate	Conf lict	Voi ce
HIV prev rate	1.00																						
Gini Coef	0.42	1.00																					
Female income	-0.51	-0.33	1.00																				
Sh of fem labor	-0.46	-0.28	0.95	1.00																			
Female enrol rate	0.45	0.58	-0.19	-0.11	1.00																		
Wage during mat leave (%)	-0.19	-0.46	0.26	0.19	-0.60	1.00																	
Mat leave (wks)	-0.27	0.04	-0.17	-0.19	-0.23	0.00	1.00																
GNI per capita	0.47	0.54	-0.37	-0.33	0.62	-0.46	0.31	1.0															
GNI per capita (lagged value)	0.45	0.57	-0.40	-0.37	0.63	-0.45	0.33	**0.99**	1.0														
Telephone lines	0.52	0.51	-0.40	-0.37	0.59	-0.31	0.28	**0.96**	0.96	1.0													
Malnut preval	-0.14	-0.56	0.44	0.34	-0.52	0.42	-0.14	-0.46	-0.51	-0.45	1.0												
Access to safe water	0.45	0.44	-0.15	-0.12	0.49	-0.13	-0.00	0.50	0.51	0.52	-0.43	1.0											
Health Care expen per capita	0.34	0.43	-0.29	-0.30	0.49	-0.30	0.27	0.85	0.83	0.80	-0.33	0.38	1.0										
Immun rate	0.15	0.36	0.29	0.29	0.62	-0.23	-0.09	0.36	0.36	0.35	-0.35	0.46	**0.24**	1.0									
Ratio PUB/PRI	-0.02	-0.33	-0.08	-0.15	-0.09	0.29	-0.01	-0.15	-0.13	-0.07	0.13	0.02	**-0.02**	-0.18	1.00								
Young people Literacy rate	0.66	0.51	-0.42	-0.34	0.56	-0.31	-0.13	0.43	0.46	0.44	-0.32	0.47	0.27	0.26	-0.04	1.0							
Television sets	0.64	0.06	-0.59	-0.65	0.03	0.15	-0.05	0.31	0.31	0.40	-0.09	0.28	0.26	-0.23	0.25	0.44	1.0						
Contraceptive prevalence	0.17	0.46	-0.19	-0.18	0.55	-0.08	0.20	0.48	0.57	0.55	-0.45	0.44	0.49	0.38	0.03	0.43	0.16	1.00					
Muslim pop (%)	0.36	-0.23	-0.32	-0.35	-0.21	0.23	-0.16	-0.02	-0.06	0.06	0.18	-0.20	-0.03	-0.40	0.19	-0.03	0.56	-0.29	1.00				
Ethnic fractionalization	0.01	0.18	-0.32	-0.35	0.03	0.19	0.09	0.22	0.23	0.29	-0.15	-0.21	0.27	-0.25	0.16	0.02	0.31	0.00	0.46	1.0			
Urb rate	0.32	0.46	-0.39	-0.38	0.19	0.08	0.39	0.59	0.59	0.62	-0.34	0.36	0.49	0.04	-0.01	0.33	0.47	0.24	0.21	0.62	1.0		
Conflict	-0.09	-0.62	-0.00	-0.03	-0.45	0.09	-0.09	-0.34	-0.36	-0.34	0.47	-0.24	-0.31	-0.58	0.20	-0.15	0.09	-0.44	0.29	-0.14	-0.36	1.0	
Voice	0.15	0.45	0.16	0.20	0.46	-0.24	0.19	0.55	0.52	0.52	-0.36	0.30	0.41	0.54	-0.27	0.03	-0.12	0.20	-0.08	0.14	0.43	-0.64	1.0

Figure A.3: Simple correlation between HIV prevalence rate and key determinants on the first sample

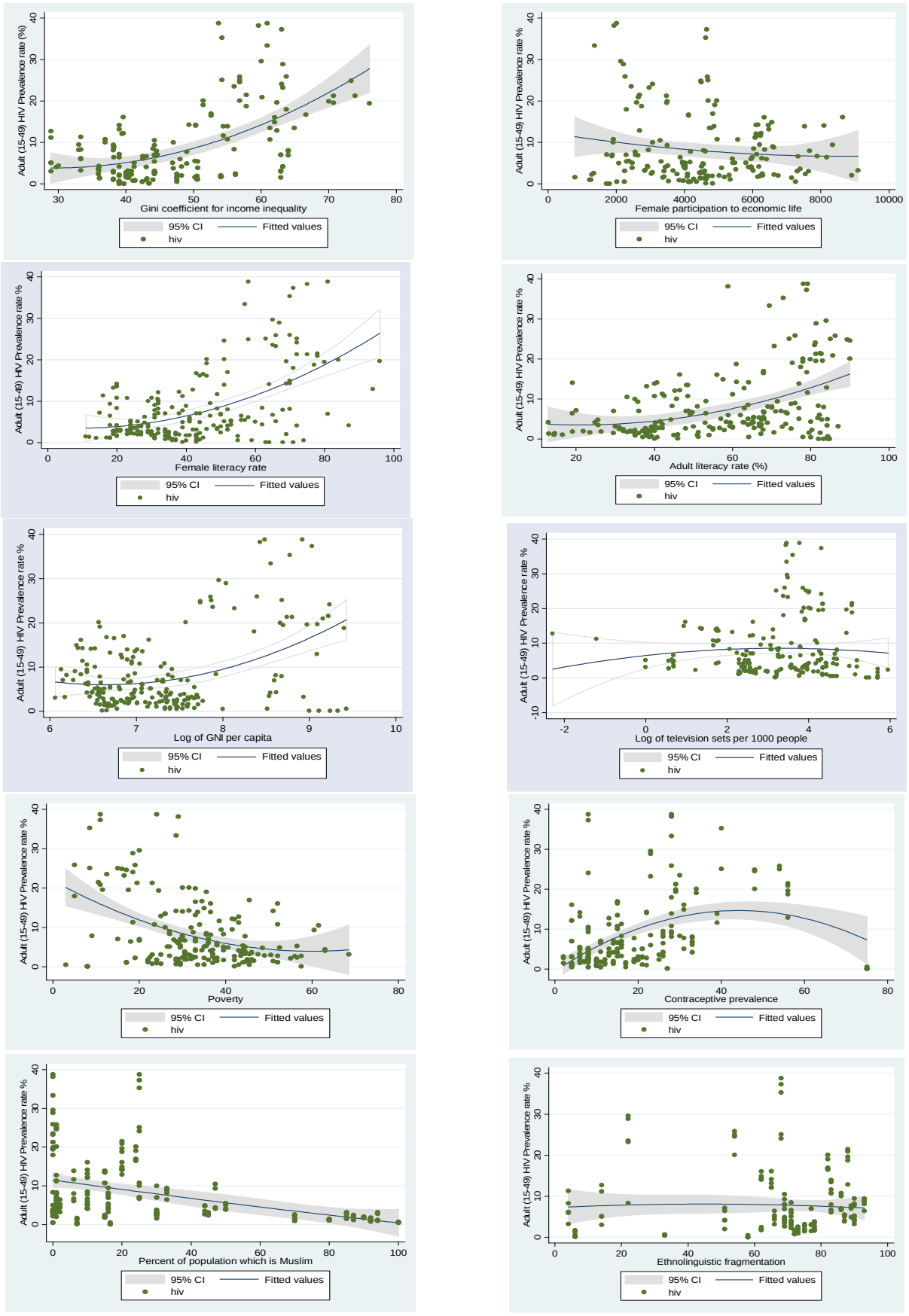
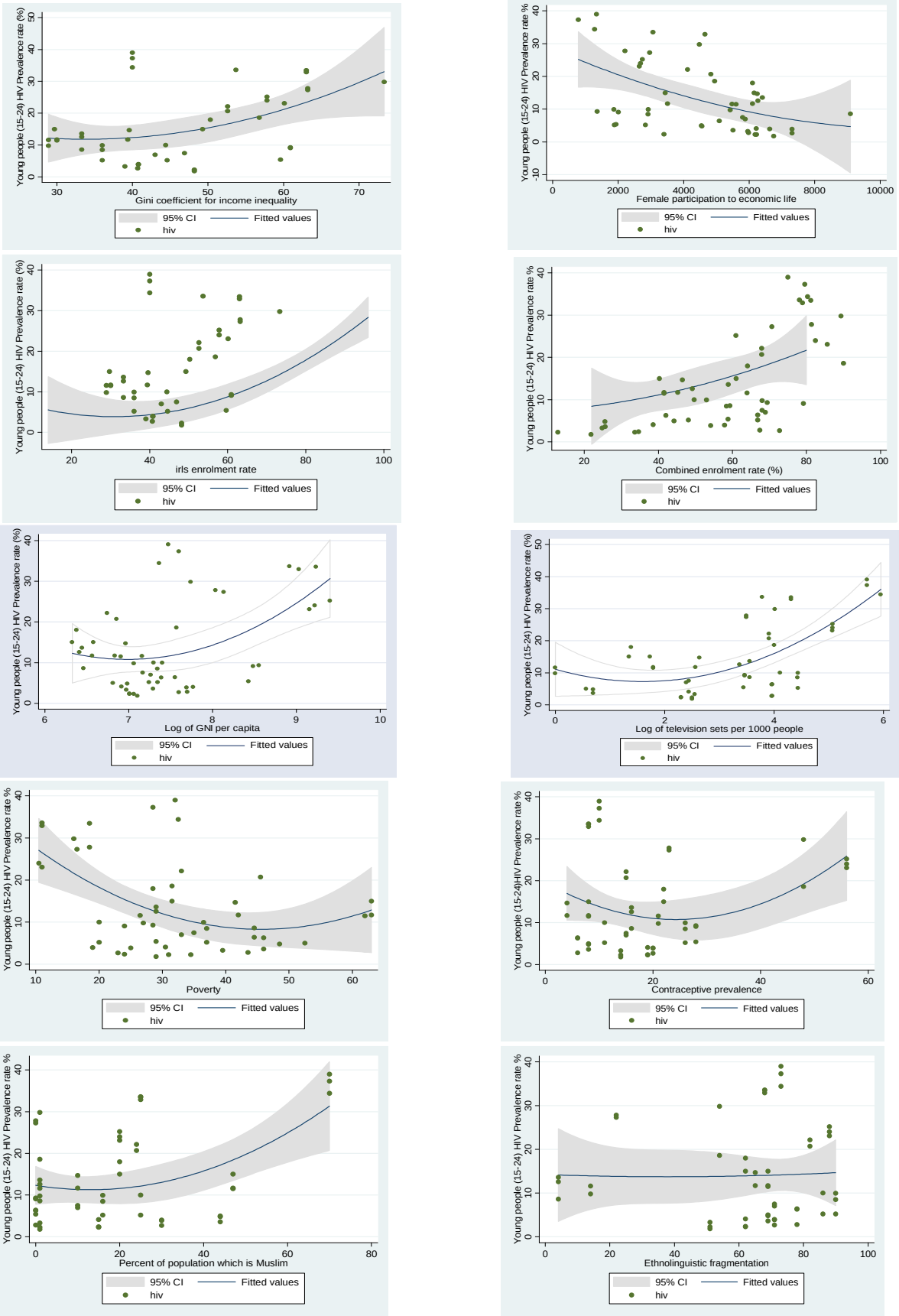


Figure A.4: Simple correlation between HIV prevalence rate and key determinants on the second sample



Appendix 4: Distribution of variables

Figure A.5: Distribution of key variables determinants on the first sample

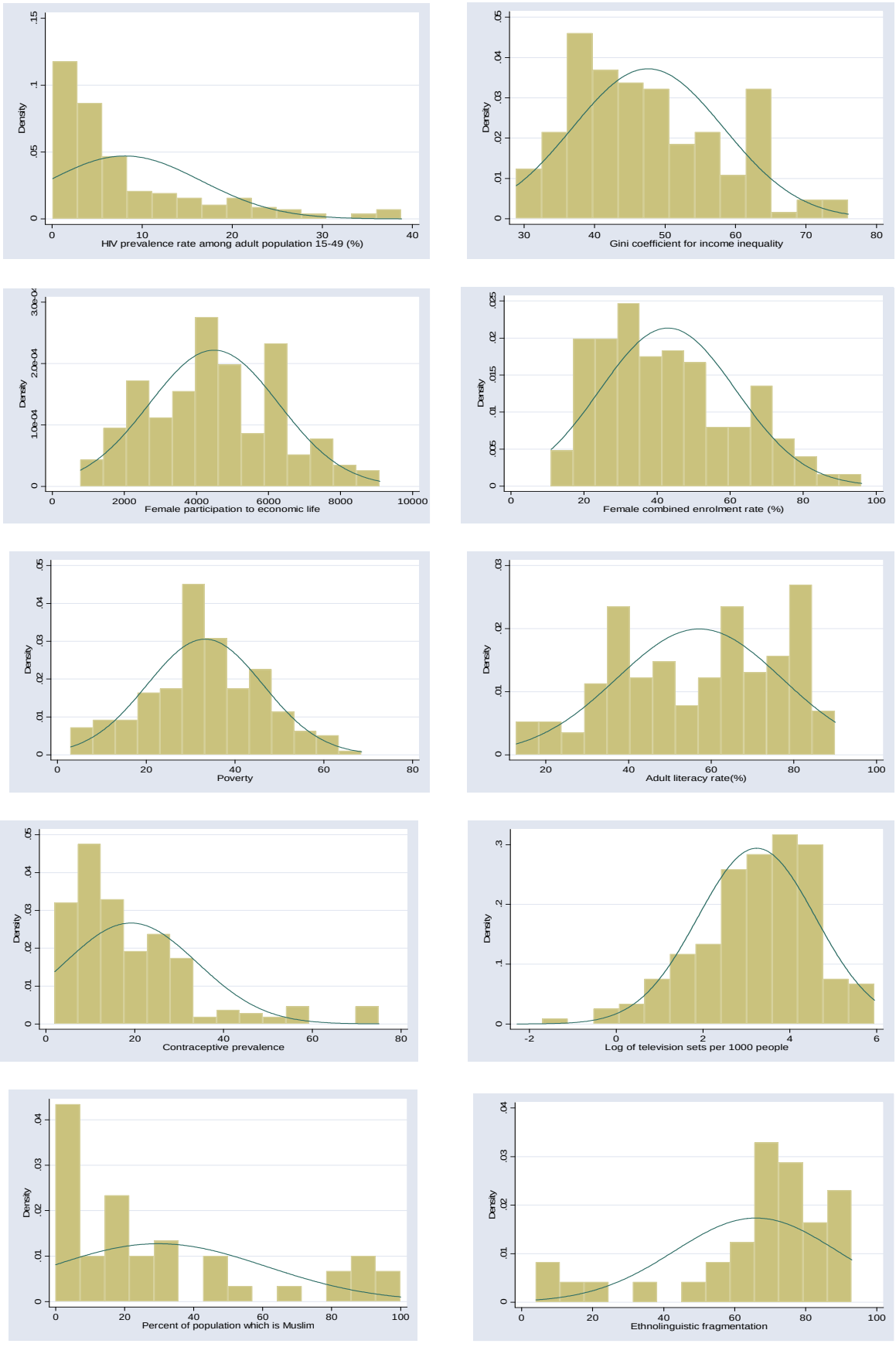
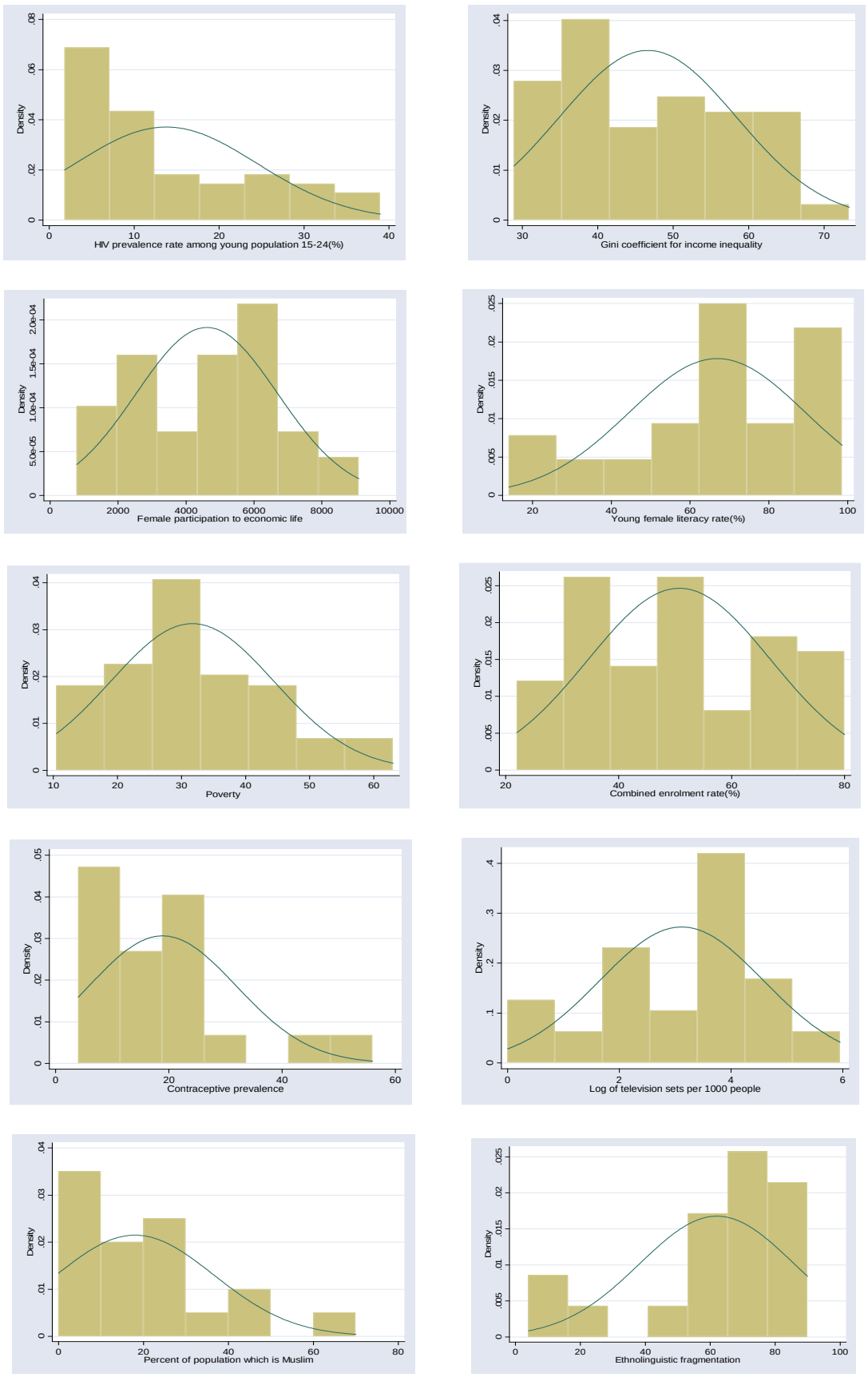


Figure A.6: Distribution of key variables determinants on the second sample



Appendix 5: Effect of a reduction of socio-economic inequalities on HIV/AIDS epidemic

Table A.3: Effect of a reduction of inequality on adult (15-49) prevalence rate in Sub-Saharan African countries

Country	year	Hiv (actual value)	HIV (predicted value)	Δ income inequality = -5% Δ Girl's enrolment rate= 0	% Change*	Δ income inequality = -5% Δ Girl's enrolment rate = +5%	% Change*	Δ income inequality = -5% Δ Girl's enrolment rate = +10%	% Change*	Δ income inequality = -5% Δ Girl's enrolment rate = + 20%	% Change*
Botswana	1999	35,30	14,02	13,15	6,19	12,45	11,16	11,79	15,90	10,55	24,73
Botswana	2003	37,30	32,58	30,72	5,71	29,39	9,79	28,09	13,77	25,60	21,40
Botswana	2005	24,10	79,44	78,00	1,81	76,88	3,23	75,71	4,69	73,28	7,76
Burkina Faso	1997	7,17	4,76	4,53	4,95	4,47	6,16	4,41	7,35	4,30	9,69
Burkina Faso	1999	6,44	6,43	6,12	4,87	6,02	6,29	5,93	7,69	5,76	10,44
Burkina Faso	2001	4,20	2,50	2,38	5,06	2,34	6,54	2,30	7,99	2,23	10,83
Burkina Faso	2003	4,20	3,14	2,94	6,18	2,89	7,79	2,84	9,37	2,74	12,46
Burkina Faso	2005	2,01	3,67	3,45	6,15	3,38	7,99	3,31	9,80	3,18	13,30
Burundi	1997	8,30	9,07	8,70	4,06	8,56	5,61	8,42	7,14	8,15	10,12
Burundi	1999	11,32	16,32	15,71	3,75	15,52	4,90	15,33	6,04	14,97	8,29
Burundi	2001	6,20	7,73	7,42	4,11	7,25	6,31	7,08	8,45	6,76	12,61
Burundi	2003	6,00	7,67	7,35	4,12	7,17	6,54	6,99	8,91	6,64	13,49
Burundi	2005	3,26	2,29	2,19	4,35	2,13	6,98	2,07	9,54	1,96	14,46
Cameroon	1999	7,73	6,32	5,93	6,09	5,74	9,11	5,56	12,05	5,20	17,65
Cameroon	2001	7,00	4,56	4,30	5,60	4,15	9,01	4,00	12,29	3,71	18,53
CAR	1997	10,77	4,65	4,33	6,92	4,25	8,50	4,18	10,05	4,04	13,07
CAR	1999	13,91	7,23	6,74	6,75	6,63	8,28	6,52	9,80	6,31	12,76
CAR	2001	13,50	11,41	10,55	7,58	10,38	9,05	10,22	10,49	9,89	13,32
CAR	2005	10,73	7,23	6,69	7,49	6,56	9,25	6,44	10,97	6,20	14,32
Cote D'ivoire	1997	10,06	5,31	5,06	4,66	4,92	7,21	4,79	9,70	4,54	14,48
Cote D'ivoire	1999	10,76	6,43	6,13	4,61	5,98	6,98	5,83	9,29	5,54	13,75
Cote D'ivoire	2001	6,70	6,22	5,88	5,54	5,73	7,96	5,58	10,33	5,29	14,90

Ethiopia	1997	9,31	8,53	8,07	5,38	7,95	6,77	7,83	8,14	7,60	10,82
Ethiopia	1999	10,52	6,87	6,49	5,48	6,32	7,95	6,16	10,37	5,84	15,02
Ethiopia	2001	4,10	3,86	3,71	3,83	3,63	6,03	3,54	8,19	3,38	12,36
Ethiopia	2003	4,40	4,42	4,25	3,84	4,14	6,19	4,04	8,49	3,85	12,93
Ethiopia	2005		1,59	1,53	3,95	1,49	6,45	1,45	8,88	1,38	13,56
Gabon	2005	7,88	18,33	17,06	6,94	16,22	11,53	15,41	15,93	13,89	24,20
The Gambia,	1999	1,96	1,97	1,87	5,09	1,81	8,11	1,75	11,04	1,64	16,63
The Gambia,	2001	1,60	1,86	1,74	6,51	1,68	9,97	1,61	13,30	1,50	19,60
The Gambia,	2003	1,20	1,87	1,75	6,17	1,68	9,80	1,62	13,29	1,50	19,87
The Gambia,	2005	2,44	1,85	1,74	6,17	1,66	10,19	1,59	14,05	1,46	21,27
Ghana	1997	2,38	2,15	2,05	4,68	1,99	7,72	1,92	10,65	1,80	16,26
Ghana	1999	3,60	1,97	1,88	4,69	1,81	7,89	1,75	10,98	1,64	16,87
Ghana	2001	3,10	1,90	1,80	5,31	1,74	8,73	1,67	12,03	1,55	18,28
Guinea	2005	1,52	1,19	1,13	5,29	1,10	8,17	1,06	10,96	1,00	16,30
Kenya	1997	11,64	14,97	14,05	6,15	13,53	9,62	13,02	12,98	12,07	19,38
Kenya	1999	13,90	17,21	16,18	6,00	15,57	9,53	14,98	12,95	13,86	19,47
Kenya	2001	8,00	16,65	15,82	4,96	15,22	8,61	14,63	12,15	13,51	18,88
Kenya	2003	6,70	14,72	14,01	4,85	13,48	8,44	12,97	11,91	11,99	18,52
Kenya	2005	6,09	7,23	6,85	5,25	6,53	9,71	6,22	13,98	5,65	21,96
Lesotho	1999	23,53	9,62	8,98	6,70	8,51	11,51	8,07	16,09	7,25	24,60
Lesotho	2001	29,60	7,71	7,15	7,30	6,77	12,17	6,42	16,80	5,75	25,38
Lesotho	2003	28,90	8,70	8,04	7,60	7,61	12,55	7,20	17,26	6,44	25,98
Lesotho	2005	23,24	8,47	7,83	7,62	7,41	12,51	7,02	17,16	6,29	25,78
Madagascar	1997	0,12	0,57	0,54	5,72	0,52	8,93	0,50	12,02	0,47	17,91
Madagascar	1999	0,15	0,90	0,85	5,70	0,82	9,22	0,79	12,61	0,73	19,01
Madagascar	2001	1,30	1,16	1,10	5,65	1,06	9,16	1,02	12,54	0,94	18,94
Madagascar	2003	1,70	1,99	1,87	6,16	1,81	9,39	1,75	12,51	1,63	18,43
Madagascar	2005	0,51	0,58	0,55	6,24	0,52	10,71	0,50	14,96	0,45	22,87
Malawi	1997	14,92	21,56	20,16	6,48	19,17	11,05	18,22	15,45	16,44	23,74
Malawi	1999	16,10	26,77	25,14	6,08	24,38	8,93	23,63	11,72	22,18	17,13
Malawi	2001	14,30	18,38	17,39	5,38	16,51	10,16	15,67	14,74	14,09	23,34
Malawi	2003	14,20	23,48	22,26	5,16	21,22	9,62	20,21	13,92	18,30	22,06
Malawi	2005	14,09	8,86	8,32	6,08	7,89	10,88	7,49	15,45	6,74	23,96
Mali	1997	1,67	2,49	2,32	6,94	2,28	8,55	2,24	10,13	2,16	13,21
Mali	1999	2,03	1,46	1,35	7,01	1,27	12,49	1,20	17,65	1,06	27,08

Mali	2001	1,90	1,49	1,38	7,01	1,35	9,11	1,32	11,17	1,26	15,14
Mali	2003	1,90	1,30	1,22	6,58	1,19	8,78	1,16	10,92	1,11	15,06
Mali	2005	1,73	1,18	1,10	6,59	1,07	9,03	1,04	11,40	0,99	15,97
Mauritania	1997	0,52	0,68	0,64	5,59	0,62	8,55	0,60	11,42	0,56	16,90
Mauritania	1999	0,52	0,45	0,42	5,60	0,41	8,65	0,39	11,60	0,37	17,23
Mauritania	2001	0,50	0,41	0,39	5,16	0,38	8,47	0,37	11,67	0,34	17,73
Mauritania	2003	0,60	0,53	0,50	5,16	0,48	8,71	0,47	12,13	0,43	18,58
Mauritania	2005	0,68	0,70	0,66	5,15	0,64	8,78	0,61	12,26	0,57	18,85
Mauritius	1999	0,08	0,21	0,20	5,26	0,19	10,51	0,18	15,46	0,16	24,57
Mauritius	2001	0,08	0,11	0,11	5,18	0,10	10,75	0,10	16,00	0,09	25,58
Mauritius	2003	0,08	0,12	0,12	5,18	0,11	10,99	0,10	16,44	0,09	26,37
Mauritius	2005	0,55	6,06	5,77	4,89	5,42	10,63	5,09	16,05	4,49	25,96
Niger	1997	1,45	1,09	1,04	4,76	1,03	5,68	1,02	6,59	1,00	8,39
Niger	1999	1,35	2,23	2,12	4,71	2,10	5,70	2,08	6,68	2,03	8,62
Niger	2003	1,20	2,22	2,07	6,52	2,04	7,90	2,01	9,26	1,95	11,92
Niger	2005	1,10	1,49	1,39	6,57	1,37	8,04	1,35	9,48	1,31	12,30
Nigeria	1999	5,08	6,48	6,14	5,13	5,94	8,34	5,73	11,44	5,35	17,35
Nigeria	2001	5,50	7,01	6,57	6,19	6,35	9,34	6,14	12,40	5,73	18,22
Nigeria	2003	5,40	3,14	2,93	6,48	2,79	10,98	2,66	15,28	2,41	23,27
Nigeria	2005	3,86	2,79	2,61	6,50	2,50	10,47	2,39	14,28	2,19	21,44
Rwanda	1999	11,21	11,92	11,51	3,42	11,16	6,36	10,82	9,21	10,17	14,69
Rwanda	2001	5,10	6,82	6,57	3,61	6,30	7,63	6,04	11,49	5,54	18,76
Rwanda	2003	5,10	6,37	6,14	3,63	5,87	7,82	5,62	11,83	5,14	19,38
Rwanda	2005	3,07	2,24	2,16	3,78	2,06	8,05	1,97	12,14	1,80	19,78
Senegal	1997	1,77	1,09	1,02	7,05	0,99	9,56	0,96	12,00	0,91	16,69
Senegal	2001	0,80	0,74	0,70	5,41	0,68	8,21	0,66	10,93	0,62	16,14
South Africa	1997	12,91	18,25	17,02	6,77	15,86	13,07	14,78	19,03	12,79	29,93
South Africa	1999	19,61	24,69	23,14	6,27	21,66	12,29	20,24	18,03	17,62	28,65
South Africa	2001	20,90	13,35	12,43	6,90	11,69	12,42	10,99	17,66	9,70	27,31
South Africa	2003	21,50	13,93	13,01	6,60	12,24	12,11	11,51	17,33	10,17	26,97
Sudan	1997	0,99	2,14	2,02	5,59	1,97	8,11	1,92	10,57	1,81	15,29
Sudan	1999	0,99	2,32	2,19	5,70	2,13	8,22	2,07	10,67	1,96	15,38
Sudan	2001	2,30	1,63	1,54	5,23	1,50	7,86	1,46	10,41	1,38	15,31
Sudan	2003	2,60	1,53	1,45	5,24	1,41	8,11	1,36	10,89	1,28	16,21
Sudan	2005	1,59	1,12	1,06	5,26	1,03	8,06	1,00	10,78	0,94	15,98

Tanzania	1997	9,42	23,58	22,65	3,92	22,16	6,02	21,67	8,09	20,72	12,14
Tanzania	2001	9,00	20,26	19,43	4,09	19,00	6,21	18,58	8,29	17,76	12,35
Tanzania	2003	8,80	17,09	16,36	4,25	15,88	7,07	15,41	9,83	14,50	15,13
Tanzania	2005	6,46	5,49	5,23	4,82	5,02	8,53	4,83	12,10	4,46	18,85
Togo	1997	9,51	5,33	5,11	4,18	4,91	7,92	4,72	11,52	4,36	18,32
Togo	1999	8,19	6,28	6,02	4,14	5,78	8,00	5,55	11,72	5,11	18,72
Togo	2001	4,30	3,90	3,72	4,62	3,56	8,86	3,40	12,93	3,10	20,54
Togo	2003	4,10	4,08	3,89	4,61	3,72	8,77	3,56	12,76	3,26	20,23
Togo	2005	3,24	1,53	1,46	4,73	1,40	8,50	1,35	12,13	1,24	18,96
Uganda	1997	8,52	13,59	12,90	5,08	12,55	7,70	12,20	10,26	11,53	15,20
Uganda	1999	5,98	12,43	11,74	5,60	11,36	8,60	11,00	11,52	10,31	17,12
Uganda	2001	5,10	11,13	10,52	5,55	9,98	10,41	9,46	15,04	8,50	23,68
Uganda	2003	4,10	9,67	9,17	5,18	8,65	10,56	8,16	15,67	7,25	25,10
Uganda	2005	6,66	9,92	9,41	5,16	8,92	10,03	8,46	14,67	7,61	23,29
Zambia	1997	19,07	14,34	13,50	5,86	13,03	9,15	12,57	12,34	11,70	18,44
Zambia	1999	20,12	17,92	16,91	5,63	16,35	8,80	15,79	11,89	14,73	17,81
Zambia	2001	16,70	31,94	30,00	6,08	29,20	8,58	28,41	11,05	26,88	15,85
Zambia	2003	16,50	23,06	21,81	5,42	21,13	8,35	20,47	11,22	19,19	16,75
Zambia	2005	16,96	8,72	8,17	6,36	7,83	10,27	7,50	14,03	6,88	21,12
Zimbabwe	1997	25,84	4,29	3,99	7,16	3,77	12,27	3,56	17,10	3,18	26,02
Zimbabwe	1999	25,06	5,88	5,47	7,05	5,18	11,87	4,92	16,44	4,42	24,93
Zimbabwe	2001	24,90	14,33	13,15	8,27	12,57	12,31	12,01	16,20	10,96	23,53
Zimbabwe	2003	24,60	13,33	12,46	6,53	11,97	10,19	11,50	13,72	10,61	20,43
Zimbabwe	2005	20,12	12,85	12,01	6,57	11,53	10,24	11,08	13,79	10,21	20,52

All values are expressed in %.
The baseline is the predicted value (fourth column).
*The changes are negative, thus values reported here are absolute values.
The results of the simulation of a reduction in gender inequality with no reduction in income inequality are not presented here, as they are exactly the same as when income inequality is reduced.

Table A.4: Effect of a reduction of gender inequality on incidence rate in Sub-Saharan African countries

Country	Year	Hiv (actual value)	HIV (predic-ted value)	Δ female earned income = + 5%	% Change*	Δ female earned income = + 10%	% Change*	Δ female earned income = + 20%	% Change*
Botswana	2003	32,9	29,06	28,21	2,95	27,37	5,84	25,73	11,46
Botswana	2005	33,5	35,62	34,99	1,77	34,36	3,52	33,13	6,99
Burkina Faso	2001	3,3	1,83	1,74	5,13	1,65	10,01	1,49	19,03
Burkina Faso	2003	2,3	2,56	2,42	5,28	2,29	10,29	2,06	19,54
Burkina Faso	2005	1,8	2,40	2,26	5,77	2,13	11,21	1,89	21,19
Burundi	2001	12,6	16,17	15,42	4,64	14,69	9,11	13,33	17,51
Burundi	2003	13,6	13,41	12,76	4,89	12,13	9,57	10,95	18,34
Burundi	2005	8,6	7,16	6,63	7,34	6,14	14,18	5,27	26,46
Cote D'ivoire	2001	10,0	9,39	9,25	1,52	9,11	3,01	8,84	5,94
Ethiopia	2001	15,0	14,88	14,49	2,60	14,11	5,15	13,38	10,07
Ethiopia	2003	11,7	16,26	15,84	2,62	15,42	5,18	14,62	10,13
Ethiopia	2005	11,5	10,55	10,08	4,42	9,63	8,67	8,79	16,67
Ghana	2001	2,7	3,86	3,62	6,14	3,40	11,92	2,99	22,47
Lesotho	2001		28,71	28,32	1,36	27,93	2,71	27,17	5,37
Lesotho	2003	27,8	32,97	32,53	1,33	32,10	2,64	31,24	5,25
Lesotho	2005	27,3	26,06	25,55	1,96	25,05	3,90	24,06	7,69
Malawi	2001	15,0	14,79	14,11	4,63	13,45	9,08	12,21	17,45
Malawi	2003	18,0	16,23	15,50	4,52	14,79	8,87	13,46	17,08
Malawi	2005		11,72	10,98	6,26	10,29	12,17	9,02	23,02
Rwanda	2001	9,8	9,67	9,25	4,32	8,85	8,48	8,09	16,31
Rwanda	2003	11,6	9,98	9,55	4,35	9,13	8,54	8,34	16,41
Rwanda	2005		6,55	6,14	6,24	5,76	12,12	5,05	22,85
South Africa	2001	23,1	22,46	22,04	1,84	21,64	3,65	20,84	7,20
South Africa	2003	24,0	23,84	23,40	1,83	22,97	3,63	22,13	7,17
Sudan	2001	34,4	34,62	34,36	0,75	34,10	1,50	33,58	3,00
Sudan	2003	39,0	35,44	35,17	0,77	34,89	1,55	34,35	3,08
Sudan	2005	37,3	35,60	35,44	0,45	35,28	0,91	34,96	1,81
Togo	2001	8,5	8,85	8,64	2,36	8,44	4,68	8,04	9,16
Togo	2003	10,0	9,10	8,88	2,37	8,67	4,68	8,26	9,17
Togo	2005	5,2	6,11	5,96	2,37	5,82	4,68	5,55	9,15
Uganda	2001	7,5	7,74	7,38	4,71	7,03	9,22	6,38	17,65
Uganda	2003	7,0	7,55	7,19	4,78	6,85	9,36	6,20	17,90
Uganda	2005		4,16	3,90	6,14	3,66	11,92	3,23	22,47
Zambia	2001		24,05	23,38	2,79	22,72	5,53	21,45	10,83
Zambia	2003	22,1	20,98	20,37	2,90	19,77	5,74	18,62	11,23
Zambia	2005	20,7	19,15	18,48	3,47	17,84	6,84	16,60	13,31
Zimbabwe	2001	29,8	29,81	28,97	2,81	28,15	5,57	26,55	10,94
Zimbabwe	2003		20,96	20,30	3,16	19,65	6,24	18,41	12,19
Zimbabwe	2005	18,6	18,18	17,53	3,59	16,89	7,08	15,68	13,76

All values are expressed in %.
The baseline is the predicted value (fourth column).
*The changes are negative, thus values reported here are absolute values.

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TABLE OF CONTENTS

Résumé

Bien que le VIH soit devenu une cause majeure de pauvreté et de décès sur le continent africain, la relation entre pauvreté et épidémie du VIH/Sida n'est pas aussi simple qu'elle ne le paraît. En effet, si à l'échelle mondiale les pays les plus touchés sont les pays pauvres, au niveau de l'Afrique Sub-saharienne, les pays les plus affectés sont les plus riches. Ces pays se trouvent également être ceux qui affichent les distributions de revenu les plus inégalitaires. Un autre fait saillant de la distribution de l'épidémie du VIH/Sida en Afrique Sub-saharienne est qu'elle est la région du monde avec la plus grande proportion des femmes infectées. Cette thèse s'intéresse à l'estimation de l'impact des inégalités de revenu et de genre dans la propagation de l'épidémie du VIH/Sida dans les pays d'Afrique au sud du Sahara. A l'aide d'un modèle de panel de données macroéconomiques de 42 pays d'Afrique Sub-saharienne sur la période 1997-2005, nous examinons le lien entre les inégalités de revenu et de genre d'une part et l'évolution de l'épidémie du VIH/Sida d'autre part en tenant compte des autres déterminants traditionnels de l'épidémie. Nos résultats montrent que l'inégalité de revenu favorise la diffusion de l'épidémie du VIH/Sida. Son impact est un impact spécifique qui ne transite pas par la pauvreté ; de plus la relation entre ces deux variables est significative et robuste à des spécifications alternatives du modèle et à une analyse dynamique. Quant à l'inégalité de genre, cette variable, appréhendée par trois indicateurs, joue un rôle important dans l'évolution de l'épidémie du VIH/Sida en Afrique Sub-saharienne. Plus précisément, il apparaît que c'est l'inégalité de genre en matière de revenu et de participation à l'activité économique qui favorise la propagation de l'épidémie au sein de la population jeune (15-24 ans), tandis que la variable de genre déterminante pour l'épidémie au sein de la population adulte dans son ensemble (15-49 ans) est l'inégalité de genre en matière d'éducation.

Abstract

Throughout African continent, HIV/AIDS epidemic has became a major cause of death and poverty. Nonetheless, the relation between poverty and HIV/AIDS epidemic is not as straightforward as it might first appear. Indeed, if at the international level the most affected regions are the poorest, in Sub-Saharan Africa however, the most affected countries also happen to be the richest. Meanwhile, these countries are also those with the least egalitarian income distributions in the world. Moreover, the distribution of the epidemic across both sexes differs according to regions, with Sub-Saharan Africa being the most *gender-affected* region: more than half of infected people there are women. Our focus in this dissertation is to assess the importance of income and gender inequalities as determinants of the spread of HIV/AIDS pandemic in Sub-Saharan Africa. Using a panel data of 42 African countries from the period 1997-2005, we examine the link between income and gender inequalities on the one hand and HIV/AIDS epidemic on the other hand by introducing these variables among the traditional determinants of the epidemic. Our results suggest that there is indeed a link between income inequality and HIV/AIDS epidemic. Moreover, the correlation remains even after we control for poverty and when we perform a dynamic analysis of the epidemic. Furthermore, women's education and economic independence appear to be critical determinants of the pandemic. Specifically, our results indicate that the component of gender inequality which drives the epidemic among young population (15-24) is gender inequality in income and participation to economic life, while it is gender inequality in education which fuels the epidemic among adult population (15-49).