

Université Rennes 1

Ecole Doctorale Vie-Agro-Santé

**Cyclic epidemics on the crops of the agro-ecosystems:
adaptation of fungi to varietal resistances**

**This manuscript is an English version, translated by L. Bousset for the purpose
of evaluation, from:**

**"Rapport pour l'obtention du diplôme d'habilitation à diriger des recherches :
Epidémies cycliques sur les cultures des agro-écosystèmes : adaptation des
champignons aux résistances variétales"**

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Rapporteur

Rapporteur

Examineur

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[...] poursuivre et prolonger la voie du maître en critiquant le maître [...]
[...] follow and extend the path of the master, while criticizing the master [...]
[C. Rovelli, Anaximandre de Milet ou la naissance de l'esprit scientifique, 2009]

La tête dans les théories... en conservant les mains dans le cambouis !
Head in theories... while getting hands dirty
[Devise d'épidémiologiste, 2014]

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Abstract

The control of fungal epidemics by the use of genetic resistances occupies a central place in my journey as a researcher. As an epidemiologist, I am studying the biology and population genetics of the fungus causing blackleg (*Leptosphaeria maculans*) in relation to the efficacy and stability of oilseed rape resistances in agro-ecosystems. As a backbone for my HDR manuscript, I propose to travel along this journey, showing why the use of the concept of “epidemics” was locking our conceptualization of epidemiologists, focussing the work on the scale of the field during the cropping season; and then how my work on natural populations (of snails) allowed me to enlarge my vision of agronomist. Such knowledge allowed me to work towards the joint consideration of epidemiology and population genetics in the study of the transmission of progenies across successive cropping seasons. In particular, I have produced knowledge on the production of inoculum at the end of the epidemics, and the dissemination across fields. Alongside the production of knowledge, my investment in theory helped me to propose a framework in which epidemiology – provided it is thought of as cyclic epidemics – can foster dialog between genetics and agronomy, between dynamics and evolution of populations. This opens the prospect of working in agro-ecosystems, on the coevolution between pathogen (the fungi) metapopulations, and host (the plants) metapopulations, under the influence of human actions.

During that journey, my implication in coordination of research was materialized by the coordination of two interdisciplinary projects, between biological disciplines (CTPS) and between biology and social sciences (ANR); and by the coordination of groups within my research unit and team. My scientific production leaned upon the supervision of students in epidemiology [st1, st2], at the interface between epidemiology and population genetics [doc1] and at the interface between epidemiology and agronomy [doc2]. It is because supervising contributes both to relay knowledge and to get confronted to the calling into question that I wish to continue this activity. This is the reason that leads me to defend this HDR, in order to continue supervising or co-supervising, which will enrich my research project.

Acknowledgements

Choosing means giving up. I will not choose whom to acknowledge. It would be too long to list all the ones whose example contributed to train me to this profession, to inspire me, to help me, to advise me and to guide my scientific journey. It would be too impolite to list the ones whose example allowed me to realise the traps I should avoid not to end up where I was seeing them. It would be too contrary to my nature to list acknowledgements of convenience, without sincerity. Thus there will be no list of names in this section: I hope I have testified my gratitude in proper time to the many ones deserving it.

Nevertheless, it does not mean that I owe nothing. All what I was able to produce results from the confluence of a professional journey, with possibilities for encounters and curiosity, with the successive contexts – favourable of adverse – in which I had the chance to walk. This manuscript relates some of the steps. I sometimes learned more from my lacks of success than from successes, but it is in the hope that the same lacks of success will not be reproduced, that I consider important to contribute to transmission.

This manuscript uses previous documents, publications or working papers. I sometimes fail to express ideas clearly in the first version of a text. I therefore revised them following advice and comments of readers, with hindsight of time, with acquisition of new pieces of knowledge, and the modification of my outlook on the topic.

For the purpose of evaluation, I have produced an English version of the French manuscript, as accurate as my limited skills allow me to. I take the responsibility for the potential translation mistakes, and encourage – whenever possible – the readers to refer to the French manuscript.

Part 1: Curriculum, productions and responsibilities

1. CURRICULUM

Lydia Bousset

born in 1972 in Lodève (Hérault)

Academic and professional curriculum

- Since 2002: **Researcher INRA** (CR2 then CR1) at UMR 1349 IGEPP Rennes, France in the Resistance and Adaptation team led by Régine Delourme
- 2001-2002: **Post-doctoral fellow in population genetics** at CEFE - CNRS in Montpellier, France supervised by P. Jarne
- 2000-2001: **Post-doctoral fellow in epidemiology** at the Justus Liebig University in Giessen, Germany supervised by J. Pons
- 1996-2000: **PhD in epidemiology** (INAPG AgroParisTech, France) at INRA Grignon supervised by C. de Vallavieille-Pope.
Engineer in agronomy from INA-PG
- 1995-1996: **Master in phytopathology** (BDAPC, University Paris XI Orsay, France). Training period at ETH Zürich (Switzerland) supervised by P. Blaise and C. Gessler
DAA Biology Applied to Agronomy (Pathologie végétale approfondie)
- 1994-1996: **Curriculum of engineer in agronomy** (INAPG AgroParisTech, France)
- 1990-1994: **Undergraduation, Licence and Master in plant biology** (University Paris XI Orsay, France)

Thematic journey

Researcher

Population biology and transmission of progenies in blackleg (*Leptosphaeria maculans*) related to the efficacy and the stability of resistances in oilseed rape

Post-doctorate 2

Local adaptation, mating systems and invasion genetics in the water snail *Physa acuta*.

Post-doctorate 1

Adaptive response of populations exposed to the selective pressure of chemical treatments, evaluation of the potential durability of a chemical inducer of resistance in barley powdery mildew (*Blumeria graminis* f.sp. *hordei*).

PhD

Adaptive response of populations exposed to the selective pressure of genetic resistances, consequences of sexual reproduction, consequences of seasonal changes in selection pressures (modelling with winter and spring varieties) in barley powdery mildew (*Blumeria graminis* f.sp. *hordei*).

Master

Resistance management strategy (variety mixtures) and consequences on the development of epidemics in apple scab (*Venturia inaequalis*). Epidemiological survey and simulations.

Skills

Theoretical fields

Epidemiology (pathotype dynamics, diversity, transmission of progenies among successive seasons). Population genetics (structuring, local adaptation, mating systems, isolation by distance, assignments), phylogeography. Adaptation processes (evolution of virulences and fungicide resistance, durability, consequences of sexual reproduction).

Laboratory techniques

Molecular markers (use of microsatellites, AFLP, RAPD), sequencing. Virulence and aggressiveness tests (gene for gene relationship, symptom quantification). Quantification of the inoculum production potential (fruiting bodies counting). Toxicological tests (fungicides and chemical inducer of resistance). Recurrent selection under controlled conditions. Crosses of powdery mildew isolates. Management of a collection of isolates.

Field experience

Prospection and sampling (fields and airborne spore trapping). Artificial inoculation of field plots. Quantification of the inoculum production potential (severity of epidemics issued from inoculation). Epidemic survey in the field and the orchard.

2. PUBLICATIONS

The names of the students that I have supervised are underlined. The numbers in brackets are used as references in the text.

1. Chronological list of the 21 A-rank publications, of which 16 as main author (as defined by Rennes1).

- [21] Delourme R, Bousset L, Ermel E, Duffé, Besnard AL, Marquer B, Fudal I, Linglin J, Chadœuf J, Brun H (2014). Quantitative resistance affects the speed of frequency increase but not the diversity of the virulent alleles overcoming a major resistance gene to *Leptosphaeria maculans* in oilseed rape. *Infection, Genetics and Evolution*, In press. doi: 10.1016/j.meegid.2013.12.019
- [20] Bousset L, Pointier JP, David P, Jarne P (2014). Neither variation loss, nor change in selfing rate is associated to the worldwide invasion of *Physa acuta* from its native North America. *Biological Invasions*, In press. doi: 10.1007/s10530-013-0626-5.
- [19] Bousset L, Chèvre AM (2013). Stable epidemic control in crops based on evolutionary principles: adjusting the metapopulation concept to agro-ecosystems. *Agriculture, Ecosystems and Environment* 165:118-129. doi: 10.1016/j.agee.2012.12.005
- [18] Bousset L, Chèvre AM (2012). Controlling cyclic epidemics on the crops of the agro-ecosystems: articulate all the dimensions in the formalisation, but look for a local solution. *Journal of Botany* Article ID 938218, 9p.doi:10.1155/2012/938218
- [17] Xhaard C, Barrès B, Andrieux A, Bousset L, Halkett F, Frey P (2012). Disentangling the genetic origins of a plant pathogen during disease spread using an original molecular epidemiology approach. *Molecular Ecology* 21:2383-98. doi: 10.1111/j.1365-294X.2012.05556.x
- [16] Travadon R, Sache I, Dutech C, Stachowiak A, Marquer B, Bousset L (2011). Absence of isolation by distance patterns at the regional scale in the fungal plant pathogen *Leptosphaeria maculans*. *Fungal Biology* 115:649-659. doi 10.1016/j.funbio.2011.03.009.
- [15] Lô-Pelzer E, Aubertot JN, Bousset L, Salam MU, Jeuffroy MH (2010). SIPPOM-WOSR : A simulator for integrated pathogen population management of phoma stem canker on winter oilseed rape. II. Sensitivity analysis. *Field Crops Research* 118:82-93. doi:10.1016/j.fcr.2010.04.006
- [14] Lô-Pelzer E, Bousset L, Jeuffroy MH, Salam MU, Pinochet X, Boillot M, Aubertot JN (2010). SIPPOM-WOSR : A simulator for integrated pathogen population management of phoma stem canker on winter oilseed rape. I. Description of the model. *Field Crops Research* 118:73-81. doi:10.1016/j.fcr.2010.04.007
- [13] Lô-Pelzer E, Aubertot, JN, Bousset L, Pinochet X, Jeuffroy MH (2009). Phoma stem canker (*Leptosphaeria maculans*/*L. biglobosa*) of oilseed rape (*Brassica napus*): is the G2 Disease Index a good indicator of the distribution of the observed canker severities? *European Journal of Plant Pathology* 125:515-522. doi 10.1007/s10658-009-9499-y
- [12] Travadon R, Marquer B, Ribule A, Sache I, Masson JP, Brun H, Delourme R, Bousset L (2009). Systemic growth of *Leptosphaeria maculans* from cotyledons to hypocotyls in oilseed rape: influence of number of infection sites, competitive growth and host polygenic resistance. *Plant Pathology* 58:461-469. doi 10.1111/j.1365-3059.2008.02014.x
- [11] Lô-Pelzer E, Aubertot JN, David O, Jeuffroy MH, Bousset L (2009). Relationship between severity of blackleg (*Leptosphaeria maculans*/*L. biglobosa* species complex) and subsequent primary inoculum production on oilseed rape stubble *Plant Pathology* 58: 61-70. doi 10.1111/j.1365-3059.2008.01931.x
- [10] Travadon R, Bousset L, Saint-Jean S, Brun H, Sache I (2007). Splash dispersal of *Leptosphaeria maculans* pycnidiospores and the spread of blackleg on oilseed rape. *Plant Pathology* 56:595-603 doi 10.1111/j.1365-3059.2007.01572.x
- [9] Aubertot JN, West JS, Bousset-Vaslin L, Salam MU, Barbetti MJ, Diggle AJ (2006). Improved resistance management for durable disease control: a case study of phoma stem canker of oilseed rape (*Brassica napus*). *European Journal of Plant Pathology* 114:91-106. doi 10.1007/s10658-005-3628-z
- [8] Henry P-Y, Bousset L, Sourrouille P Jarne P (2005) Partial selfing, ecological disturbance and reproductive assurance in an invasive freshwater snail. *Heredity* 95:428-436. doi 10.1038/sj.hdy.6800731

- [7] Bousset L, Henry P-Y, Sourrouille P, Jarne P (2004) Population biology of the invasive freshwater snail *Physa acuta* approached through genetic markers, ecological characterization and demography. *Molecular Ecology* 13:2023-2036. doi 10.1111/j.1365-294X.2004.02200.x
- [6] Bousset L, Pons-Kühnemann J (2003) Effects of acibenzolar-S-methyl and ethirimol on the composition of a laboratory population of barley powdery mildew. *Phytopathology* 93:305-315. doi 10.1094/PHYTO.2003.93.3.305
- [5] Bousset L, de Vallavieille-Pope C (2003). Effect of sexual recombination on pathotype frequencies in barley powdery mildew populations of artificially inoculated field plots. *European Journal of Plant Pathology* 109:13-24. doi 10.1023/A:1022034829401
- [4] Bousset L, de Vallavieille-Pope C (2003) Barley powdery mildew populations on volunteers and changes in pathotype frequencies during summer on artificially inoculated field plots. *European Journal of Plant Pathology* 109:25-33. doi 10.1023/A:1022073224259
- [3] Bousset L, Hovmøller MS, Caffier V, de Vallavieille-Pope C, Østergård H (2002) Observed and predicted changes over eight years in frequency of barley powdery mildew avirulent to spring barley in France and Denmark. *Plant Pathology* 51:33-44. doi 10.1046/j.0032-0862.2001.00654.x
- [2] Bousset L, Schaeffer B, de Vallavieille-Pope C (2001) Effect of early infection on pathotype frequencies in barley powdery mildew (*Blumeria graminis f. sp. hordei*) populations in field plots. *Plant Pathology* 50: 17-324. doi 10.1046/j.1365-3059.2001.00532.x
- [1] Délye C, Bousset L, Corio-Costet MF (1998). PCR cloning and detection of point mutations in the eburicol 14 α -déméthylase (CYP51) gene from *Erysiphe graminis f. sp. hordei*, a "recalcitrant" fungus. *Current Genetics* 34:399-403. doi 10.1007/s002940050413

Submitted manuscripts

- [ms1] Bousset L, S. Jumel S, Picault H, Domin C, Lebreton L, Ribulé A, Delourme R [submitted]. An easy and quick but accurate method to quantify disease severity: application to phoma stem canker leaf spots. Submitted to *Plant Pathology*, October 2013. In revision (February 2014).

2. Journals without review and papers destined to a wider audience.

- [v9] Bousset L (2012). Travail d'ébauche interdisciplinaire : peut-on parler simplement d'un processus complexe ? *Nature, Sciences, Sociétés* 20:448-454. doi: 10.1051/nss/2013058
- [v8] Bousset L, Brun H, Chèvre AM, Delourme R (2011). Quel lien entre l'épidémiologie, la création variétale et le potentiel de durabilité des résistances ? Illustration chez le phoma du colza. *Innovations Agronomiques* 15:29-43. www.inra.fr/ciag/content/download/4230/34111
- [v7] Evans N, Fitt BDL, van den Bosch F, Eckert M, Huang YJ, Pietravalle S, Karolewski Z, Rouxel T, Balesdent MH, Ross S, Gout L, Brun H, Andrivon D, Bousset L, Gladders P, Pinochet X, Penaud A, Jedryczka M, Kachlicki P, Stachowiak A, Olechnowicz J, Podlesna A, Happstadus I, Renard M (2004). SECURE - possibilities for durable resistance to stem canker? *IOBC/WPRS Bulletin* 27:17-21.
- [v6] Pinochet X, Mestries E, Penaud A, Delourme R, Chevre A-M, Renard M, Brun H, Bousset L, Balesdent MH, Rouxel T, Aubertot JN (2003). Towards a durable management of genetic resistances to *Leptosphaeria maculans*. *Oléagineux, Corps Gras, Lipides* 10:208-211
- [v5] Kellerhals M, Mouron P, Graf B, Bousset L, Gessler C (2003). Mischpflanzung von Apfelsorten: Einfluss auf Krankheiten, Schädlinge und Wirtschaftlichkeit. *Schweiz. Z. Obst-weinbau* 13:10-13
- [v4] Bousset L, Østergård H, Hovmøller MS, Caffier V, de Vallavieille-Pope C (2000). Effectiveness of powdery mildew resistance genes in autumn-sown and spring-sown barley crops. *Acta Phytopathologica et Entomologica Hungarica* 35:153-159.
- [v3] Bousset L, Schaeffer B, de Vallavieille-Pope C (2000). Evolution of barley powdery mildew pathotype frequencies in field populations between sowing and overwintering. *Acta Phytopathologica et Entomologica Hungarica* 35:383-386.
- [v2] Caffier V, Bousset L, de Vallavieille-Pope C (1999). L'oïdium de l'orge. Evolution des virulences de ce pathogène vis-à-vis des résistances variétales en France. *Phytoma* 521:54-59
- [v1] Bousset L, Blaise P, Kellerhals M Gessler C (1997). Mixtures of apple cultivars in orchards: effect of the scab epidemics. *IOBC/WPRS Bulletin* 20:42-48.

3. Communication in conferences. (listed in Annex 1)

4. Diploming theses

- [R1] Bousset L (1996). Influence de la stratégie de mélange variétal de pommiers sur une épidémie de tavelure (*Venturia inaequalis*). DEA, INA-PG, 25p.
- [R2] Bousset L (2000). Evolution saisonnière de populations d'oïdium de l'orge (*Blumeria graminis* f.sp. *hordei*) face aux résistances des variétés d'orges d'hiver et de printemps. Thèse, INA-PG, 122p.

5. Working papers

- [D1] Bousset L (2011). Point de vue sur l'articulation de concepts disciplinaires : application à la gestion de la maîtrise des épidémies sur les cultures dans les agro-écosystèmes, INRA, Document de travail pp. 76p. – ProdINRA B-1669.
- [D2] Bousset L (2012). Maitriser les épidémies dans les agro-écosystèmes : quelques concepts et liens pour la rencontre de disciplines scientifiques, INRA, Document de travail pp. 58p. – ProdINRA 173531.

3. SUPERVISION

Main supervisor of 2 Master students and co-supervisor of 2 PhD students

Masters

- [st1] Travadon Renaud (2005). Etude de la dispersion par la pluie des spores de *Leptosphaeria maculans* issues de multiplication asexuée ; conséquences potentielles sur la phase initiale du contournement des résistances du colza. Master 2, Agrocampus Rennes. Supervisors Bousset L, Sache I, Brun H.
Publication : [10]
Future: PhD IGEPP (2005-2008)
- [st2] Colonges Thomas (2009). Initiation et caractérisation d'épidémies induites par les ascospores et les conidies de *Leptosphaeria maculans* sur le colza Master 2, Agrocampus Rennes. Supervisor Bousset L.
Future: PhD SupAgro Montpellier

Co-supervision of PhDs

- [doc1] Travadon Renaud (2005-2008). Facteurs épidémiologiques contribuant à l'adaptation des populations de *Leptosphaeria maculans* aux résistances spécifiques de *Brassica napus* : dispersion des pycnidiospores et des ascospores et progression systémique du champignon. Doctorat Agrocampus Rennes. Co-supervisors Bousset L, Sache I, Brun H (resp. HDR). <http://hal.archives-ouvertes.fr/docs/00/33/34/77/PDF/Final2.pdf>
Publications: [10], [12], [16]
Future: Post-doc (2009-present) Department of Plant Pathology, University of California, Davis USA
- [doc2] Lô-Pelzer Elise (2005-2008). Modélisation des effets des systèmes de culture et de leur répartition spatiale sur le phoma du colza et l'adaptation des populations pathogènes responsables de la maladie (*Leptosphaeria maculans*) aux résistances variétales. Doctorat AgroParisTech. Co-supervisors Bousset L, Aubertot JN, Jeuffroy MH (resp. HDR). <http://pastel.paristech.org/3792/>
Publications : [11], [13], [14], [15]
Future: Post-doc (2008-2009) AgroParisTech Unité EcoInnov, then hired (2009) CR2 at INRA UMR of Agronomy, Grignon.

Participation to boards of examiners and PhD supervision comities:

- 2008 Renaud Travadon "Facteurs épidémiologiques contribuant à l'adaptation des populations de *Leptosphaeria maculans* aux résistances spécifiques de *Brassica napus* : dispersion des pycnidiospores et des ascospores et progression systémique du champignon" Participation to the board of examiners (Supervision).
- 2011 Amandine Lê Van "Potentiel évolutif du pouvoir pathogène de *Venturia inaequalis* en lien avec la domestication du pommier et l'utilisation de résistances quantitatives en amélioration variétale" Participation to the board of examiners (Examinator).
- 2011 Constance Xhaard "Influence des processus démographiques sur la structure et les caractéristiques génétiques des champignons phytopathogènes, cas de l'agent de la rouille du peuplier *Melampsora larici-populina*" Participation to the board of examiners (Examinator).
- 2009-2012 Laure Hossard "Co-construction et co-évaluation de scénarios d'organisation spatiale des systèmes de culture pour accroître la durabilité des résistances au phoma chez le colza" Participation to the supervision comity.

4. COORDINATION

Coordinator of two projects (ANR and CTPS), in charge of animation of a collective construction in the Unit, in charge of the animation of one of the three objectives of the research team.

Coordination of research projects

2005-2009 ANR ADD Project Cèdre "How to durably exploit plant resistances". I took care of the project building with Anne-Marie Chèvre, followed by the co-coordination of this interdisciplinary project between biological and social sciences. It included 16 research teams, plus 10 partnerships with professionals. Funded by the French National Agency for Research.

2005-2008 "Adaptation of phoma stem canker populations to oilseed rape resistances: characterisation of partial resistance, biology of populations during the loss of efficacy or resistance and inoculum production at the end of the cropping season". I took care of the project building and coordination of this project including plant pathology and genetics, agronomy and CETIOM technical institute (4 teams). Funded by CTPS.

2012-2015 Coordination of Workpackage 6: "Production of candidate scenario for the durable resistance deployment in wheat and oilseed rape" in the project ANR Agrobiosphère GESTER. "Management of crop resistance to diseases in agricultural landscapes as a response to new constraints on pesticide use". Interdisciplinary project between biology and social sciences (9 teams) coordinated by C. Lannou (BIOGER, INRA Grignon)/ F. Coléno (SADAPT, INRA Grignon).

Coordination within my research Unit and team

June 2011-June 2012. Animation of the "Integrated Crop Protection" collective construction in UMR IGEPP. Collective construction of the scientific issue.

2012-present. In charge of the animation of the objective "Development of cyclic epidemics" within the Resistance and Adaptation team

Participation in research projects:

2012-2015 INRA SMaCH Action PRESUME. Projet K-masstec : Knowledge-driven design of management strategies for stem canker specific resistance genes. Projet interdisciplinaire entre sciences biotechniques et sciences sociales (6 équipes) coordonné par MH Balesdent (BIOGER, INRA Grignon).

2008-2011 INRA SPE Projet "Polyétisme des maladies aériennes et mobilisation de l'inoculum". Projet d'épidémiologie (2 équipes) coordonné par I Sache (BIOGER, INRA Grignon).

2007-2010 Réseau d'excellence européen ENDURE "the European Network for the Durable Exploitation of Crop Protection Strategies". (NoE) 18 organisations dans 10 pays. (Coord. P Ricci, INRA Antibes).

2005-2008 : Projet innovant INRA SPE "Inférence de la taille effective et des capacités de dispersion des champignons pathogènes à partir des outils et méthodes récentes de génétique des populations". Projet de génétique des populations, coordonné par C. Dutech (INRA Bordeaux).

2002-2006 : Projet Européen SECURE "Stem canker of oilseed rape: molecular tools and mathematical modelling to deploy durable resistance"

2002-2004 : INRA Action transversale "Impact et gestion des innovations variétales", Projet "Gestion durable des résistances du colza oléagineux à deux maladies (phoma et hernie) coordonné par S Lemarié (INRA Grenoble) et M Renard (INRA Rennes)

January to December 2009 Participation à l'atelier de réflexion prospective ANR ADAGE "ADaptation au changement climatique de l'AGriculture et des Ecosystèmes anthropisés" coordonné par JF Soussana

Teaching

Teaching 2012 AgroCampus Ouest. Master Pro BioVigPa. Cours (3h) intitulé "Epidémies dans l'agro-écosystème : l'éternel retour ? »

Invited talk in the summer school 2012 "Co-construire et organiser une école-chercheurs INRA. Une démarche, des savoir-faire" Construire l'interdisciplinarité (2 sessions)

Reviews:

Crop Pasture and Soil Science (2009), Journal of Phytopathology (2011), Microbial Ecology (2011), Agriculture, Ecosystems and Environment (2012), Plant Pathology (2004; 2012), European Journal of Plant Pathology (2013)

Box 1: Agro-ecosystems, agronomy and breeding

Agriculture aims at production

An ecosystem is defined as "a dynamic complex of plant, animal, and microorganism communities and their non-living environment interacting as a functional unit" (Millennium Ecosystem Assessment, 2005). Thus in the broadest sense an "agro-ecosystem" includes all managed and unmanaged environments, domesticated and wild communities as well as human communities (Loucks, 1977). In agro-ecosystems, plant populations are organized as crops, established and managed towards production. This has two implications.

The first implication is the anchoring of the cultivated field into human society. To agriculture are anchored human alimentation, trade (food, textiles, and supplies), movement (powered by animals, then fuels). This differentiates agro-ecosystems from natural ecosystems. Natural ecosystems can reach equilibriums based on closed cycles, including the return of nutrients issued from the decomposition of plants on the place where they lived. On the contrary, agriculture also includes open cycles: products are exported from the field and not all of them are returned, depending on the fate of what man withdrew.

The second implication is the modification by men of the structure and dynamics of plant populations. Cultivation towards production implies a set of "management practices" – a set of interdependent actions at the scale of the field – that can be summarized by four aims. First aim is to establish a productive plant cover (by the modification of soil structure, seeding at controlled density, pruning, weeding). Second aim is to maintain this cover and provide it with nutrients required for growth and flowering (supply of water, fertilizers including nitrogen catching intercrops). Third aim is to harvest products and co-products (roots, leaves, stems, fruits or seeds). Fourth aim is to ensure the decay of plant parts not exported from the field.

Thus, an agro-ecosystem is not simply a place where plants grow, it is a place where men optimize the exploitation of resource production by plants, which he withdraws for its uses, and partly returns.

Why to protect crops?

It is because pathogens damage and/or divert to their benefit a part of plant resources, that the plant is affected in its growth and/or development. In the context of crops intended for production, it is when the disease has a negative impact on quality and/or quantity of products that one will aim at reducing this limiting factor. Thus, it is seldom one individual that is treated, but in most cases a population of plants. On this population, one aims at limiting the development of epidemics that depend on the interaction in time and in space between host plants, pathogens, environment and human actions (Agrios, 2005).

Continuities and discontinuities: man modifies plant populations by selection

Human choices exacerbate homogeneities and heterogeneities in host plant populations. Agro-ecosystems are characterized by the conjunction of discontinuities in space (fields) in time (seasons) and in genetic properties (compatibility or not, depending on the host-pathogen interaction; see Box 3). In natural ecosystems, only those of the plants that succeeded in producing seed (I use seed in a generic way; seeds, tubers, cuttings...) can participate in the next generation. There is thus adaptation of the plants to the environments in which they live, and selection for the capacity to evade destruction by other species coexisting with them (pests, parasites).

Since men cultivate plants, its choices are added to natural selection, to assort the next generation of plants. In the beginning, it was each farmer who, unconsciously first, then in purpose, has selected plants. If at the beginning it was just a part of the harvest that was kept as seed for the following year. As soon as man kept "the best kernels" (subjective and local) to sow them, he became involved in selection. The current diversity of cropped plants and their diversification among cropping regions bears the trace of this process, recurrent over numerous generations. It bears it in association with the footprint of the diversity of environments and human societies.

For the farmer, the aim of selection is to orient production towards the desired criteria. These criteria can for example concern quality (colour, shape, taste, and conservation capacity), yield, or criteria rendering the plants easier to harvest (for example the fact that seeds remain in kernels, and do not germinate during storage). Criteria are also these that contribute to the stability of the production, to avoid fasting years. Either selected on purpose or indirectly, plant resistance to disease contributed to these selection aims.

Along the way [historians could probably date the moment] the breeder switched from "choosing among the progenies" to "choosing the parents to combine, in order to enhance the production of progenies closer to the selection aim". Since the work of Mendel on the genetic basis of trait segregation in crosses, the choice of parents and progenies lean upon knowledge about genetics. At each selection cycle, there is: choice of the parents; crosses to maximize the diversity of traits associations or to enhance the combination of desired traits; choice of the progenies bearing the desired traits and thus used for the next generation. This relies on the existence of observable criteria to make these choices. In addition to genetics, breeding for plant resistance relies on the availability of knowledge in agronomy, plant pathology and epidemiology.

Source: Adapted from Bousset L, 2012. INRA, Working paper.

Part 2: Summary of research work

Cyclic epidemics on the crops of the agro-ecosystems: adaptation of fungi to varietal resistances

1. Introduction and context

Introduction: knowledge to "protect" crops in agro-ecosystems

As an epidemiologist, I am studying the biology and population genetics of the fungus causing blackleg (*Leptosphaeria maculans*) in relation to the efficacy and stability of oilseed rape resistances in agro-ecosystems. The word agro-ecosystem itself relates the context: "agro" reminds that man crops plants to satisfy its needs; "ecosystem" says that species are interacting (Box 1). In this context, it is because epidemics reduce yield and/or quality that man aims at controlling them, by "protecting the crops" against epidemics in an efficient¹ and stable² way. The control of fungal epidemics by plant genetic resistances occupies a central place in my journey as a researcher.

As a backbone for my HDR manuscript, I propose to travel along this journey, showing why the use of the concept of "epidemics" was locking our conceptualization of epidemiologists, focussing the work on the scale of the field during the cropping season; and then how my work on natural populations (of snails) allowed me to enlarge my vision of agronomist. Such knowledge allowed me to work towards the joint consideration of epidemiology and population genetics in the study of the transmission of progenies across successive cropping seasons. In particular, I have produced knowledge on the production of inoculum at the end of the epidemics, and the dissemination across fields. Alongside the production of knowledge, my investment in theory helped me to propose a framework in which epidemiology – provided it is thought of as cyclic epidemics – can foster dialogue between genetics and agronomy, between dynamics and evolution of populations. This opens the prospect of working in agro-ecosystems, on the coevolution between pathogen (the fungi) metapopulations³, and host (the plants) metapopulations, under the influence of human actions.

Context: adaptation⁴ of pathogen populations to varietal resistances

It is upon my production of epidemiological knowledge, principally on fungi (Box 2) that I came to considerations more general and more theoretical. Fungal biology allows genetical studies, even if population genetics studies are complicated by the alternation of sexual reproduction and asexual multiplication. One can also be faced to the alternation of biotrophic and saprophytic phases, or the alternation of survival phases and phases of amplification of populations, all the way to large population sizes during epidemic phases.

In the host plant as well as in the pathogen, there is variation among individuals within populations and this influences the compatibility of the interaction between the two partners. Compatibility is a property of the interaction between one plant individual and one pathogen individual (Box 3). In the case of a fungus, starting from a germinating spore, it grows outside the host, infects, grows at the expense of the host and then sporulates (i.e. produces and releases spores that the next generation is made of). These steps will follow each other, in a more or less efficient manner and more or less rapidly, depending on the compatibility of the interaction with the plant. For the plant, the less the interaction is compatible, the less the pathogen is able to divert plant resources and/or damage plant organs.

¹ Efficacy is the capacity to produce an effect at one point in time and space; it depends on pathogens' biology and population size

² Stability is the maintenance of this efficacy over time and over space; it depends on the adaptation dynamics in populations

³ As opposed to a population (unique pool), a metapopulation consists of disjoint demes, connected by flows of individuals. Extinction and recolonisation events may occur locally.

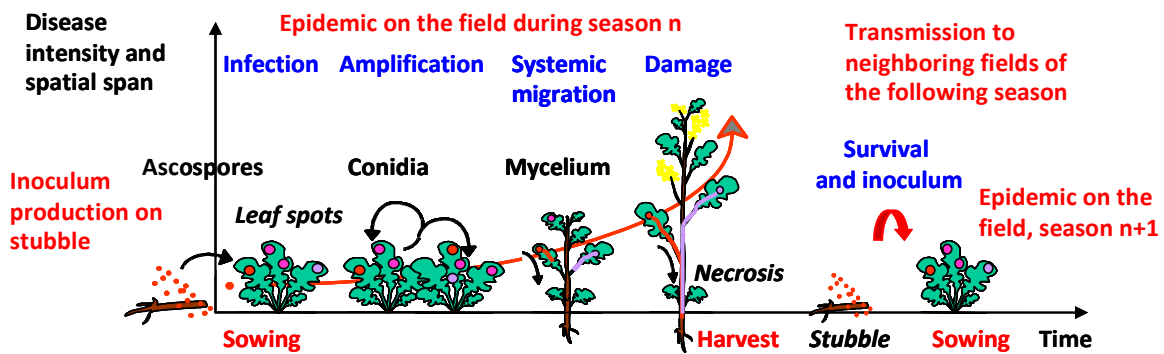
⁴ At the scale of a population, I will talk of adaptation of the population to its environment when the composition of the population changes across generations in a directional manner, depending on the environment.

Box 2: Fungi

An outdated taxonomy

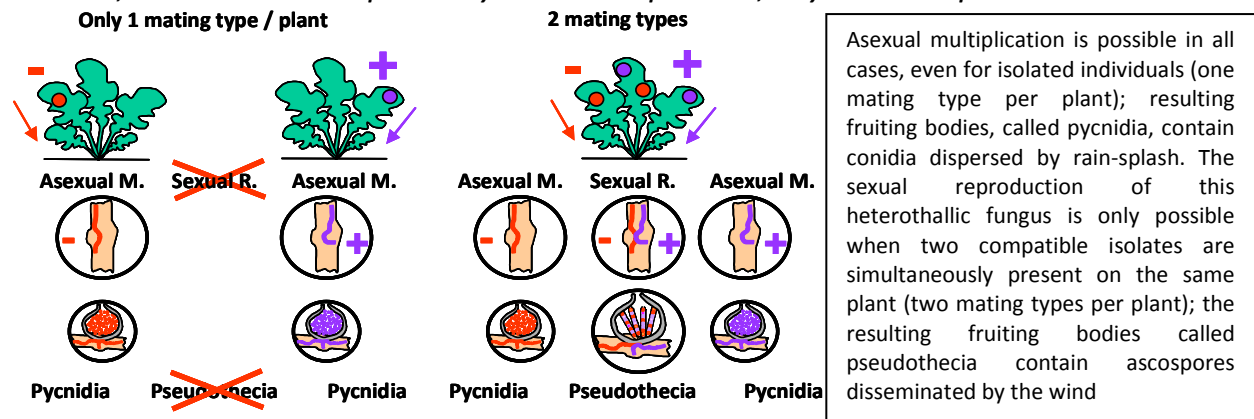
The word "fungus" became ambiguous because it designates an obsolete taxon. This term encompasses both *Fungi* (or *Mycota*), *Oomycota*, *Chytridiomycota* and *Mycetozoa*. Fungi were long considered vegetables, due to their immobility and to the presence of a thickened cell wall. They were designed « cryptogams » because of the absence of flowers. But fungi constitute a proper kingdom, because they differ from plants and algae by several characteristics. Fungi are uni or pluricellular Eukaryotes (bodies that have cells, and which chromosomes are within a nucleus). Their cells, with a chitinous or cellulose cell wall, are immobile and feed by absorption of organic molecules directly in the surrounding medium. The cell(s) have neither chlorophyll nor plasts, and these bodies are heterotrophic towards carbon. Without roots, stems or leaves, their vegetative part is called mycelium, consisting of a mass of branching, thread-like hyphae. (Source : Wikipédia, article "champignons", décembre 2013)

Lifecycle of *Leptosphaeria maculans*, ascomycete fungus causing blackleg on oilseed rape

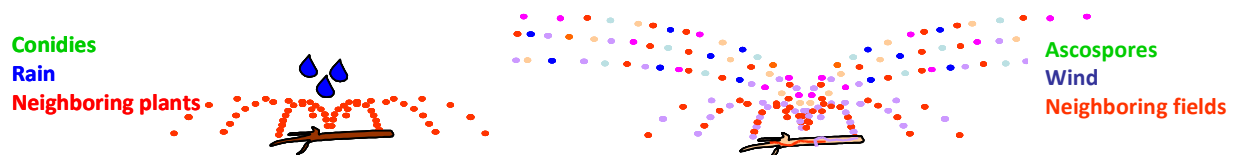


Blackleg is caused by a fungal species complex including *Leptosphaeria maculans* (Mendes-Pereira et al., 2003). Epidemics are initiated in autumn on leaves. On the resulting leaf spots, the fungus can produce conidia (also called pycnidiospores) dispersed at short distance by rain splashing [10]. From autumn to the following spring, the fungal mycelium reaches veins and progresses in a systemic way towards the plant crown, where it decays tissues causing a necrosis (Hammond et Lewis, 1986). This necrosis hampers water nutrition of the plant – possibly causing plant breakdown – occasioning yield losses. The fungus survives over summer – and the following years until complete stubble decay – as mycelium on stubble left on the field after harvest. Survival on seeds has been demonstrated, survival on other cruciferous species has been postulated, but their effective contribution to epidemics has not been proved in Europe (West et al. 2001). It is from the mycelium on stubble that the fungus produces fruiting bodies in autumn.

On stubble, *L. maculans* inoculum is produced by heterothallic reproduction, or by asexual multiplication



Leptosphaeria maculans airborne spores are disseminated either by wind, or by rain

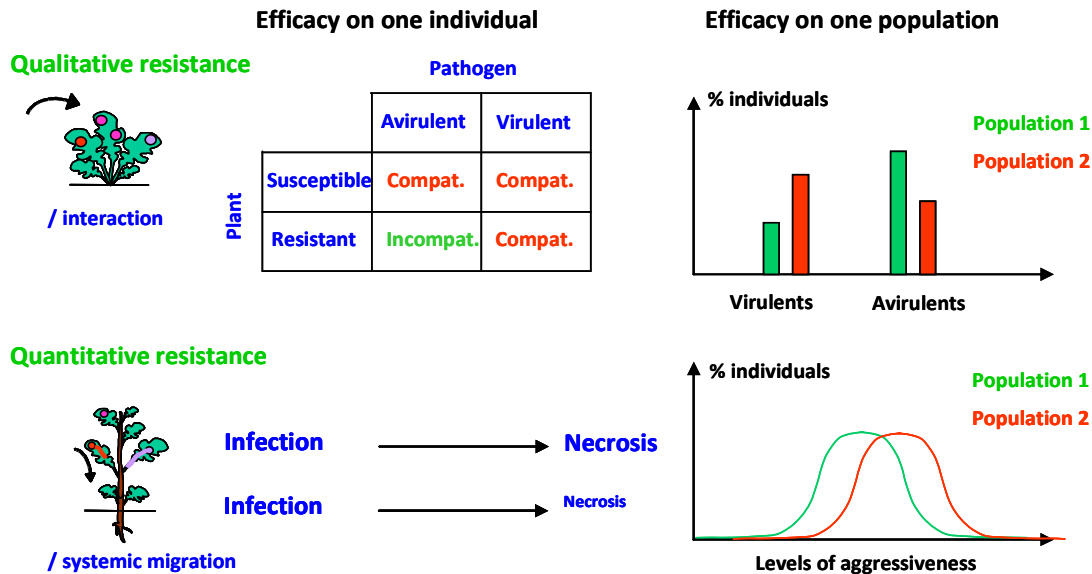


Source: Adapted from Bousset et al. 2011 *Innov. Agronomiques*.

Box 3: How do plant resistances act?

Qualitative and quantitative resistance act in a different manner on the compatibility of individuals and populations

In the plant, we call resistance the factors that reduce the compatibility of the interaction with the pathogen. For qualitative resistance, the confrontation with an isolate, called "avirulent", discriminate plant individuals into only two categories : these termed "susceptible", for which the interaction is fully compatible ; these termed "resistant", for which the interaction is fully incompatible. The compatibility of the interaction changes in an all-or-nothing manner, depending on the plant host individual – pathogen individual couples. For quantitative resistance, the confrontation with whatever pathogen individual discriminate plants into numerous categories, or quantitative resistance levels. Thus, the compatibility of the interaction changes in a gradual manner, depending on the plant host individual – pathogen individual couples.



Efficacy and stability: two different notions

The control of epidemics includes two aspects: efficacy and stability of the strategies. Their efficacy – i.e. the capacity to produce an effect at one point in time and in space, depends on the biology of the pathogens, and their population sizes. Their stability – i.e. the maintenance of efficacy over time and space – depends on the adaptation dynamics in pathogen populations.

Durability potential

In the last decades, joint efforts were aiming at an increase in efficacy, but they failed to achieve stability. In agro-ecosystems, deploying a small number of resistant host varieties induces the invasion of the pathogen populations by compatible individuals, thus causing their loss of efficacy. The loss of efficacy (also called "overcoming") of resistance genes in varieties deployed at wide scale, is due to the invasion of the regional population by individual virulent on the corresponding resistance gene (for qualitative resistances) or more aggressive (for quantitative resistances). The pathogen continuously adapts to the mosaic of host crops deployed at the scale of the landscape. This implies (i) the apparition of individuals virulent or more aggressive, (ii) the production of progenies by these individuals, (iii) the contribution of these progenies to the inoculum for the following cropping seasons. The durable management strategies aim at slowing down the adaptation of the regional pathogen population, by acting on these three mechanisms. They aim either at reducing disease severity on the field, and/or the inoculum production, and/or the transmission of progenies from a cropping season to the following one. This can be achieved both by breeding for resistant varieties [v8] and by the spatial deployment of varieties and cropping practices ([14], Hossard et al., 2010).

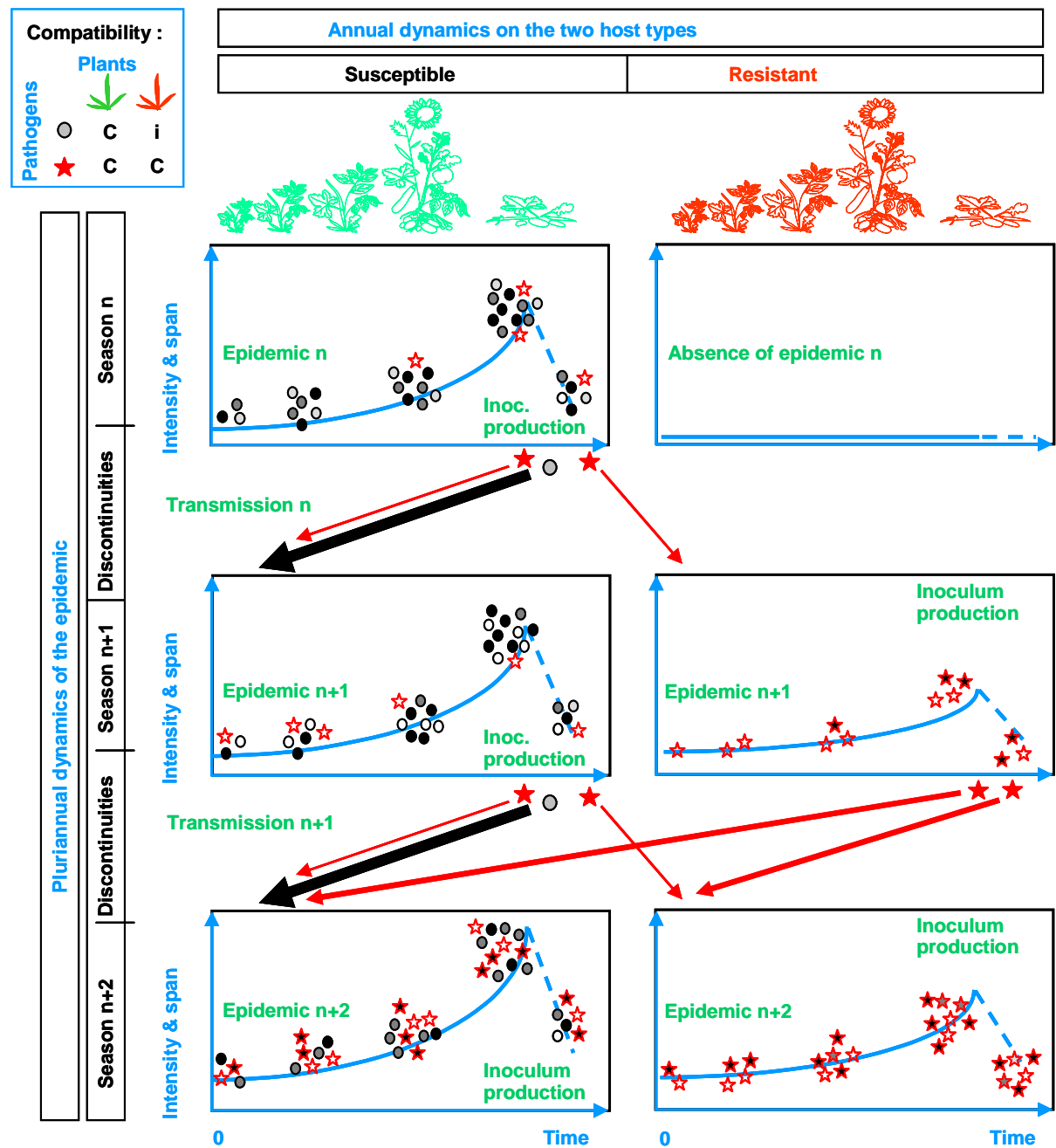
Clarifying the terminology and completing the previous definitions, Johnson (1984) wrote, insisting on the descriptive – and not causal – nature of the term, "Durable resistance to a disease is resistance that remains effective during its prolonged and widespread use in an environment favourable to disease". At the very heart of this definition is engraved the fact that durability results from the interaction in time and in space between the host plant, the pathogen, the environment and humans. Resistances are not "durable" by themselves, by relatively to a context. While appealing for practical reasons, and despite repeated attempts, the delimitation and the evaluation of an intrinsic property giving "durability" to the resistance, failed because it is impossible to simultaneously remove all the interactions.

As an alternative to the durable resistance concept, that became confuse, it appeared important to us for breeding, to reintroduce in the language the existence of these interactions. When talking *a priori*, before the resistance is deployed, we propose not to talk of "durable resistance" (referring to a unique number) but of "potential for durability" (i.e. the Esperance of the phenomenon, which realisation is conditional to a given agro-ecosystem [v8]. It is indeed possible to improve the potential for durability of a variety, but the achieved durability will also depend on the characteristics (the pathogen, the environment, man, time and space) of the agro-ecosystem in which it will be deployed.

Source : Adapted from Bousset L, 2012. INRA, Working paper, and Bousset et al. 2011 Innov. Agronomiques

Figure 1: Pluriannual population dynamics on sensitive and resistant hosts

The adaptation process can be formalised when two types of hosts are present. The simplest case is the discrete case of qualitative resistance, which establishes a clear distinction between "susceptible" and "resistant" hosts. When a qualitative resistance is efficient, the proportion of compatible individuals is low in the inoculum disseminated onto the field. This generates a small size of initial population, if any. In the first cropping season, this allows for a limitation of the epidemics, and induced damages on the resistant variety. Virulent individuals appear by mutation in the pathogen populations where they occur, that is on the susceptible variety. The simultaneous deployment of susceptible and resistant varieties in the agroecosystem confers an automatic advantage to the virulent isolates, being the only ones compatible on all the acreages, while avirulent isolates are stuck on the susceptible varieties. Noteworthy, if virulent individuals incur a "fitness cost" on their multiplication or their survival, it will only be expressed in competition on the susceptible variety, because on the resistant one there is no coexistence with avirulent individuals. As soon as the resistant variety will contribute progenies to the inoculum of the following season, the frequency of virulent isolates will increase in the regional population. As a consequence, this change in composition of the population will lead to the loss of efficacy of the resistance towards the modified pathogen population. It should be noted that the resistance is still fully efficient against avirulent isolates, but inefficient against virulent ones. (Adapted from [19]).



Sources: Adapted from Bousset L, 2012. INRA, Working paper, and Bousset and Chèvre 2013 AEE

Thus, the plants less affected by pathogens have more resources to reproduce and bear seeds for the next generation. In the plant, genetic factors reducing the interaction compatibility are named resistance.

Two kinds of resistances are opposed (Box 3). Qualitative resistance discriminate pathogen individuals into the two categories "avirulent" and "virulent", and plant individuals into the two categories "susceptible" and "resistant". The interaction compatibility changes in an all-or-nothing way, given the plant individual – pathogen individual couples. On the contrary, for quantitative resistance the confrontation with whatever pathogen individual discriminate the plant individuals into numerous categories or levels of "quantitative resistance", and fungal individuals into "levels of aggressiveness". Thus, the interaction compatibility changes in a gradual manner, given the plant individual – pathogen individual couples. All pathogen individuals are able to infect the quantitatively resistant plant, but their lifecycle is limited as compared to that on a susceptible plant.

At the scale of populations, the population resistance level depends on the number of compatible interactions. This number is linked to the composition (pathotype⁵ frequencies) and the size (number of individuals) of pathogen populations. Deploying susceptible and resistant varieties in the agro-ecosystems gives an automatic selective advantage to the virulent isolates (Figure 1). Indeed, virulent individuals are the only ones compatible on all acreages, whereas avirulent isolates are restrained to the susceptible varieties. As soon as the resistant variety contributes inoculum to the following season, virulent individual frequency will increase. As a consequence, this change in composition will lead to the loss of efficacy of the corresponding resistance gene, towards the modified population. In current agro-ecosystems, where crop protection is thought of only as a tactical action concerning one field during one cropping season, the pathogen metapopulation continuously adapts to its hosts, while the other way around does not occur. It was necessary to develop a theoretical framework to envision the crop protection strategies as a means to maintain the pathogen metapopulation locally non-adapted to host crops.

To tackle this topic, I had to combine knowledge and researches at the interface between epidemiology, plant pathology, population genetics, and agronomy. The aim of my researches is to formalize, link and quantify the processes involved in the adaptive response of a fungal metapopulation on a network of fields in which selection pressures are not homogeneous. For this, I am interested in dissemination, survival, transmission, and in the integration of these elements in models allowing for the simulation of strategies (combination of crop protection tactics) at the scale of a network of fields. I combine conceptualization, participation to modelling, experimentation in field plots or survey of farmers' fields, and studies of processes under controlled conditions.

2. Doctorate and post-doctorate formation: epidemiology on crops; metapopulations in natural ecosystems

On the crops, the concept of "epidemic" reaches its limits when faced to the discontinuities of time and space

In the nineties, the dynamics of compatible (virulent) and incompatible (avirulent) individuals on resistant plants was thought, analysed and modelled mainly in terms of differential fitness during competition (Johnson, 1984; Pedersen and Leath 1988; Wolfe 1985; 1993; Leonard 1993; Garrett and Mundt 1999). The frame of this competition was an homogeneous pool (population) in which individuals were carried-over from one generation to the next without constraints. With such hypotheses, simulating situations with equilibria, in which the deployment of resistant varieties was not followed by the increase in frequency of

⁵ Following the confrontation of a fungal isolate with a differential set of varieties or lines, each of them expressing known resistance genes, the resulting combination of virulence / avirulence interactions for the genes included in the differential set is called pathotype

Box 4: The notion of adaptation, declined across different scales

"Adaptation" means two notions: that of change, and that of adequacy. These notions are declined in a different manner at the scale of an individual, a population, or a set of populations.

At the scale of a living individual, we talk of adaptation when the expression of its characters is modified in response to the characteristics of the environment. This is possible the metabolism (the set of chemical reactions within it) of the individual can vary depending on the environment in which it lives. For a large part, the expression of the characters of an individual is determined by the information (genetic and epigenetic) that it inherited from its parents, and by the partial stabilisation of this genetic information. However, there remains some variation in the expression of characters. We talk of environmental "plasticity" to describe the range of the variation (reversible and not inheritable) possible for the phenotype issued from the expression of a given genetic information (stable and inheritable). To make it short, the key point is that we consider that this variation cannot be transmitted to the progenies of the individual, even if reality can be more complex (modification of the epigenetic imprinting depending on the environment).

At the scale of a population, we talk of adaptation of the population to the environment in which it lives when its composition changes in a directional manner, depending on the environment, across generations. I here define a population as a set of individuals of the same species, coexisting in the same place and reproducing themselves together across generations. Directional refers to a change that is not only the result of random fluctuations of its composition: across generations, an increase in population's performance (defined as the product of individual frequencies by their respective performances) in the given environment. This implies three conditions. First, changes in the populations are only possible if there exist variation between individuals (they are not all identical). Second, directional change is only possible if this variation is inherited (at least partly) across generations. Third, response to the environment is only possible if the interaction between the characteristics of the individuals and these of the environment have an impact on the composition and/or the hereditary transmission (some of the individuals contribute more than others to the composition of the future population). It should be noted that the population can also contribute to change the environments in which it develops (using some resources and producing others).

At the scale of a set of populations, we talk of adaptation of the set of populations to the set of environments when its composition changes in a directional manner, depending on the environment, across generations. Following from the definition of "population", one cannot extend spatial scales indefinitely. Indeed, a population is based on the uniqueness of place (coexistence) of reproduction (the reproduction happens "together") and of environment (exerting the same influence). Across wide scales, especially is the environment is not suitable everywhere (it is then called "fragmented") it is no longer true that each individual has a equivalent probability to reproduce with the others in all points of space. If the environment is differentiated depending on places, it is no longer true that it exerts the same influence at all points. However, it is not necessarily true either that the populations in different environments are independent from each other: if some individuals are dispersed among them, this may influence their respective futures. To think on these "sets of interacting populations", the formalism of metapopulation has been proposed. This formalism also allows taking into account the abrupt changes that climate seasonality imposes on scales of time. In the formalism of metapopulations (Gilpin and Hanski 1991; Hanski 1999), the set of individuals is fragmented into sub-sets called "demes". Due to the finite size of demes, each of them does not necessarily contains all the existing types of individuals. The interaction occurs at the local scale, between one deme of the considered species and the environment in which it lives, but the discrete demes (as opposed to the notion of continuity) exposed to the local selection are interconnected by the migration of individuals. The process of adaptation occurs across several seasons, over which the average level of compatibility on the whole set of environments results from the local interactions – between one local deme of the pathogen species and its local environment made by the host plant – and migration flows between demes. The survival of the types of individuals does not need to be local, locally there can exist events of extinction and recolonisation. It is important to keep in mind that the directional change in composition can occur only if there is homogeneity and/or recurrence of selection pressures.

Talking of "adaptation of pathogens to plant resistances thus includes processes at different scales: the individual, the population, the set of populations.

Source: Adapted from Bousset L, 2012. INRA, Working paper

virulent isolates, implied the addition of a "fitness cost" supported on susceptible varieties by the individuals-compatible-on-resistant-varieties (Lannou and Mundt 1997 ; Shaw and Østergård 1998 ; Vera Cruz et al. 2000 ; Leach et al. 2001). This was focussing the work on experimental demonstration of such a fitness cost, and its impact on pathotype frequency changes during the epidemics within the cropping season.

My studies on pathotype dynamics in barley powdery mildew allowed me to confirm that when individuals coexist on the same population, the application of a selection pressure (i.e. of a fungicide or a chemical inducer of resistance in a recurrent selection experiment) can lead to a change in pathotype frequencies across generations [6]. However, in the case of adaptation to qualitative resistances, the resistances act as a filter during infection [R2]. Incompatible individuals are excluded, thus there is no longer coexistence once the populations are established on the resistant fields. The hypothesis of differential selection during competition in an homogeneous pool is not sufficient to represent the pathotype dynamics in response to deployment of resistant varieties; it is necessary to take into account the flow between resistant and susceptible fields, thus also to differentiate the dynamics on each of them. These dynamics cannot be limited to the epidemic phase on the field during the cropping season.

My studies on pathotype dynamics in barley powdery mildew also allowed me to question the relevance of focussing knowledge production on the amplification phase within the field during the annual epidemic – indeed the most accessible experimentally. To conceptualize the dynamics of pathogen population composition change on cultivated plants, the term epidemic was used. Starting within this concept, I had the opportunity to show that pathotype frequencies depend on the time when individuals arrive [2] then remain stable throughout the cropping season [2, 5]. Changes are mainly due to sexual recombination at the end of the epidemic [5] and to the variability during the transition to volunteers [4] and subsequent contribution of populations to the regional aerial inoculum pool [3]. These findings allowed me to realize that the concept of epidemic, referring to an increase in the intensity and/or spatial span of the disease (Zadoks and Schein 1979) is adapted to continuous time and space. It is not sufficient to represent the dynamics on a network of fields on which selection pressures are not homogeneous, in presence of discontinuities. It became necessary to have a formalism to link the epidemics of one cropping season to that of the following cropping season, knowing the existence of discontinuities in time (seasonality, sexual reproduction, survival) and in space (changes in the fields in which the given plant species is cultivated).

In natural ecosystem: population genetics in fragmented environment

Working as a post-doctoral fellow on natural populations of the freshwater snail *Physa acuta* allowed me to get complementary skills in population genetics. *P. acuta* is hermaphrodite and lives in an environment consisting of ponds (closed) and rivers (open), more or less connected by flows, and more or less affected by extinction and recolonisation events during the seasonal droughts and floods. This allowed me to analyse the interaction between local adaptation, migration and mating systems [7, 8]. Furthermore, *P. acuta* is a species that recently extended its distribution area. This allowed us, using a phylogeography approach, to study invasion genetics, and more specifically the possibility to detect the trace of past events in the genetic structure of current populations [20].

Figure 2: Inoculum production potential for *L. maculans*

2a. Direct quantification, by counting

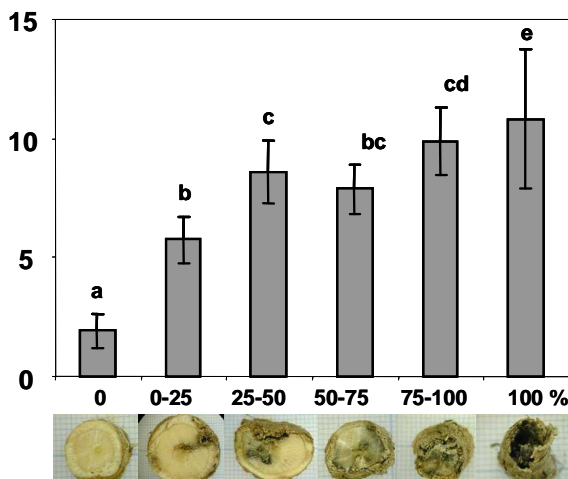
In *L. maculans*, the production of inoculum for the season n+1 occurs on stubble from season n. The positive relationship between the severity of necrosis and the production of inoculum has been quantified by the counting of pseudothecia obtained following incubation of stubble pieces under natural conditions [11].

Pseudothecia



Ascospore

Number of pseudothecia counted / cm²



Number of *L. maculans* pseudothecia per cm² of winter oilseed rape stubble, depending on the severity of necrosis at the time of harvest (proportion of the area with necrosis on a transversal section). Error bars represent the 95% confidence interval. Histograms with different letters are significantly different.

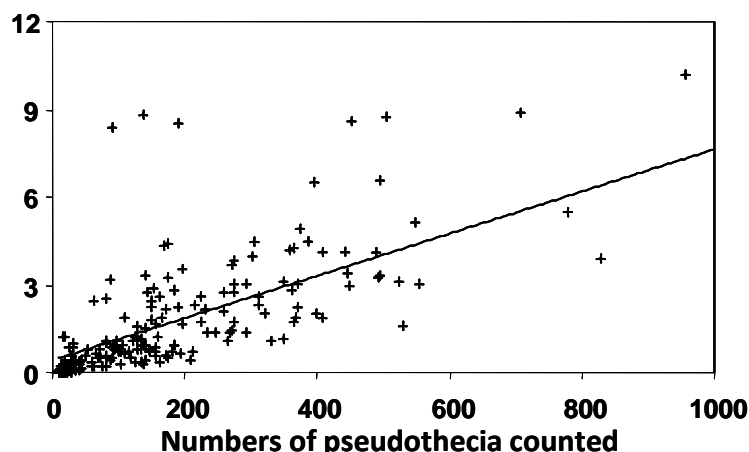
Necrosis severity classes

These results show that previous year's disease severity can be used for the prediction of the available inoculum for the initiation of the following epidemic.

2b. Approached quantification, by measurement of areas

The time required for the exhaustive fruiting bodies counting under magnifying lenses can hamper the evaluation of varieties on this criterion. However, it has been shown that an estimator can be obtained by quantifying the area occupied by the fruiting bodies, which is technically easier and less time consuming [11]. This opens the prospect of varietal characterisation for their potential for inoculum production.

Surface occupied by pseudothecia (cm²)



Relationship between numbers of pseudothecia counted and the evaluation of the area occupied by pseudothecia (correlation coefficient $R^2=71\%$). Quantification obtained from 180 stubble pieces, representing the natural range of disease severity.

Sources: Adapted from Lô-Pelzer et al. 2009 Plant Pathology and Bousset et al. 2011 Innov. Agronomiques

Working on such an organism, which dynamics at large scales can be thought in terms of metapopulations more than in terms of homogeneous pool, allowed me to acquire the skills I was lacking to link the topic of adaptation to plant resistances to the topic of coevolution in metapopulations (Gilpin and Hanski 1991; Thompson and Burdon 1992; Burdon 1993; Hanski 1999). Noteworthy, this formalisation was already largely used to think, analyse and model adaptation to host plants in natural ecosystems for pathogens (Burdon et al. 1995; Thrall and Burdon 1997) or pests (Gandon et al. 1996), simply little used for cultivated plants (Damgaard 1999). Furthermore, the notion of adaptation refers to different meanings depending on the scales considered, from individuals to groups of populations (Box 4).

3. Research program: epidemiology in metapopulations in agro-ecosystems

This former knowledge shaped the orientation of my research program. My aim was to formalise, link and quantify the processes involved in the adaptive response of a fungal metapopulation on a network of fields on which the selection pressures were not homogeneous. I especially wanted to work on the phases when population genetics influences their dynamics. This is the case in particular for the production of progenies and their transmission from one cropping season to the next. I faced two main locks. The first lock was the lack of epidemiological knowledge for the lifecycle phases other than the amplification on the field during the cropping season. I therefore focussed my work and that of the students I have supervised, on the production of inoculum at the end of the epidemic, and on dissemination among fields. The second lock was the lack of links between scattered pieces of knowledge. What first appeared as a simple question of combination of units, finally turned out into the need for an investment in theory, in particular to reciprocally adjust the concepts, rendering possible to transpose the theories existing in natural ecosystems, to the specificities of agro-ecosystems.

Transmission of progenies among successive cropping seasons: production of knowledge in epidemiology

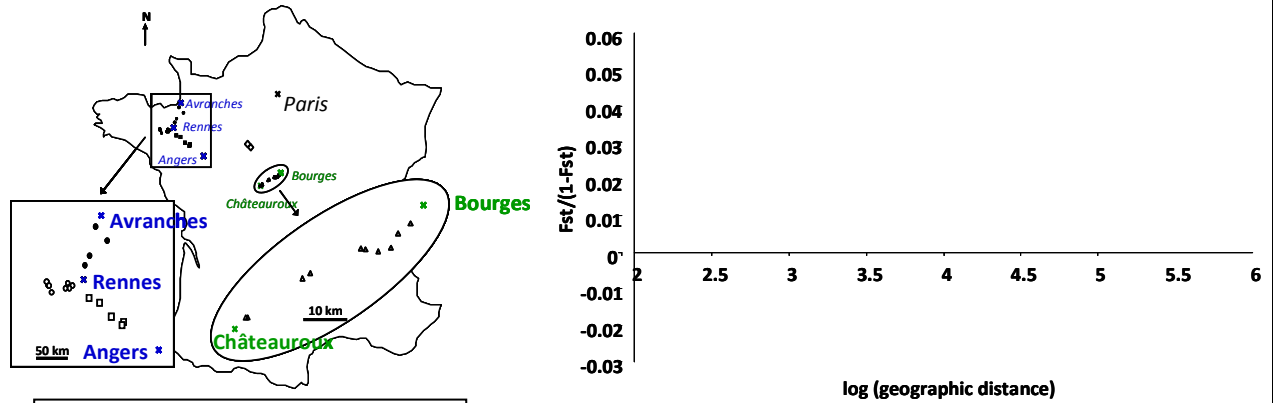
Concerning inoculum production in *L. maculans*, it was known that the fungus survives and then set fruits on oilseed rape stubble. From stubble, the fungus is disseminated, and contaminates new plants (Hall 1992; Box 2). Spores infect leaves in autumn, causing leaf spots. During winter and spring, from the leaf spots, the fungal mycelium reaches the leaf veins, grown in a systemic way down the petiole and the stem, towards the plant crown. At the crown, it produces a symptom of necrosis. The role of ascospores, produced by heterothallic⁶ sexual reproduction at the end of the epidemic, was known, but the intensity of their productions had not been quantified. For the first time in a plant pathogenic fungus, L^ô-Pelzer [doc2] has established the relationship between on the one hand disease severity at the end of the cropping season, and in the other hand the inoculum production potential for the following season, measured by the number of fructifications [11; Figure 2a]. This result opened the prospect of pluri-annual modelling of epidemics, and of characterizing varieties for this criterion (Figure 2b).

Concerning the impact of oilseed rape resistances on inoculum production, it was known that necrosis size was reduced on quantitatively resistant varieties (Pilet et al. 1998; Delourme et al. 2006). The consequence on inoculum production was not known. Renaud Travadon [st1; doc1] showed that systemic phase success depends on the number of infection points and on the quantitative resistance level of the variety [12]. This opened the prospect of studying the constraints on encounter of mates for sexual reproduction.

⁶ Sexual reproduction possible only between two individuals of different mating types; there are two mating types in *L. maculans*

Figure 3: Dissemination of spores and consequences on population structure

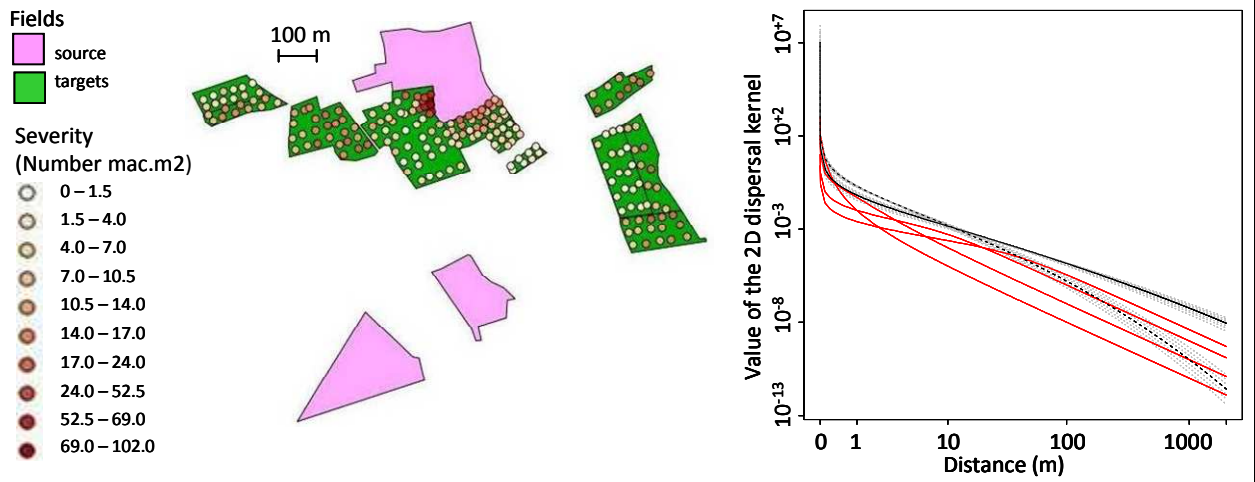
Indirect approach: isolation by distance



3a. Localisation of the 29 populations sampled (o) in regions Bretagne and Centre.

3b. Genetic differentiation among *Leptosphaeria maculans* populations. Multilocus estimates (θ) of genetic differentiation F_{ST} expressed as $F_{ST} / (1 - F_{ST})$ are plotted against logarithm of geographic distance (km) for each pair of field populations (maximum distance between two populations is 364.2 km; the regression is $y = 1.13 \cdot 10^{-4} x + 0.0015$; $r^2 = 1.7 \times 10^{-4}$; $P = 0.35$), F_{ST} was estimated according to Weir and Cockerham (1984).

Direct approach: bayesian estimation of the dispersal kernel



3c. Map of the observed blackleg severities in autumn 2012 on farmers' fields near le Rheu (anonymised representation).

3d. Posterior median of the value of the 2D dispersal kernel at increasing distances for the 2009-2010 dataset (solid black line) and the 2011-2012 dataset (dashed black line). Red lines are examples of kernels used in the simulator of L -Pelzer et al. (2010) with wind speed equal to 0.01, 0.10, 1.00 and 4.00 meters per second.

Sources: Adapted from Travadon et al. 2011 Fungal Biology

Since we showed that stubble bearing only pycnidia are sufficient to initiate an epidemic [st1; st2; 10], it is thus necessary to take into account not only survival via sexual reproduction, but also survival without access to sexual reproduction.

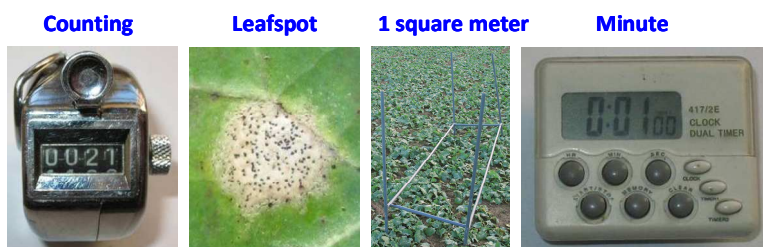
Concerning inoculum transmission, it was known that ascospores could be disseminated by wind over large distances, up to 8km in Australia (Bokor et al 1975; Petrie 1978; Gladders and Musa 1980). However, these data were not sufficient for the modelling of the transmission from many sources in a heterogeneous landscape. Due to the lack of precise data on the dispersal gradient (Marcroft et al. 2004), it was postulated possible to use the relationship parameterized on lupine anthracnose, extrapolated from data collected at short distance (Diggle et al. 2002). To obtain data on dispersal, we used two successive approaches.

The first approach was to use the current population structure for the inference of flow intensity, by an indirect population genetics approach. If local dispersal predominates, it is expected that the neutral differentiation pattern follow an "isolation by distance" or IBD model (Rousset, 1997, Slatkin, 1987, Wright, 1943). Assuming (i) that neutral genetic structure is mainly due to drift; (ii) that it is counterbalanced by the dispersal of alleles (in a two dimensional environment); (iii) that it reflects the dispersal capacities of the studied species, a linear correlation is expected between the logarithm of geographical distance, and the ratio $F_{ST} / (1 - F_{ST})$ estimated between population pairs (F_{ST} being an index of genetic differentiation between populations; Wright, 1943). The detection of an IBD pattern requires a precise framework, under the mutation-drift equilibrium, considering geographical distances in the range between σ and 20σ , where σ^2 is the second moment of the dispersal distance between parents and offspring. The analysis performed by R. Travadon [doc1] using molecular markers has not allowed detecting an IBD pattern at regional scale [16; Figure 3ab], and thus it is not possible to deduce information about dispersal distances.

The second approach that I develop actually in collaboration with Samuel Soubeyrand (BioSP, INRA Avignon) for spatial statistical analyses is a direct approach of dispersal gradient characterisation in farmer's fields [in progress; n19]. The modelling of long distance dispersal relies on the extrapolation of data acquired over short distances, from punctual sources. To consolidate these data, our aim was to collect data in commercial situation, and to develop methods for their analysis. In autumn, *L. maculans* ascospores issued from stubble on fields harvested in the previous summer (sources) are disseminated onto fields newly sown to oilseed rape (targets) (Box2). We have quantified blackleg severity on sources in 2009 and 2011; and on targets in 2010 and 2012 (Figure 3c). For the estimation of the *L. maculans* dispersal kernel between two successive cropping seasons (Figure 3d), we have built a dispersal model in which 1/ the sources are described as continuous in the space made of the source fields, by a spatial lognormal process; 2/ the following year disease severities are described as continuous in the space made of the target fields, by a convolution of the sources and the dispersal function. In this model, the descriptions of the sources and the disease severities on the targets are continuous, whereas data are punctual. Using a Bayesian inference algorithm, nine different versions of this model were fitted to the data. The most adapted version (in the sense of Bayes factor) to each of the two data sets contains a fat tail dispersal function (power exponential, with a shape parameter lower than 0.25). These data are the first obtained on distance from 0 to 1000 m, from a set of non-punctual sources. They open the prospect of improving the existing simulators, or to produce maps of risks.

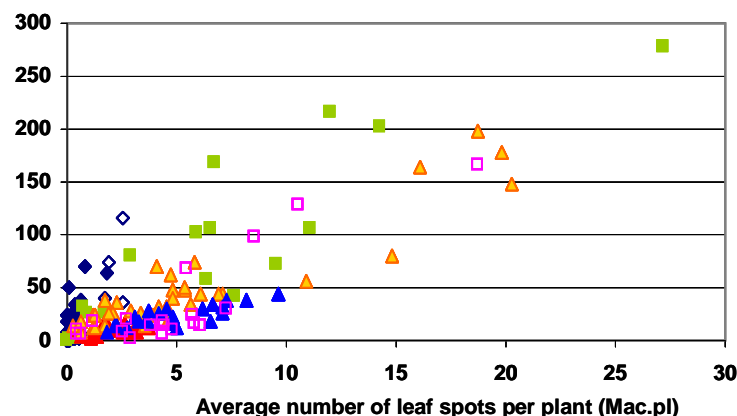
Figure 4: Methodological developments: optimizing the cost-precision ratio

Estimation of the severity (numbers of individuals)



4a. A one square meter (0.57 x 1.75 m) observation area, delimited with a portable PVC pipes structure, placed at canopy height. During one minute, the observer moves at regular speed lengthwise along this area. He observes the whole area, without recounting leaf spots.

Average number of leaf spots observed on one square meter within one minute (Mac.m2)

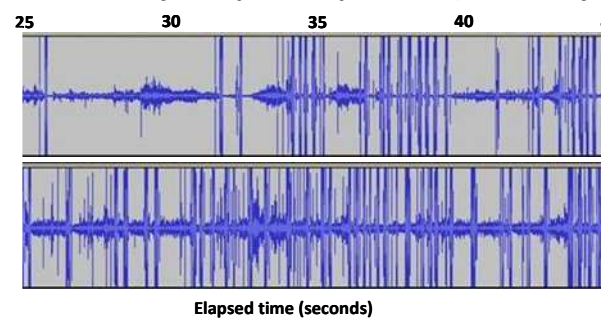


Correlation (IDC 95%)

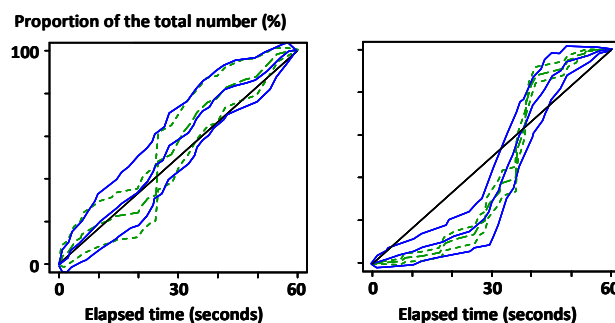
◆ Exp. 1	0.77 (0.60 - 0.87)
◇ Exp. 2	0.86 (0.68 - 0.94)
■ Exp. 3	0.90 (0.74 - 0.96)
▲ Agr. 1	0.38 (-0.08 - 0.70)
△ Agr. 2	0.93 (0.86 - 0.96)
▲ Agr. 3	0.81 (0.60 - 0.92)
□ Exp. 4	0.87 (0.71 - 0.95)

4b. Relationship between the average number of leaf spots per plant (Mac.pl) and the average number of leaf spots seen in one minute on the one square meter area (Mac.m2) for 179 points issued from field plots of four experiments (Exp. 1 à 4) and areas delimited in farmers' fields (Agr.1 à 3). Pearson correlation coefficients and their 95% confidence intervals are indicated.

Estimation of the spatial repartition (location of the individuals)



4c. Examples of visualisations for two sound records corresponding to contrasting situations. Each wide and sound-saturating vertical band corresponds to one bip generated upon the observation of one leaf spot. The bips are unevenly distributed in time in the upper record, and more evenly distributed in the lower record.



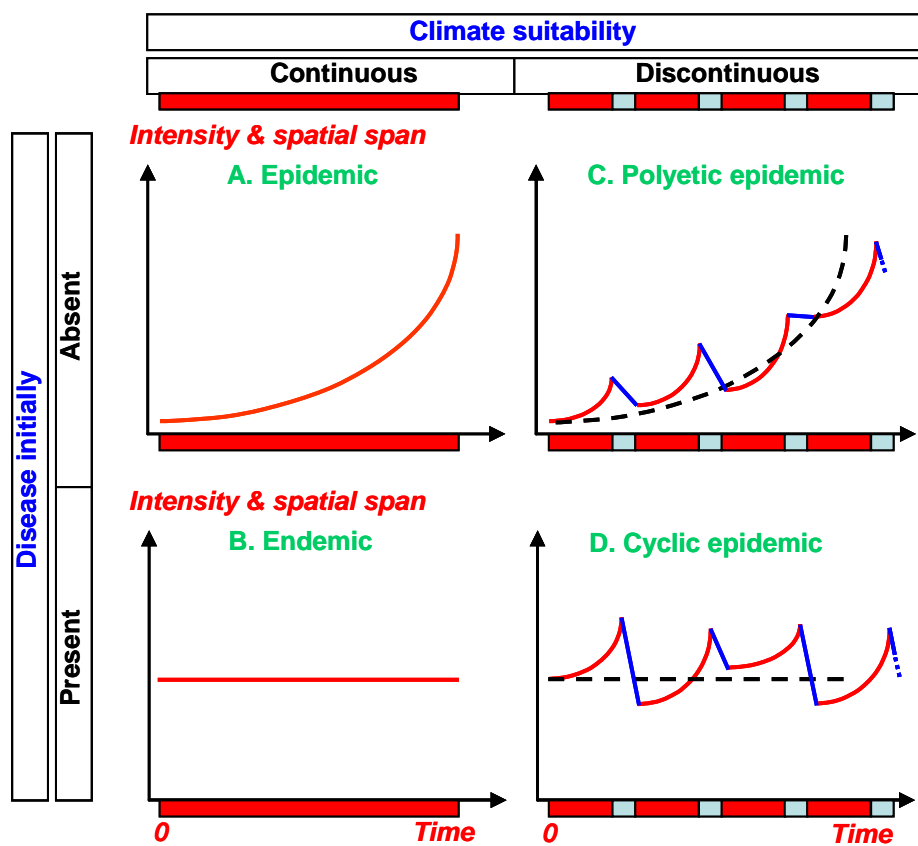
4d. Examples of repartitions of individuals observed on two experimental plots with contrasting disease symptom aggregation. The diagonal corresponds to uniform repartition of individuals observed per time unit. Counts on geo-localised plants (solid blue line) and time of detection by symptom sampling (green dashed lines) are indicated, with calculated confidence intervals. In the left panel, the spatial symptom repartition is uniform, as indicated by the diagonal falling within confidence interval for both methods. In the right panel, the symptom repartition is significantly different from the uniform repartition. The congruence between the two methods can be deduced from the overlap of confidence intervals.

Sources: Adapted from Bousset et al. Manuscript [ms1] submitted to Plant Pathology

Box 5: What is a cyclic epidemic?

Four different types of epidemics, illustrated according to their description by Zadoks and Schein (1979)

Depending on whether the disease is initially present or not, and the climate is continuously suitable or not, four types of epidemics can be contrasted: A. When the disease is initially absent, then initiated by the mobilisation of external inoculum (arriving from outside), then its intensity and spatial span increase over time, we talk of an epidemic "The noun epidemic refers to an increase of intensity as well as extensity of the disease". B. When the disease is present since the origin, and its intensity and spatial span are constant over time, we talk of an endemic. When suitable periods alternate with periods unfavourable to the disease – for example in case of climate seasonality – the increase of the disease can include discontinuities on short time scales, with the alternation of epidemic phases and survival phases. Depending on whether the dynamics on long time scales are epidemic or endemic, we talk either of C. polyetic epidemic "The newer term *polyetic epidemics* [...] is limited to epidemics whose increase in intensity takes many years"; or D. of a cyclic epidemic "If an epidemic flares up and dies down periodically, annually for instance, it is termed a *cyclic epidemic*". This is the case of numerous diseases that increase in frequency during the cropping season, but neither of their intensity or spatial span increase over years at the scale of the landscape. The dynamic of cyclic epidemics can be formalised for pathogens on crops of the agro-ecosystems [18; Figure 5].

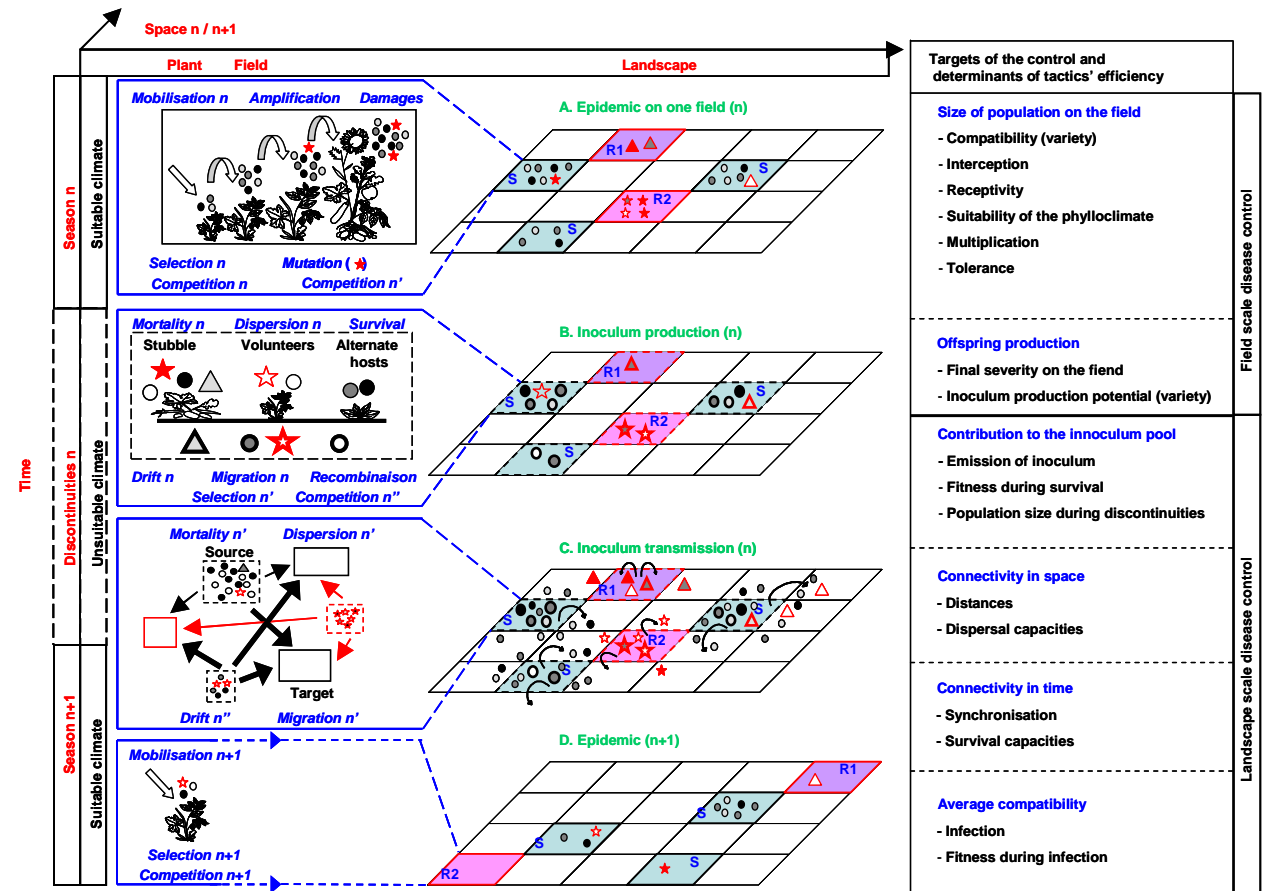


What are the phases of a cyclic epidemic?

The alternation of continuities and discontinuities on scales of time and space allows summarising the pluri-annual dynamics by three phases: the epidemics on the field; the production of inoculum on the field, and the transmission of inoculum among fields. Within each phase, the processes affecting population size or structure can be summarised at the lower scales of space (the field, the plant or the organ) and of time (the cropping season, the duration on one generation of the pathogen) (Figure 5). In the agricultural ecosystem, the prospect is open, to maximise the efficacy of disease control by using human actions to interfere with the cyclic epidemic, both during the epidemic and survival phases.

Sources: Adapted from Bousset L, 2012. INRA, Working paper, and Bousset and Chèvre 2012 Journal of Botany

Figure 5: Determinants of the cyclic epidemics control efficacy



Determinants of disease control efficacy on one field and in the landscape based on a schematic representation of pluri-annual dynamics of cyclic epidemics of plant diseases in an agro-ecosystem. The agro-ecosystem is defined on scales of space and time by the alternation of homogeneities and heterogeneities induced by agricultural practices. The "cyclic epidemics" – represented for seasons (n) and (n+1) – is defined as the succession of A. within field epidemics, B. production of inoculum on the field, C. transmission of inoculum from source to target fields, D. within field epidemics in the following season. The processes defined by life cycle and genetics affect pathogen population size or structure (above and below encapsulated panels, respectively). Different letters (n, n', n'') indicate that the successive realisation of processes can be independent. For each component of the pluri-annual epidemics, a limited number of attributed aims (listed on the right) determine the efficacy of disease control tactics. The diversity of individuals within pathogen populations is represented by the different shading of the symbols, illustrated with respect to plant resistance. The efficacy of disease reduction on susceptible (S) and resistant varieties (R1, R2) depends on the host and pathogen populations properties (listed on the right) in the landscape that it is possible to manage.

Sources: Adapted from Bousset L, 2012. INRA, Working paper, and Bousset and Chèvre 2012 Journal of Botany

To undertake such studies, I first had to develop an alternative method to quantify disease severity, based on the counting of leaf spots seen while observing a one square meter area during one minute (Mac.m2) [ms1]. Among the current methods, there is a trade-off between precision and cost: precise methods are time consuming, while rapid ones yield categories, too imprecise for the ranking of numerous observations. The new method has been tested on blackleg, in field plots and farmers' fields. Precision has been tested by evaluation correlation with a usual disease severity index, the number of lesions per plant (Mac.pl); we have confirmed that Mac.m2 values are correlated to Mac.pl values (Figure 4ab). Repeatability has been evaluated by testing the correlation between repeated observations, and we have confirmed the correlation between successive observations on the same area, or by two different observers, for Mac.m2 and Mac.pl. We have tested the ease to implement the method by comparing the rankings of multiple observers, and confirmed that the ranking of field plots with contrasting severities were coherent among observers. We have also identified the limits of the method. Used with care, it opens the prospect of performing studies previously hampered by the time of realisation.

In collaboration with Joël Chadœuf (GAFL, INRA Avignon), we are exploring the capacity of a derived method, to provide information not only on population size, but also on the spatial repartition of the individuals. The principle is to use the time of detection as an estimator of the spatial localisation of individuals (Figure 4cd). We used artificial inoculation of experimental field plots with *L. maculans* to create contrasting situations with respect to symptom aggregation. The aim of our study was to compare two methods in their abilities to discriminate the contrasting situations. The first method was an exhaustive localisation of symptoms (number of lesions per geolocalized plant). The second method was a symptom sampling, using the time of detection as an estimator for the localisation on space. As previously described, the observer progressed along the one-square-meter area at constant speed. But instead of counting the leaf spots he generated a sound signal (bip) for each leaf spot observed. The bips were recorded with a portable voice-recorder. The procedure was repeated 4 times per plot, and confidence intervals were calculated. (Figure 4cd). The repartitions observed either by exhaustive geo-localised counts, or by the symptom sampling method were compared. This method is complementary to that of "transect sampling" used in ecology, and opens the prospect of studying disease symptom aggregation much more rapidly than with the usual counting methods.

Reciprocal adjustment and weaving links between concepts: cyclic epidemic dynamics

To optimise the combination of disease control tactics, we used modelling. Previously, there were models taking into account either spatial aspects for epidemics very polycyclic⁷ during the cropping season (Zadoks 1989; Shi-Mai 1991; Holt and Chancellor 1999), or the temporal aspects for polyetic⁸ epidemics (Jeger 1986; Bailey and Gilligan 1997; Filipe and Gibson 2001; Gilligan 2002; Bailey et al. 2004) but no models were considering there two discontinuities and several control tactics. The topic of adaptation to resistances, focussed on adaptation, was little linked to models considering the impact of cropping practices on yield losses (Savary et al. 2006). In her PhD co-supervised at the interface between epidemiology and agronomy, Elise Lô-Pelzer [doc2] has produced a simulator allowing for the ranking of disease resistance management strategies, depending on their economical cost, their cost in loss of efficacy, and their environmental cost [14, 15]. This work is the first to take into account both population dynamics and genetics at pluri-annual scale over a network of fields, and allowing for the combination of disease control tactics based on cropping practices, on genetics, on pesticide use, and on the landscape structure. In particular, it has opened the prospect of considering jointly the biology and the actors' strategies at the scale of a territory (Hossard et al. 2013).

⁷ Epidemics in which several cycles (generations) of asexual multiplication or sexual reproduction follow each other.

⁸ Epidemics for which the increase in intensity and / or spatial span take several years

From the epidemiological studies, the aim to develop this simulator and my previous knowledge of the topic and the current researches on adaptation to resistances, the need for networking between several scientific disciplines has rapidly emerged. This was materialised by the ANR-ADD Cèdre (2005-2009) project, bringing together biology and social sciences. We realised that an additional work was needed for the formalisation of the efficacy of epidemic control on the crops of the agro-ecosystems, and its stability over time, in order to consider jointly population dynamics and genetics. Spatial and temporal scales needed to be explicit, and accounting for disease control tactics issued from different disciplines should be possible. This work started during the coordination of the project ANR-ADD Cèdre, in collaboration with several colleagues, and then continued with Anne-Marie Chèvre. This work has allowed proposing a formalisation for the pathogens' dynamic of cyclic epidemics on the crops of the agro-ecosystems.

This theoretical work obliged me to search for the concepts existing in the different disciplines, in order to weave links between them. It also made me realise that in epidemiology, the concept of cyclic epidemic⁹ – ignored even if published in 1979 by Zadoks and Schein – was much more suited than that of epidemic to represent the dynamics of the many crop diseases that increase in intensity and spatial span during the cropping season, but neither intensity nor span increase over years at the scale of the landscape (Box 5). This dynamics can be summarised by three phases: the epidemic on the field, the inoculum production on the field, and the transmission of inoculum among fields [18]. This allows connecting the epidemics of a cropping season to that of the following one, and opens the prospect to study the coevolution between hosts and pathogens at the scale of a network of fields. In order to study the coevolution between host plants and their pathogens in metapopulations, the geographic mosaic of coevolution theory (Thompson 2005) has been formulated for natural ecosystems. I had to explicit the specificities of agro-ecosystems – discontinuities and human actions – in order to be able to adjust this theory to the adaptation of pathogens to cultivated plants. The framework we developed allows connecting the efficacy of control tactics to the epidemiology [18; Figure 5] and the stability to the adaptation dynamics [19]. This provides a conceptual basis to combine several tactics, and to jointly study population dynamics and genetics, with some formalisms adjusted to the specificities of agro-ecosystems. The long delay before having this work published ([18] was the 8th submission of the manuscript) can be explained by the conjunction of several limits needing to be overcome: to form a clear representation of the processes, to allow oneself to express it and write it clearly, to link it to the existing literature in order to render it acceptable to the reviewers even if it goes against the current practices of the theoreticians well recognised in epidemiology. I here point to the crucial role of the collaboration with Anne-Marie Chèvre, because without an external look, following that way is even more difficult. Moving the focus from the field to the landscape scale was long mentioned as a desirable aim. Behind the technical difficulty was in fact hidden a theoretical problem [18; 19]. This conceptual work offered me the opportunity to think more broadly about interdisciplinarity and to the means to make it live [v9].

⁹ " If an epidemic flares up and dies down periodically, annually for instance, it is termed a *cyclic epidemic*" (Zadoks et Schein 1979)

4. Perspectives: ecology of contagion at landscape scale

In the actual agro-ecosystems, the pathogen metapopulation continuously adapts to its hosts, but the other way around does not occur. While the management of weed infestations is conceived over a succession of years, the protection of crops against epidemics is considered a tactical action, concerning one field in one given season. Having the theoretical framework of the geographic mosaic of coevolution opens a different perspective. Crop protection can become the combination of actions on the metapopulation of plants, and direct actions on the pathogen metapopulations, with the aim of maintaining the latter locally non-adapted on the crops. The prospects that I wish to develop concern the ecology of contagion and the combined use of immunisation and reduction of contagion at the scale of landscapes.

Thematically, I wish to explore these questions under the point of view of contagion between fields in the agro-ecosystems. Traditionally, and perhaps because healing animals and humans answers to an ethical injunction, the collective health is considered as the recovery of health in diseased individuals. Thus, one considers the infected individuals, focussing on the disease cycle from infection to infectiousness, by processes specific to each plant-pathogen pair. In view of the cyclical nature of epidemics, I chose a different point of view: focussing on the processes that allow (or not) the transmission of disease from infectious individuals to infected individuals. I use the term "contagion" referring to disease transmission from an infected host to a new host. Contagion can only occur with an infected host, a contact process (in case of direct contagion) or a process of vection (in case of indirect contagion), and a receptive host.

Prospect 1: Ecology of contagion to explore new levers of action

The theoretical framework that we proposed [18; 19] allows placing the field during the cropping season within the dynamic of cyclic epidemics, and thus to look for levers of action outside of the epidemic phase. In agro-ecosystems, the mobility of plant hosts during their "life" can be managed. This opens the prospect of managing contagion, aiming at the three possible targets: the infectiousness of products issued from the field; the mobility of seeds between seasons; the vection by abiotic (wind, water) and human means. In line with my previous work, I wish to explore these linked to host infectiousness, and to direct and indirect transmission.

Reducing host infectiosity in L. maculans

We have shown the positive relationship between disease severity at the end of the cropping season, and the production of fruiting bodies. As opposed to cropping practices sometimes difficult to implement, acting on host infectiousness by their genetics offers simplicity to the end user. I thus wish to explore the possibility of selecting varieties with a low potential of inoculum production, looking for genotypes that, at a given severity at the end of the epidemics, would produce less fruiting bodies than others. A preliminary request is to demonstrate the existence of some variability for this trait within genotype collections. In collaboration with Regine Delourme, we have set up experiments to explore the genetic variability that exist for the production of inoculum, and proposed a supervision project.

Reducing direct contagion in L. maculans

We have estimated the ascospore dispersal kernel, using the severity observed in autumn. For this, we quantified leaf spots both on sources (autumn n-1) and on targets (autumn n). It has been shown, for weed dispersal, that it is possible to develop spatial inference models simultaneously using several kinds of data (Bourgeois et al. 2012). In collaboration with Samuel Soubeyrand, I wish to continue by exploring if adding information on the severity of cankers (that is after the systemic migration of the fungus from leaf spots down to stem bases; Box 2) on sources, on cropping practices during summer, and on differential receptivity depending on varieties on the target fields, would allow to obtain better fit of the model to the data. Already, the dispersal kernel as we have estimated it, allows for calculation the contagion by contact

between neighbouring fields at the scale of a landscape, to develop maps of risk (Haran et al. 2010; Magarey et al. 2011; Rivas et al. 2012), to improve the existing simulators and to achieve simulations at the landscape scale (11; Hossard et al. 2013; Papaïx et al. 2013).

Quantifying indirect contagion at the scale of agro-ecosystems

In my previous works, I mainly focussed on direct contagion (by contact between hosts, via aerial dispersal). Conceiving cyclic epidemics in terms of contagion between hosts allows asking the question of indirect contagion. The impact of anthropogenic activities as a support to vection is attested in invasion biology. For example, invasion frequencies increasing with the intensity of human exchanges along roads or maritime ways (Hume 2009) and an impact of human activities on weed dissemination (Hodkinson et Thompson 1997) have been observed. As numerous tools move between fields, are they vectors for plant pathogens? The soilborne organism in their resting forms can show a great resistance. For example, it has been shown that nematode cysts survive to the digestion by cattle (Kontaxis et al. 1976) or the process of sewage plants (Spaull et al. 1988). In collaboration with Eric Grenier and Sylvain Fournet (UMR IGEPP) we have proposed projects (ANR, INRA métaprogramme SMAcH) to describe the anthropogenic vection of root beet cyst nematodes, and its consequences on population structure. For me, this work would be a basis for a wider reflexion on connectivity at landscape scale, and on the relationship between the production of knowledge and its use by actors.

Prospect 2: Immunising versus preventing contagion: connectivity at the landscape scale

The studies of interactions between metapopulations in natural ecosystems indicate an interplay between ecological and evolutionary processes (Nemri et al. 2012; Tack et al. 2012; Thrall et al. 2012; Tack and Laine 2013a) with a strong impact on the phase between epidemics on the dynamic of epidemics (Tack and Laine 2013b) and an interaction that is dynamic over time (Burdon and Thrall 2013). For the interaction between metapopulations in agro-ecosystems, these facts incites to take into account all the processes, natural as well as anthropogenic [18; 19] especially for dissemination. The interaction between landscape structure and the stability of resistances' efficacy (Papaïx et al. 2013) reflects the synergic action of immunisation of the fields by the deployment of resistances, with the reduction of contagion between the fields of the landscape.

The control of cyclic epidemics by the structure of the agro-ecosystem at the landscape scale is based on the hypothesis that contagion intensity is variable. One of the descriptors used is connectivity. The notion of connectivity in ecology is specified in the case of active dispersal of individuals, with choice of preference for some of the environments. It has been appropriated by plant pathogen epidemiology, both on data simulated at the scale of a network of fields (Papaïx et al. 2013) or on data aggregated at the scale of administrative districts (Margosian et al. 2009). Does the notion of connectivity have a meaning in the case of passive dissemination? How to simultaneously take in to account natural dissemination and anthropogenic dissemination? For agricultural ecosystems, several approaches are actually developed in metapopulations, focussed on diffusion by contact processes (Parnell et al. 2010; Ndeffo-Mbah et Gilligan 2013; Papaïx et al. 2013) or by exchanges within networks (Moslonka-Lefebvre et al. 2011; Kleczkowski et al. 2012).

This corresponds to two distinct manners to conceive connectivity, either as an ecological contact process rendered possible by the physical contiguity of suitable environments, or as a process of contact within a network of relationships. The first leads to the proposal of indices to describe the landscape in ecology (Burel and Baudry 2005). The second leads to consider the social networks (Keeling and Eames 2005; Martínez-Lopez et al. 2012). The topic of epidemics on crops relies on these two notions. Optimising the impact of cropping systems at the landscape scale has to be designed towards all kinds of diseases, whatever their mode of dissemination, natural or anthropogenic. It is thus necessary to obtain the convergence between these two components in order to represent all processes. The fields are connected both by their physical

location in the landscape (geographic proximity of fields, aerial dissemination) and by their belonging to common farms (anthropogenic dissemination by tools and machinery). The locations of fields (Thénail et al. 2009) as well as the choice of cropping systems (Fargue-Lelièvre et al. 2009) depend on the organisation within farms. It is thus important to weave the link between the « physical » (location of fields) and « human » (belonging of the fields to farms) landscapes. For this, I wish to use the concept of connectivity at the scale of landscapes in agro-ecosystems, and if necessary to adjust it in order to represent its spatial, temporal and genetic components. Even if our research proposals submitted (ANR, INRA métaprogramme SMaCH) have not yet been accepted, I will continue to prepare research proposals towards this aim.

As a summary, I chose three keywords to describe the direction of my prospects: epidemiology, interdisciplinarity, theory. The exact pathway will also depend on the context, opportunities, successes and failures. These elements will also be considered to weight my investments in the different ways that it would be possible to explore, while preserving coherence and avoiding scattering.

5. Paths for reflexion: from the ecology of contagion to questioning in ethics

The following paragraphs do not deserve the status of prospects. Their span is too wide; their development requires further maturation, including the elaboration of interfaces between disciplines. However, as defending HDR is the occasion to take the time to look at one's research topic from a bit further, it is a pleasure for me to share in this part some questions about my job as a researcher in plant disease epidemiology. They concern some paths to refine the theory for the control of epidemics, and to develop ethical questioning around the production of knowledge and its use at the service of human societies.

Path 1: Immunising versus reducing contagion: theoretical convergences?

The control of epidemics applies to numerous organisms, pathogens or parasites, both in humans, animals or cultivated plants. However, and even though the questions asked share similarities on a theoretical point, the scientific communities which study them remain disjointed and do not model the processes in a similar manner. Establishing a common reflexion implies to first render the objects commensurable. This would then allow developing a common approach of the weighting between immunisation and reduction of contagion.

The choice of a scale.

Will plants, animals and humans eternally remain incommensurable as individuals? I don't think so. In the opposite, I consider that the epidemics that affect them can be studied in a same way despite the numerous processes of different nature that act at different scale (Burdon and Thrall 2013; Nemri et al. 2012). Instead of establishing the equivalence between one animal individual and one plant animal, for the crops in agro-ecosystems I propose to establish it between one animal individual and the vegetation of one cultivated field, of which the plants would be the "cells" (Box 6). The host individual would then be composed of the whole of the plants of the field, with a "life" spanning from sowing to the complete decomposition of the products issued from that field. For the plant host, this offers the advantage of taking into account the homogeneities in the destiny and the generation between the plants of one field, issued from a common pool but not generating each other.

The spatial (fields) temporal (cropping season from sowing to harvest) and genetic (mono-varietal fields) discontinuities characterise the individuals. This allows tracking the intra and inter-host processes, affected by human actions [19]; For example, the host metapopulation can include the anthropogenic movement of seed batches. For the pathogens, this allows clarifying the intra and inter-host reproduction and dissemination modes. This makes apparent the horizontal (between hosts of the same generation) and vertical (between parents and progenies) transmission. The coevolution around host-pathogen compatibility is studied both for

Box 6: A way to link plant, veterinary and human epidemiology

Instead of establishing the equivalence between one animal individual and one plant animal, for the crops in agro-ecosystems I propose to establish it between one animal individual and the vegetation of one cultivated field. The "fields" of the agro-ecosystem are neither mobile nor "mortal" – as pieces of land – but the "host individual" is represented by the whole of the plants of one species that grow at that location, sown in a given season. I rely on the consideration that the plants of a same field have destiny as linked as that of the cells on one individual (sowing and cropping practices synchronised, collective contribution to the following generation). By mentally transposing plants into "cells", and the vegetation of one field into an "individual", the "life" of that "individual" spans the duration from the germination of the seeds sown, to the harvest of products and co-products.

This individual can become infectious, after the establishment and the propagation of disease within it (the plant-to-plant transmission is then equal to cell-to-cell propagation). The production of inoculum on the field becomes "multiplication within the infected host". The transmission between hosts can occur in a vertical manner (from parents to progenies, via infected seeds) or in a horizontal manner (between hosts, via "contagion" between individuals). Formalising the dynamics of the most plant metapopulation allows to proceed up to (i) the parents of the seeds to "track" vertical transmission (from parents to progenies) of inoculum and (ii) the neighbouring fields of the previous or ongoing season to "track" horizontal transmission (between individuals but due to contagion by contact and not by descent) [19]. After the complete destruction of stubble, infectiousness becomes null for the host, but some infectious units can persist either free in the a-biotic environment, or in the seeds or the products issued from that field. Human actions can be included in the representation.

This individual can reproduce, and its progenies are then the batches of seeds issued from the field. This allows getting the filiations between cropped fields between seasons across generations. In this filiations, it is possible to represent the selection of plants, the production of seeds and their trade [19].

The health of this individual can be modulated by human actions. The use of plant genetic resistance finds its parallel in the "immunisation" of individuals. In humans, a distinction is made between the vaccines preventing infection, and these limiting the multiplication within the infected hosts. Is it possible to draw the parallel further, with qualitative resistance preventing the infection of the "cells" and quantitative resistance limiting the multiplication within the host by reducing the extension "from cell to cell" and the progression towards the infectious stage? Is it possible to get support to the parallel between spraying a pesticide on a field and giving an antibiotic treatment to someone? Are the cropping practices targeting the destruction of stubble analogous to the prophylaxis – with which they already share the denomination in the phytopathological literature?

Weaving the link in this way, "host-individuals" in crops and animals are not longer that much incompatible. It will be necessary to explore the legitimacy of these transpositions, by examining in details each of the formalisms used in the theories developed in medical epidemiology, and, if necessary, to adjust them. The advantage of this parallel is to propose a common conceptual basis for the theoretical reflexion in epidemiology of plants, animals and humans.

Source: Adapted from Bousset L, 2011. INRA, Working paper and Bousset and Chèvre 2013

Box 7: And what if, in areas of endemism, the immunisation was not sufficient?

A documented case, the eradication of smallpox. Against smallpox, a collective management strategy has been decided. The modalities have been elaborated based on the modelling of epidemics, which allowed determining the necessary immunisation ratio. Immunisation at this ratio allowed achieving eradication in the areas where the disease had an epidemic behaviour. However, in the areas of endemism, the immunisation alone failed, despite the fact that the vaccination ratio chosen (in a centralised manner, according to epidemiological models) had been obtained (Foege et al. 1975). In the areas of endemism, it is by associating decentralised and local actions – techniques of monitoring and control of disease foci – that the eradication has been obtained, sometimes at a date earlier than the model predictions, or even before the time when vaccines were delivered (Foege et al. 1975).

Some specificities. Smallpox is a contagious human disease, caused by a pox virus. It includes a latency phase, a first stage with fever, followed by a second stage with eruption of skin pustules. This renders possible the reduction of contagion, in cases when infected individuals are detected early, before becoming highly infectious. An efficient communication has been set up, to promote the detection of infected people by their relatives, and to teach the practices preventing contagion.

Some lessons to be learned? The aim of eradication cannot be transposed to all diseases. However, these facts raise two points for reflexion. The first is the illustration of the potential of the methods reducing contagion. In this case, reducing contagion has a synergic effect with the immunisation, and allowed reaching the aim. This is an incitation to explore (for the diseases of crops for which the symptomatic hosts can be detected before becoming infectious) in which cases the cultural practices reducing contagion do have such a synergic effect with the use of plant genetic resistance. The second point is that optimal strategies may not be the same for epidemic areas and for areas of endemism, in which the control based only on immunisation may not be sufficient.

Source: Adapted from Bousset L, 2011 ; 2012. INRA, Working paper

pathogens and parasites of humans and animals (Shim and Glavani 2009), the deployment of plant genetic resistance (Damgaard 1999; Papaix et al. 2013) and of pesticide treatments on plants, humans and animals (Koella et al. 2009; REX Consortium 2013). To test the validity of the conceptual parallel (Box 6) I wish to participate to the dialog between these scientific communities, for example by participating as a biologist to the formalisation of mathematical models.

Weighting immunisation and reduction of contagion

Until now, I have mainly worked on the adaptation of fungi to plant genetic resistance. To date, there exists no theoretical basis to the combination of levers acting on the different phases of the cyclic epidemic [18; 19]. In parallel, theoretical studies of the epidemiological and evolutionary consequences of immunisation highlight the importance of the within-host multiplication processes (André and Gandon 2006) and of transmission (Litvak-Hinenzon et Stone 2009) on the dynamics. Exploring this topic by generic theoretical studies, to understand the determinants of success and the keys for optimisation, appears as a pre requisite for the case-studies. Taking into account the cyclic nature of epidemics is already initiated in human and animals (Litvak-Hinenzon and Stone 2009) and for pest outbreaks (Jepsen et al. 2009). But are there studies introducing an anthropogenic bias term in the interaction between metapopulations? Models centred on the epidemic phase allowed to propose thresholds based on the basic reproduction coefficient R_0 . (Gilligan and van den Bosch 2008). Others centred on the proximity between hosts allowed to propose thresholds based on the percolation theory (Poggi et al. 2013). Could we propose thresholds based on the "cyclic epidemic" object as such and in totality? If the aim is to used human actions, in a directed manner, to interfere with the cyclic epidemics both during the epidemic phases (cropping season on the continuous space of one field), during survival phases (outcome of the inoculum produced at the end of the cropping season) and by reducing the transmission (reducing the spatial and temporal connectivity as well as compatibility): when and how to combine the actions? Optimal control (Forster and Gilligan 2007; Ndeffo-Mbah and Gilligan 2010; 2013) and multi-attribute optimisation (Sadok et al. 2009) could inspire such studies. Developing multi-agent simulation models (Roche et al. 2008; An 2012) could be a way to model the interaction between metapopulations.

Path 2: from the ecology of contagion to the questioning in ethics

Thinking over the optimisation of strategies, questions more deeply the contribution of modelling to the management of crop protection.

Contagion and education to health

In practice, two main types of management strategies can be observed for the control of epidemics and plant or animal species expansion. The first one is based on the large scale deployment of strategies planned in a centralised manner, aiming at stamping down the epidemic dynamics, either by mass-immunisation (humans and animals) or by eradication of individuals (plants and animals). It is mainly applied to lethal diseases, with a high sanitary or economic impact and to species which extension is considered detrimental. In these strategies, actors are told to act, without choice of the implemented modalities. The second one is based on advice and collective education to prophylaxis. It is mainly applied to diseases and species which impact is not consider critical, often endemics. In these strategies, actors are educated or incited to act, leaving them the choice of the modalities to implement.

Crop protection falls within these two types of strategies: some diseases are of obligatory detection, declaration and eradication (quarantine, destruction, certification of the sanitary status); others rely of practices taught or advised (Labarthe 2009; Labarthe and Laurent 2013). Among these practices lies the use of pesticides under free access (as opposed to antibiotics) even if their application is regulated. In highly homogeneous agricultural landscapes, pesticide use combines the triple advantage of ensuring the protection of one field neither having to take into account the combination of several methods, nor the spatial environment of the field

(neighbouring fields), nor the temporal environment of the field (previous and next crops in the cultural succession). None of the alternative methods cumulates these three advantages. Reducing pesticide use thus implies to re-appropriate time, space and the combination of methods. The majority of pesticides are spread in order to control cyclic epidemics, not lethal, and already present locally (Aubertot et al. 2005). Nevertheless, the implicit hypothesis of current theoretical studies concerning the optimisation of strategies is that of an action chosen in a centralised manner, and resting upon modelling. Beyond their cost – sometimes associated with a contestation of their ethical value (Wood 2004) – these approaches are highly dependent upon the data used for the mathematical modelling (Charleston et al. 2011; Ndeffo-Mbah and Gilligan 2011).

Modelling implies the simplification of the reality. The balance between the simplification / generality and the adjustment to local specificities will need to be explored. Should researchers simplify the reality upstream, to model and look for a unique strategy, and then transmitted in a centralised manner to the actors that will have to implement it? Should simplification be downstream, transmitting to actors not a strategy, but guidelines for action so that they decide locally taking into account the local constraints, allowing for the differentiation of strategies by the actors? As I previously stated elsewhere [D1, D2] admitting the cyclic nature of the epidemics leads to wonder whether a management specific to these diseases, in areas of endemism, shouldn't be studied (Box 7). At a time when a privatisation of farm advisory services is observed (Labarthe 2009; Labarthe and Laurent 2013), opposite dynamics occur in human health (with in France l'INPES¹⁰ which is a dedicated public institute) and in ecology for example with the education towards decentralised management (Braun et al. 2006). In my opinion, a theoretical investment on such strategies is necessary. Further, as the absence of ethical injunction to heal plants would allow it, it would be possible (as opposed to animals) to experimentally compare the efficacy of both types of strategies. I do not have the skills to undertake such studies on my own, but I could contribute to the elaboration of interdisciplinary groups, or to the drafting of documents that could enable the actors to appropriate the topic of cyclic epidemics' dynamics.

Acting at the scale of agro-ecosystems: what ethical frame?

The scientific discipline "agronomy" is built upon the use of biological knowledge to serve human societies. The extension of the possibilities scales leads me to wish taking the time to develop ethical questioning in parallel to the production of knowledge. From a biological point of view, it becomes possible to intentionally modify agro-ecosystems, in a directed manner. However, speaking of agriculture is speaking of farmers, and more widely of human collectives. Modifying agro-ecosystems thus cannot be done without impacting the human groups that live there and make a living out of them (Mazoyer and Roudart 2002). Is it desirable to aim at the optimisation of an ecosystem just as fields were optimised? Is it desirable to optimise the acting on humans? Are the modalities of collectives' decision and law suited to go with such changes?

Defining aims such as "Initiate, foster, guide and support the change" are in agreement with the applied aim of agronomy, but does the corresponding ethical frame exist? For the acting on humans groups, the possibilities expand, by the development on knowledge allowing lowering the reluctance towards adoption of techniques, for example building upon the analysis of actors' attitudes (Breukers et al. 2012) or of the relationships of reciprocal power among them (Vanloqueren and Baret 2008 ; 2009 ; Fares et al. 2012). But what is desirable? The acting on non human beings and living materials can refer to ethical theories of Nature. The acting on human groups can refer to economic and social ethical theories. But what to refer to, when both

¹⁰ National institute for prevention and education for health (Institut national de prévention et d'éducation pour la santé)

get confronted? Which of the choices generate a conflict? The institutional aim of optimising at the scale of landscapes generates these questions.

Our responsibility as researchers is engaged in the knowledge that we chose to produce, in the equity of its diffusion to actors, and the modalities of collective decision regarding risks, know or unknown at the time of the decision (Baudoin 2009). If the ethics of knowledge production has already been approached for the production of human artefacts (for example weapons of bioterrorism; Ehni 2008), the questioning is neither over for moral dimensions (Baudoin 2009; Bourg and Papaux 2007) or organisational applications (the case of our topic). I wish to take some for this questioning, concerning the control of epidemics on crops.

Part 3: Link between researches, coordination and supervision

1. Coordination

During my journey, my involvement into coordination was materialised by my implication in the coordination of two interdisciplinary projects from 2005 to 2008, and continues with the responsibility for a workpackage (ANR 2012-2015). At the local scale, I was responsible for the coordination of a collective (June 2011-June 2012) during the structuring of our research Unit, and I am currently responsible for the animation of one of the three objectives of my research team. These activities clearly enriched my way to get involved in my profession of researcher, have fed my theoretical reflexion, and offered me to think of interdisciplinarity [v9].

Coordination of research projects

My previous journey allowed me to get involved early in project coordination. The main lock was the weaving of links between scattered pieces of knowledge about the topic of crop protection by varietal resistances. The projects that I led contributed to collectively build and enrich the topic. We have produced knowledge (scientific publications) and contributed to the training of students (Masters and PhD training periods). We have contributed to share knowledge among colleagues, towards our institutions and funding agencies, and towards the actors. These productions are expected for every project. As interdisciplinary projects, we have also contributed to elaborate the interfaces between scientific disciplines.

From 2005 to 2008, I took care of the setup followed by the coordination of the project "Adaptation of the populations of blackleg to the resistances of oilseed rape: characterisation of partial resistance, functioning of populations during the loss of efficacy of the resistance, and production of inoculum at the end of the cropping season" funded by the CTPS¹¹. This project brought together plant pathology, plant genetics, agronomy and the CETIOM¹² technical institute (4 teams). It has allowed to produce pieces of knowledge that can be used in breeding, but also to contribute to their diffusion by the redaction of a publication destined to a wider audience [v8].

From 2005 to 2009, in collaboration with Anne-Marie Chèvre, I took care of the setup and then the coordination of the ANR ADD¹³ project Cèdre "How to durably exploit varietal resistances". During this time, Anne-Marie Chèvre taught me how to coordinate a project, and I am grateful to her for that. This interdisciplinary project between biotechnical and social sciences brought together 16 research teams, plus 10 partnerships with technical institutes, regional services and associations of growers. This project was based on a pluri-disciplinary approach in order to merge human, social and economical analyses to the biotechnical studies. It was intended to obtain the convergence of modelling, experimentations, site surveys and

¹¹ Comité Technique Permanent pour la Sélection Végétale / Permanent technical comity for plant breeding

¹² Centre Technique Interprofessionnel des Oléagineux Métropolitains / Interprofessionnal technical center for metropolitan oilseed crops

¹³ Programme "Agriculture et Développement Durable" de l'Agence Nationale pour la Recherche / Program Agriculture and sustainable development of the French national research agency

inquiries on a set of representative pathosystems (concerning different hosts – broad acre crops, fruit trees, vegetables; and pathogens with contrasted biological characteristics). In addition to the production of disciplinary knowledge, the main result lies in the disciplinary interfaces that have been created. This translates into publications that have renewed the topic: formalisation of a conceptual framework [18; 19], production of a simulator [14, 15], appropriation of the topic by sciences of management (Fargue-Lelièvre et al. 2011) and economics (Ambec et Desquilbet 2012). These collaborations gave birth to continuations as new projects after the project duration. The project produced numerous communications and academic publications, or publications for a wider audience of practitioners. It has allowed the training of students (21 training periods) and the production of tools to simulate spatio-temporal dynamics. At the end of this project, I have coordinated the setup of the interdisciplinary collective for the project "Modelling of the spatial allocation of varietal resistances and transmission of epidemics at the landscape scale (MARTEP) " proposed in September 2009 to the call from the Ministry (MEEDAT¹⁴). This project has not been funded, but it has built several of the bases of the project GESTER in which I am currently involved.

From 2012 to 2015 I am in charge of the Work package 6 "Production of candidate scenario for the durable resistance deployment in wheat and oilseed rape" in the project ANR Agrobiosphère GESTER. "Management of crop resistance to diseases in agricultural landscapes as a response to new constraints on pesticide use". This interdisciplinary project between biotechnical and social sciences (9 teams) is coordinated by Christian Lannou (Bioger, INRA Grignon) and François Coléno (SADAPT, INRA Grignon). I also contribute to the project by experimentations in epidemiology.

Scientific animation within my research unit

Within the INRA politics of merging units, the unit I belong to (INRA SPE¹⁵ department) has been merged with a unit with which we were collaborating in genetics of plant resistances to fungi (INRA GAP¹⁶ department) to become the UMR IGEPP¹⁷ research unit. During this process, I was in charge of the collective construction of the scientific topic around integrated crop protection. I am currently responsible for the animation of one of the three objectives of my research team.

From June 2011 to June 2012, I was in charge of the "Integrated Crop Protection" action within IGEPP. Following the perturbations caused by the AERES¹⁸ evaluation of the project concerning integrated crop protection, this action allowed the collective construction of the scientific topic. To achieve reciprocal knowledge between colleagues, I have associated times for collective exchanges, individual exchanges with colleagues, exchanges with the direction of IGEPP, and participated to exchanges with our INRA SPE and GAP departments. This reflexion produced a collective consideration for the structuring of researches on integrated crop protection in the unit. The collective framework was used by several colleagues, when redefining their research projects. It served as a basis for the application for reinforcement.

Since my actual research team was created (Resistance and Adaptation, led by Régine Delourme) I am in charge of the scientific animation of one of our three objectives: "Development of cyclic epidemics".

¹⁴ Ministère de l'Ecologie, de l'Energie, du Développement durable et de l'Aménagement dur Territoire / Ministry for the ecology, energy, sustainable development and landscape planning

¹⁵ Santé des Plantes et Environnement / Plant health and environment

¹⁶ Génétique et Amélioration des Plantes, devenu Biologie et Amélioration des Plantes depuis / Plant genetics and breeding, that later became Plant biology and breeding

¹⁷ Institut de Génétique, Environnement et Protection des Plantes / Institute for genetics, environment and plant protection

¹⁸ Agence pour l'Evaluation de la Recherche et de l'Enseignement Supérieur / Agency for the evaluation of research and teaching

The coordination of research projects has allowed me to acquire the necessary hindsight to position my research within a wider interdisciplinary framework; the animation of local collectives has allowed me to contribute to the collective structuring of research projects.

2. Supervision

My production of scientific knowledge has leaned upon the supervision of students in epidemiology [st1, st2], at the interface between epidemiology and population genetics [doc1] and at the interface between epidemiology and agronomy [doc2].

Which aims to supervision?

The aim that I set to my supervision activities is to prepare the student, with confidence, to formulate research questions, to explore new paths and to communicate the results obtained. It is allowing the student to work in a frame comfortable enough to build confidence, but open enough to leave some space for curiosity, thinking and questioning theoretical bases.

Transfer knowledge to students, training them to research by researching, implies for me that four conditions are combined. The first is the identification of a lock in available knowledge. The second is the existence of a clear research aim to lever this lock. The third is thus the existence of a conceptual framework solid enough to lean the questioning on it. The fourth is the existence of some prior knowledge and methodologies, to ensure the feasibility of the project. There is thus a balance to find, so that it exist paths to explore for students, sufficiently cleared (conceptually and methodologically) to allow some self-assurance; sufficiently open and new to leave space for the expression of their creativity in the way they tackle the topic.

What consistency in the supervisions that I have realised.

These conditions being combined, I was able to propose, or to contribute to the elaboration of some subjects, and then to supervise or co-supervise students from 2005 to 2009 [st1; st2; doc1; doc2]. These training periods were focussed on the two locks that I had identified: on the one hand, acquire epidemiological knowledge on inoculum production and dispersal; on the one hand, weave links between pieces of knowledge in order to study strategies at the scale of a network of fields. During the co-supervision [st1; doc1] my colleague epidemiologist Ivan Sache taught how to supervise, in a rigorous manner, I acknowledge him for that.

The capacity of the *L. maculans* fungus to produce conidia by asexual multiplication was known, but their contribution to the dynamic of epidemics and to the population functioning was not. During his Master thesis, Renaud Travadon confirmed the dispersal of conidia, and characterisation of the resulting dispersal gradient under controlled conditions [st1; 10]. This allowed me to propose to Renaud Travadon to follow up in PhD [doc1], at the interface between epidemiology and population genetics, co-supervised with Ivan Sache, in interaction with Hortense Brun. On the one hand, he used a population genetics approach to quantify ascospore dispersal [16]; on the other hand, he studied the systemic migration phase of the fungus, under the angle of a potential limitation of the mate encounter in order to achieve sexual reproduction [12]. Renaud followed his was (post-doctoral fellowship, 2009-present) by joining a team of epidemiologists (UC Davis, USA) to which he brought his skills to tackle epidemiology with the help of population genetic approaches. The topic has been continued by the Master thesis of Thomas Colonges, who continued the characterisation of the dispersal gradient of conidia in the field, as compared to that of ascospores [st2]. In the mean time, Elise Lô-Pelzer [doc2] has established the relationship between disease severity at the end of the cropping season, and the potential for inoculum production for the following season, measured by the number of fruiting bodies [11].

In collaboration with two colleagues agronomists (JN Aubertot and MH Jeuffroy), I co-supervised the PhD thesis of Elise Lô-Pelzer at the interface between epidemiology and

agronomy. Starting from the available knowledge and the results that she has produced [11; 13] Elise Lô-Pelzer has written the SIPPOM simulator [14] and realised its analysis of sensitivity [15]. Elise has followed her way (post-doctoral fellowship 2008-2009 at INRA Ecoinnov) and she is now researcher in agronomy. I kept following the development of these projects by participating in the supervision comity for the following PhD (Laure Hossard, 2009-2012) and to the reflexion of which strategies to simulate (ANR GESTER 2012-2015).

What prospects for supervision?

The results produced by the students that I have supervised from 2005 to 2009 have rendered unstable the theoretical bases, questioned the ways of observing or moved the topic towards studies for which methodologies needed to be developed. I have thus chosen not to propose subjects right after this period, the time for me to rebuild a theoretical coherence [18.19] and to develop the methods [ms1].

Now that I have again a clear theoretical framework, that the methodologies produced render the projects feasible, I started again to identify aims that I could propose to students. I am quite sure that the future collaborations will lead to shake the actual equilibrium. Supervising will oblige me to formulate questions clearly, to develop argumentations, to accept questioning. Co-supervising will be the opportunity to enrich my questioning from different points of view, including these of younger colleagues (not yet having HDR). This will contribute to my research project, and this is the reason that leads me to defend the HDR. Being able to co-supervise students, but without depending completely on them, will allow me a reasonable amount of distance from the current certainties.

Conclusion: why to defend HDR?

Training future researchers by the practice of research implies that students can work in a framework secure enough to build self confidence, but sufficiently open to leave enough space for curiosity, wondering and questioning of theoretical bases. If the previous successes are enjoyable, it is mainly from the un-successes that one learns. By moving away from main streams, I have encountered numerous un-successes (experimental failures, successive rejection of publications and projects) being careful that they affect my journey and not the students' projects. I modestly hope now to have sufficient hindsight to find the best balance between on the one hand, creativity and risk; on the other hand feasibility of the projects, readjusting them if necessary.

I am far from being through my research topic, and the path to explore are many, which leaves ample space for projects managed by students, complementary with the ones I will carry. I thus would like being able to propose to students to come along the way on my research topic. In order for them to learn identifying locks in the current knowledge, or disagreements among theories and observations, formulating research questions, exploring them and communicating the results obtained.

I owe a lot to the colleagues that trained me to the role of supervisor. I also enjoyed a lot the confrontation between points of view during co-supervision. Defending HDR will allow me to explore new co-supervisions, including some with younger colleagues (not yet HDR).

Within my research team and unit, defending this HDR will enlarge our supervision capacities, and especially to diversify the topics on which we will be able to propose some subjects for PhDs. Training tomorrow's researchers to question our own today's certainties, allows renewing the current ways of thinking, it is allowing them to explore the current topics in a different manner, or to explore different ones.

I will conclude by simply referring to the physician Carlo Rovelli:

"Au cœur de la force de la pensée scientifique est la remise en question continuelle des hypothèses et des résultats antérieurs ; remise en question qui, néanmoins, repose avant tout sur la reconnaissance profonde de la valeur de connaissance contenue dans ces mêmes résultats. " (Rovelli 2009. Anaximandre de Milet ou la naissance de l'esprit scientifique p83)

that I translate as "At the heart of the strength of the scientific thinking is the continuous questioning of hypotheses and previous results; questioning that nevertheless rests on the deep acknowledgement for the knowledge contained in these results. " (Rovelli 2009. Anaximandre de Milet ou la naissance de l'esprit scientifique p83).

This leads him to write a little further

" (...) follow and extend the master's path, while criticising the master (...)".

I appreciate this sentence for several reasons. On the one hand, it marks the way for our production of knowledge: pushing further the limits of knowledge, in continuation to the ones before us. On the other hand, it tells what we have to pass to and teach our students: daring tomorrow doubting of what we today consider as the most firmly based certainty, as the most appropriate method, as the most appropriate formalisation. It lies in our hand to teach those who will come after us, so that this questioning always keeps going.

The only addition I will make to this sentence is that following and intermingling several points of view greatly enrich the reflexion. It is thus numerous masters that one should listen to!

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ANNEX 1: list of conference communications

(The names of the students that I have supervised are underlined. Speaker's name is underlined. The numbers in brackets are used as references in the text).

International conferences:

- [i29] **Bousset L**, Jeuffroy MH, Aubertot JN, Lô-Pelzer E (2011) Adaptation to plant resistance genes in agricultural ecosystems: a model system to study disease emergence. *Conférence Jacques Monod "New and Emerging Fungal Diseases of Animals and Plants: evolutionary aspects in the context of global changes"*. June 25-29, 2011, Roscoff (Brittany), France (Poster).
- [i28] **Hossard L**, Lô-Pelzer E, Bousset L, Delourme R, Souchere V, Jeuffroy MH (2011). Assessment of quantitative resistance level impact on durability of associated qualitative resistance using model simulations. Example of phoma stem canker. *13th International Rapeseed Congress June 5-9 (Prague, République Tchèque)*. (Poster).
- [i27] **Brun H**, Chèvre AM, Ermel M, Bousset L, Besnard AL, Huteau V, Marquer B, Eber F, Renard M, Delourme R (2011). Durability assessment of oilseed rape resistance to blackleg: eight years of recurrent selection of *Leptosphaeria maculans* populations in field experiments. *13th International Rapeseed Congress June 5-9 (Prague, République Tchèque)*. (Poster).
- [i26] Lô-Pelzer E, Aubertot JN, Bousset L, Pinochet X, Jeuffroy MH (2009). SIPPOM, a Simulator for Integrated Pathogen POPulation Management: A model to help design innovative cropping systems for integrated crop management strategies at the regional scale. *Farming System Design August 23-26 2009, Monterey, USA* (Oral communication).
- [i25] Lô-Pelzer E, Boillot M, Aubertot JN, Bousset L, Jeuffroy MH, **Pinochet X** (2009). SIPPOM-WORS, Simulator for integrated pathogen population management : un outil d'aide à la conception et à l'évaluation de stratégies durables de contrôle du phoma sur colza d'hiver à l'échelle régionale. *9ème conférence internationale sur les maladies des plantes (AFPP), Tours*. (Poster)
- [i24] **Boillot M**, Sauzet G, Pelzer E, Penaud A, Pic E, Carpezat J, Pinochet X, Aubertot JN, Bousset L, Jeuffroy MH (2008). Sustainable management of rapeseed blackleg by modelling and monitoring of a pilot production area. *Diversifying Crop Protection - Endure La Grande-Motte, France* (Communication orale).
- [i23] **Bousset L**, Chèvre AM, Desquilbet M, Chauvin JE, Sache I (2008). The CEDRE project: a multidisciplinary evaluation of durable management of plant resistance. *Diversifying Crop Protection - Endure La Grande-Motte, France* (Oral communication).
- [i22] **Brun H**, Besnard AL, Huteau V, Ermel M, Marquer B, Bousset L, Eber F, Renard M, Delourme R, Chèvre AM (2008). Combination of resistance factors increases the resistance durability of oilseed rape to blackleg. *Diversifying Crop Protection - Endure La Grande-Motte, France* (Oral communication).
- [i21] Lô-Pelzer E, Aubertot JN, Bousset L, Salam MU, Jeuffroy MH (2008). SIPPOM-WOSR: simulator for integrated pathogen population management for blackleg on canola. *Diversifying Crop Protection - Endure La Grande-Motte, France* (Oral communication).
- [i20] **Travadon R**, Sache I, Marquer B, Ribulé A, Delourme R, Brun H, Masson JP, Dutech C, Bousset L (2008). Preserving durable resistance to Phoma stem canker in oilseed rape : epidemiological key factors. *Diversifying Crop Protection - Endure La Grande-Motte, France* (Oral communication).
- [i19] **Chèvre AM**, Eber F, Vallée P, Letanneur JC, Domin C, Bousset L, Brun H, Delourme R (2008). Are varietal associations of *Brassica napus* a way to manage efficiently specific resistance genes to *Leptosphaeria maculans*? *IOBC oilseed rape meeting OILB Colza, Paris, France* (Communication orale).
- [i18] Lô-Pelzer E, Boillot M., Aubertot JN, Bousset L, Jeuffroy MH, Pinochet X (2008). SIPPOM-WOSR, Simulator for Integrated Pathogen POPulation Management: a tool to help design and evaluate sustainable strategies to control phoma stem canker on Winter OilSeed Rape at the regional scale. *IOBC oilseed rape meeting OILB Colza, Paris, France* (Oral communication).
- [i17] **Travadon R**, Sache I, Marquer B, Ribulé A, Delourme R, Brun H, Masson JP, Dutech C, Bousset L (2008). Facteurs épidémiologiques contribuant à l'adaptation des populations de *Leptosphaeria maculans* aux gènes de résistance spécifiques de *Brassica napus* : dispersion des pycniospores et des ascospores et progression systémique du champignon. *11e Journées du réseau biotechnologies végétales et améliorations des plantes de l'agence Universitaire de la Francophonie - Biotech2008 "Biotechnologies végétales et gestion durable des résistances face à des stress biotiques et abiotiques"*. Rennes, France. (Oral communication).
- [i16] **Travadon R**, Sache I, Marquer B, Ribulé A, Delourme R, Dutech C, Bousset L (2008). Epidemiological factors contributing to the adaptation of *Leptosphaeria maculans* populations to specific resistance genes in *Brassica napus*: Access to the site of sexual reproduction and ascospore dispersal. *5th ISHS International Symposium on Brassicas and 16th Crucifer Genetics Workshop Lillehammer, Norvège*. (Oral communication).

- [i15] Lô-Pelzer E, Boillot M, Aubertot JN, Pinochet X, Jeuffroy MH, Salam MU, Bousset L (2008). SIPPOM-WOSR, a Simulator for Integrated Pathogen POPulation Management, to Help Design Sustainable Strategies to Control Phoma Stem Canker on Winter OilSeed Rape. *5th ISHS International Symposium on Brassicas and 16th Crucifer Genetics Workshop Lillehammer, Norvège*. (Poster).
- [i14] Boillot M, Sauzet G, Pic E, Carpezat J, Pelzer E, Bousset L, Aubertot JN, Pinochet (2008). Méthodologie de gestion durable du phoma du colza. *11e Journées du réseau biotechnologies végétales et améliorations des plantes de l'agence Universitaire de la Francophonie - Biotech2008 "Biotechnologies végétales et gestion durable des résistances face à des stress biotiques et abiotiques"*. Rennes, France. (Poster).
- [i13] Bousset L, Chèvre AM, Desquilbet M, Aubertot JN, Sache I, Chauvin JE (2008). Comment exploiter durablement les résistances variétales ? *11e Journées du réseau biotechnologies végétales et améliorations des plantes de l'agence Universitaire de la Francophonie - Biotech2008 "Biotechnologies végétales et gestion durable des résistances face à des stress biotiques et abiotiques"*. Rennes, France. (Poster).
- [i12] Lô-Pelzer E, Aubertot JN, Bousset L, Salam MU, Jeuffroy MH (2008). Mise au point de stratégies de gestion durable des résistances du colza au phoma : SIPPOM-WOSR, un modèle pour hiérarchiser des systèmes de culture combinant lutte génétique, culturale et chimique à l'échelle régionale et pluriannuelle. *11e Journées du réseau biotechnologies végétales et améliorations des plantes de l'agence Universitaire de la Francophonie - Biotech2008 "Biotechnologies végétales et gestion durable des résistances face à des stress biotiques et abiotiques"*. Rennes, France. (Poster).
- [i11] Lô-Pelzer E, Aubertot JN, Bousset L, Salam MU, Jeuffroy MH (2008). SIPPOM-WOSR: a Simulator for Integrated Pathogen Population Management to Design Control Strategies against Phoma Stem Canker on Winter Oilseed Rape, Maintaining the Efficiency of Specific Resistances. *10th congress of the European Society of Agronomy. Bologne, Italie*. (Poster).
- [i10] Travadon R, Marquer B, Ribulé A, Sache I, Brun H, Delourme R, Bousset L (2008). Systemic progress of *Leptosphaeria maculans* from cotyledons to the stem of *Brassica napus*. *9th International Congress of Plant Pathology, Torino, Italie*. (Poster).
- [i9] Evans N, Fitt BDL, van den Bosch F, Eckert M, Huang Y-J, Eckert M, Pietravalle S, Demon I, Papastamati K, Karolewski Z, Rouxel T, Balesdent M-H, Ross S, Fudal I, Gout L, Brun H, Andrivon D, Bousset L, Besnard AL, Gladders P, Pinochet X, Penaud A, Jedryczka M, Kachlicki P, Irzykowski W, Stachowiak A, Olechnowicz J, Podlesna A, Happstadius I, Renard M, (2007). The SECURE project - Stem canker of oilseed rape: molecular tools and mathematical modelling to deploy durable resistance. *12th International Rapeseed Congress, 26-30 March 2007, Wuhan, Chine. p187* (Poster)
- [i8] Lô-Pelzer E, Aubertot JN, Bousset L, Jeuffroy MH, Salam MU (2007). Modelling for durable resistance of canola to blackleg: combining management strategies for a collective disease control at a regional scale. *APS-SON joint meeting. San Diego, USA Phytopathology 97 p S91* (Oral communication).
- [i7] Travadon R, Bousset L, Saint-Jean S, Brun H, Sache I, (2007) Rain-splash is an effective mechanism of dispersal of blackleg (*Leptosphaeria maculans*) pycnidiospores. *12th International Rapeseed Congress, 26-30 March 2007, Wuhan, Chine. p154* (Poster)
- [i6] Evans N, Fitt BDL, van den Bosch F, Eckert M, Huang Y-J, Pietravalle S, Rouxel T, Balesdent M-H, Ross S, Gout L, Brun H, Andrivon D, Bousset L, Galdders P, Pinochet X, Penaud A, Jedryczka M, Kachlicki P, Stachowiak A, Olechnowicz J, Happstadius I. (2004). Stem canker of oilseed rape: molecular tools and mathematical modelling to deploy durable resistance. *Proceedings of the BCPC "Crop Science and Technology 2004" Seminar series, SECC, Glasgow, 1-3 November 2004*
- [i5] Evans N, Fitt BDL, van den Bosch F, Eckert M, Huang Y-J, Pietravalle S, Karolewski Z, Rouxel T, Balesdent M-H, Ross S, Gout L, Brun H, Andrivon D, Bousset L, Galdders P, Pinochet X, Penaud A, Jedryczka M, Kachlicki P, Stachowiak A, Olechnowicz J, Podlesna A, Happstadius I, Meyer J, Renard M (2004). Stem canker of oilseed rape: molecular tools and mathematical modelling to deploy durable resistance. *Proceedings of the 7th Conference of the European Foundation for Plant Pathology & British Society for Plant Pathology Presidential meeting Meeting 2004, 5-10 September 2004*.
- [i4] Evans N, Fitt BDL, van den Bosch F, Eckert M, Huang Y-J, Pietravalle S, Karolewski Z, Rouxel T, Balesdent M-H, Ross S, Gout L, Brun H, Andrivon D, Bousset L, Galdders P, Pinochet X, Penaud A, Jedryczka M, Kachlicki P, Stachowiak A, Olechnowicz J, Podlesna A, Happstadius I, Meyer J, Renard M (2003). Stem canker of oilseed rape: molecular tools and mathematical modelling to deploy durable resistance. *Proceedings of British Society for Plant Pathology Presidential Meeting 2003, 15-18 December 2003*.
- [i3] Bousset L, Østergård H, Hovmøller MS, Caffier V (1999). Population dynamics in barley powdery mildew. *Workshop on disease resistance and cereal leaf pathogens beyond the year 2000. Martina Franca (Italie), 11-12 Novembre 1999*. (Oral communication).

- [i2] **Bousset L**, de Vallavieille-Pope C (1999). Founder effect and selective advantage of a barley powdery mildew genotype in a field. *The first international powdery mildew conference. Avignon, 29 août-2 septembre 1999*. (Oral communication).
- [i1] **Bousset L**, Blaise P, Kellerhals M, Gessler C (1996). Mixtures of apple cultivars in orchards: effect on the scab epidemics and comparison to computer simulations. *4th Workshop on Integrated Control of Pome Fruit Disease. Croydon, England, August 19-23 1996*. (Oral communication).

National conferences:

- [n19] **Bousset L**, Jumel S, Garreta V, Picault H, Soubeyrand S(2014). Transmission de *Leptosphaeria maculans* entre parcelles de saisons culturales successives. *Journées Jean Chevaugeon, Aussois, 27-31 janvier 2014*. (Oral communication).
- [n18] **Bousset L**, Chèvre AM (2012). Adaptation des agents pathogènes aux résistances des plantes : un cadre conceptuel interdisciplinaire pour stabiliser la maîtrise des épidémies sur les cultures à partir des principes évolutifs. *Rencontre annuelle du REID, Rennes, 6-8 février 2012*. (Oral communication).
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