



VetAgro Sup



Colloque Santé-Biodiversité 27&28 octobre 2014 à VetAgro Sup

Relations biodiversité
microbienne-devenir des
pathogènes dans
l'environnement: de la
description à la
compréhension/prédiction

Xavier Le Roux

DR1 INRA

*Laboratoire d'Ecologie Microbienne
(UMR5557 Lyon1-CNRS/INRA/ENVL)*

Coordinateur de BiodivERsA

Introduction:

- Je ne suis pas un spécialiste des relations biodiversité – santé!
- Je suis un écologue travaillant sur les relations biodiversité-fonctionnement et services écosystémiques

Straw men don't get Lyme diseases to Wood and Lafferty

Richard S. Ostfeld¹ and Felicia Keesing^{1,2}

¹ Cary Institute of Ecosystem Studies, Box AB, Millbrook, NY 12545, USA
² Biology Program, Bard College, Annandale-on-Hudson, NY 12504, USA

Wood and Lafferty [1] (hereafter WL) attempt a synthesis of two views of the ecological factors underlying variable Lyme disease (LD) risk. LD emerged during the 1970s following the post-agricultural reforestation of the north-eastern USA, which provided the habitat required by the blacklegged tick vector (*Ixodes scapularis*) and many of its hosts [2]. However, within the large and growing North American LD zone, risk and incidence vary enormously. To explain LD risk, WL contrast a 'traditional' perspective, in which forestation is associated with high risk, and a 'dilution effect' perspective, in which loss of vertebrate diversity is associated with high risk. Unfortunately, this dialectic confuses the objectives of each perspective and distorts relevant evidence.

WL conflate 'forestation' and 'biodiversity', epitomized by their repeated use of the term 'forestation and/or biodiversity' ([1] pp. 240 and 244). Although forest is required for blacklegged tick populations, host diversity within these forests and associated landscape varies dramatically. Reforestation during the 20th century of agricultural land has been linked to LD emergence, but more recent forest fragmentation has been linked to increased LD risk (reviewed in [2]).

WL engage in fallacious reasoning, arguing that, because LD would disappear if all biodiversity were eliminated, increasing biodiversity amplifies LD. Indeed, their characterization of the 'traditional' approach leads them to the untenable position that the most effective means of reducing LD risk is to deforest the landscape, an option that they find 'unadvisable' ([1] p. 246). It also leads them to state ([1] p. 246) that, 'most evidence currently available points to a monotonic increase in disease risk with increasing biodiversity', a statement utterly devoid of support (and unneeded). WL are critical of the 'dilution effect' perspective, contending that it is part of a growing effort to market conservation actions based on the utilitarian services that biodiversity can provide for human society' ([1] p. 246). We disagree that efforts to use scientific understanding to inform policy should be considered 'marketing'.

WL's discomfort with the dilution effect stems from a series of mischaracterizations. First, they contend that the dilution effect 'is premised on the unreasonable belief that biodiversity must always benefit human society' ([1] p. 243). On the contrary, the dilution effect literature clearly shows that biodiversity can either dilute or amplify disease risk, specifying the conditions under which each would be

expected [3]. Furthermore, it is strong/universal to abundant/remain in a stable/decline little a Monhe [8], so adult/island comp relat or a rich arg me is com ric a in is a t

Ostfeld and Keesing's rebuttal [1] to our published review [2] does not question our overall synthesis that Lyme disease (LD) transmission is a complex balance between dilution and amplification. Ostfeld and Keesing do rebut some details, critique conclusions by authors cited in our review, question whether deer are important hosts for deer selves introduced into the literature (quoting biodiversity with forestation). Ostfeld and Keesing confuse 'reductio ad absurdum reasoning' with a deceptive 'straw man'. The consideration of extreme end points, such as zero biodiversity (our *reductio ad absurdum* reasoning), is common when making theoretical predictions. Because there will be no zoonotic disease transmission when biodiversity declines to zero, the relationship between biodiversity and zoonotic disease risk must pass through the origin, leading to positive, positive asymptotic, or hump-shaped associations between biodiversity and disease. Therefore, a disease can never be the whole story. This leads to the core types – from urban lands to pristine forest – risk of LD must first rise as the extent of forestation increases and then, within forested habitat, might fall with increasing vertebrate biodiversity, depending on the biological details.

Given how often researchers repeat the claim by Ostfeld and Keesing that 'current evidence that high diversity dilutes far more often than it amplifies, at scales from local to global, is strong' [3–5], we shift our focus to the strength and generality of the dilution effect beyond LD.

Corresponding authors: Lafferty, KD (kdlafferty@stanford.edu); Wood, CL (clwood@stanford.edu)

References

1. Wood, C.L. and Lafferty, K. (2013) Biodiversity and disease: a synthesis of ecological perspectives on Lyme disease transmission. *Trends Ecol. Evol.* 28, 269–287.

© 2013 The Authors. Journal compilation © 2013 British Ecological Society,
any medium, provided the original work is properly cited.

Letters

2. Ostfeld, R.S. (2013) *Lyme Disease: The Ecology of a Complex System*. Oxford University Press.
3. Ostfeld, R.S. and Keesing, F. (2000) Biodiversity and disease risk: the case of Lyme disease. *Conserv. Biol.* 14, 722–728.
4. Keesing, F. et al. (2006) Effects of species diversity on disease risk. *Ecol. Lett.* 9, 489–498.
5. Ostfeld, R.S. and Keesing, F. (2012) Effects of host diversity on infectious disease. *Annu. Rev. Ecol. Syst.* 43, 187–192.
6. Cardinale, B.J. et al. (2012) Biodiversity loss and its impact on terrestrial gradient in income. *PLoS Biol.* 10, e1001456.
7. Bonds, M.H. et al. (2013) Disease ecology, biodiversity, and the latitudinal gradient in income. *PLoS Biol.* 10, e1001456.
8. Rand, P.W. et al. (2004) Abundance of *Ixodes scapularis* (Acari: Ixodidae) after the complete removal of deer from an isolated offshore island, endemic for Lyme disease. *J. Med. Entomol.* 41, 779–784.

9. Levi, T. et al. (2012) Deer, predation, and the emergence of Lyme disease. *Proc. Natl. Acad. Sci. U.S.A.* 109, 10943–10947.
10. LaDalle, K. et al. (2012) The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proc. Natl. Acad. Sci. U.S.A.* 109, 8472–8477.
11. Ostfeld, R.S. and LoGiudice, K. (2003) Community disequilibrium, biodiversity loss, and the erosion of an ecosystem service. *Ecology* 84, 1421–1427.
12. Ostfeld, R.S. et al. (2009) Biodiversity and ecosystem function: perspectives on disease. In *Biodiversity, Ecosystem Function, and Human Well-Being: An Ecological and Economic Perspective* (Naeem, S. et al., eds), pp. 203–216. Oxford University Press.

© 2013 The Authors. Journal compilation © 2013 British Ecological Society,
any medium, provided the original work is properly cited.



It's a myth that protection against disease is a strong and general service of biodiversity conservation: Response to Ostfeld and Keesing

Kevin D. Lafferty¹ and Chelsea L. Wood²

¹ US Geological Survey, Western Ecological Research Center, c/o Marine Science Institute, University of California, Santa Barbara, CA 93106, USA
² Department of Biology, Stanford University, Stanford, CA 94305, USA

Westart by examining the three papers Ostfeld and Keesing cite for their conclusion that diversity dilutes infectious diseases more often than it amplifies them: Cardinale et al. [6], Bonds et al. [3], and Ostfeld and Keesing [4]. Here, we show that these authors provide inadequate, limited, or opposing evidence for the claim by Ostfeld and Keesing.

Although Cardinale et al. did find a general negative association between plant diversity and plant pathogens in their quantitative review [6], they observed that 'evidence on the effect of plant diversity on pest abundance is also mixed, with four available data syntheses showing different prevalence of animal disease is mixed, despite recent claims [5] that biodiversity generally suppresses disease'. In other words, Cardinale et al. are critical [6], not supportive, of the claim by Ostfeld and Keesing.

In a fascinating study on the feedback between ecosystems and disease, Bonds et al. observed a negative association between biodiversity and human infectious disease, but this residual effect emerged only after controlling for major factors that affect biodiversity in the first place (e.g., latitude, tropical vs temperate region) [3]. A more relevant point is that the raw data obtained by Bonds et al. show that disease prevalence is much higher in areas with high biodiversity, as other studies have found [7]. This broader-scale pattern directly contradicts the claim by Ostfeld and Keesing.

The third citation by Ostfeld and Keesing [4] discusses case studies with evidence of a dilution effect. This derives from a study by Keesing et al. [8], who listed 12 example diseases for which one or more studies claim that biodiversity loss can increase transmission. Keesing et al. conclude that their case studies represent most disease

Hypotheses to be tested with why with (Murray et al.,

Biodiversity and disease: a synthesis of ecological perspectives on disease transmission

Chelsea L. Wood¹ and Kevin D. Lafferty²

¹ Department of Biology, Stanford University, Stanford, CA 94305, USA
² US Geological Survey, Western Ecological Research Center, c/o Marine Science Institute, University of California, Santa Barbara, CA 93106, USA

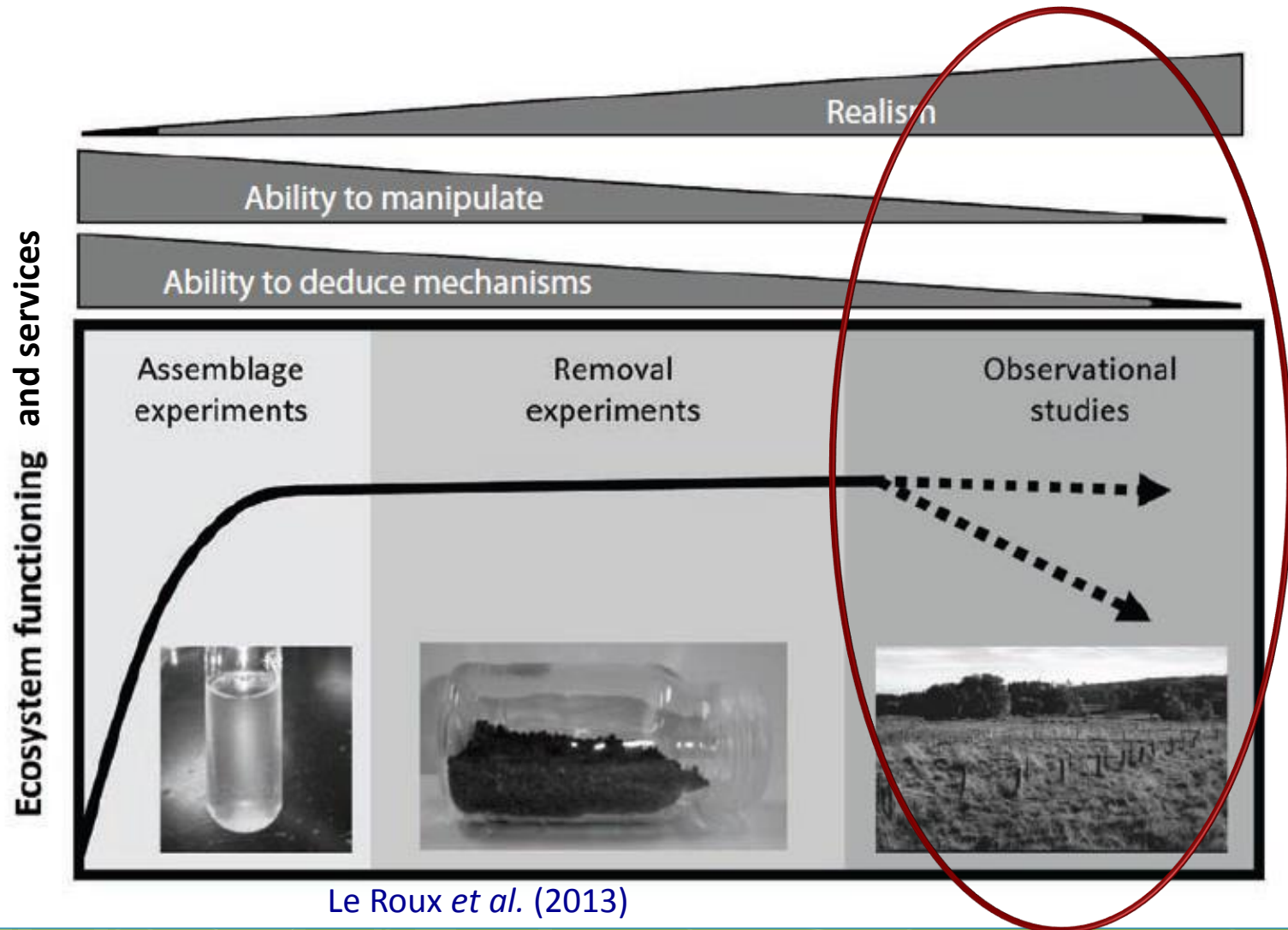
Recent reviews have argued that disease control is among the ecosystem services yielded by biodiversity. Lyme disease (LD) is commonly cited as the best example of the 'dilution' effect of biodiversity on disease transmission, but many studies document the opposite relationship, showing that human LD risk can increase with forestation. Here, we unify these divergent perspectives and find strong evidence for a positive link between biodiversity and LD at broad spatial scales (urban to suburban to rural) and broad temporal scales (from the early 20th century to the present). This finding suggests that biodiversity within forests, this finding suggests that biodiversity within forests, the biodiversity-disease relationship is scale dependent and complex.

Can biodiversity protect humans against disease? Does anthropogenic biodiversity loss generally increase or decrease zoonotic disease transmission? This is a contentious question in disease ecology, and its answer is not only of theoretical interest, but also reveals whether biodiversity conservation could be deployed as an effective approach for disease control. LD has become a major focus in this controversy. Understanding how to interrupt transmission of LD (which is caused by the spirochete pathogen *Borrelia burgdorferi* and vectored by ticks) is important because the disease affects thousands of people in the northeastern USA and, if not treated quickly, can lead to serious health complications [1]. However, published studies differ considerably in their estimation of the most effective approach for LD control. Early work on the ecology of LD focused on the relationship between forestation and land use: epidemiological studies found positive spatial correlations between forested land and human LD cases (e.g., [2–10]), and proposed clearing of vegetation, controlled burns, and culling of reservoir hosts as the most effective ways to reduce transmission [11,12]. Later, the 'dilution effect' hypothesis emerged, which proposed that, by limiting the relative abundance of highly competent reservoir hosts, biodiversity ecosystems could 'buffer' LD transmission [13–19]. This body of work led to calls for the use of forest conservation as a tool for reducing human LD risk [15,19,20], as well as the risk

LD transmission: The complex The pathogen is in the forest and in the mouse (P. most prod (Odom's nymphal mounds, and the

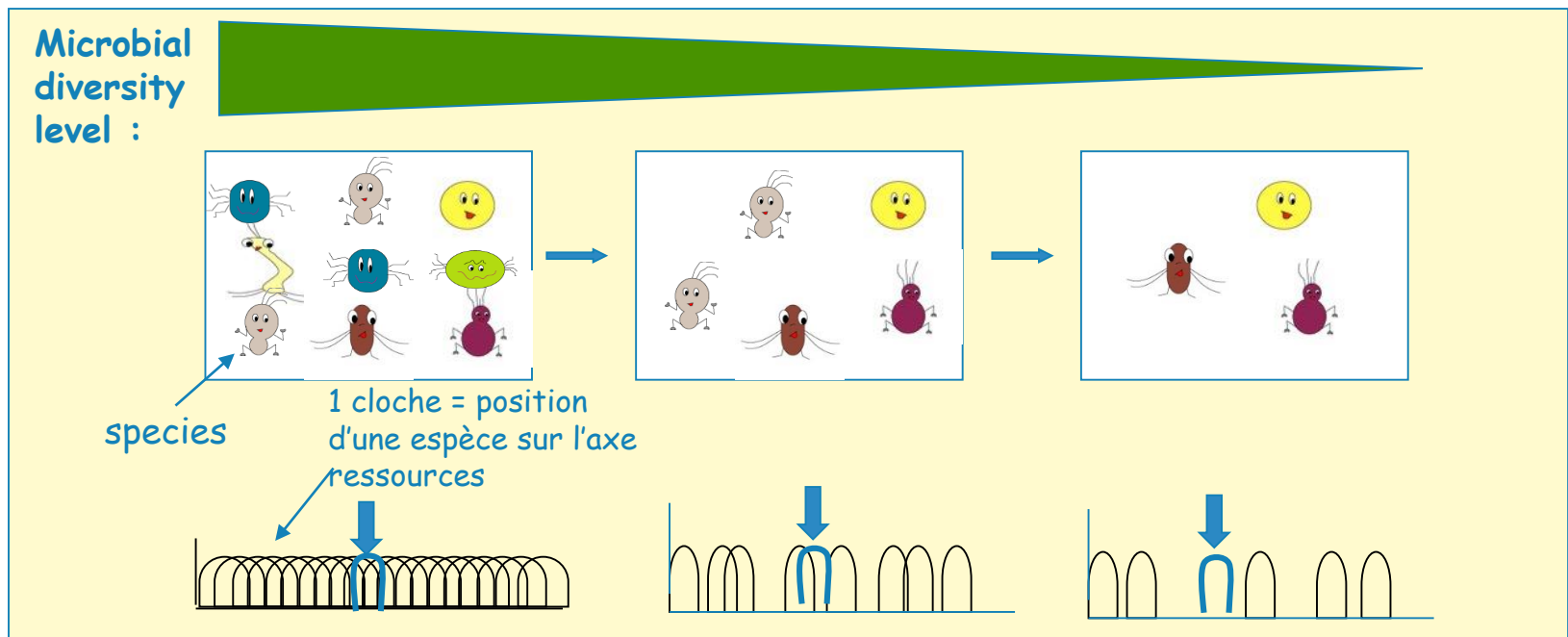
Corresponding authors: Ostfeld, R.S. (rostfeld@caryinstitute.org); Keesing, F. (keesing@caryinstitute.org)

La plupart des études restent descriptives et corrélatives
Peu de manipulation de la biodiversité
Encore moins de démonstration des mécanismes



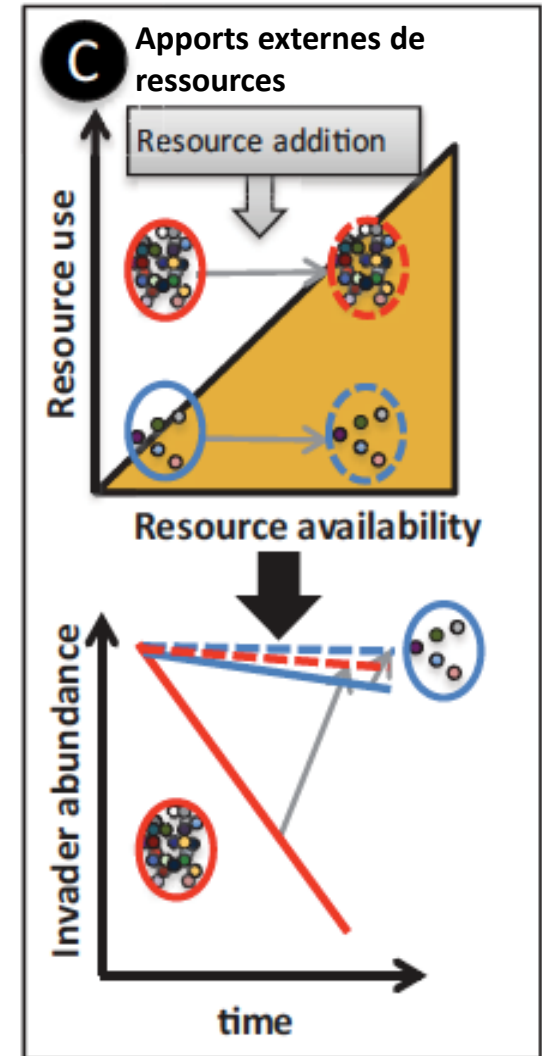
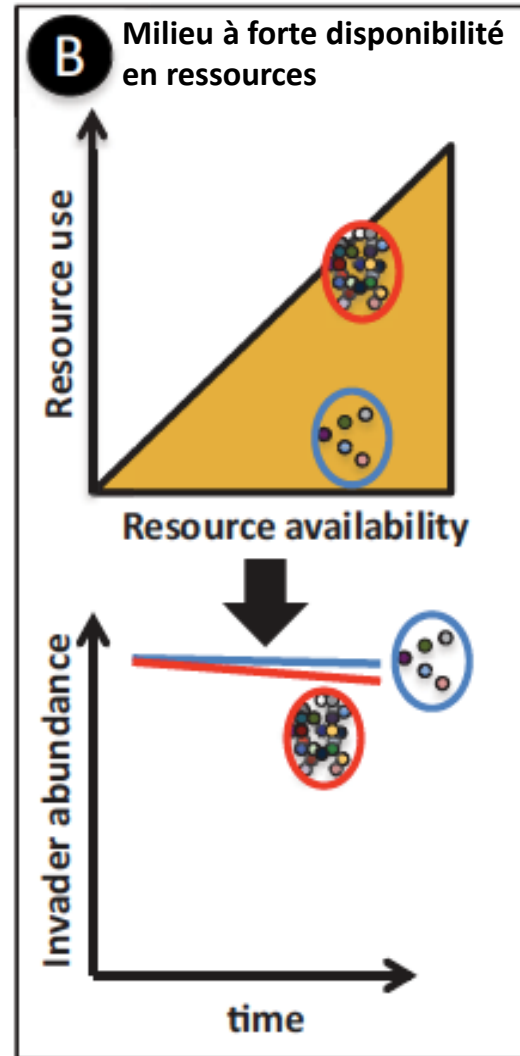
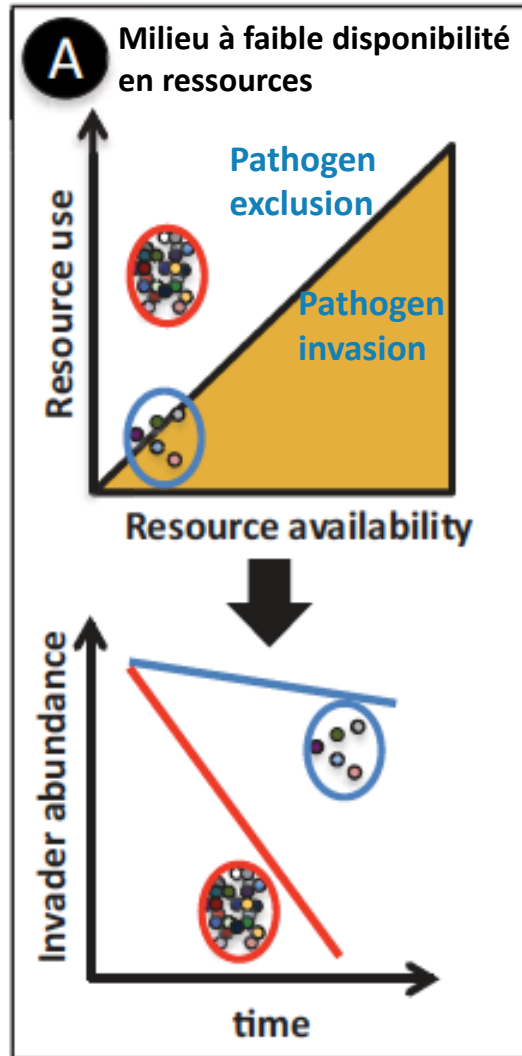
Ici, une question: peut-on comprendre et prédire les relations entre biodiversité & devenir d'un pathogène dans l'environnement?

- Test de l'hypothèse d'exclusion compétitive de pathogènes potentiellement invasifs par la communauté microbienne en place



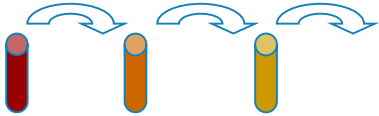
- Prise en compte de la disponibilité en ressources dans le milieu

Prédiction de l'effet de la biodiversité microbienne et de l'effet ressources sur le devenir d'un pathogène



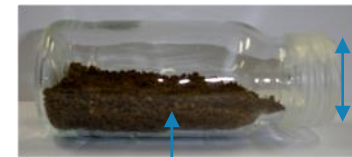
Méthodes: manipulation de la biodiversité microbienne (1)

1. Suspension/dilutions à partir d'un sol: perte des taxons les moins abondants

-  etc..
Soil serially diluted in water
(10 fold)

Inoculation →

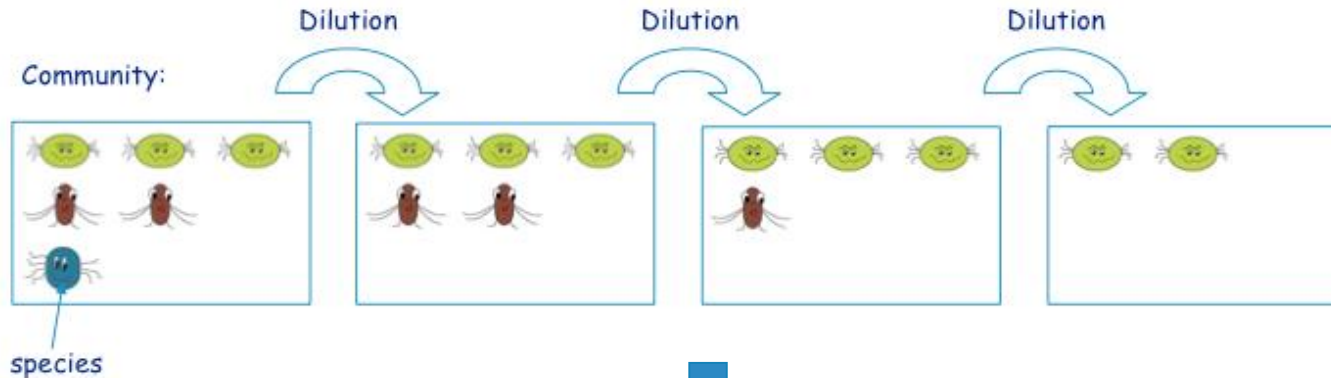
Sterile soil microcosms:



parafilm

γ-ray sterilized soil
30 g equivalent dry soil, 2 mm sieved
moisture : 50 % saturation

- Dilution levels imposed:
From 10^{-1} to 10^{-10} g non sterile soil g^{-1} sterilized soil



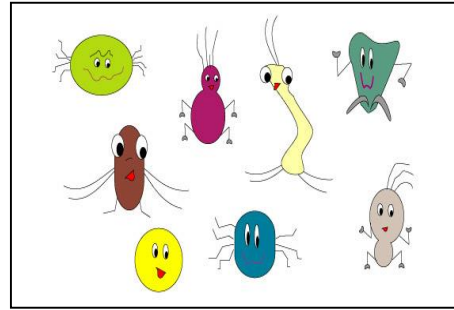
2. Colonisation → même abondance pour chaque cas ; puis apport d'*E. coli*

3. Devenir d'*E. coli* en fonction de la diversité de la communauté en place ?

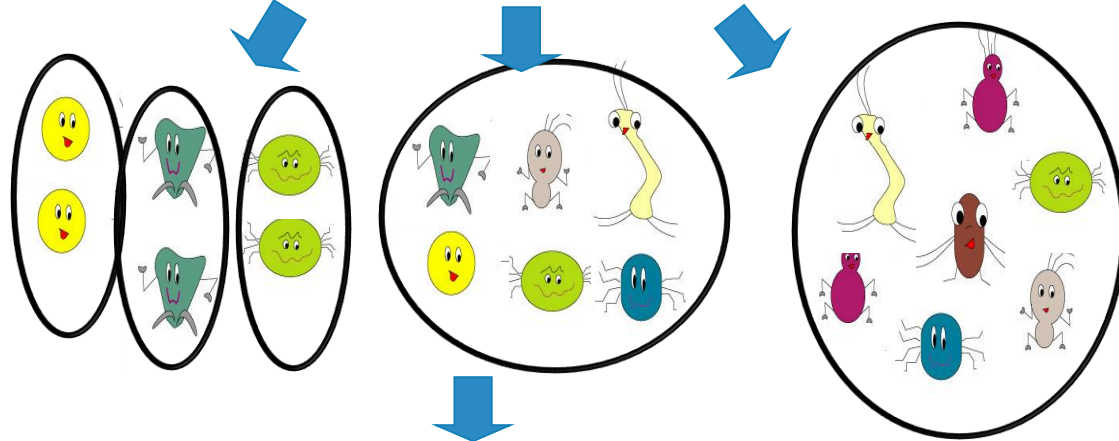
Méthodes: manipulation de la biodiversité microbienne (2)

1. Inoculation de sol avec des communautés bactériennes assemblées à partir de souches

Pool de souches
bactériennes du sol



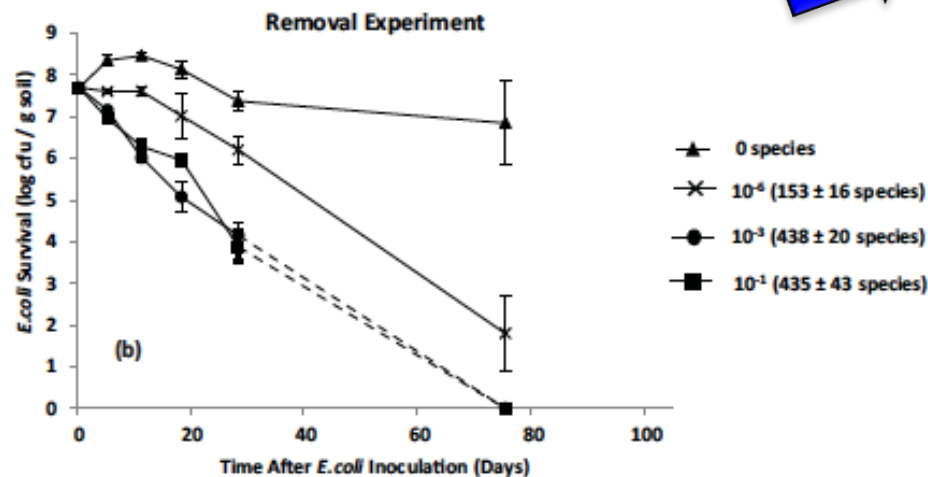
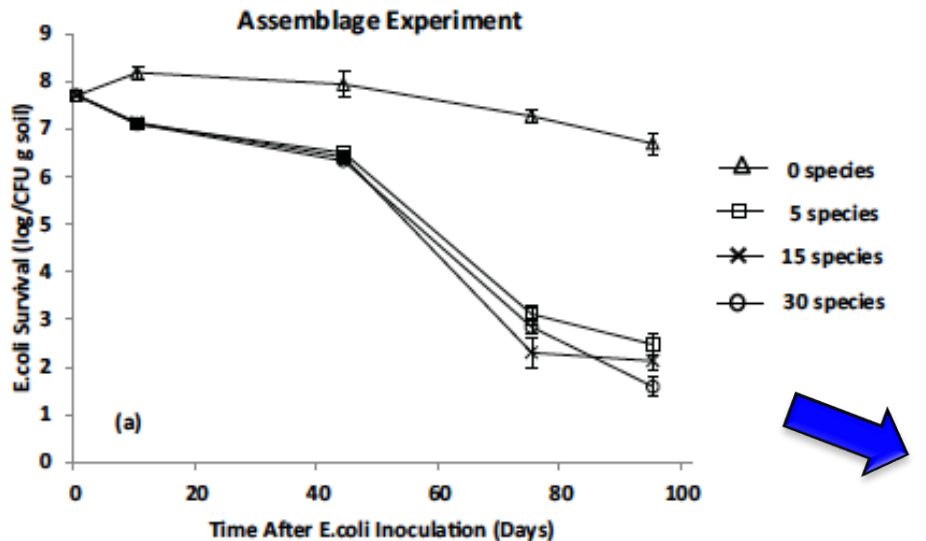
Création de
communautés
bactériennes
contrastées en
terme de diversité



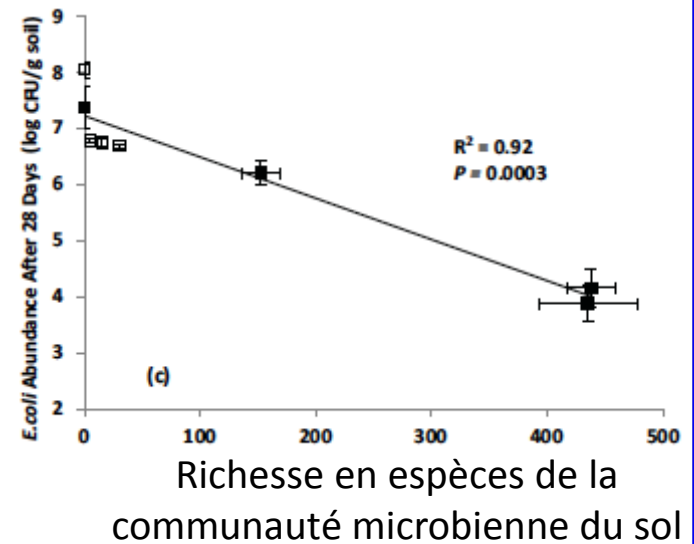
2. Colonisation du sol → même abondance pour chaque cas ;
puis apport d'*E. coli*

3. Devenir d'*E. coli* en fonction de la diversité de la communauté en place ?

Résultats: devenir de *E. coli* en fonction de la biodiversité de la communauté en place



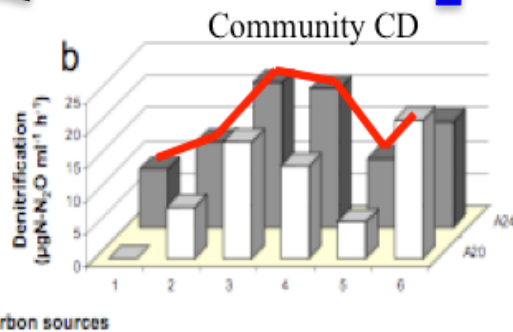
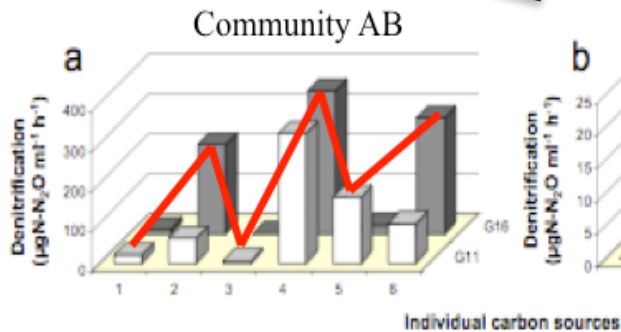
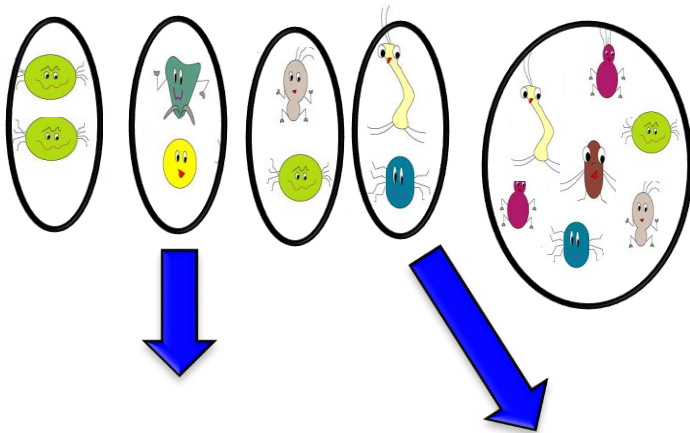
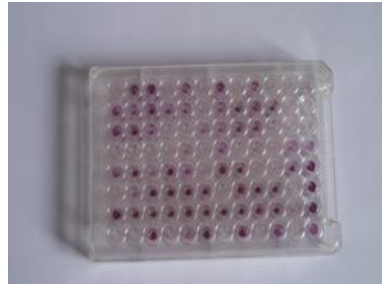
Niveau de maintien
de *E. coli* dans le sol



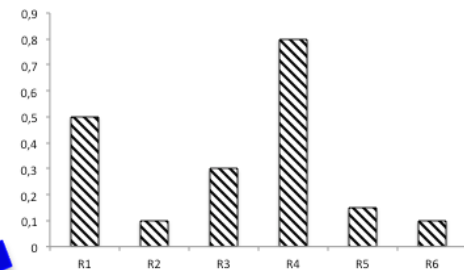
Grande diversité de la
communauté en place
➔ difficulté du
pathogène à s'implanter

Peut-on passer de la description à la compréhension de ces relations?

Caractérisation de la niche écologique de chaque communauté en place

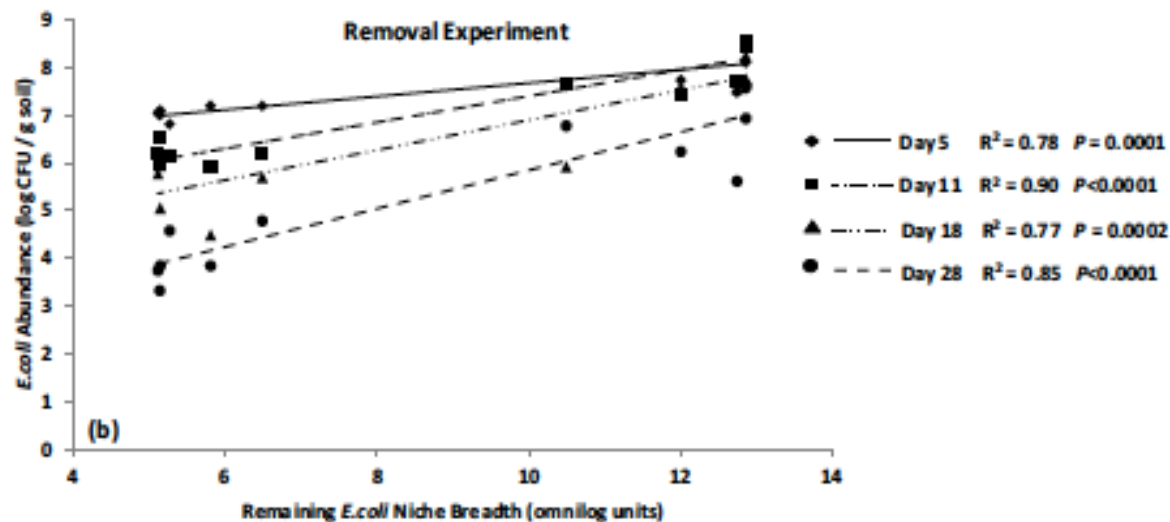
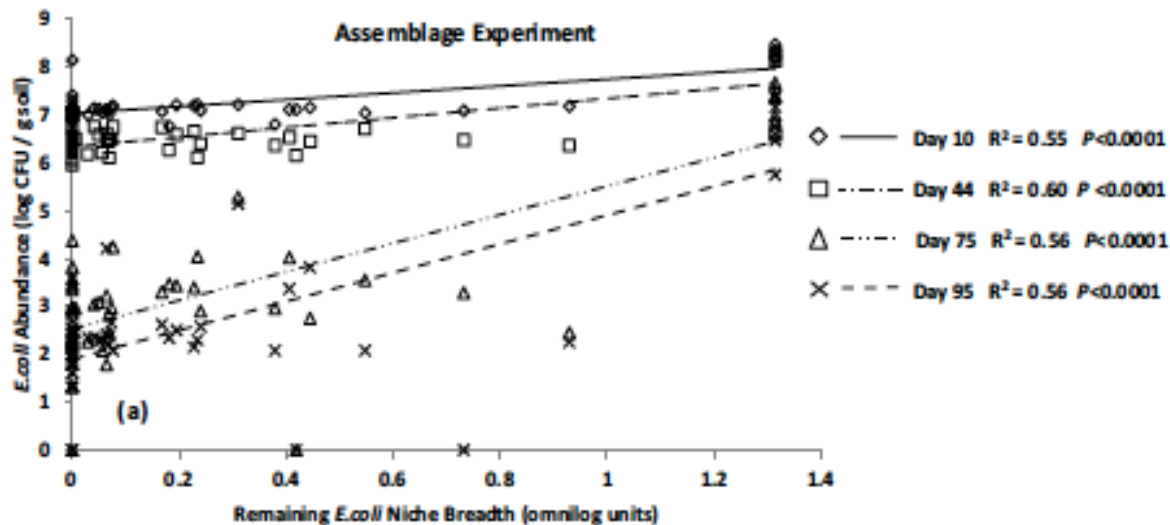


Caractérisation de la niche écologique de *E. coli*



Fraction de la niche d'*E. coli* non déjà occupée par la communauté en place
Niche effective restant pour *E. coli* = bon prédicteur de sa capacité à se maintenir?

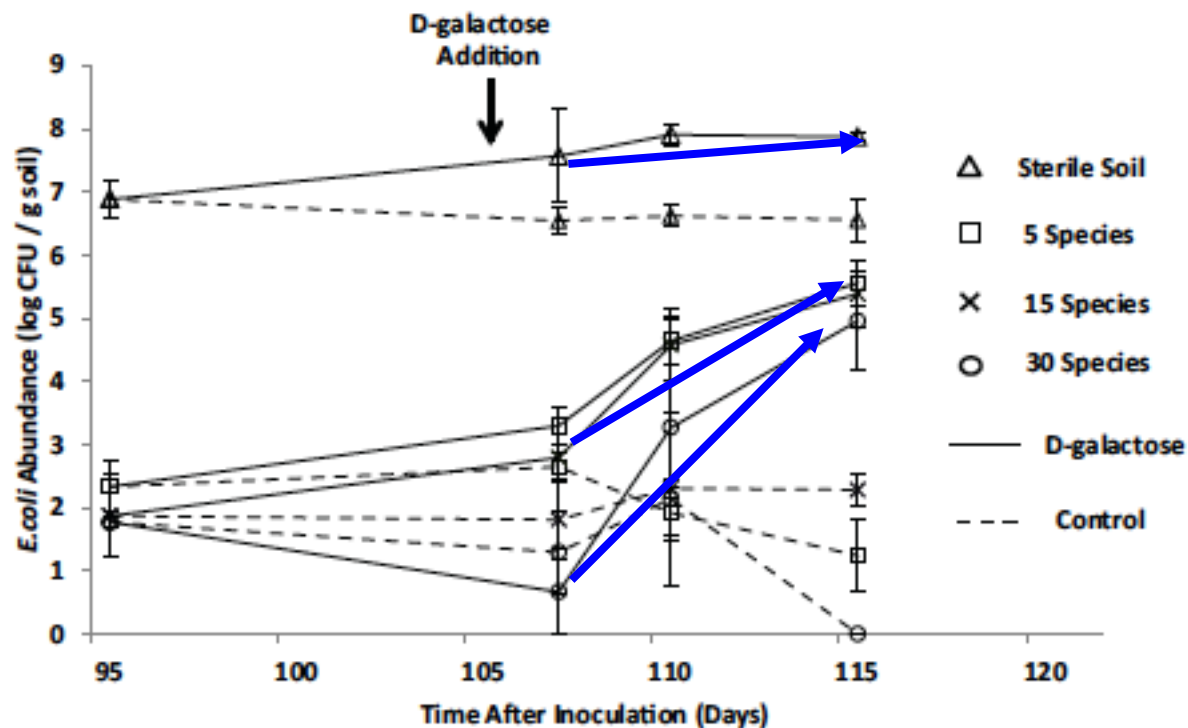
Résultats: devenir de *E. coli* en fonction de la biodiversité de la communauté en place



Niche effective restant à *E. coli*

Grande diversité de la communauté en place
 → niche effective restant à *E. coli* réduite
 → difficulté du pathogène à s'implanter

Résultats: effet direct d'un apport de ressources (ici C organique) sur le devenir de *E. coli* en fonction de la biodiversité de la communauté en place



Capacité du pathogène à se maintenir...
... même face à une communauté diverse ...
... si des ressources sont amenées

Title: Ecological niches of an invader and the invaded communities determine the invasion success in a soil microbial ecosystem

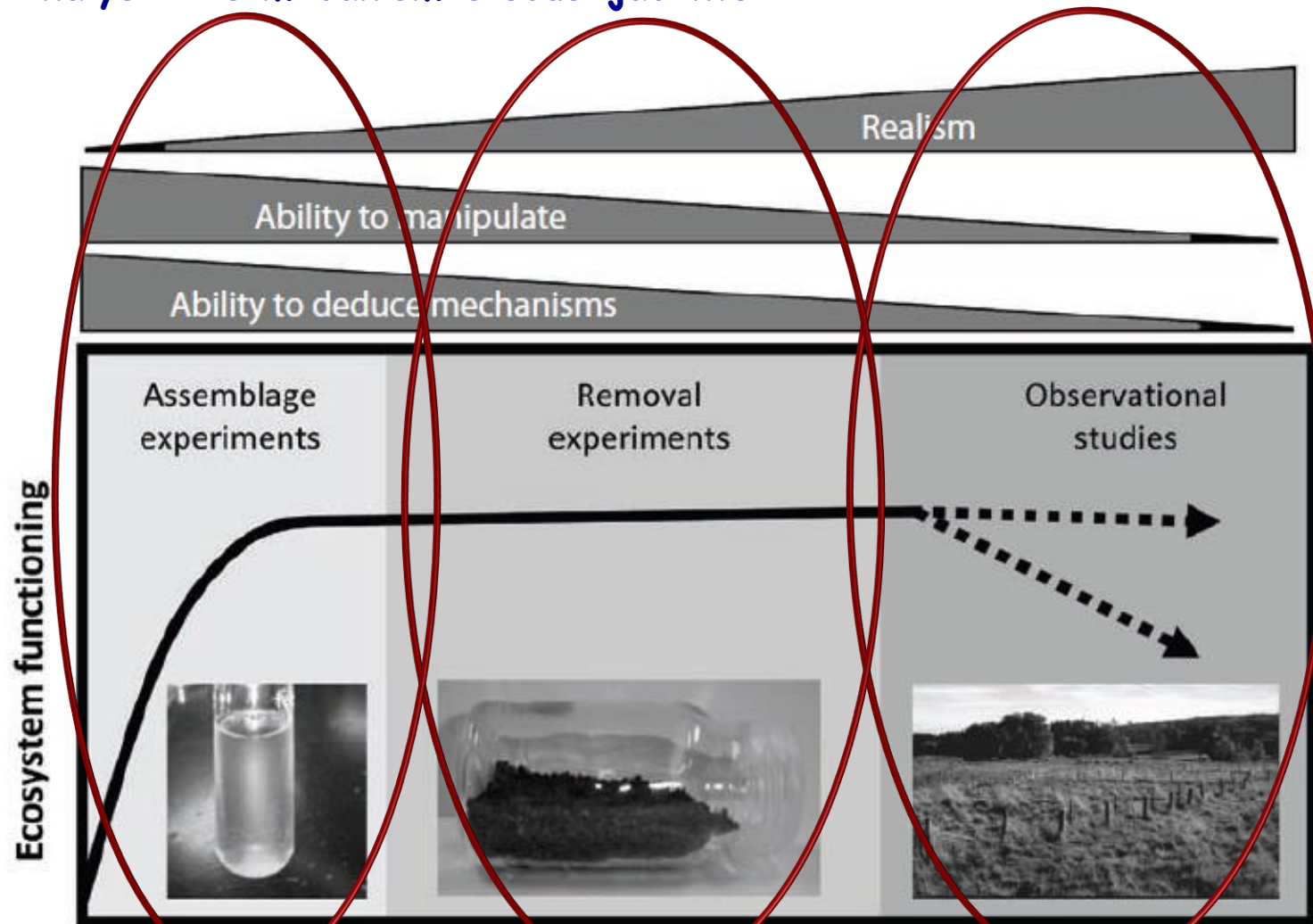
Authors: Cyrus A. Mallon¹, Franck Poly², Xavier Le Roux², Irene Marring¹, J.D. van Elsas¹, and Joana F. Salles¹

¹Department of Microbial Ecology, Center for Ecological and Evolutionary Studies, University of Groningen, Nijenborg 7, 9747 AG, Groningen, the Netherlands ²Université de Lyon, INRA, CNRS, Université Lyon 1, Laboratory of Microbial Ecology (UMR 5557 CNRS, USC 1364 INRA), 43 blvd du 11 Novembre 1918, 69622, Villeurbanne, France.

Ecology (in press)

Apport de l'écologie aux études biodiversité microbienne-santé

- Approches directes de manipulation expérimentale de la biodiversité
- Analyse des mécanismes sous-jacents



Le Roux et al. (2013)