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Demographic Bifurcations and Hysteresis in Ecological LCA: Why Linear Dose-Effect Models Fail to Predict Colony Collapse and Eutrophication

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> *This article belongs to the research program "Waste as dynamical systems" (Piens, in preparation). Its sections are numbered independently; the few §x.y references not resolved in the text point to the companion articles, namely the theoretical Manifesto (axioms S1 to S4, theorems T1 to T4), the anaerobic digester, the toxicology of emerging contaminants, environmental logistics and public decision-making, or to the book-length manuscript.*

Abstract

Ecotoxicology and territorial LCA read a stressor's effect as a regularly rising dose-response, where less cause gives less effect, and this linearity governs no-effect doses and restoration plans. We apply critical-transitions theory (Scheffer) to three cases where the nonlinearity lies not in soil chemistry but in population *demography* and ecosystem *hysteresis*; in all three, reducing the cause does not reduce the effect in proportion, because the system holds several regimes and history decides. With neonicotinoids and bees, a sublethal dose impairs forager homing (return down by 10 to 32 %; Henry et al. 2012), and colony-level demographic feedback turns this graded effect into a *bifurcation*: doubling the dose flips a living hive to a dead one (S2). With lake eutrophication, two stable states, clear and turbid, are separated by *hysteresis*, and cutting phosphorus does not restore the lake at the tipping load because the stabilizing feedback keeps running (S3). With Agent Orange, a persistent lipophilic flux (TCDD) couples soil, groundwater, food chain and generations, and its unpredictability is structural, not a measurement gap. For such nested systems the costliest error is the silent assumption that path does not matter; in a world of regimes, going back is never the outbound path in reverse.

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1. Introduction

A linear assessment holds a reassuring promise: reduce the cause and you reduce the effect, in the same proportion. This article shows, on three ecological cases, that the promise is false as soon as the system holds several regimes. The nonlinearity lies there not in the chemistry of a soil but in the demography of a population, the hysteresis of a lake, the multi-scale coupling of a persistent compound. We mobilize critical-transitions theory (Scheffer et al. 2001; Scheffer 2009) and two axioms of static evaluation, linear characterization (S2) and path independence (S3), established in the companion manifesto. The early-warning signals, the constructive counterpart of these tipping points, are treated in the companion article on the anaerobic digester, to which we refer.

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2. Neonicotinoids and bees: a population bifurcation, colony collapse (Henry et al. 2012)

Here is a case where non-linearity lies not in the chemistry of a soil but in the demography of a population. A sublethal dose of thiamethoxam (a neonicotinoid insecticide) does not kill the bee; it impairs its navigation. The forager leaves, forages, and does not find the hive again. Henry et al. (2012) measured this effect directly: depending on the concentration, forager homing success falls by 10 to 32 %. At the scale of the individual, the effect is graded, almost innocuous.

At the scale of the colony, it is not. A hive is a demographic system endowed with a feedback: the foragers bring back the resources that allow tomorrow's foragers to be raised. As long as the loss stays small, the colony compensates. But there exists a threshold (on the order of 30 to 50 % of cumulative losses over a season) beyond which the population no longer renews itself fast enough, and collapse sets in, irreversible within the season. This is not a dose-effect response that rises steadily; it is a *bifurcation*.

A minimal demographic model makes it visible. Starting from a forager population subject to a natural loss and to an additional dose-dependent loss, and placing the collapse threshold at 35 % cumulative losses, one obtains a clear behavior: at zero or low dose, the colony stays stable around its headcount; but when the dose crosses a critical value (a mere factor of two suffices in our units), it tips all at once toward complete collapse, the hive lost mid-season. To double the dose does not produce a twice-as-large effect: it shifts from a living colony to a dead one (Figure V6).

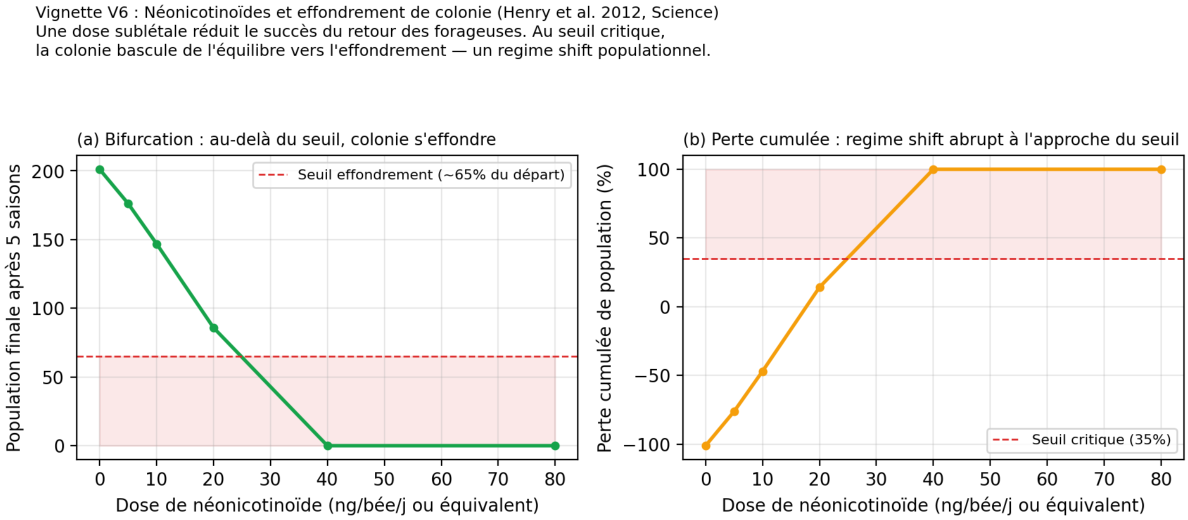

Figure V6. Neonicotinoids and bees: between a stable colony and a collapsed one, the switch plays out on a factor of two in dose. A regime change that the linear "dose → effect" response does not foresee.

This is the exact ecological analogue of the digester's bistability (§7.5): two regimes, the living colony and the extinct colony, an abrupt switch between them, and a linear "dose → effect" model unable to anticipate it, since it can only announce a proportional decline. The word "sublethal" is here the very trap. It reassures, because no dose kills; but it is their silent addition that, one day, crosses the line. One cannot sum small effects as if they stayed small: past the threshold, their sum does not grow, it changes in nature, and a hive still humming the day before is, the next day, only an empty box of wax.

Falsifiable. Follow colonies exposed to increasing doses and measure not individual mortality but the demographic trajectory of the hive: the regime reading predicts a tipping threshold, not a gradual decline.

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3. Eutrophication and lake bistability: hysteresis, insufficient P reduction (Scheffer 2001)

The last vignette is the purest demonstration of axiom S3 (path independence) caught at fault, and it comes from the ecology of lakes (Scheffer et al., 2001). A shallow lake can exist in two stable states. The *clear* state is dominated by rooted aquatic plants, which fix the sediments and sequester phosphorus; the water there is limpid. The *turbid* state is dominated by phytoplankton, which clouds the water, deprives the plants of light and makes them disappear; deprived of their anchorage, phosphorus is released from the sediments, which feeds the phytoplankton further. Each state sustains its own cause: they are two self-stabilized regimes.

As long as the phosphorus load stays low, the lake is clear. When it crosses a threshold, it tips abruptly toward the turbid. So far, just a threshold. The decisive point is what happens next: if one *reduces* the phosphorus load to repair, the lake does not become clear again at the same threshold. It stays turbid well below it, because the feedback that maintains it (turbidity, plant death, release of sedimentary phosphorus) keeps running. There is therefore a whole range of loads where both states are possible, and the one the lake actually occupies depends not on the present load, but on history: by which way one arrived. This is hysteresis. It has the shape of a stiff door: you push it, it gives way all at once; but to close it again, it is not enough to stop pushing, you must pull, and pull well past the point where it gave way. This is S3 defeated in the costliest way there is (Figure V8).

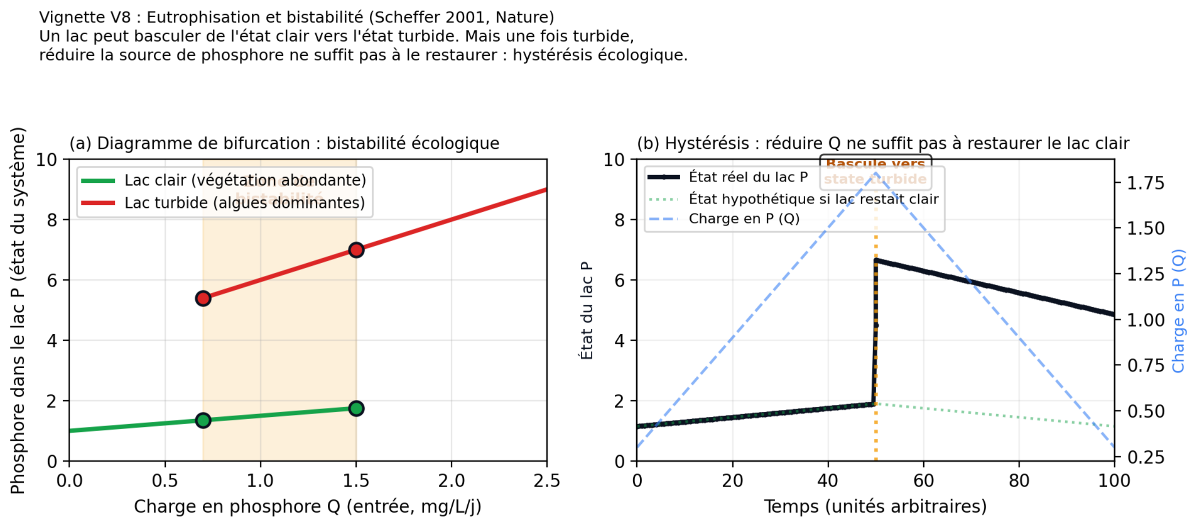

Figure V8. Hysteresis of eutrophication: as the phosphorus load is increased, the lake follows the clear branch then tips toward the turbid branch; as it is reduced, it stays turbid well below the tipping threshold. Between the two thresholds, the occupied state depends on history, not on the present load alone.

The practical consequence is bitter for whoever funds restoration plans. To reduce the phosphorus source (what a linear assessment naturally prescribes, where "less cause" gives "less effect") *does not suffice* to restore an already tipped lake. One must either reduce it drastically, well below the return threshold, or intervene on the very structure of the system: remove the sedimentary phosphorus, manipulate the trophic web. An LCA that had credited the phosphorus reduction with a proportional benefit would have erred not on the magnitude, but on the *nature* of what was at stake. This is, to close the series, the illustration that the costliest error is not in the number: it is in the silent assumption that the path does not count. To destroy an equilibrium costs a single crossing; to restore it costs several, and sometimes none suffices. This is perhaps the soberest lesson of this whole part: in a world of regimes, going back is never the outbound path traveled in reverse.

Falsifiable. On a real lake, compare the phosphorus load at which it tips toward the turbid with the one at which it returns to the clear: the regime reading predicts two distinct thresholds (hysteresis), the linear reading a single one.

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4. Nested systems and emergence

The nine vignettes are not nine independent anomalies; they are nine views of one and the same structure. A field is a system nested in a catchment, itself nested in a climate; the environmental logistics of §6 operates within these nestings; and the waste body is itself a sub-system with its own internal dynamics. This is the image described by hierarchy theory (Allen & Starr, 1982), panarchy (Gunderson & Holling, 2002) and ecosystems seen as complex adaptive systems (Levin, 1998). The lesson holds in one formula, Anderson's (1972): *more is different*, the behavior of the whole does not reduce to the linear sum of the parts. And it holds even when the waste is neither living nor self-reproducing: as soon as matter interacts with matter across these scales, exchanges, thresholds and regimes emerge that are read on none of the parts taken in isolation. The ontology of the static unit is, at bottom, only the linear-summation hypothesis (S2) applied to a hierarchy that, precisely, does not sum. The vignette that follows gives the most dramatic case of it.

4.1 Agent Orange: a flux that couples water, soil, air, life

No case shows this multi-scale coupling better than Agent Orange. Between 1962 and 1971, Operation Ranch Hand sprayed over Vietnam (and, by spillover, over Laos and Cambodia) on the order of 74 to 80 million liters of tactical herbicides, of which about 60 % was Agent Orange, an equal-parts mixture of 2,4-D and 2,4,5-T. The 2,4,5-T was contaminated by a synthesis by-product, the dioxin 2,3,7,8-TCDD, of which some 170 kg are estimated to have been disseminated (Stellman et al., 2003). These figures are not the object of the vignette; they fix its scale. The object is what this mass becomes once it has entered the soil–groundwater–life hierarchy.

And what it becomes illustrates, writ large, the prediction limit of the program. A few years after the spraying, the system stabilizes enough for an average residual contamination to be estimated; but in the meantime, and well beyond, unpredictability dominates, because TCDD does not obey a simple transport. It is a strongly *lipophilic* molecule: its octanol-water partition coefficient is about 6.9 in logarithm, its solubility in water is minute, and its elimination half-life in humans is on the order of seven to eight years (estimated on the Seveso cohort). It therefore does not dilute, it *concentrates*: in the fats of organisms, then along the trophic chain by biomagnification, so that the dose that counts is not the soil's but the one, multiplied, that climbs back up to the predator and to the human, sometimes into the next generation, via maternal fat reserves.

A point of chemistry is needed here, for field intuition mixes two mechanisms that must be distinguished. The first is the one just described: a lipophilic molecule flees water to lodge in fats, persists and accumulates there, this is TCDD, and it is already a source of unpredictability, for these reserves release slowly, long afterward. The second is more devious: certain persistent substances are at once *hydrophobic and lipophobic*, fleeing water *and* fat. This is the signature of the perfluorinated compounds, the PFAS of §7.1, and it is precisely the property sought in non-stick coatings: to repel water and oil at once. Now a molecule that neither water nor fat will host is a molecule the organism struggles to eliminate by either route, and which, heavy and stable, accumulates for lack of an exit. Both mechanisms lead to persistence in the living, but by opposite paths; to confuse them is to mistake the compartment. Rigor therefore requires separating the lipophilic route, that of the dioxins, from the amphiphobic route, that of the perfluorinated compounds, both defying the assumption of a simple fate, but not in the same way.

To this biological coupling is added a coupling with the groundwater that the static apparatus ignores just as much. A very hydrophobic molecule does not dissolve in groundwater; it associates with organic matter and, above all, with non-aqueous oily phases, a fuel remnant, a soil hydrocarbon. Lighter than water, these phases float above the water table and trap the contaminant there in a fringe, between earth and water. As long as the water table is stable, the stock seems dormant; but a rain, a seasonal rise, a fluctuation of the level brings the water back into contact with this fringe and remobilizes the contaminant (toward the surface or toward the aquifer) in jolts, years after deposition. This is a textbook case of path dependence (S3) doubled by chemical logistics (§6.5): the same quantity, in the same place, is inert or mobilized depending on recent hydrological history alone. Even knowing exactly what was spilled, one cannot predict either the moment or the magnitude of these releases. Unpredictability, once more, comes not from a lack of measurement, but from the coupled structure of the system itself.

It must now be named what these equations hide, for it is here that the argument of this book ceases to be technical. A lipophilic, persistent molecule does not merely endure: it travels through time. Lodged in fats, transmitted from mother to child, concentrated at each link of the food chain, it carries its dose forward onto beings who were not born when it was spread. Vietnam still counts, two generations later, malformations and cancers that epidemiologists link to these sprayings; the detail of the figures remains debated, the general sense no longer. This is the gravest illustration of our thesis. When static evaluation errs on a persistent compound, the error is not an accounting imprecision to be corrected next year: it is a debt that is passed on. The number is entered today; it is paid tomorrow, by others, and it no longer corrects. To assess a waste is never only to measure a quantity. It is, sometimes, to mortgage a future. This is why Agent Orange closes this part: it reminds us that the regimes we speak of are not abstractions of dynamicists, but the real frame in which lives are at stake.

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5. Conclusion

Three systems, three regimes, one and the same lesson. A bee colony, a shallow lake, a territory contaminated by a persistent compound do not answer a stressor with a proportional decline: they hold one state until they tip, then hold the other. Critical-transitions theory names these tipping points and, through the early-warning signals, grants the foreboding of them. For assessment, the consequence is severe: to credit a reduction of cause with a proportional benefit is to err not on the magnitude but on the *nature* of what is at stake. To destroy an equilibrium costs a single crossing; to restore it costs several, and sometimes none suffices. This is, for this ecological branch of the program, the soberest lesson: in a world of regimes, going back is never the outbound path traveled in reverse.

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Supplement

Code and figures are reproducible (Python; numpy/scipy/matplotlib) and openly deposited (Zenodo DOI at the first stable version). The factors quoted are not cherry-picked points but representative values over a plausible parameter space, with robustness sweeps.