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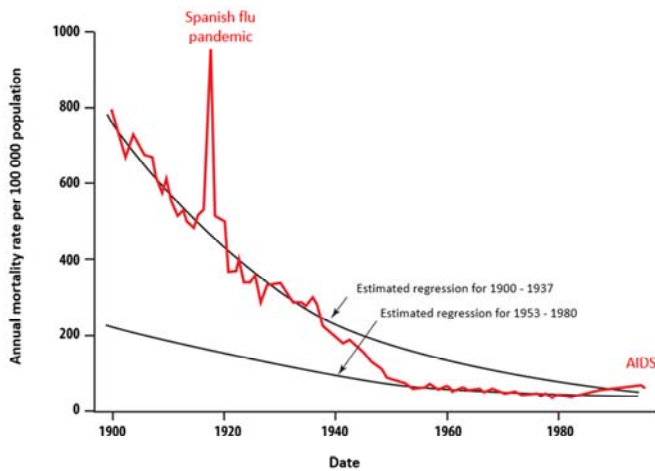
# **Biodiversity and Health**

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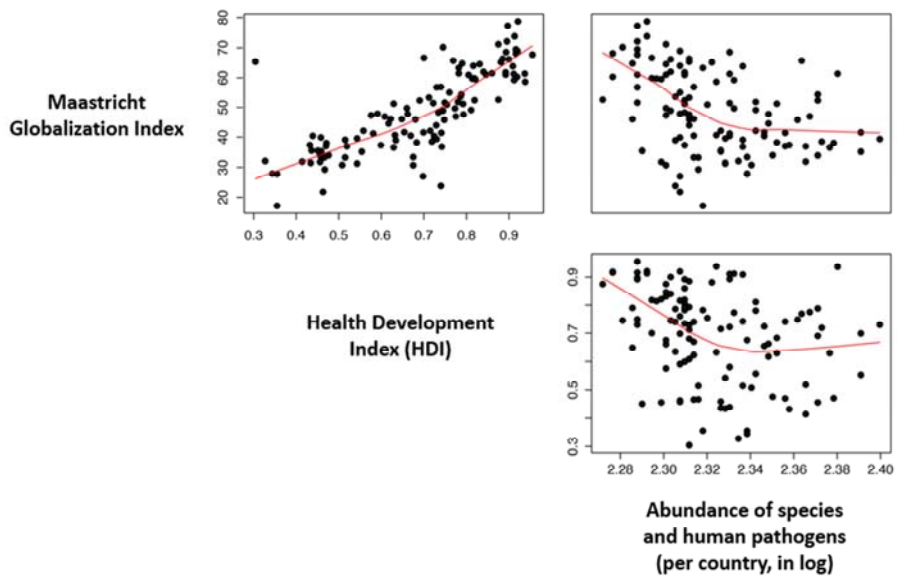
*Linking Life, Ecosystems and Societies*

Serge Morand  
Claire Lajaunie

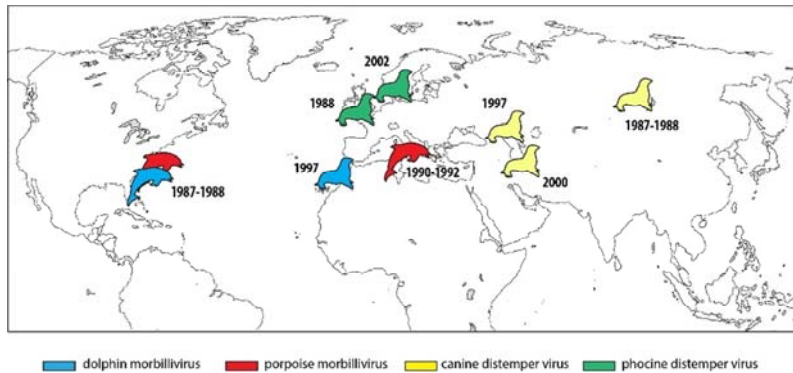
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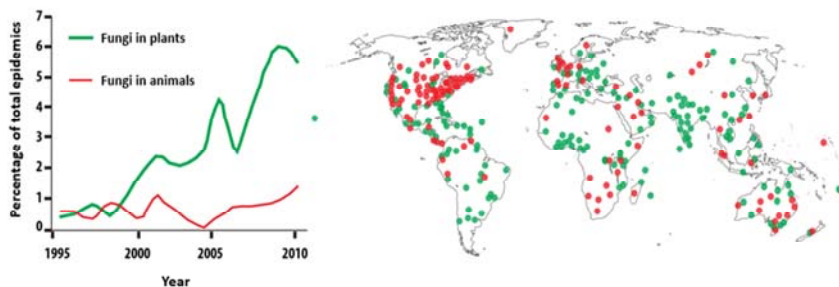
**Figure 3.1.** *Decline of infectious diseases in the United States during the last century [ARM 99]. Between 1938 and 1952, the mortality rate due to infectious diseases decreased by 8.2% per year, and from 1953 to 1980 the decline was reduced to 2.3% per year. This was followed by an increase of 4.8% from 1980 to the end of the 1990s (taken from [ARM 99])*



**Figure 3.2.** Relationships between the Maastricht Globalization Index, the Health Development Index and the abundance of infectious and parasitic diseases by country. Data on the Maastricht Globalization Index are from Figge & Martens [FIG 14] and data on the Health Development Index and the number of infectious and parasitic diseases are from Morand et al. [MOR 14b]

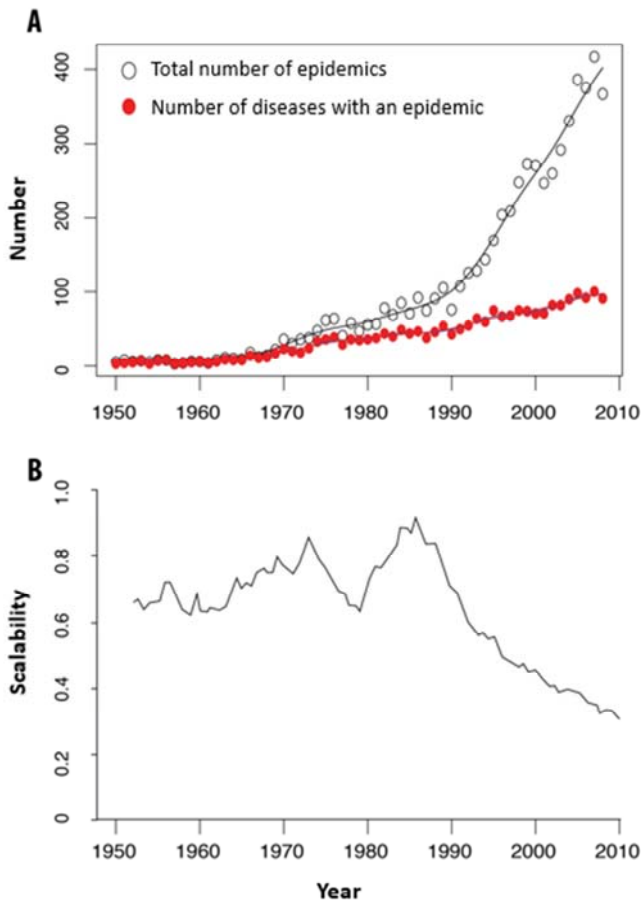


**Figure 1.** Examples of events of mass mortality of marine mammals caused by infection from morbilliviruses (according to [FRI 06])

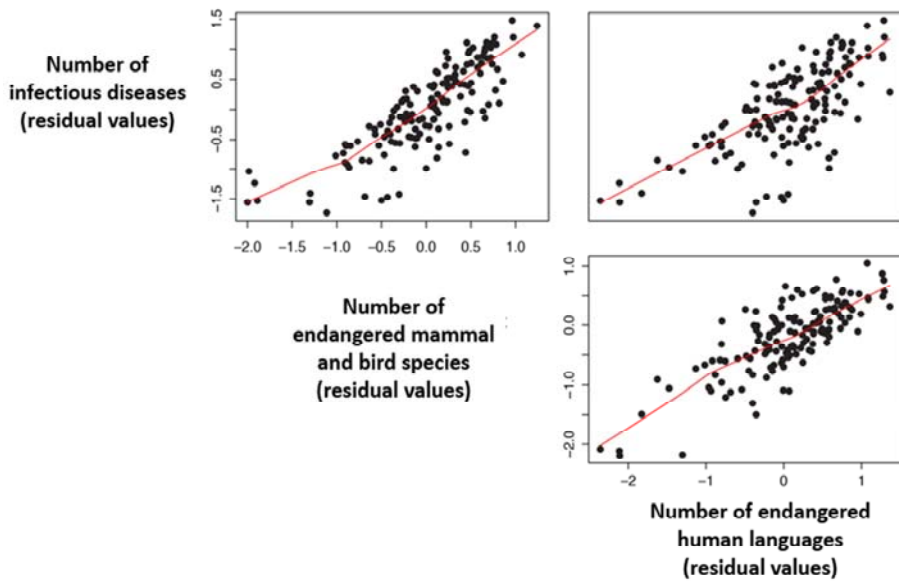


**Figure 2.** Increasing epidemics in fungal diseases in plants and animals: on the left, percentage of total number of cases recorded over time; on the right, spatial representation (taken from [FIS 12])

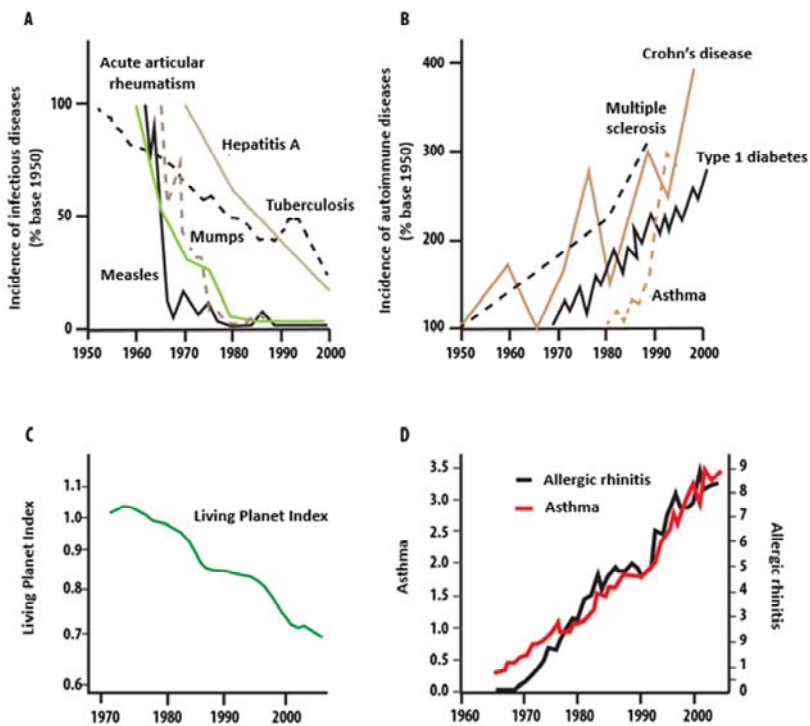
### Box 3.2. Emergences in wild animals



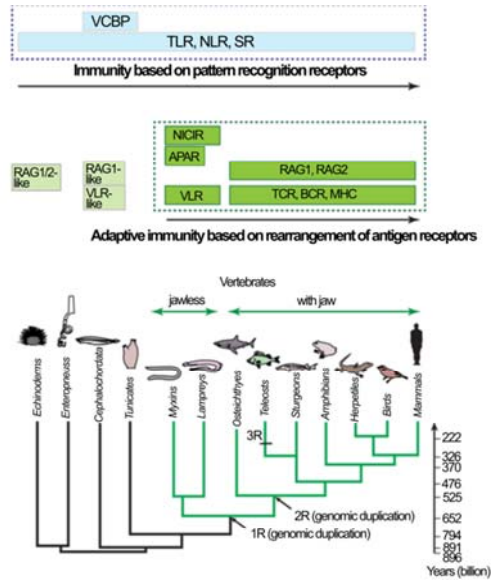
**Figure 3.4.** (A) Evolution of the total number of infectious disease epidemics and of the number of different infectious diseases that had at least one epidemic in a given year on a global scale over the last six decades (based on data from [MOR 14b]). (B) Analysis of the modularity of epidemic networks that are shared between countries showing that, per year, the number of countries that share epidemics for the same infectious diseases decreases from the 1980s onwards while the total number of epidemics increases (A). Increasingly, more countries are sharing epidemics (reprinted from [POI 15])



**Figure 3.6.** Correlations between the number of pathogens and parasites, the number of endangered mammals and birds (according to IUCN criteria) and the number of endangered languages (according to UNESCO criteria). The variables are corrected for geographical area, demographic size of the country and wealth (GDP per capita) (data supplemented by [MOR 14a])

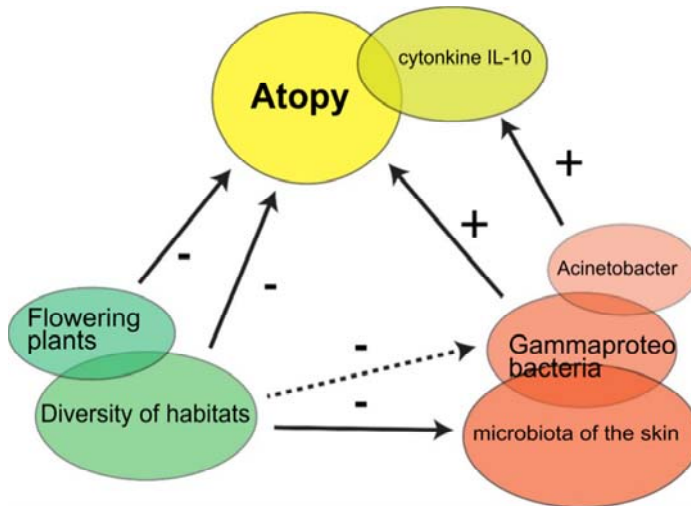


**Figure 4.1.** (a) Infectious diseases in the US over time, (b) Different autoimmune diseases in Europe over time, (c) “Living Planet Index” over time, and (d) Asthma and rhinitis allergies in Europe over time (reprinted from [BAC 02, VON 11])

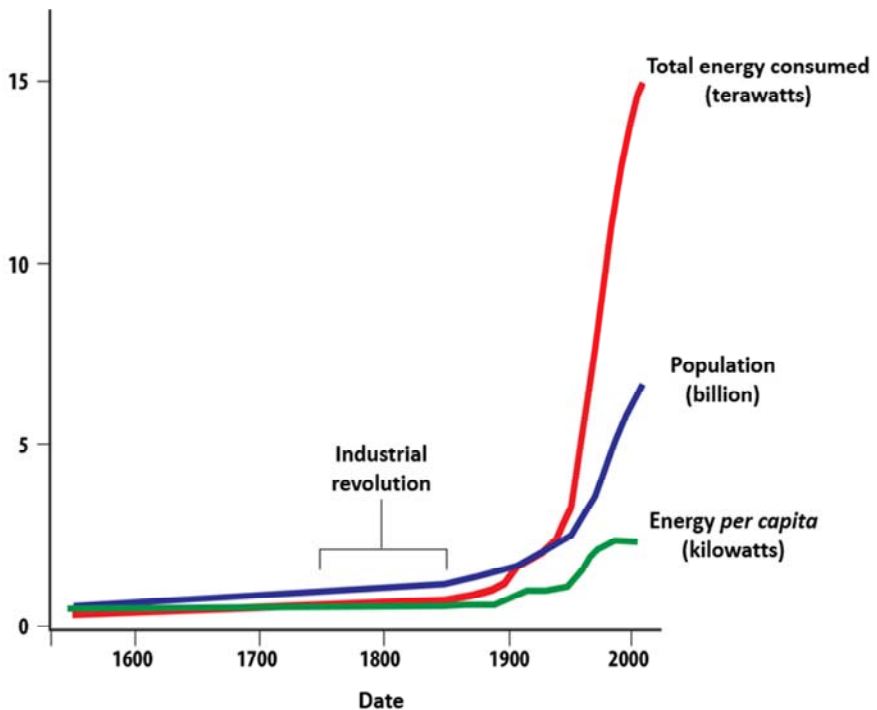


**Figure 4.2.** Evolution of immune system genes in vertebrates (jawed and jawless) and in some invertebrate groups

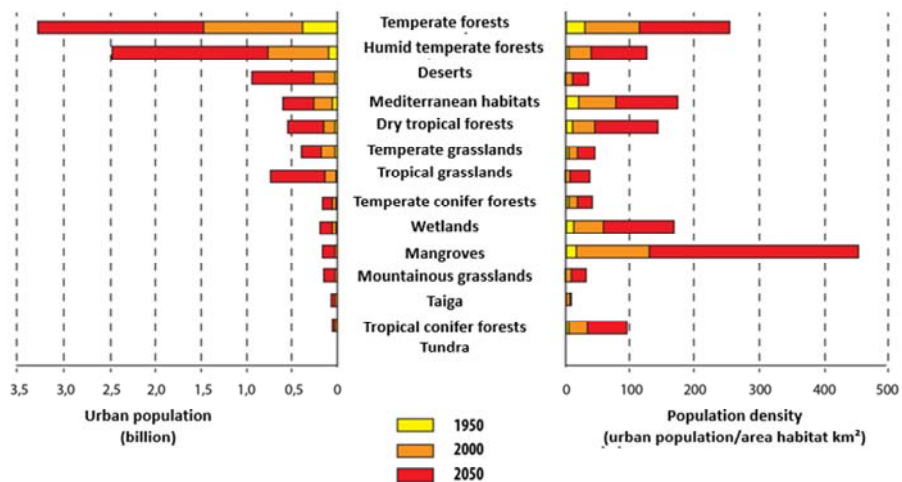




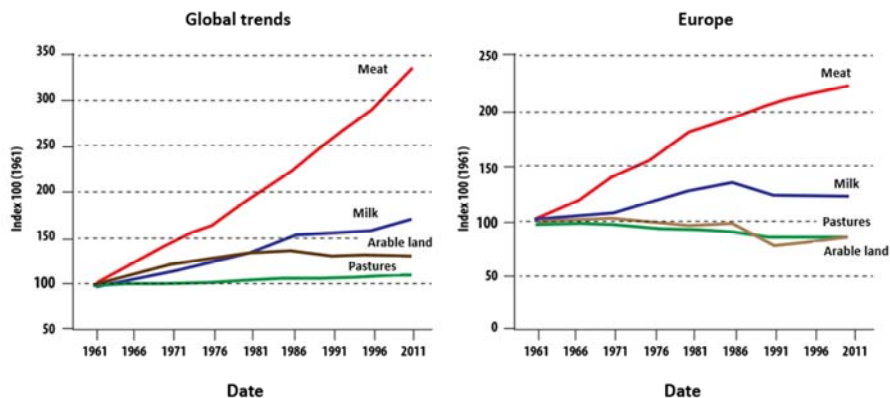
**Figure 4.5.** According to the “farm hypothesis”, a reduction of contact between people and biodiversity can negatively affect the human microbiota and its immunomodulatory capacity. Hanski et al. [HAN 12] analyzed atopic sensitization (allergic dispositions) in a sample of adolescents living in a heterogeneous region in terms of forest, agricultural and urban habitats. The environmental biodiversity around the dwellings of the adolescents in the study influenced the composition of bacteria in the skin. Compared to healthy subjects, atopic individuals living in a low biodiversity environment harbored significantly lower Gammaproteobacteria diversity on their skin. These have a functional role, through *in vitro* measurements show the expression of an anti-inflammatory cytokine that is key in immunological tolerance: IL-10. In healthy subjects, the expression of IL-10 is positively correlated with an abundance of the Gammaproteobacterial *Acinetobacter* genus, which is not seen in atopic subjects (taken from [HAN 12])



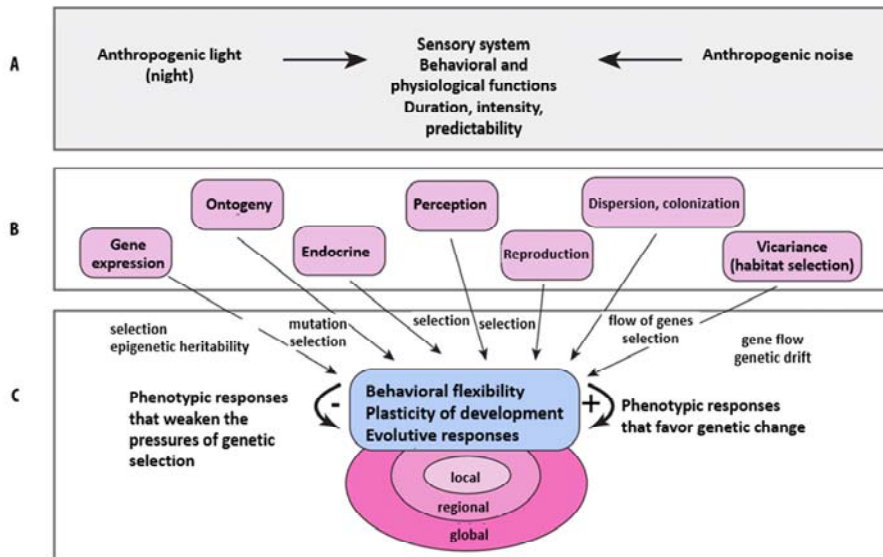
**Figure 5.1.** Human population and energy consumption has been increasing since the 17th Century. Let us note the decrease in individual energy consumption in recent decades, which highlights the increase in disparities (taken from [EHR 12])



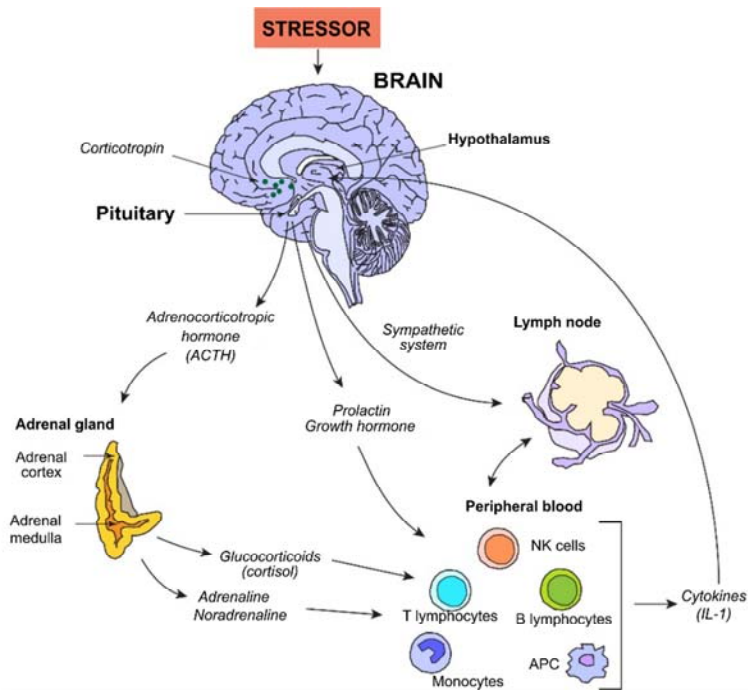
**Figure 5.2.** Urban population size (left) and population density per total habitat area (right) by major type of biome (center). The main types of biome are from the World Wildlife Fund (WWF). Urban population data are from the Global Urban/Rural Mapping Program (2000 data). Urban population data for 1950 and 2050 are based on growth rates estimated by the United Nations Population Division (taken from [MCD 13])



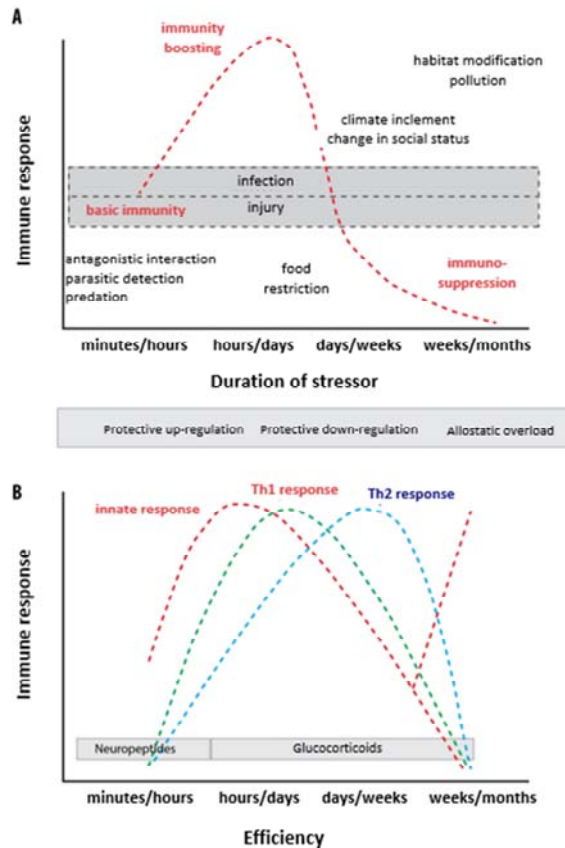
**Figure 5.3.** Evolution of land use globally (left) and in the EU (right) for livestock, meat and milk production, and arable land for forage crops (base 100 in 1961) (reprinted according to the FAO [FAO 06])



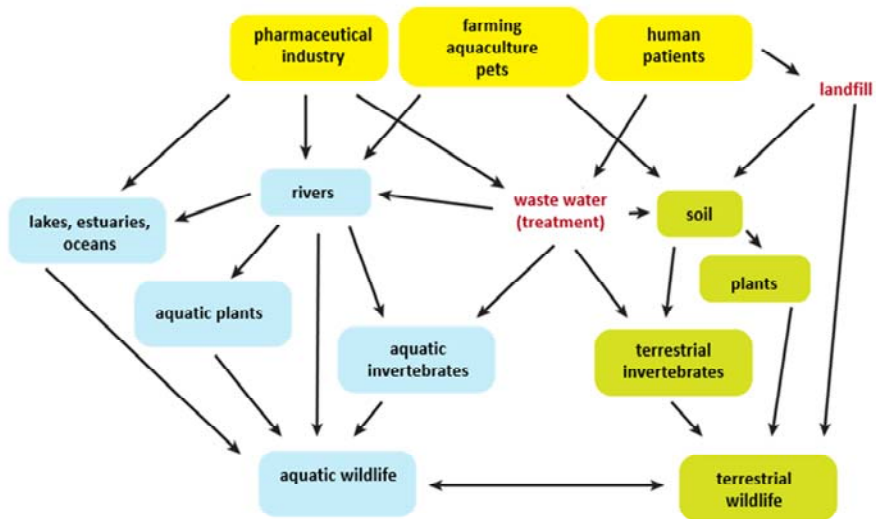
**Figure 5.5.** Nocturnal lighting and anthropogenic noise influence the ecology and evolution of organisms. (A) The characteristics of noise and light act independently or synergistically as factors of ecological and evolutionary change as they affect various levels of biological organization (B) through (C) short-term behavioral flexibility, development plasticity and micro-evolving responses (taken from [SWA 15])



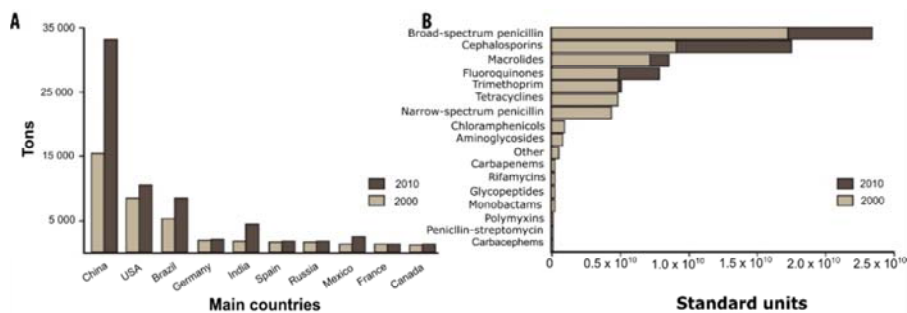
**Figure 5.6.** Responses and associated modulations of the hormonal response to stress by the central nervous system. The experience of a stressful situation is perceived by the brain, resulting in stimulation of the hypothalamic-pituitary-adrenal axis (HPA) and the sympathetic-adrenal-medullary axis (SAM). The production of adrenocorticotrophic hormone (ACTH) by the pituitary gland leads to the production of glucocorticoid hormones (such as cortisone). The SAM axis can be activated by stimulation of the adrenal medulla to produce catecholamines, adrenaline and noradrenaline, and by innervation of the sympathetic nervous system and lymphoid organs. Immunity is affected by the presence of receptors for stress hormones on the surface of leukocyte cells. Noradrenaline can also modulate immune cell activity. These interactions are bidirectional because cytokines, which are produced by immune cells, can modulate hypothalamic activity (APC: antigen-presenting cell, IL-1: interleukin-1, NK: natural killer) [GLA 05]



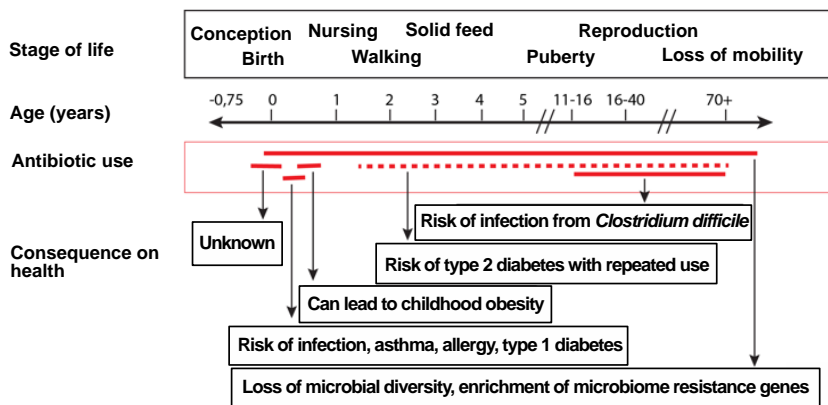
**Figure 5.7.** Effects of stressors (habitat, pollution, climate, social status, infection, food restriction, predation, aggression) on (A) immune responses (protective up- and down-regulation, allostatic overload) relative to the time span of the stressors. The shaded area represents the long-term variability of the sensitivity to stressors resulting from individual experience. (B) Changes in innate immune responses, Th1 (pro-inflammatory T cells) and Th2 (antibody response) relative to stressor duration. The repeated increase of innate immunity in response to long-term stressors would be responsible for the emergence of autoimmune diseases. The main mediators of these immune modulations are peptides derived from the central nervous system and glucocorticoids (see Figure 5.6) (from [MAR 05a])



**Figure 5.8.** Dispersion of pharmaceutical products throughout the environment. The sources of pharmaceutical products are the industrial manufacturer, livestock, aquaculture, pets and human patients. Pharmaceuticals disperse directly into the environment or through sewage treatment plants. Their dispersal in aquatic and terrestrial environments directly contaminates invertebrate and vertebrate wildlife. Food webs favor their bioaccumulation (adapted from [ARN 14])

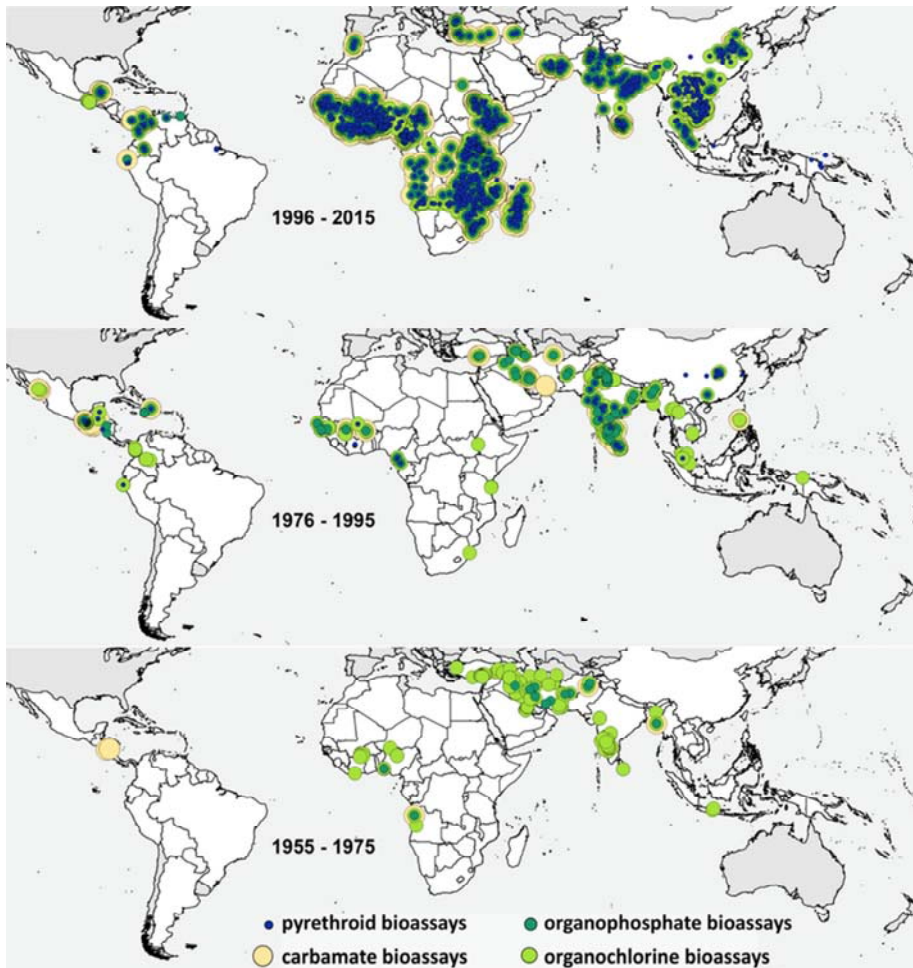


**Figure 5.9.** (A) Consumption of antibiotics in livestock production in 2010 and expected trend in 2010 for the ten heaviest consumer countries (from [SIL 08]). (B) Evolution of the consumption of antibiotics in human health between 2000 and 2010 [VAN 14]

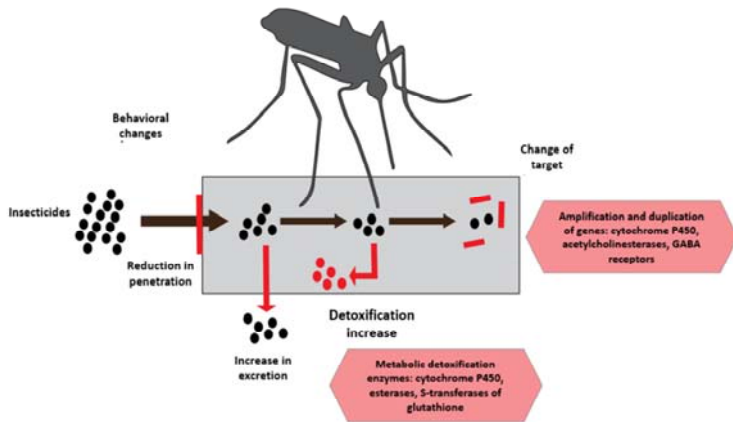


**Figure 5.10.** Effects of antibiotics on the human microbiome from early life to adulthood. A single dose of antibiotics can have consequences on longer or shorter timeframes, as shown by the solid red lines. On the other hand, repeated doses of antibiotics over time, shown by the dotted red line, are necessary to see a link, as in the case of type 2 diabetes (from [LAN 16])

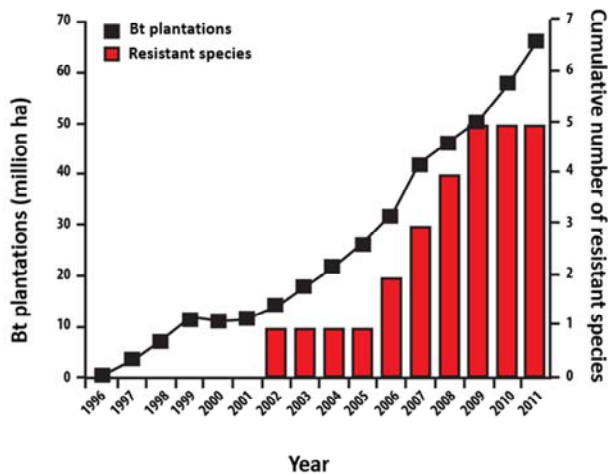




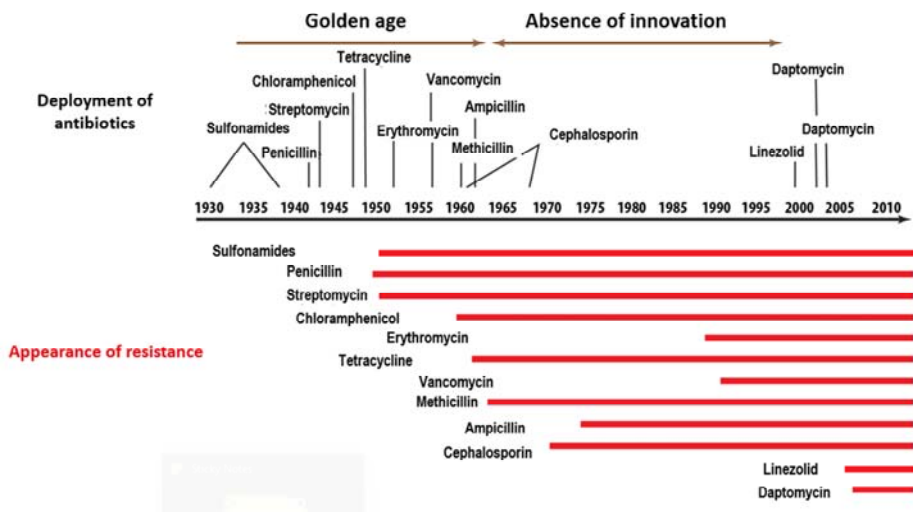
**Figure 6.2.** Evolution of resistance to insecticides (carbamates, organochlorines, organophosphates and pyrethroids) of 74 species of *Anopheles* mosquitoes and their species complexes from 1955 to 2015 in 71 malaria endemic countries [COL 17]



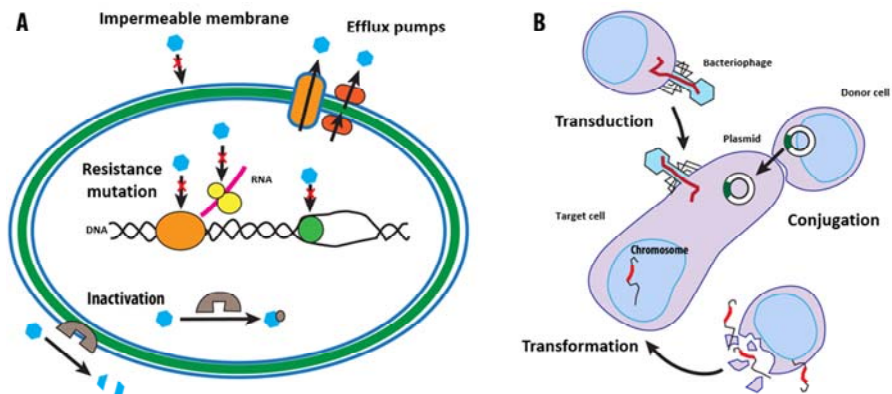
**Figure 6.3.** *Mechanisms of insect resistance to insecticides (from [COR 13, LIU 15])*



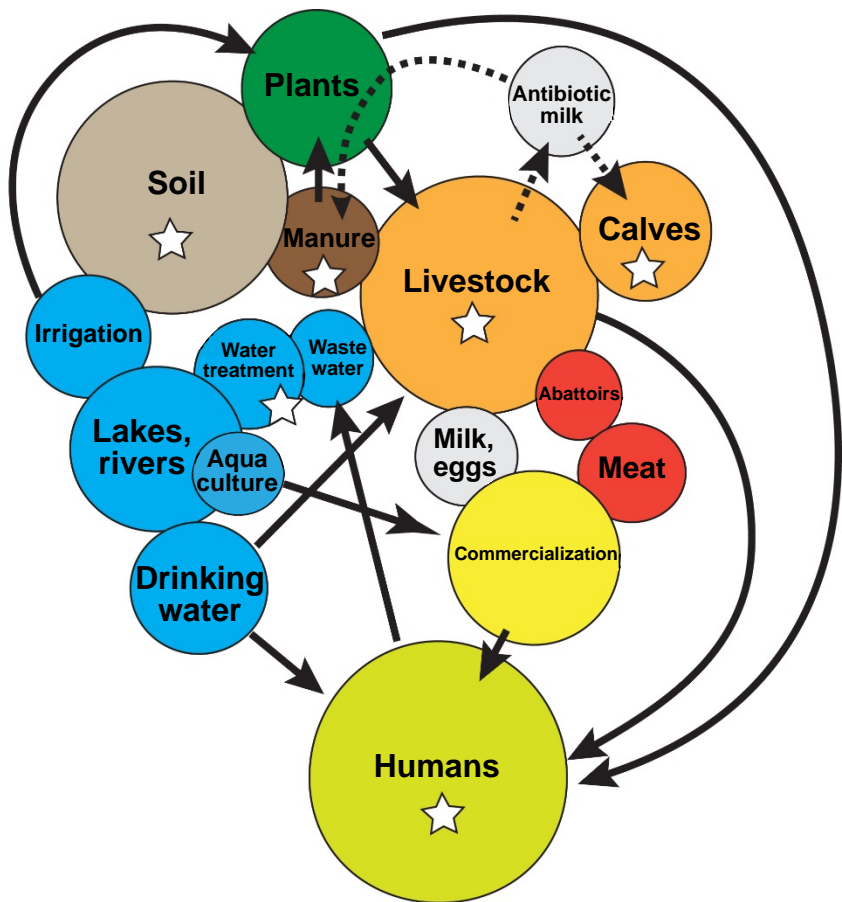
**Figure 6.4.** *Bt transgenic crop plantation on a global scale and resistance seen in the field, in cumulative numbers of insect species with reduced resistance or reduced efficacy as evaluated in the field. Bt cultivation areas increased from 1.1 million hectares in 1996 to 66 million hectares in 2011 (from [TAB 13])*



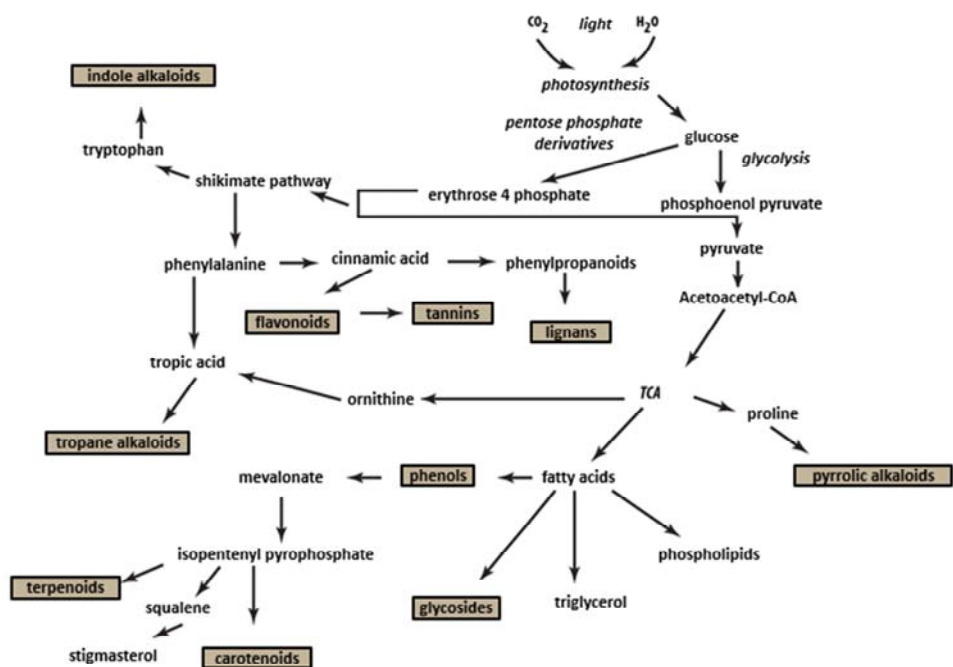
**Figure 6.5.** Development and deployment of antibiotics followed by appearance of resistance (from [CLA 07])



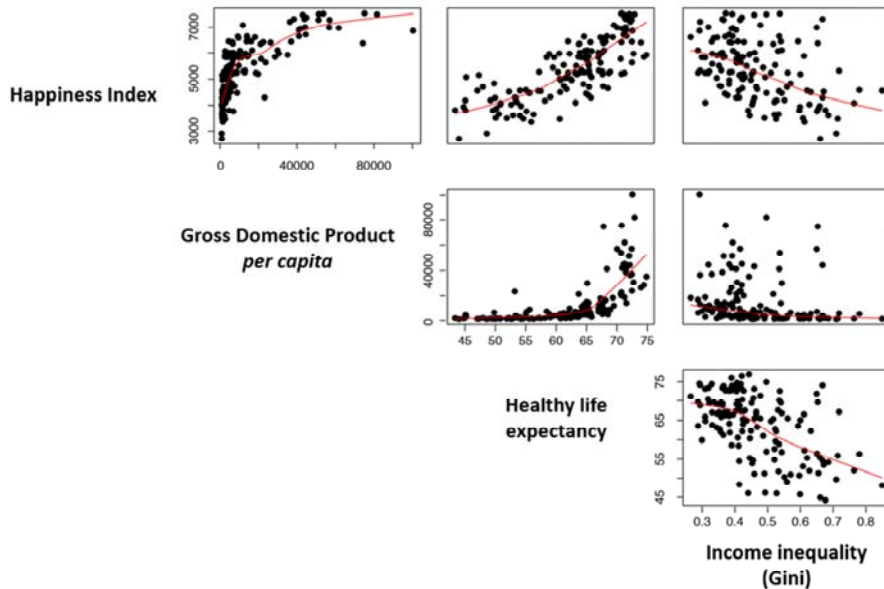
**Figure 6.6. Mechanisms of resistance: (A) and transfer of resistance genes; (B) in gram-negative bacteria.** (A) Antibiotic resistance mechanisms are caused by: (i) membranes that are impermeable to certain antibiotics (or not targeted by the antibiotic); (ii) efflux pumps, which reject antibiotics (RND, Resistance Nodulation Division or MFS, Major Facilitator Superfamily); (iii) resistance mutations that disable the antibiotic binding site on the target protein; iv) inactivation of the antibiotic by modification or degradation (from [ALL 10]). (B) Genetic material is transferred between bacteria through three main pathways: (i) transformation, with environmental DNA recombination; (ii) transduction, where the genetic material is transferred by bacteriophages; (iii) conjugation with plasmid transfers (from [HOL 16])



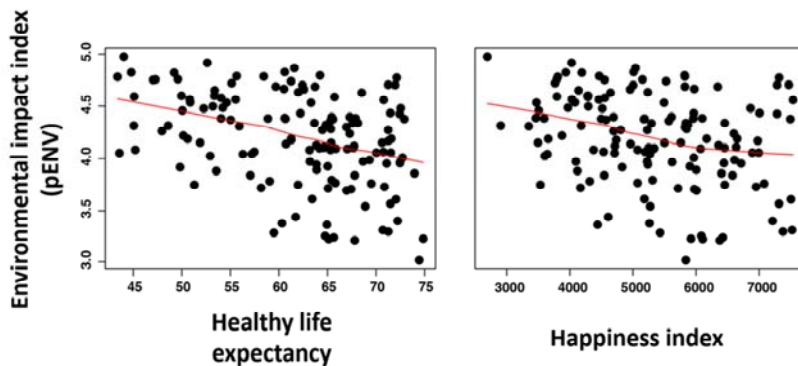
**Figure 6.7.** Diffusion of antibiotics and antibiotic resistance genes in agriculture, the environment and the agri-food industry. The different colors define different groups of reservoirs. Stars indicate the hot spots of antibiotic resistance genes that depend on high bacterial densities and selective pressure, as induced by antibiotics in livestock and human metagenomes, manure storage facilities, sewage treatment plants and the rhizosphere (from [THA 16])



**Figure 7.1.** Primary and secondary metabolism of plants, the main types of metabolites are in boxes (from [ALV 14])

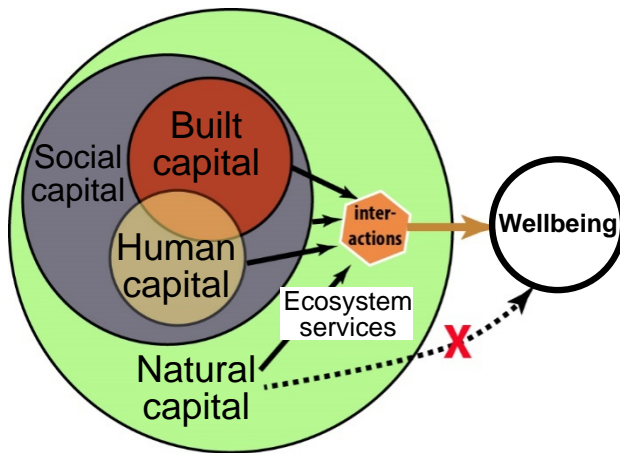


**Figure 8.1.** Relationship between the Happiness Index from the World Happiness Database and three objective measures of well-being: economic level as measured by GDP per capita (in USD, World Bank data), healthy life expectancy (in years, WHO data) and the Gini Income Inequality Index (World Bank data) (all of these relationships are statistically significant)

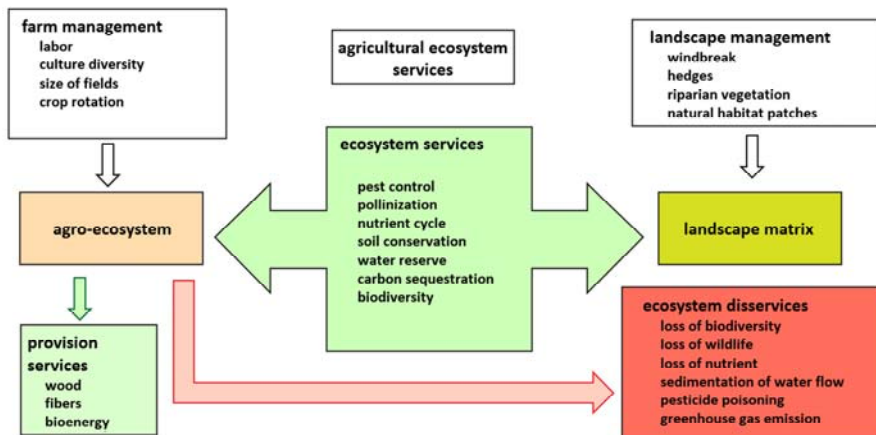


**Figure 9.1.** *Correlation between impact on the environment and human well-being at the country level. The environmental impact is estimated by the proportional composite environmental index, which incorporates loss of forest habitat, natural habitat transformation, fisheries, use of agricultural inputs, water pollution, proportion of endangered species and greenhouse gas emissions (CO<sub>2</sub>). Well-being is valued by healthy life expectancy (in years, WHO data) and the subjective happiness index (World Happiness Database). There is a negative correlation between the environmental impact index and, on the one hand, healthy life expectancy ( $P < 0.001$ ), and on the other hand, the happiness index ( $P < 0.001$ )*

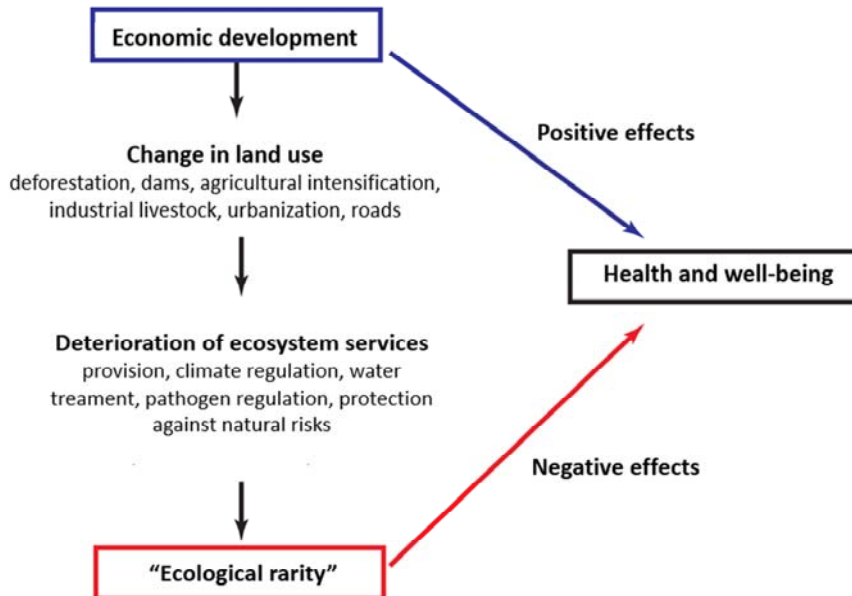




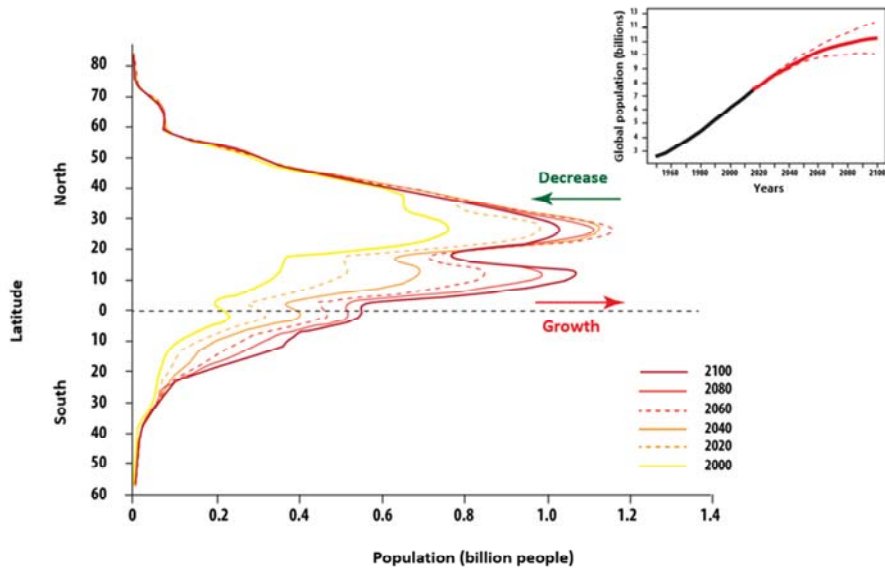
**Box 9.1.** *Natural capital and ecosystem services (according to Costanza in [JØR 10, RUH 07])*



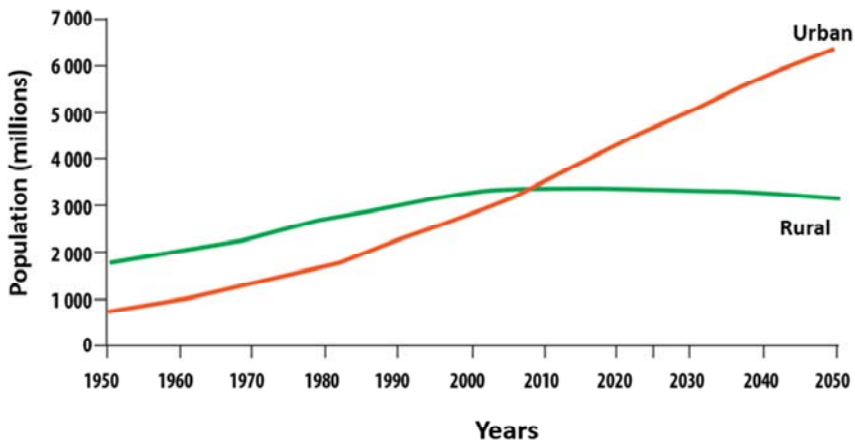
**Figure 9.3.** *Impacts of agricultural management on the flow of ecosystem services and disservices [POW 10]*



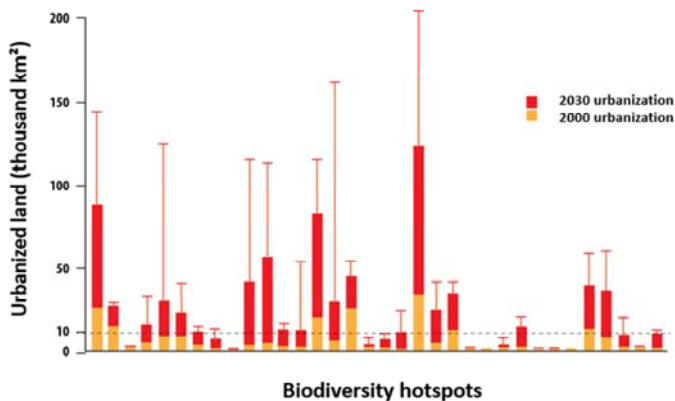
**Figure 9.4.** Effects of economic development on ecosystems, with the beneficial effects of economic development on health and the negative effects of its impacts on ecosystem services and increase in ecological shortage (from [BAR 11])



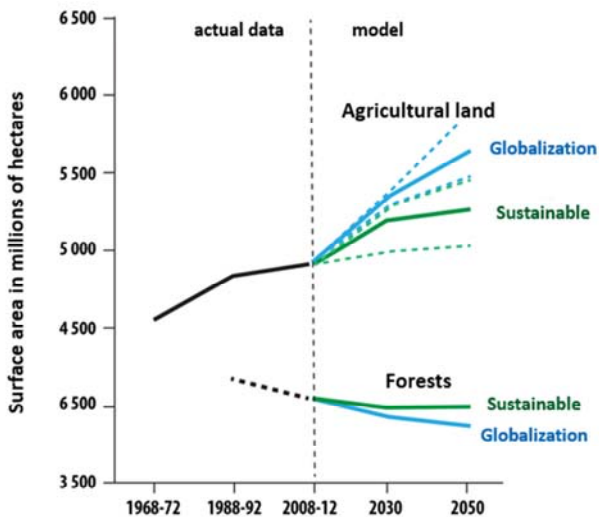
**Figure 10.2.** Projections of changes in world population growth according to latitude (taken from McDonald et al. [MCD 13], based on the 2010 United Nations data). There is a decrease in population in the temperate zones of the Northern hemisphere and an increase in population in the tropical zones across the globe. The top right graph gives estimates of population growth by 2100 [UNI 15]



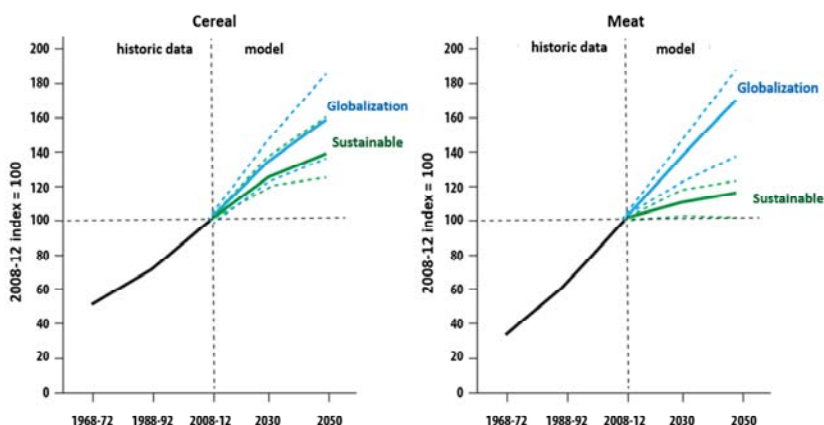
**Figure 10.3.** Projection of rural and urban populations (from [UNI 14]). Global growth will affect urban populations



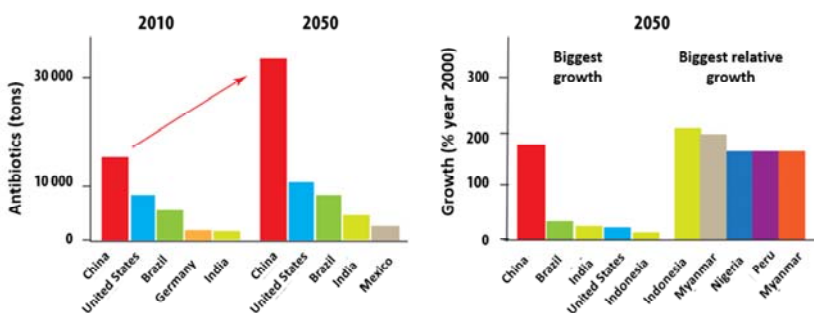
**Figure 10.4.** Urban expansion projected in the 34 biodiversity “hotspots” by 2030. Over half of these hotspots will be significantly affected by the increase in urbanization (redesigned based on McDonald et al. [MCD 13])



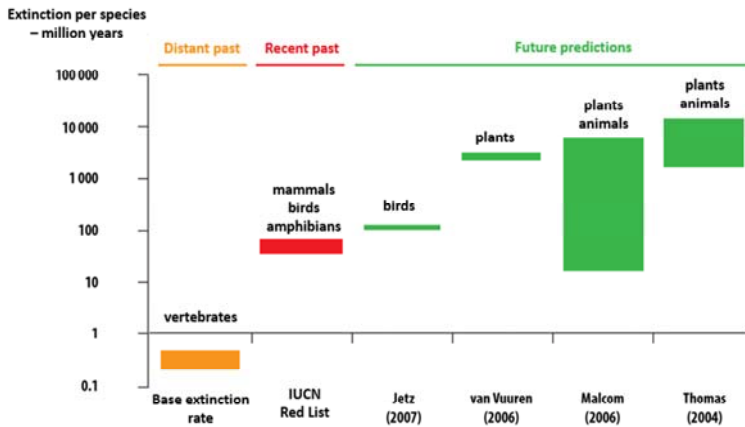
**Figure 10.5.** Evolution of agricultural and forest land cover between 1970 and 2050. Data on changes between 2010, 2030 and 2050 are derived from modeling in various scenarios: the scenario of economic globalization is in blue, sustainable development is in green (simulation values are given by the dotted lines) (from [OEC 15, FAO 12] historical data)



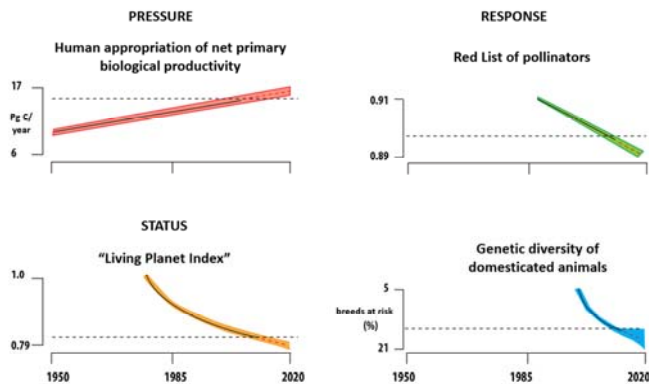
**Figure 10.6.** Evolution of cereal and meat production. Historical data are based on physical quantities, while the modeled data are based on production volumes valued at constant prices (2008–2012). The range of the simulation values is given by dotted lines (from [OEC 15])



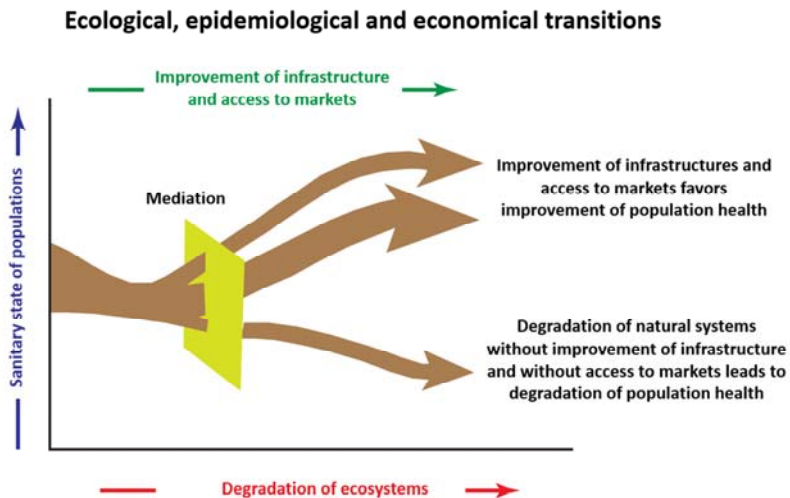
**Figure 10.7.** Evolution of antibiotic consumption in livestock between 2010 and 2050 for the biggest antibiotic-consuming countries. Data shown in tons (left) and percentage increase (right) in absolute or relative data, showing the countries that will present the greatest increases in use of antibiotics in animal husbandry (taken from [VAN 14])



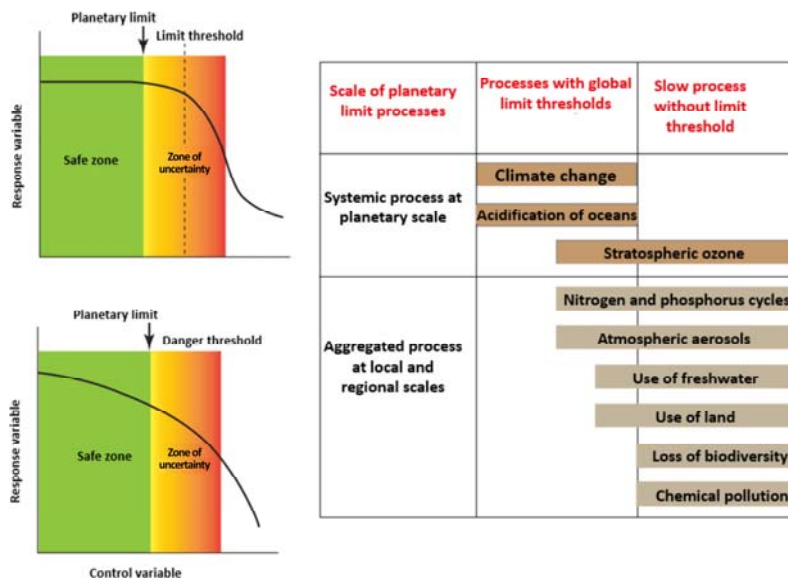
**Figure 10.8.** Estimated extinction rates for the past (fossil record), recent times (IUCN Red List data) and for the 21st Century. These rates are estimated as extinctions per million species-years. Different global scenarios are presented for the future: for birds ([JET 17], for the period 2000 to 2050), vascular plants ([VAN 06], for the period 1995 to 2050), and various plant and animal taxa ([THO 04], for the period 2000 to 2050, [MAL 06], for the period 2000 to 2100) (taken from [LEA 10])



**Figure 10.9.** Projections to 2020 of four indicators for Aichi objectives: pressure on biodiversity (human appropriation of net biological productivity), status of biodiversity (Living Planet Index) and responses in terms of benefits linked to biodiversity (terrestrial domestic animal breeds, Red List of pollinators). The model corresponds to the red lines with 95% confidence intervals (colored areas). Significant differences with the 2010 estimates are given by the horizontal dotted line (from [TIT 14])

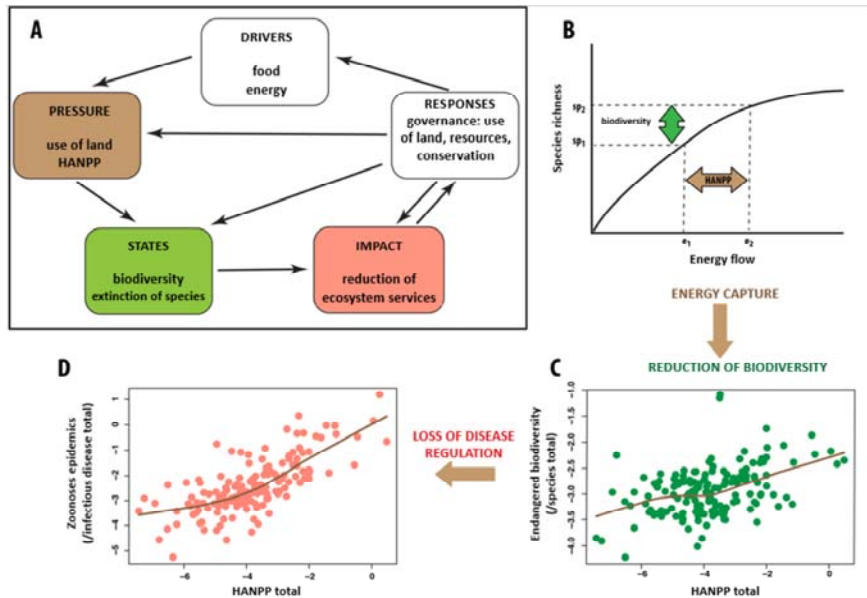


**Figure 10.10.** Conceptual diagram of the link between population health and ecological transition, adapted from Myers et al. [MYE 13]. People are moving from a primary dependency on natural systems for health-related ecosystem services to a state where these populations depend on engineering infrastructure for these services and market access. The trajectory of transition depends on many factors that can alter the vulnerabilities and health levels of populations such as economic equity, quality of governance and environmental characteristics (mediation space) (modified from [MYE 13])

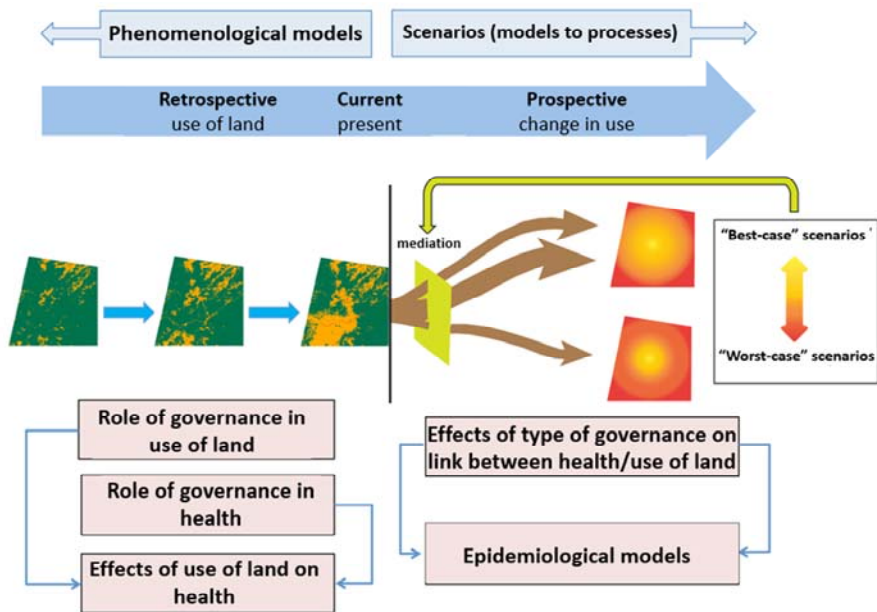


**Figure 10.11.** Description of planetary thresholds and limits according to Rockström et al. [ROC 09]. The planetary limit is designed to be a boundary beyond which a critical threshold at the local, regional or global scale affects a process in the terrestrial system (climate change, land-use change), as given in the table on the right. Insufficient knowledge of threshold dynamics generates an uncertainty zone and positions the planetary limit. Exceeding the planetary limit causes threshold effects, which can lead to tipping points

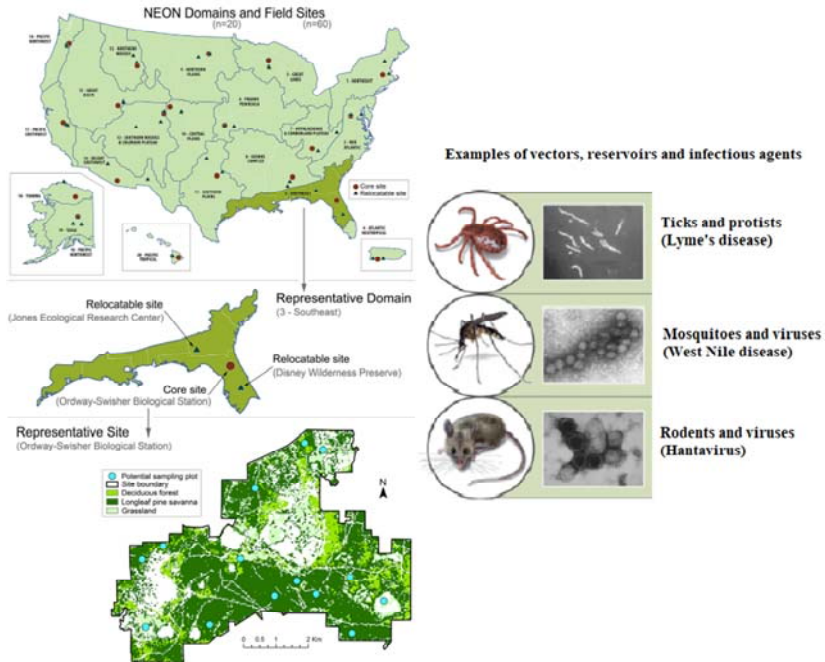




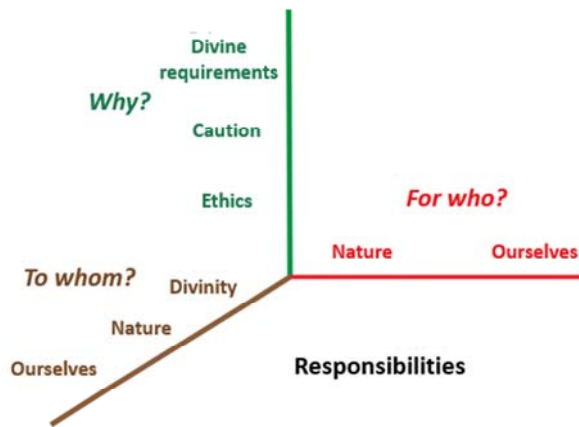
**Figure 10.12.** Conceptual framework of dynamic interactive network that links “drivers” (food consumption, energy), pressures (land use, HANPP), states (biodiversity change and extinction of species), impacts (reduction of quality of ecosystems) and responses (governance, land planning, conservation) (modified from [PLU 16]). This framework is based on the species-energy hypothesis [VIT 86, WRI 83, WRI 90], where a decrease in biological productivity, notably through human appropriation (HANPP), translates to loss of biodiversity ( $\Delta$  biodiversity). This conceptual framework finds empirical support in the correlations which we observe between HANPP ([IMH 06] data, <http://sedac.ciesin.columbia.edu/data/collection/hanpp>) and the number of endangered species (IUCN Red List data), and between HANPP and the number of zoonotic disease epidemics (data from GIDEON, [MOR 14a])



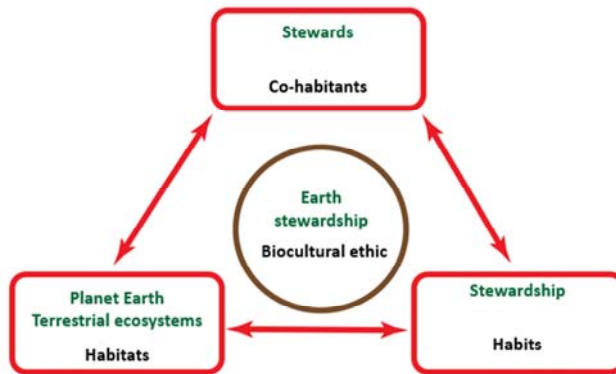
**Figure 10.13.** Conceptual framework to produce retrospective modeling and predictive scenarios for the links between land use (agriculture, conservation, urbanization) and public health (including veterinary and phytosanitary). Retrospective modeling that is based on phenomenological/statistical models allows us to analyze the determinants and interactions of factors of change and their consequences on public health. Prospective scenarios are based on models that incorporate processes (such as multi-agent models, epidemiological models, etc.), which integrate external conditions (climate change, global/local economy, socio-demography) and produce potential scenarios for land-use change in the mediation of local/national governance. These “worst-case” or “best-case” (i.e. desirable) scenarios are incorporated into mediation to produce new scenarios. The process is iterative and requires close collaboration with the different elements in the socio-ecosystem



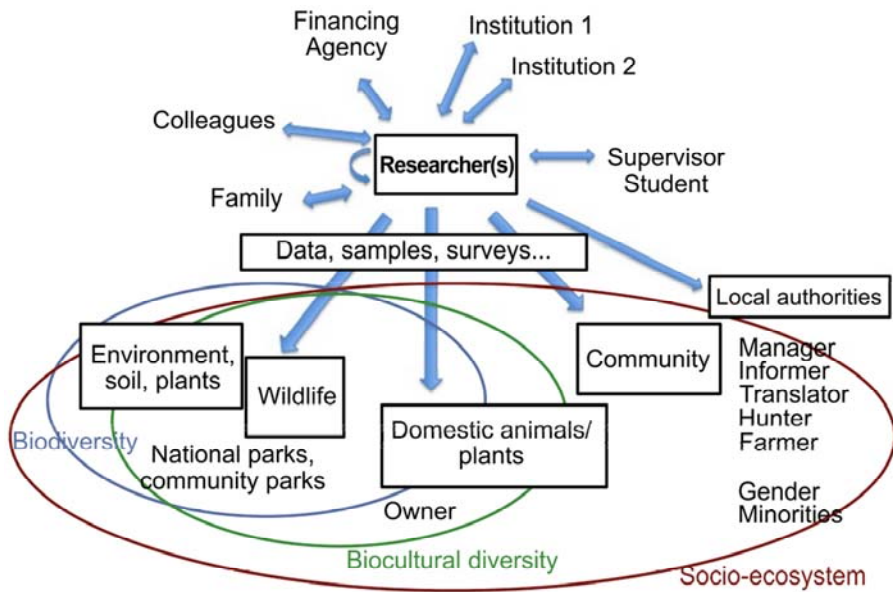
**Figure 10.14.** NEON and zoonotic diseases [SPR 16], based on the hierarchical spatial structure of NEON (National Ecological Observatory Network) [SPR 16]. On the left, top to bottom: boundaries of the 20 eco-climatic domains and the locations of associated sites; example distribution of a central site and two sites in a representative domain with multiple sampling plots distributed among the vegetation types. On the right: a network of the collections and sampling of vectors and reservoirs and infectious agents (images from the CDC Public Health Image Library in the United States, from [SPR 16])



**Figure 12.2.** *Notions of responsibility towards nature, as described by Passmore (1980, quoted by [HOO 92]) (Figure 12.2). We have responsibilities towards nature and for ourselves (“for whom?” axis) and these responsibilities are for ourselves, a divinity or nature itself (“to whom?” axis), because it is in our personal interest or it is necessary from an ethical stance (“why?” axis)*



**Figure 12.3.** *Biocultural ethics are connected through three essential components of Earth stewardship: habitats and planet Earth, stewardship and practices, stewards and cohabitants (from [ROZ 15])*



**Figure 12.4.** *The network of ethics and responsibilities in health/biodiversity research and "One Health" (see text)*