Epistolution: How a Systems View of Biology May Explain General Intelligence

C. S. Munford(✉)

1610 Robert E. Lee Blvd., New Orleans, LA 70122, USA

Abstract. The genes-first view of life provides a theory of traits interacting with ecological niches, and of genes as determinants of these traits, but fails to link the two with a logic of physiology. How are genes selected for expression? It is on this level of physiology that intelligence appears. In this paper, I propose a formula by which *epistemology*, the sources of knowledge, and *evolution* might be united—an "epistolution" that offers in principle a testable synthesis to predict organismic behavior. Perhaps organisms and their microbiota, through allostasis, mediate between their ecological niches and their DNA. Perhaps they form networks nested within networks that are sensitive enough to synchronize with their niches using the formula: *if used, then reinforce; else mutate stochastically*.

Keywords: Epistolution · Downward causation · Sleep · Synchrony · Niche · Allostasis · Artificial general intelligence

1 Downward Causation

In the mid-twentieth century, evolutionary biology arrived at the DNA-centered view of life known as Neo-Darwinism. The theory of evolution since Darwin's time had described organisms as bundles of traits and noticed that the traits can be selected by differing rates of survival and reproduction inside an ecological niche. After the mechanisms of DNA inheritance were worked out, these traits were conceptually linked back to genes, that is, to sequences of DNA that code for specific proteins. But this linkage, although rational, still skips a level. We still have not worked out the logic of the middle level, *physiology*. How do we get from a protein to a trait? Even more mysteriously, how does the cell determine which genes to express, and when? The selection of which DNA to use to solve a cellular problem appears almost purposive; the cell "knows" as if by magic. I will argue in this paper that this physiological logic is in fact where purposiveness resides, that intelligence consists in the sensitivity of all the parts of a complex system to its larger contextual niche.

This middle level, physiology, is where we can observe general intelligence in humans who react to their circumstances by building knowledge. The study of the sources of knowledge is called *epistemology*. In order to explain life, epistemology must be linked to evolution without skipping any levels, so that the consequences of both big situations (niches) and small molecules on the configuration of living bodies can be predicted. We need a testable synthesis—an *epistolution*—a logic of physiology. Ingesting a new experimental medicine comprises a tiny alteration of the body

B. Goertzel et al. (Eds.): AGI 2021, LNAI 13154, pp. 163–173, 2022.
https://doi.org/10.1007/978-3-030-93758-4_17

system as a whole, yet the consequences are often quite unknown. Currently, the only way around this is to conduct randomized controlled trials. Imagine if small, molecule-level alterations to an airplane's design required launching thousands of trial airplanes to determine safety and efficacy. The difference is that we know the logic of the airplane's "physiology," so we can predict the effects of small changes to the complex system, and we can build airplanes by designing them from scratch. Not so with organisms—even simple single-cellular life forms operate by principles that baffle us.

A gene has never expressed itself; it requires a cell and a regulatory network. It is true that DNA sequences are often held in such a way as to make them easier or harder to express given the architecture of regulatory networks, and expression levels can often be partially predicted from such positioning [1]. This does not isolate causation because the regulatory networks are themselves never isolated from their environment, and that environment also partially predicts gene expression. A good example is the sex determination of crocodilians through egg temperature [2]. The production level of a given protein in adjacent cells of the same type can vary by as much as three orders of magnitude [3]. What makes one cell overexpress the protein and the adjacent one underexpress it? How do the cells determine the right average level of production?

In order to work together as a coordinated multicellular organism, cells must exert influence on one another. A typical cell must interact with others in a way that promotes the survival of the organism as a whole and not its destruction from, say, cancer. But the nature of this causal influence is still murky. The possibilities of gene expression are nearly endless. If a trait can arise from any number of genes, the number of ways that the 30,000 or so genes in human cells could be combined to produce traits amounts to a number near 2×10^{72403} [4, 5]. But the total number of particles in the universe is estimated at only 3.28×10^{80} [6]. This shows that it is impossible, even in the long history of life, for evolution to have explored even a tiny fraction of all traits. Instead, the cell is exercising what a naïve observer would be tempted to call "choice" in deciding what genes to express.

None of these facts fit the "blueprint" metaphor which has sometimes been used in biology. If life is an emergent consequence of DNA, why are organisms not *systematically* interpreting their DNA codes one by one, like a carpenter with a blueprint? Or alternately, why are cells not *randomly* exploring these possibilities for gene expression? If a trait can arise from any combination of genes, then there must be some systematic logic at work that selects combinations of genes. As the math I've just referenced suggests, the possibilities for expression are far too vast to be unguided.

If this logic of gene expression were encoded quite inflexibly in the genes, then cells couldn't influence one another at all. If it were encoded in the genes such that it might be expressed in many different ways given a variety of triggers, then these triggers would control every functional pattern. I presume that this is the working assumption of many biologists today. In this case the physiological logic of gene expression may be a vast field of meta-instructions *other than the laws of physics*, built up by the DNA into the structure of its regulatory network. In this case, in order for us to fully understand the logic of the human body, we would have to map out all the possible internal states of each epigenetic regulatory network, and then map all the possible physical conditions faced by each cell that might lead to these internal states. We would have to do this in all the 35 trillion or so human cells in the body. Bear in

mind these cells diversify into roughly 200 cell types…skin, blood, neurons, bones, muscle, and so on, and that they change as they undergo all the phases of growth, development, and senescence. This is indeed the work of the sciences of genomics, transcriptomics, proteomics, and metabolomics, but they have so far not produced a model of the system as a whole. If we miss just a few of these meta-instructions in building our map, it seems possible that these unaccounted-for codes might throw the whole model off.

The explosion of complexity does not end there. Research over the past two decades has revealed that the human body is really a superorganism, composed not only of human cells but also of many trillions of prokaryotes, viruses, and very small eukaryotes that may outnumber human cells [7]. This microbiota functions not only as a digestive organ and regulator of metabolism, but as an integral part of a healthy immune system, and as a vital component of the cognitive process [8–12]. This community of nonhuman cells with unique genes is not inherited along with the germ cell from the parent, but acquired from the environment after birth in a somewhat haphazard way, resulting in significant differences in microbiota even in identical twins [13]. Dethlefsen et al. write that "at the species and strain level the microbiota of an individual can be as unique as a fingerprint [14]." There *are* internal organelles in eukaryotic cells with their own genetic material that are inherited from the germ cell, but the symbiogenesis thesis suggests that these were once separate organisms that have long ago been incorporated [15]. This may be evidence that this flexible partnership with external cells with foreign DNA is not only very ancient indeed, but that it is nearly ubiquitous among eukaryotes, and is vital to normal function [16].

The existence of a microbiome means trouble for the promise of understanding physiological function through the genes-first view of life. If it were correct that gene expression was determined by a meta-program that was encoded in the DNA, then compatible programs would also be required in the tens of trillions of diverse cells of the microbiota as well. These microbiota might be expected to contain wildly different genomes and meta-instructions, yet as a community they would have to instruct macroconditions that supported the survival of the host. And in order to understand that host and its survival we would have to map all these microbiotic genes and meta-instructions just as precisely as the host cell genome and meta-instructions. The fact that the microbiome of an individual is reorganized by diet, sleep, exercise, and other variables [17], yet maintains its long-term stability [18], suggests that another level of logic is present. Organisms spontaneously assemble themselves into functional ecosystems, as the species-area curve in the biogeography of islands attests [19].

There is one plausible alternative, testable in principle, that might suffice. Unfortunately in order to understand it one has to rearrange most of the philosophical furniture of Western civilization. This is the idea that ecological niches may structure the interactions of organisms directly. In order to entertain this hypothesis, we have to set aside the aversion to downward causation that has accompanied serious biology since the nineteenth century. I should say that this is *not* an argument for intelligent design. This idea is compatible with a materialistic cosmology, and with the empirical observations that have underpinned Neo-Darwinism. I have no doubt that DNA evolves by natural selection, and that having the right DNA is vital for life. I am only suggesting that on the level of physiology, organisms may be sets of interlocking

networks that are sensitive enough to their niches that they take their instructions from those niches. Just as the upward logic of Neo-Darwinism requires only mutation and differential selection, this downward logic may only require a similar basic universal formula.

2 Finding a Niche

What is a niche, exactly? A niche is a set of orderly physical patterns that allow an organism to remain intact to live and reproduce. A human can live only in a narrow band of conditions, in air with sufficient oxygen, at mild temperatures, in regular cycles of light and dark, with gravity of a certain strength, with fresh water and nutritional solids, in areas free from large predators, parasites, viruses, storms, and excessive radiation. All these conditions are vital for our survival and are not ubiquitous in the universe but highly concentrated in a very delicate area between the sea, land, and sky of one particular planet. How do we know where a niche is and where it is not? We can guess, but we do not know precisely, because we cannot see niches directly…the only niche-detection device ever invented is an organism. There may be many more niches than there are organisms to fill them. Jakob von Uexküll called it the *Umwelt* [20]. A niche is a place with a special form of order; a niche is not just *anywhere*.

How do we stay in this niche and not drift into deadly hazard, drowning in the bathtub or falling off the balcony? We do this by our *actions*. It is intuitive for us to see ourselves as independent intelligent agents in the world we live in. When we reflect on ourselves, we see a loose part, an "I", that drives the whole system by our choices, rather than being driven by it. But if we say that a human, as part of the larger system in a human niche, acts independently, this description separates what flows from our minds from what contributed to it. When we attempt to investigate this empirically, we get caught in an infinite regress trying to find the "I" in the neurons. It's as if we are asking of a clock, "What part of the clock keeps the time?" We are looking at each spring and gear, noticing which of them impairs timekeeping most when removed, and deriving from this a reductive account of where the essential timekeeping function lives. We describe actions as *purposive* at the level of the organism, rather than at the level of brain cells or at the level of the biosphere as a whole. But our body system is is completely enmeshed in interaction with both the environment and with itself at all times. The whole clock mechanism keeps the time, of course.

This illusion of agency is reinforced by the fact that there are a tremendous number of possible ways a human organism *could* interact with its niche. The environment can change markedly without impacting the health of the organism, a fact which suggests it may have little causal influence. But surprisingly, research suggests that many genes can also be deleted with no harmful effect. For example, 80% of roughly 6000 gene knockouts in an entire yeast genome were found to be silent under normal conditions [21]. So there appears to be considerable buffering in either direction. One possible conclusion we can draw from this is that the causal chain in an organism runs from the DNA up to the niche and back down again, in a continual loop. This is what the authors of the Santiago theory of cognition called "a circular form of organization [22]." In this case the organism could be seen as a process *mediating* between its genes and its niche.

Claude Shannon wrote of information that "the important aspect is that it be a message selected from a set of possible messages" [23]. This could describe the A,G,T, and C of the nucleobases. Cells may be using genes much as we use *memes* in the human niche, as tools drawn from a library of possible templates for solving problems.

3 Niche Synchrony

One well-known natural process that takes chaotic materials and assembles them into orderly structures is synchrony [24]. This process has proved very difficult to study, perhaps because mathematical models of the nonlinear phenomena involved are hard to develop. But nevertheless the phenomenon exists in many forms in Nature, from the orbits of moons to the chirping of crickets to the formation of crystals. Synchrony brings chaotic energy and matter into orderly or rhythmic motion. Many metronomes, placed on a tabletop but set to different rhythms, gradually synchronize [25]. In this example, it is easy to see that there are only two forms of change that matter, either changes toward synchrony or away from it (Fig. 1).

Fig. 1. Positive and negative feedback drives synchronization of connected networks

The pressure of the metronomes on the left as they swing exert a strong pull on the metronome on the right, which gradually forces it to accede to their same rhythm. In this case, and in all cases of synchrony, an object caught in the synchronizing system only has two ways to change, either away from the system's rhythm or towards it. The physical dynamics of synchrony simply make it a bit harder for the object to move out of synchrony and a bit easier to move into it. This is what gives synchrony its eerie "drift" that can be so beautiful to observe. The result is the coordination of forces that seemed disconnected into a seamless dance of elegant fluid motion.

Perhaps organisms do much the same. Homeostasis is the process by which organisms maintain their physiology within certain parameters…salinity, temperature, pH, and so forth, by interacting with their environment, and *allostasis* is a term which recognizes that there is a "drift" to this process. These are the actions that every living cell carries out to solve its problems by selecting genes for expression. Perhaps we can think of the allostatic process as a form of intelligent "agency," keeping the organism inside its niche. At a basic level, all matter is a network of dynamic energy quanta held in a certain pattern by physical interactions. This means that everything living, too, is made of networks. Organic molecules are networks, proteins are networks, organs are networks, and whole animals are networks. Matter-energy passes in and out of these networks, but the networks cycle and reconfigure themselves.

If we keep in mind that there are only two cardinal forms of change in a synchronizing system, then it follows that the only thing necessary to produce approximate synchrony would be some active process in each network that distinguished between them. If a network is moving toward synchrony it must take some form of reinforcement, and if it is moving away from synchrony it must take discouragement or undergo degradation or mutation or some kind. What cue would there be when a network is approaching synchrony? The network would be stimulated or triggered by the niche. It would be *used*. If it remained unused, then it may be departing from synchrony over time.

As a general rule, all structures in the body experience some breakdown or atrophy if they are both unused and alive for a long period of time. With disuse muscles, tendons, even organs like the heart and brain become gradually weaker and shrink in size [26]. Structures that are used vigorously, on the other hand, become stronger. We can keep ourselves more physically fit through exercise, a fact that is hard to explain from the perspective of Neo-Darwinism. Likewise, neural pathways that are exercised become more active, and those that are disused fall into degradation more rapidly. We forget far more than we remember. The epistolution formula for adjusting the networks to drive niche synchrony might simply be: ***If used, then reinforce; else mutate stochastically***. Perhaps organisms have no explicit, inalterable instructions for function anywhere, any more than the dust particles in the rings of Saturn have special-purpose algorithms. Instead of algorithms, organisms may have *habits* influenced by the genetic tools available to them but structured by the niche.

4 Artificial General Intelligence

One way to experimentally refute the causal theory of Neo-Darwinism would be to show that lifelike behavior could be produced by a niche without an organism inside it, and without strictly coded instructions, simply through epistolution. We suspect that naked DNA alone in a petri dish remains inert forever; it never produces life. But perhaps niches can produce lifelike activity without cells.

How would we know if an experimental device was interacting with a niche in a lifelike way? If the niche and the device were created in a computer simulation, the niche itself would be highly artificial and bear little resemblance to the chaotic conditions of the real world. It would be impossible to tell if the device was really behaving

as a living cell would, solving problems, or instead in a way that just superficially resembled problem-solving. How would we determine what comprised real problems for this simulated device?

In practice the easiest niche to examine empirically may be the niche of the entire human being, simply because this is the niche of the examiner. Behavior that is lifelike, if it appeared in a nonhuman or artificial niche, would be hard to recognize as such. This is because if this conjecture is correct, the key feature of lifelike behavior is not any particular set of actions but rather the quality of using actions to solve problems using creativity. This quality could only really be recognized by an observer who was himself sensitive to the contextual problems of a similar niche. Since the device would have, in many particulars, slightly different problems than a human no matter how carefully it was constructed, the evaluation of those solutions by the examiner would always be a matter of some intuitive judgement. We may recognize intelligence, for example in an octopus, though we can't currently say precisely what intelligence *is*.

The premise that intelligence consists in exquisite sensitivity to and synchrony with a niche may be supported by the observation that higher intelligence seems to require organisms to sleep. The function of sleep is no longer considered to be rest, or torpor, but rather comprehensive repair [27]. Why should maintenance of the networks of higher animals require a holistic repair cycle in which the animal is prone, unconscious, and vulnerable for hours at a time? Why can we not repair on-the-go? Evolution should have surely selected against this dangerous adaptation unless there were a tremendous benefit involved. Sleep has convergently evolved both in bilaterians like us (fish, reptiles, mammals) and also in intelligent mollusks, further suggesting that it is indispensable to intelligence [28, 29]. The primary symptom of sleep loss is cognitive impairment. Without any sleep at all, cognition eventually becomes impossible.

I propose that sleep may be the cycle within which highly complex multicellular organisms make a concerted effort to apply the first command of the epistolution formula to their networks: ***if used, reinforce***. Stochastic mutation can happen in many ways, including the passive degradation of complex particles at body temperature, but repair and reinforcement requires coordinated effort. This might explain sleep.

5 Testing Epistolution

If an artificial network could be designed which was a) complex enough to store as much knowledge as the human body, b) adjustable according to the epistolution formula, and c) sensitive to many of the same stimuli with which a human body interacts, the device might serve as an empirical test both of Neo-Darwinist causation and of inductivist epistemology. Inductivism holds that learning occurs by building theories from observations, but some theorists, such as the physicist David Deutsch, advance the contrary view of Karl Popper that knowledge is built through conjecture and refutation [30]. A Popperian view of the body might suggest that our thoughts could be considered anticipatory hallucinations, punctuated by corrections from our niche. Expectations are conjectures, in other words, while surprises are refutations. For example, one might never notice the skin on the outside of one's left pinky for years until one day one finds that a glove has a hole in it in just that tiny location. The skin in

that little patch had been sending sensory signals continually for years, but they only reached one's awareness when those signals violated a hallucinatory set of expectations (Fig. 2).

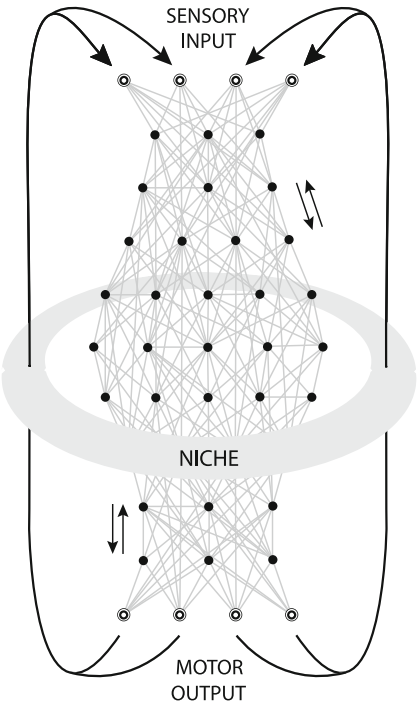

Fig. 2. Expect-actions take on the rhythms of a niche because they are causally connected.

Advances in hardware and software have only recently brought this test into the range of technical feasibility. In order for a human-like niche to be engaged, it would be necessary for the test device to possess the robotic equivalent of arms and fingers to handle objects, temperature, vibration and pressure sensors, and robotic eyes, ears and larynx. It is our general body design and sensitivities that activate the human niche. This provides the frame of reference within which our individual problems make sense to one another as humans, allowing communication and coordinated problem-solving.

To model a Popperian nervous system in software, a complex set of nodes might be linked to sensory input and to motor output. A flow of energy moving down a pathway between nodes could serve both as anticipation of the patterns of excitement coming from the niche and also as an impulse to motor action. Since motor action would cause sensory input to change, the flows of energy through the system would be causally linked to the rhythms of the niche. They would be both expectations and actions, or "expect-actions." New motions would be triggered only when surprises occurred, otherwise patterns of energy would simply repeat.

Rhythms require timing. This may be why every known cell has at least one internal oscillator, why organ systems have peripheral clocks, why all large organisms have a coordinating circadian rhythm, and why brain activity manifests as "waves. These myriad biological rhythms are very sensitive to stimulation such as light, temperature, feeding, exercise, and hypoxia [19]. Each of the test nodes should possess, like neurons, both a set of adjustable connections to other nodes and an adjustable endogenous clock that allows the system as a whole to evolve synchrony. Picture the neural network as complex strings of dominoes that fall in rhythmic patterns but reset themselves after a few milliseconds. If each domino didn't have an adjustable clock, they could not sync with the rhythmic patterns of input emerging from the niche. After it fires, a neuron has a recovery period during which it cannot fire again [31]. Though

imprecise at first, over time this type of network should evolve into a better anticipation of the stimuli in the niche, simply by mutating away errors and reinforcing successes. Like heritable genetic evolution, this process would select functional connections and more accurate rhythms over time, which may comprise creativity.

This form of embodied cognition would not be statistical prediction, Bayesian or otherwise. Motor interactions would be necessary to incorporate causal knowledge into the training dataset; statistics is not enough. As the computer scientist Judea Pearl puts it, *experiment* is necessary to rise higher on the "ladder of causation," [32]. A human is not simply an inductive statistical machine; we expect and act on many things we have never encountered before, such as our own death, or marriage, or climate change. In this network, learning would arise from surprises to the hallucinatory expectations embedded in the pattern of connections and their rhythmic firing. These interactions may be universally translatable in a way that Bayesian predictive computations are not. Consider words like "light" and "heavy." They are metaphorical. As Nietzsche suggested, all our truths may be of this sort [33].

Would this device have motivations? Yes. It would have mismatches between its hallucinatory expect-actions and the flow of its sensory input, and these would drive new interactions to develop. These may be the same sort of contextual problems that we experience in trying to understand our world. The evolution of new interactions that more correctly synchronize to anticipate those problems may be the source of creativity in all higher animals. If this robotic niche synchrony worked approximately at a high level in the human niche, this would provide one possible explanation for the physiological logic of gene expression in all living organisms.

This experiment might also resolve the AI control problem [34]. If human-recognizable knowledge consists in rhythmic responses developed in the context of the human niche, then only machines trained with that human-niche-like dataset could be recognizably generally intelligent to human observers. If the epistolution conjecture is correct, any such device would be as sensitive to the moral norms of humans as we are.

In this view of life, an organism would be a mediator that adjusts between two vast reservoirs of possibility, one above and one below, by applying the epistolution formula to its networks in cycles of periodic adjustment, through sleep. It would be a process that harmonizes the possibilities of its genome with the possibilities of its niche. As a result, its solutions would always be approximate solutions, adjusting between vast opportunities in either direction. This might be the source of our spontaneity, our impetuous, inventive creativity. In this world, there would be no such thing as a final correct answer. Far from an abstract search for absolute truth in Plato's cave, life would be revealed as a dynamic contest of embodied contingent truths, a struggle between the body and the inherently partial world it can sense. As Heraclitus said, "the way upward and downward are the same."

Acknowledgements. Peter Bierhorst, Mike Skiba, Ella Hoeppner, Drew Schimmel, David Gorin, Anna Viertel, Brett Hall, Jake Moritz, Johnathan Bard, Barrie McClune, Matt Roe, Ryan Moragas, James Collins, Laurence Lowe, Denis Noble.

References

1. Zrimec, J., et al.: Deep learning suggests that gene expression is encoded in all parts of a co-evolving interacting gene regulatory structure. Nat. Commun. **11**(1), 6141 (2020)
2. Crews, D.: Sex determination: where environment and genetics meet. Evol. Dev. **5**(1), 50–55 (2003)
3. Noble, D.: Lecture to Cancer and Evolution Symposium, Cellular Darwinism: Regulatory Networks, Stochasticity, and Selection in Cancer Development (2020)
4. Feytmans, E., Noble, D., Peitsch, M.: Genome size and numbers of biological functions. Trans. Comput. Syst. Biol. **1**, 44–49 (2005)
5. Noble, D.: The Music of Life: Biology Beyond the Genome, vol. xiii, p. 153. Oxford University Press, Oxford, New York (2006)
6. Bennett, T.: How Many Particles are in the Observable Universe? Popular Mechanics online (2017)
7. Sender, R., Fuchs, S., Milo, R.: Revised estimates for the number of human and bacteria cells in the body. PLoS Biol. **14**(8), e1002533 (2016)
8. Zoetendal, E.G., et al.: The human small intestinal microbiota is driven by rapid uptake and conversion of simple carbohydrates. ISME J. **6**(7), 1415–1426 (2012)
9. Kostic, A.D., et al.: The dynamics of the human infant gut microbiome in development and in progression toward type 1 diabetes. Cell Host Microbe **17**(2), 260–273 (2015)
10. Smith, P.M., et al.: The microbial metabolites, short-chain fatty acids, regulate colonic Treg cell homeostasis. Science **341**(6145), 569–573 (2013)
11. Desbonnet, L., et al.: Microbiota is essential for social development in the mouse. Mol. Psychiatry **19**(2), 146–148 (2014)
12. Sudo, N., et al.: Postnatal microbial colonization programs the hypothalamic-pituitary-adrenal system for stress response in mice. J. Physiol. **558**(Pt 1), 263–275 (2004)
13. Goodrich, J.K., et al.: Genetic determinants of the gut microbiome in UK twins. Cell Host Microbe **19**(5), 731–743 (2016)
14. Dethlefsen, L., McFall-Ngai, M., Relman, D.A.: An ecological and evolutionary perspective on human-microbe mutualism and disease. Nature **449**(7164), 811–818 (2007)
15. Sagan, L.: On the origin of mitosing cells. J. Theor. Biol. **14**, 255–274 (1967)
16. Douglas, A.E.: Fundamentals of Microbiome Science: How Microbes Shape Animal Biology, vol. viii, p. 236. Princeton University Press, Princeton (2018)
17. David, L.A., et al.: Diet rapidly and reproducibly alters the human gut microbiome. Nature **505**(7484), 559–563 (2014)
18. Faith, J.J., et al.: The long-term stability of the human gut microbiota. Science **341**(6141), 1237439 (2013)
19. Xie, Y., et al.: New insights into the circadian rhythm and its related diseases. Front. Physiol. **10**, 682 (2019)
20. Uexküll, J.V.: Streifzüge durch die Umwelten von Tieren und Menschen. Springer, Verlag von Julius (1934)
21. Hillenmeyer, M.E., et al.: The chemical genomic portrait of yeast: uncovering a phenotype for all genes. Science **320**(5874), 362–365 (2008)
22. Maturana, H.R., Varela, F.J.: The Tree of Knowledge: The Biological Roots of Human Understanding, p. 269. Rev. ed. (1992)
23. Shannon, C.: A mathematical theory of communication. Bell Syst. Tech. J. **27**, 379–423, 623–656 (1948)
24. Strogatz, S.H.: Sync: The Emerging Science of Spontaneous Order, vol. viii, 1st edn., p. 338. Hyperion, New York (2003)

25. UCLA: YouTube video Spontanous Synchronization (2013). https://www.youtube.com/watch?v=T58lGKREubo
26. Harris, T.C., de Rooij, R., Kuhl, E.: The shrinking brain: cerebral atrophy following traumatic brain injury. Ann. Biomed. Eng. **47**(9), 1941–1959 (2018). https://doi.org/10.1007/s10439-018-02148-2
27. Royo, J., Aujard, F., Pifferi, F.: Daily torpor and sleep in a non-human primate, the gray mouse lemur (Microcebus murinus). Front. Neuroanat. **13**, 87 (2019)
28. Frank, M.G., et al.: A preliminary analysis of sleep-like states in the cuttlefish Sepia officinalis. PLoS One **7**(6), e38125 (2012)
29. Libourel, P.A., et al.: Partial homologies between sleep states in lizards, mammals, and birds suggest a complex evolution of sleep states in amniotes. PLoS Biol. **16**(10), e2005982 (2018)
30. Deutsch, D.: The Beginning of Infinity: Explanations that Transform the World, vol. vii, p. 487. Viking, New York (2011)
31. Bachatene, L., et al.: Electrophysiological and firing properties of neurons: Categorizing soloists and choristers in primary visual cortex. Neurosci. Lett. **604**, 103–108 (2015)
32. Pearl, J., Mackenzie, D.: The Book of Why: The New Science of Cause and Effect, vol. x, 1st edn., p. 418. Basic Books, New York (2018)
33. Nietzsche, F.: On Truth and Lies in a Nonmoral Sense (1896)
34. Bostrom, N.: Superintelligence: Paths, Dangers, Strategies, vol. xvi, 1st edn., p. 328. Oxford University Press, Oxford (2014)