

Is information the other face of causation in biological systems?

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Abstract: Is information the other face of causation? This issue cannot be clarified without discussing how these both are related to physical laws, logic, computation, networks, bio-signaling, and the mind-body problem. The relation between information and causation is also intrinsically linked to many other concepts in complex systems theory such as emergence, self-organization, synergy, criticality, and hierarchy, which in turn involve various notions such as observer-dependence, dimensionality reduction, and especially downward causation. A canonical example proposed for downward causation is the collective behavior of the whole system at a macroscale that may affect the behavior of each its member at a microscale. In neuroscience, downward causation is suggested as a strong candidate to account for mental causation (free will). However, this would be possible only on the condition that information might have causal power. After introducing the Causal Equivalence Principle expanding the relativity principle for coarse-grained and fine-grained linear causal chains, and a set-theoretical definition of multiscale nested hierarchy composed of modular \subset -chains, it is shown that downward causation can be spurious. It emerges only in the eyes of an observer, though, due to information that could not be obtained by “looking” exclusively at the behavior of a system at a microscale. On the other hand, since biological systems are hierarchically organized, this information gain is indicative of how information can be a function of scale in these systems and a prerequisite for scale-dependent emergence of cognition and consciousness in neural networks.

Keywords: causal chains; information; hierarchy; emergence; self-organization; downward causation; coarse-graining

1. Introduction

On any account – physical, biological, statistical, or philosophical – causation is about a canonical cause-effect relationship. Meanwhile, in the relevant literature, “cause” and “effect” both are intuitive concepts. These have never been properly defined excepting that a notion of causation requires that causes and effects are well-defined local factors and that there is an asymmetry in determination of their relationship (Hitchcock 2007; Kutach 2013). To put it another way, the local factors are observable events, whereas the relationship between them is time-irreversible. The latter seems to immediately divorce causation from fundamental physical laws that are time-reversible. Physical laws cannot thus be called “causal” in respect to the arrow of time. Instead, it is usually said that everything in the universe evolves causally by physical laws (which, in principle, might work in the reverse order as well). Thus, some intrinsic intimacy between causation and physical laws is still preserved, though not explained. For example, one might argue that causal order is well compatible with the second law of thermodynamics which also respects the arrow of time. But this law is rather an exclusion from the general picture. There must be something that links our notion of causation with fundamental laws.

First of all, the laws of physics, as we know them, are extracted from multiple observations of physical regularities, and then inferred logically. In particular, it explains why there is also a striking resemblance between our notion of causation and formal logic that is somehow inherent to us (i.e., to neural networks “causally made” in our brains). The cause-effect relationship is very similar to how we make logical inference: $a, a \rightarrow b$, hence b . Since the very laws are based on observation of events as these are naturally presented to us in everyday life, a point of their convergence can indeed be found. It is that both our notion of causation and our making logical inference are scale-independent.

Fundamental laws, e.g., conservation laws or Newton laws are formulated with no respect to a spatial scale – whether it is an atomic or a cosmological scale. Regardless of a scale, the laws will be universally applicable to any system of interest. Accordingly, general equations based on these laws will be scale-independent as well. Even the Schrödinger equation – as an extreme example – designed to describe the evolution of quantum system can (though arguably) be applied to the whole universe (Everett 1957; t’ Hooft 2016). This is just the way where causation acquires the property of scale-independence: we believe that even at these two extreme scales causation will be valid. Mathematical models do not specify exactly what scale of description they refer to. Causation is, thus, universal property of matter at any scale of observation and mathematical description.

Its scale-independence becomes trivial by realizing that the concept of causation is inferred by us from observation of events. At the same time, we agree the universe with all its stuff and laws exists independently of our observation. Causation analyzed in this paper is about phenomenal events resulting from temporal dynamics of systems, governed by physical laws, not about statistical correlations in controlled interventions (Pearl 2009; Woodward 2003), or proximate and ultimate causes in Mayr’s (1961) dichotomy, or four causes (material, formal, effective, and final) in Aristotelian classification, applied then to biological mechanisms (Rosen 1991; Hofmeyr 2017; Farnsworth 2022).

But what is event? At what scale do events appear? We equally call “event” very different things – be it a wavefunction collapse, a fired neuron in the brain, or the explosion of a supernova. Even the whole universe is supposed to appear from a single event, the big bang. As being observer-dependent, the notion of event depends on the spatiotemporal resolution an observer can provide. It happens that events are ubiquitous in the universe from quantum to cosmological scales. They are scale-independent like fundamental laws.

However, their phenomenal nature can generate much confusion. A collision of two bodies – whether it is atoms or planets – brings about an event at a corresponding scale of observation. Thus, we do not ascribe any particular scale to our notion of event, thereby making it a scale-independent phenomenon. If one strikes fire from flint at some moment t , it is a physical event that just occurred at that time. However, if one says that

there is no smoke without fire, one does not refer to an event but only to one's logical (counterfactual) reasoning. At the best, "smoke" means a continuous physical process which indeed can be considered as a temporal succession of events at a corresponding (atomic) scale. Thus, it is not correct to say that fire is a cause of smoke beyond an appropriate frame of reference.

On the other hand, as noted above, there is a resemblance between causation and laws of logic as these are inferred from mental reasoning in very abstract ways. These laws are also scale- and time-independent like physical laws to which they naturally apply. However, historically, laws of logic, which are somehow inherent to our mental reasoning, precede the formulation of physical laws. Strictly speaking, an immediate observation of two successive events in nature is not sufficient to establish a cause-effect relation between them, unless the logical inference is involved. When their causal relationship is experimentally confirmed (e.g., via controlled interventions), physical laws can be proposed for generalization of successive events observable many times. That explains why physical laws have historically been formulated as scale- and time-independent. Their universality is only a byproduct of logical inference from observable events, consistent with a successful theory of physical reality.

The next step in evolving science was the appearance of information theory and computer science with its logic gates. These both in turn had given rise to machine learning and artificial intelligence. In particular, they have also shed light on why logic and information are intrinsic to our consciousness and cognition emerging both from structure of neural networks and their dynamics over time. Nonetheless, within those entangled relationships between observed phenomena and natural sciences, the relation between causation and information will remain most puzzling (Figure 1).

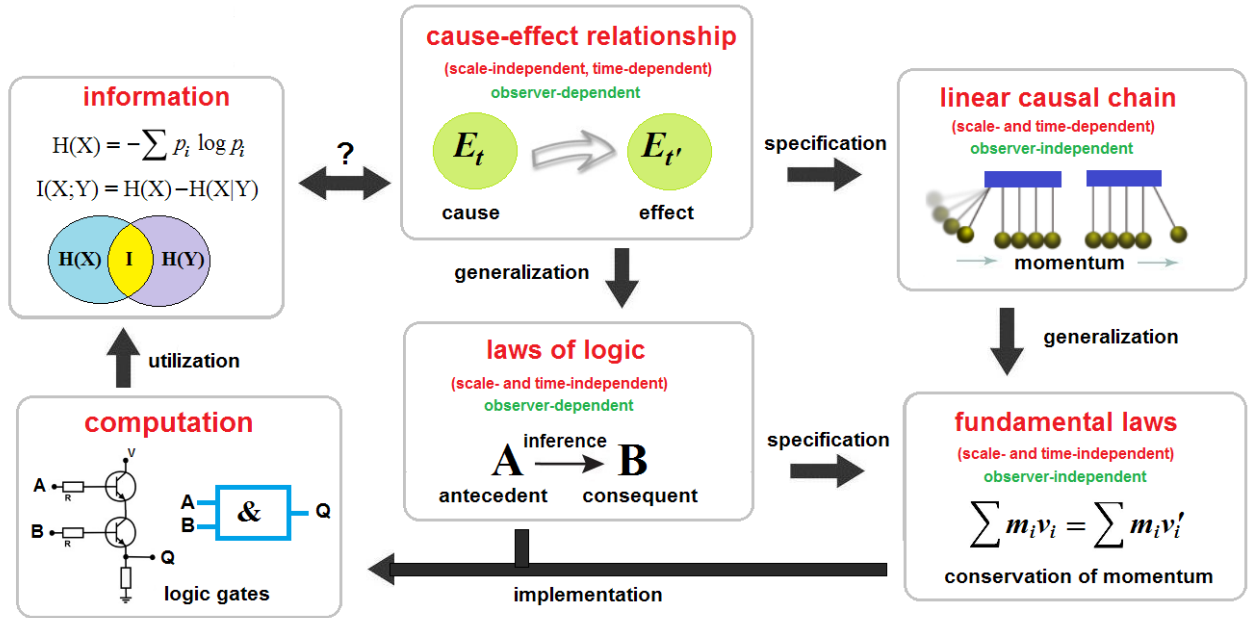


Figure 1. Causation-information relationship. The comparison between fundamental notions is made in terms of their dependence on spatial scales, the arrow of time, and observation, with the bold (black) arrows indicating how they are interconnected.

In fact, our intuitive notion of causation is not innate, but derived from information we gain by observing events at various scales across the world. Is then information the other face of causation? This issue, involving events, logic, physical laws, and computation, is debated over decades (Schrodinger 1944; Maturana and

Varela, 1972; Jablonka and Szathmáry, 1995; Landauer 1996; Maynard Smith 2002; Haken 2006; Kauffman 1995; Corning 2012; Rosas 2018) and even termed “the hard problem” of life (Walker and Davies 2016) because life seems to be distinct from other physical systems in how information processed by biological systems affects the causal world (Farnsworth et al. 2013). The problem was encapsulated by Davies (2019) in the slogan: Matter + Information = Life. This raises the question about the status of information not as a concept but as a physical entity. Does it imply that information can affect matter, or even underlies physical reality (Wheeler’s “It from bit”)? Can information have its own causal power in biological systems?

The aim of this paper is to show that information has not its own causal power besides that of matter. Intuitively, information theory (coupled with thermodynamics, given their mathematical relationship) should be derived from causation theory we are lacking now. The rationale is based on two arguments:

1. Causation can produce (and erase) information, but causal chains cannot go across spatial scales.
2. Assuming that information can flow over many spatiotemporal scales, it cannot produce causation.

Information can be collected, stored, transmitted, and copied, but causation cannot. In biology, the organism develops from a parent cell through cell-division cycles that also multiply information encoded in genes. Thus, information can naturally spread over scales. Does it mean that causation involved in these processes can go over scales as well? The concept of causation has different meanings in physics, biology, statistics, and philosophy, all in their own right but sometimes incompatible. So, causation in Relativity has little to do with four classes of causes (material, formal, effective, and final) in Aristotelian classification, adopted some biologists, and these both in turn have little to do with causation in statistics.

2. Causation and Causal Chains

2.1. Cause and reason

The concept of “cause” as a physical event must not be confused with the concept of “reason” that covertly dominates in philosophical writings, statistical analysis, and causal modeling. A physical cause is a unique event that happens in some point of spacetime. A reason is a general explanation, inferred from the observation of many particular events, unified then by their similarity. Humans can observe a sunrise over millennia and make various (geocentric or heliocentric) inferences about its physical reason, but the sunrise is not the same event every day. We can never return to the same event in time. The primary difference between cause and reason is the fact that cause is an event that directly leads to something, whereas reason is a logical explanation for why something happened in the past and can happen in the future. What follows is that causal reasoning requires counterfactuals of the sort “what if” while every particular cause occurs in the observer’s present (“just now”) and is unique in the physical world that has no counterfactual worlds. Thus, we must distinguish between an ontological *cause* (a matter of being) and an epistemic *reason* (a matter of knowledge) inferred logically.

In fact, the causal modeling literature often confuses “cause” and “reason” inferred from repeated observations with an explanatory purpose. Clearly, if it can be predicted that the occurrence of A always entails the occurrence of B under controlled conditions, there is a causal relationship between them (though, as stated above, if A and B are events, these can never occur twice over time). Thus, a reason can propose a reliable logical (time-independent) explanation of why a phenomenon is so but the explanation is not the way of how causal chains had produced it by going on their own by physical laws over time. A canonical example of counterfactual reasoning is the chicken-egg dilemma where each part is the reason but not the physical cause for the other. One possible solution to the chicken-egg problem in modern biochemistry is offered by the discovery of ribozymes, which could potentially code for their own replication and serve as catalysts for their

replication as well. Whatever ultimate solution biology can suggest in the future, we believe that evolution had somehow solved this problem without resorting to instantaneous causal loops or retrocausation (like that in the grandfather paradox).

In evolutionary biology, the phenotypical traits of a species, e.g., their size, weight, form and other morphological features had been selected as most fitted to the environment they inhabit. For example, the aerial environment requires a lightweight body to allow birds to fly (Branchi 2022). Does this reason explain causal chains that are solely responsible for evolving the bird body, or does it confuse logic with causation? From the perspective of reason, one can speak of environmental (large-scale) constraints acting on organisms in a downward fashion (Noble et al. 2019; Ellis 2020). From the ontological perspective, causal chains that produced a lightweight body had been favored by natural selection over the evolutionary timescale without requiring downward causation.

Another more striking example of confusing physical causation (its ontology) with counterfactual reasoning in statistical analysis of causation is saying that smoking cigarettes can cause lung cancer. None of them is an event. The former is a bad habit, while the latter is a persistent state of health, both related to a particular individual. On a strict account, thus, causal analysis must distinguish between events as actual (unique) phenomena and counterfactuals as these are involved in causal reasoning (Pearl 2009), which identifies causation with the effects of controlled interventions. In particular, Ellis discusses the relation between smoking and lung cancer ontologically by arguing: “It can certainly be redescribed at the physics level, but the key concepts in the correlation - smoking, cancer - cannot. Therefore, starting off with an initial state described at the microphysics level, one cannot even in principle determine the probabilities of cancer occurring on the basis of those variables alone, let alone when death will occur as a result of the cancer, because death also cannot be described at that level” (Ellis 2020).

Ellis suggests this argument to somehow account for downward causation, based on the idea that higher level (large-scale) events or entities constrain, modify or form a context for the lower level events or entities (Auletta et al. 2008). But it is not the case. What is shown is that reductionism is not valid in biology, neuroscience, and social sciences. It is impossible to observe living systems and their large-scale properties at the atomic scale. There are only interacting atoms at that scale. A hypothetical observer, knowing only the microphysics level, might scarcely predict that one or another pool of atoms packed in some configuration generates an organism. Moreover, that “atomic” observer might not have even a guess about the very existence of organisms at that scale (not mention smoking, cancer, and death), because organisms are complex systems that emerge at larger scales.

The world around us is full of regularities we use for counterfactual reasoning to discover the physical causes of events (e.g., sunrise whose *reason* is the rotation of earth around its own axis). There appears room for confusing “cause” as a unique event in spacetime with “reason” inferred logically from regularities observed many times. No doubt, any physical reason must necessarily be causally implemented, but reason and cause are not the same. In practice, a unique event is of little use to us, and we are mostly interested in discovering reasons when we make causal analysis of biological systems. When we ask how biological systems adapt to the environment and how they had evolved over time, e.g., in Mayr’s dichotomy of proximate/ultimate causation, is it about cause or about reason? In causal analysis, made in the context of function and purpose and confusing causal linear chains with counterfactual reasoning, circular causation for feedback control, large-scale environmental constraints, and downward causation become admissible. On a strict physical account, however, they are either impossible or problematic.

2.2. Causal chains in spacetime

A natural way to specify the concept of cause is to adopt the formal approach to causation, which is now canonical in the Causal Set Approach that has a long history in physics (for review see e.g. Surya 2019). This is determined by the very nature of spacetime in Relativity. In Lorentzian manifolds, in particular, in a flat Minkowski space, the causal relations are defined over events. First of all, all events are unique as occur at particular points of spacetime to which the past and future lightcones apply. These determine the absolute past and absolute future of an event. The events that are causally linked have a timelike interval between them, where “timelike” means that the speed of causal action is less than the speed of light. Two events separated by a spacelike interval are independent as the causal action between them would violate the second principle of Relativity (be faster than light). These causal relations give the causal structure of a spacetime. This is summarized in the slogan: Causal Order + Spacetime Volume = Lorentzian Geometry (Bombelli et al. 1987).

Theoretically, all causal chains with timelike links originate from the singular event in the big bang, and pervade the whole spacetime inside the future lightcone of this event. Thus, these chains are extracted from the global causal set, restricted only by the global horizon of the universe (Figure 2). The events, which are separated by spacelike intervals, belong to different chains but have, at least, one common cause in the big bang. If some event C is caused by two (or more) events A and B, there are two (or more) different chains that converge to C at some point of spacetime, where C occurs. A chain containing C can also bifurcate if C causes two mutually independent (spacelike separated) effects A and B.

Mathematically, the Causal Set Approach is based on the fact that the lightcone in a Minkowski space forms a set whose order relation is a usual (transitive and irreflexive) causal order (Sorkin 1991). Since all observers themselves are placed within the global lightcone, their particular position at a given moment of time can be depicted by a lightcone, attached then to an arbitrary frame of reference (where their observations and measurements will be made).

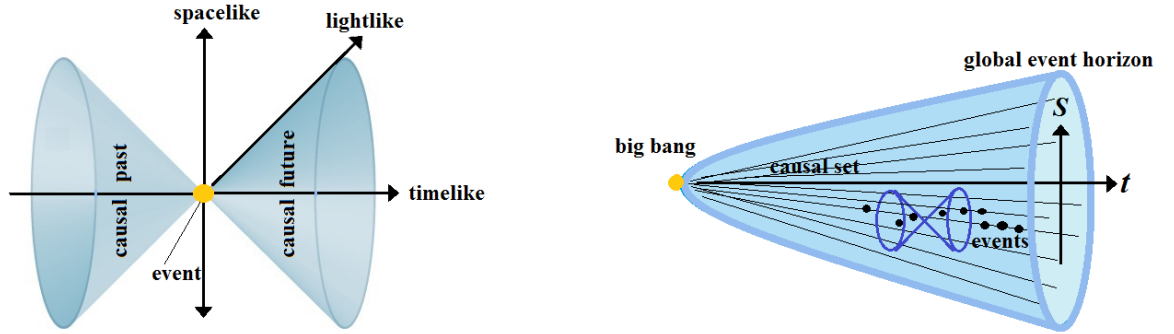


Figure 2. In Relativity, the past and the future lightcones of an event, associated with a particular frame of reference, uniquely determine its causal past and causal future. Any event inside the past lightcone might affect this event, which in turn might affect any event inside its future lightcone. Within the lightcone, only events with timelike intervals between them can be causally connected. Events with spacelike intervals cannot be so connected since the interaction between them would then be instantaneous (faster than the speed of light). In big bang cosmology, all causal chains originate from the big bang, thereby making the universe causally closed.

Definition. A causal set is a partially ordered set $S = (N, <)$ where N is a set of events in one’s observation, with a binary relation $<$ for causal chains, which satisfies the two conditions:

- (i) transitivity: $(\forall x, y, z \in N) x < y \& y < z \Rightarrow x < z$;
- (ii) irreflexivity: $(\forall x \in N)(x \nless x)$.

(1)

The causal set S can be viewed as a directed acyclic graph $G = (N, E)$, where the set of edges $E \subseteq N \times N$ symbolizes a timelike links $<$, with a “parent-descendant” relationship. Despite the similarity of formalisms, the interpretation of the causal set as a graph is totally separate and different from Pearl’s (2009) approach to interpret probability distributions on G as representations of causal structure in counterfactual reasoning. The G is then turned into a Bayesian network by ascribing random variables to nodes, with edges that represent the conditional probability for the variables.

In particular, circular causation, discussed in the context of feedback in networks or in functional space of biological mechanisms, is impossible in the physical spacetime, where a cause precedes an effect. Accordingly, causal chains with timelike links are by definition irreflexive: instantaneous causal loop or retrocausation are strongly forbidden. Functional circular causation can be admissible in counterfactual reasoning when applied to bodies or processes (separate chains), schematized as $A \rightarrow B \rightarrow C \rightarrow A$, where a chain A affects another chain B via some common event x , and so on. On the other hand, this schema, if applied to events properly, is always unfolded in time over a lag Δt as a causal chain $A(t) \rightarrow B(t + \Delta t) \rightarrow C(t + 2\Delta t)$. C cannot affect the event A which has already been in its past lightcone.

Now let $\{x_i\}_n$ be a segment of length n for an arbitrary causal chain in $S = (N, <)$, where x_i and x_{i+n-1} are the first and the last events of the segment respectively. Let the segment be compressed as a pair $x_i < x_{i+n-1}$ by neglecting all the intermediate events. Intuitively, we reduce a temporal resolution of our observations without loss of causal relationship. Again, these compressed segments in S will still satisfy the above conditions (i) and (ii), i.e., they themselves are transitive and irreflexive:

$$\{x_i\}_n \nless \{x_i\}_n \quad (2)$$

The compression of segments of a chain with timelike links changes only the temporal scale but not a spatial one. In practice, experiments we make have a certain temporal resolution going over some temporal lag Δt conditioned by our observations and measurement devices. But this alone is not sufficient, because our measurements are also made over many variables (molecules, cells, brain domains, and so on) to study the dynamics of a system of interest. It implies coarse-graining that requires both temporal and spatial compression.

Indeed, the spatial compression of S is also possible due to simultaneity. Simultaneous events (with spacelike links between them) cannot be causally (timelike) connected for the simple reason that a cause should necessarily precedes an event. Formally, a causal set S can easily be represented as a multilayer structure Σ by endowing it with a set L of layers, $\Sigma = (S, L, <)$. In the spatiotemporal context, the relation $<$ means the temporal order, where each layer $l \in L$ contains all events with spacelike intervals in Σ at a moment t . Thus, the layers provide a partition of S by temporal slices $\{l\}_t$, each consisting of *causally* independent events, i.e. any two of them belong to different causal chains. Theoretically, each slice comprises all simultaneous events inside the lightcone, associated with a certain observer-dependent frame of reference. In practice, this is bounded by us to the events we are mostly interested within the scope of our observation.

Again, the conditions (i) and (ii) are preserved: the relation $<$ over slices $\{l\}_t$ will still be irreflexive and transitive over some unitary lag 1 (ideally, related to dt),

$$\{l\}_{t-1} < \{l\}_t < \{l\}_{t+1} \quad (3)$$

Thus, both temporal and spatial compression (presented within the lightcone by “horizontal” and “vertical” lines respectively) can embrace the causal set S completely, thereby making coarse-graining scientifically legitimate. Importantly, they are not applied to a particular system but to spacetime itself. This “spacetime compression” provides the general formalization that explains why causal chains are scale-independent as depending on the events that by their phenomenological conceptualization can be observed at any scale (from quantum to cosmological).

This scale-independence of causal chains can be formulated by analogy with the equivalence of all inertial frames of reference in Relativity. The first principle of special relativity postulates that the laws by which the states of a physical system undergo change are independent of how a particular frame of reference is chosen. Observations made in one inertial frame can be converted to observations made in another frame by the Lorentz transformation as related to the speed of light. However, this does not specify at what scale observations should be made, implicitly assuming that observers initially agree on the scale of description and even on how exactly to make measurement. However, it does not imply that the fundamental laws of nature are scale-dependent and different measurements can violate them. Likewise, causation remains invariant across spatiotemporal scales as related to the laws governing the behavior of physical systems regardless of their size.

The causal equivalence principle (CEP). *Coarse-grained and fine-grained spatiotemporal variables must yield the same dynamics and/or make consistent predictions on the dynamics of a system of interest, excluding the scope of detail.*

The CEP is an expansion (or a specification) of the relativity principle: not only all frames of reference are equivalent but all scales of reference in the frames are equivalent. In other words, the CEP argues for a stratification of causation by spatiotemporal scales. Intuitively, one must make a difference between events with respect to a scale of observation to detect linear causal chains. There cannot be a cause-effect relationship between scales, for example, between an atom and a planet. Although we can theoretically consider a collision between these two, there is no “atom-planet” causal relationship but only a collision of a free atom with one of trillions of atoms the planet consists of. Another example from physics is the neutrino, a famously causally inert particle that can pass through a planet without having interaction with normal matter. Whatever reason of that can be, it can be said that this particle does not produce linear causal chains with other particles at the subatomic scale (let alone larger scales).

Physical entities in the universe, their existence and properties do not depend on observation. Causation is a ubiquitous property of matter’s behavior in spacetime. We find events and their causal relationships everywhere in nature. Accordingly, we can grain causal chains of events at any scale of description without affecting causation itself. Coarse-grained and fine-grained descriptions are like different frames of reference in special relativity, where spatiotemporal dimensionality reduction plays the role similar to that of the Lorentz transformation, which is a linear transformation that connects the spatial and temporal coordinates of an event as measured by an observer in each frame. Likewise, spatiotemporal dimensionality reduction transforms the linear causal chains at one scale to the linear causal chains at another scale (Figure 3A). In fact, the CEP amounts to a trivial statement: the temporal evolution of a system does not depend on the scale of description.

The first corollary is that not only the microscale is causally closed (as reductionism claims), but every scale must also be. Thus, the CEP is not reductionist principle. Reductionism requires a privileged scale for causation without explaining why the concept of causation, inferred by us from observation of events in different scales, should obey it, whereas the very state of affairs tells us that every science is in its right to study causal processes of a system of its interest at the scale that is most relevant to the size and dynamics of the system. As noted above, biological properties of living systems (e.g., smoking, cancer, death) could not even reasonably be

described at the atomic scale. Ironically speaking, an observer, knowing only the atomic (or quantum) scale, could not in principle become a biologist.

The second corollary entails that genuine (linear) causal chains with timelike links are valid only at the same scale of observation/description. Causal chains of one scale cannot interfere in causal chains of another scale. According to the relativity principle, if two observers in different frames of reference measure the momentum of a collision of two bodies, it does not double the momentum. According to the CEP, the observations of a system made at different “scales of reference” do not multiply causation. Thus, causal chains across scales upward and downward are forbidden.

The third corollary argues that spatiotemporal dimensionality reduction of observations via coarse-graining many microstates (and their associated variables) to single macrostates is not only methodologically legitimate but also ontologically valid. In the physical world, causal linear chains unfold on their own (theoretically, since the big bang) across the whole spacetime. They permeate both the multiscale structure of a system and its environment at all relevant scales. Thus, observations of the system can be ontologically valid not only in any frame of reference but also at any scale with that frame.

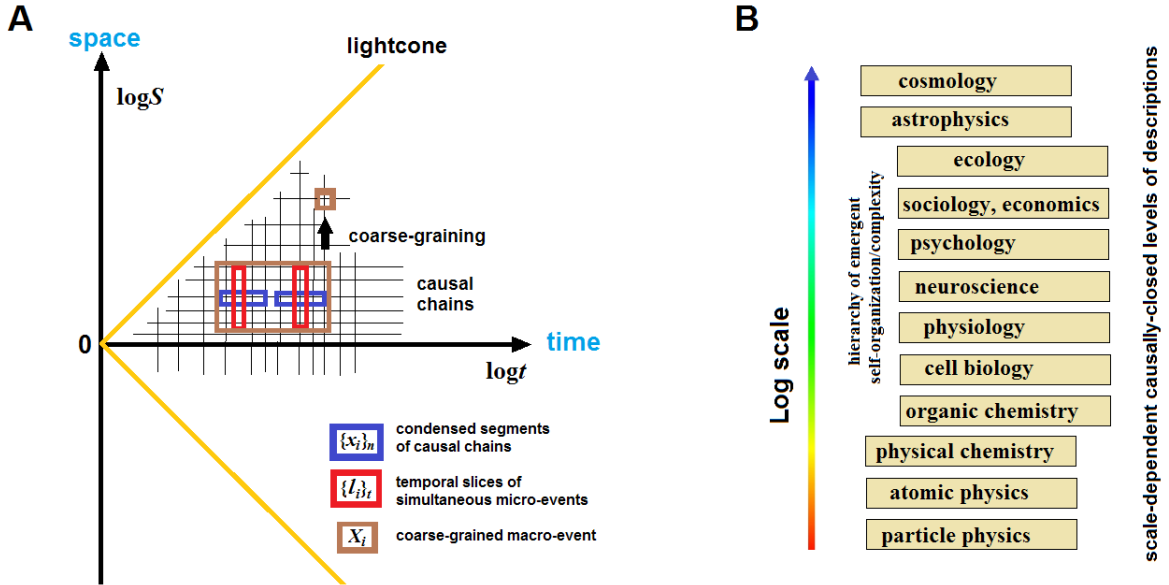


Figure 3. (A) Coarse-graining as the spacetime compression. Here, condensed segments of causal chains with timelike intervals are depicted as horizontal blue boxes, and temporal slices of simultaneous (independent) micro-events with spacelike intervals are depicted as vertical red boxes. This schematic of spatiotemporal dimensionality reduction is illustrative since it does not correctly present spacetime. The segments and slices are represented in linear scale inside a future lightcone, associated with a particular observer (a frame of reference), whereas coarse-grained macro-events in a brown box above are referred to log scale. (B) The hierarchy of sciences. Unlike radical reductionism that holds that the microscopic (quantum?) scale is the only scale that is causally closed, the CEP argues that any scale separately provides the same causal closeness. For example, Newtonian mechanics is causally legitimate while being historically formulated without any knowledge about linear causal chains at the atomic or quantum scale. The CEP also takes into account that biological sciences deal with self-organizing (information-processing) systems to which physics is blind.

3. Reductionism, Holism, and Emergence

3.1. *Reductionism and the CEP*

In its most general form, reductionism is a physics-grounded postulate that the parts unilaterally determine the behavior of the whole. This is based on the three assumptions (Kim 1999): (i) once the microscale properties of a system are fixed, its macroscale properties are fixed too (supervenience); (ii) causal power resides fully at the microscale (micro-causal closure); and (iii) if all the causal work is done at the microscale, there is no room for any causal contribution at the macroscale (macro-causal exclusion).

Relativity holds that causal (temporal) order in all frames of reference, defined with respect to the speed of light, cannot be violated: if A causes (precedes) B in one frame, it is so in all frames. The speed of light has a privileged status, but nothing is said about a scale of observation. Thus, reductionism can still be admitted. The CEP clarifies this issue by asserting that causal linear chains at one scale of description must be consistent with causal linear chains at any other scale of description, with respect to the smallest scale of causal analysis. Ultimately, this smallest scale should be related to the Planck scale. This scale is fundamental physically in the sense that everything arises from it, but it has not a privileged status causally as soon as our notion of causation is entirely based on the observation of events. Although every particular event appears at a corresponding scale, the very notion of event is scale-independent as no privileged scale can be ascribed to this notion. Saying that the Planck scale is causally fundamental would resurrect reductionism the CEP has abandoned.

As stated above, it would be in principle impossible to explain large-scale phenomena of biological systems (e.g., smoking, cancer, and death) from a perspective of quantum interactions. In particular, to formulate a complete causal theory of consciousness at the ultimate scale of physics there should appear Laplacian demon capable of fixing an enormous set of quantum events and their configurations to trace the spontaneous emergence of consciousness in the brain hierarchy across the progression of scales. One question would still persist: Might that demon be able to observe these large-scale emergent phenomena of life?

On the other hand, it is practically impossible to study a system's structure and dynamics at the scales much larger than its own. For example, at the cosmological scale even millennia in humans' history on the planet they inhabit are negligibly small to make a difference. In practice, a preferable scale of causal analysis or, more generally, of any scientific research appears spontaneously as the elementary basis for a given system of interest. This implicitly involves the hierarchy of scales a system physically spans, beginning with the chosen elementary basis and referring to all the scales above the span as "environments." Within the span of a system, these scales are commonly divided into three: a microscale for the elementary basis, a macroscale for the upper bound, with a mesoscale placed between them.

In this sense, the CEP allows us to choose an elementary basis of description as most appropriate to a system of interest. For example, molecular biology operates on the molecular scale. In the case of neuroscience, the elementary basis is usually placed at the neuronal level where most significant causal events occur. The meso- and macro-scales are then related to modular interactions and to the whole-brain dynamics respectively. Accordingly, everything outside the brain is taken as the environment. In the case of cognitive psychology, most interested in mental states of individuals, the elementary basis is shifted up to the scale of brain regions and functional systems, whereas social psychology operates on the basis of personal interactions where the social environment takes the place of a macroscale. Sociology and economics go further to the scale of large human communities across the world.

3.2. *Holism and the CEP*

In contrast to reductionism, holism, grounded in biology, neuroscience, and social sciences, argues that the whole is more than its parts: once the importance of complexity is recognized, there is an additional difference between observations of system components at the microscale and observations of the system at the macroscale (Anderson 1972, Bar-Yam 2004).

Holism in turn is conditioned on the concept of emergence in its two “weak” or epistemic and “strong” or ontological versions (Chalmers 1995; Turkheimer et al. 2019). The former refers to the difficulty to understand the relationship between microscopic parts and their collective macroscopic behavior (Bedau 2002). The latter describes properties that are unique to the behavior of a system and cannot be identified through any observations of its parts (Sperry 1991). Strong emergence claims the appearance of genuinely novel system properties at some non-fundamental level, while weak emergence refers to greater explanatory or predictive power at some higher level of description.

Water is a weakly emergent property of hydroxides. Two or more hydroxide molecules are not sufficient to produce water. Their number must be large enough in order to cause emergent self-organization (very primitive but above Brownian motion in gas) that is responsible for fluidity and viscosity. Replace now water by a cellular substance, and then much more complex forms of emergent self-organization will appear there. From the evolutionary perspective, the very these forms had created a cell from scratch. Organisms composed of the sets of specialized cells represent more complex forms of emergent self-organization. Thus, the hierarchy of scales as these naturally unfold in spacetime provides the hierarchy of levels for more and more complex emergent self-organized systems. These in turn determine the classification of sciences, where physics spans two extreme spatiotemporal scales of observation, ranged from the quantum to the cosmological scale, but has a difficulty with explaining biological and social sciences (Figure 3B). In this sense, weak emergence can be specified via scale-dependent self-organization.

Strong emergence agrees that emergent phenomena are scale-dependent but requires causal power for large-scale phenomena. Ultimately, the crucial feature to discriminate between weak emergence and strong emergence is downward causation (Kim 1999). Otherwise the philosophical debate about their large-scale properties and the predictive power of lesser scales would lead us to a conclusion that the difference between the weak and strong forms of emergence may be in principle impossible (Corning 2012; Schmickl 2022). The CEP rejects downward causation and, hence, holism based on strong emergence, but not on weak emergence. As noted above, the observations, made at the atomic scale, could not even detect the existence of complex biological systems. Their emergent properties can be observed and described only at an appropriate scale. On the other hand, causation cannot be multiply accounted for by changing the scale of observation to allow for downward causation.

So, Bedau (2002) argues for the ordinary nature of downward causation by saying: “To see this, choose some micro piece of the macro effect and note that the macro cause is also responsible for the consequent changes in the micro piece of the macro effect.” If it would be true, both upward and downward causation were indeed ordinary and ubiquitous in nature. The CEP separates the scales of observation. For example, if we apply a force to a moving ball, we change causal chains at all scales the ball occupies. It follows from the homogeneity of spacetime over scales. The dynamics of a system and of its parts are continuous in spacetime: they all have its own past and future that do not vanish in one point and re-appear in another point. However, it does not affect microscales by downward causation, if we take into account that the macro-cause itself (derived from discretized events) can be represented by different, causally closed scales. Thus, when we intervene in a system, the very intervention can be decomposed at a microscale.

Recently, a series of papers (Rosas et al. 2020; Luppi et al. 2021; Mediano et al. 2022) has proposed to reconcile weak and strong emergence, thereby claiming to legitimize downward causation via information-based measures. This is made in the way very similar to that in Hoel et al. (2013), based on effective

information, where “macro beats micro.” However, their mathematical construct does not affect genuine linear causal chains (see the Discussion section). According to the CEP, all scales are causally closed so that both upward and downward causation will inevitably lead to double causation or what Kim (2016) called “overdetermination” (Figure 4A). At the same time, the CEP gives no support to both “micro-causal closure” and “macro-causal exclusion” advocated by Kim (1991).

3.3. Coarse-graining and the double causation fallacy

Notwithstanding that the elementary basis for a system of interest can be chosen, with the corresponding span of micro-, meso-, and macro-scales, causal analysis at the microscale can still impose prohibitive computational costs on the analysis. In science, dimensionality reduction via spatial stratification and temporal condensation of linear causal chains (Figure 3A) takes place in the form of coarse-grained variables. Generally, dimensionality reduction produces low-dimensional representations of high-dimensional data (at a microscale), where the representation is chosen to retain or highlight some meaningful properties of the original data. Here, dimensionality reduction is of our interest mainly with respect to how it relates to scale-dependent emergent phenomena in their weak and strong forms.

A canonical example of dimensionality reduction is statistical methods, such as principal component analysis, that provide a parsimonious description of statistical features of interest by discarding some aspects of the data as noise. These can result from redundant and spurious causal correlations. Another example of spatiotemporal or phenomenological dimensionality reduction is a phase-space plot when a set of variables

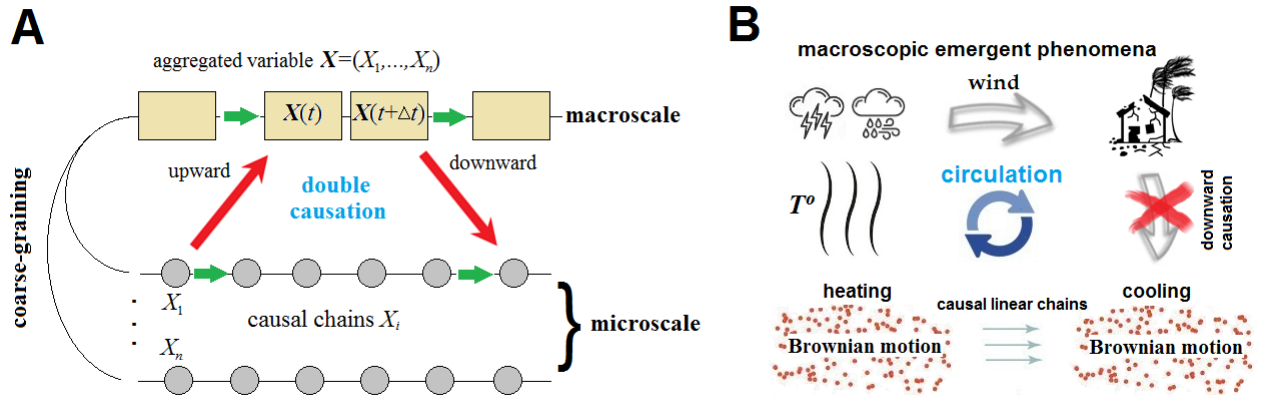


Figure 4. Coarse-graining and double causation. (A) Coarse-graining allows to reduce data from a high-dimensional space into a low-dimensional space by aggregating many micro-variables into a single macro-variable, modeled often probabilistically as Markov chains. In physical spacetime, these variables should represent causal chains, each evolving at a corresponding scale by its own cause-effect relation (via green arrows). Double causation arises if the temporal evolution of a chain at one scale is assumed to be affected by a chain from another scale (via red arrows). (B) Weather forecasting is a particular (relatively simple) example of coarse-graining. Macroscopic emergent phenomenon such as a hurricane cannot go over scales to destroy air and water molecules since causal chains remain valid only at the same scale of description. Here, circulation of air and water molecules via heating and cooling is a dynamical process, without involving upward or downward causation. Even circular causal loops at the same scale cannot be involved there if viewed in the context of physical cause. Circular causation can be admissible only in the context of

reason. In this context, Brownian motion, resulting from random interactions of particles, can be full of temporally unclosed loops.

associated with individuals in two (or more) populations is reduced to population counts, e.g., in Lotka-Volterra (prey-predator) or Wilson-Cowan (excitation-inhibition) models. Overall, any manipulations with data (as being extracted by discretizing events from some physical processes) can be viewed exclusively in the terms of spatiotemporal dimensional reduction imposed upon them by coarse-graining many (un)observable microstates to a single macrostate (Figure 4A).

Weather exemplifies macroscopic emergent phenomena, where coarse-graining becomes necessary in forecasting. These phenomena are caused ultimately by solar radiation that heats the surface of Earth. The heating results in an increase of temperature due to an increase in the average translational kinetic energy of air and water molecules. Thus, solar radiation produces macroscopic emergent phenomena such as wind, rain, and the ocean current which in turn move huge volumes of air and water across spacetime, causing hurricanes and storms. Is there multiscale causation, e.g., upward from solar photons to storms, or downward from hurricanes to air molecules? Although a hurricane can destroy macroscopic objects like buildings, both observed at the same scale of macro-causation, all causal chains arising there are of the same spatial scale. A hurricane cannot destroy air and water molecules in a downward fashion because it is just caused by their own collective motion (Figure 4B).

For chaotic systems such as weather the coarse-grained variables predict the local future of a system better than the variables of its fluctuating microscopic components because it requires fewer degrees of freedom. However, biological systems show complexity of another kind. In this case, coarse-graining not only allows to predict their behavior by observing and detecting macroscopic emergent properties of their self-organization, which could not be observed at a microscale taken for the elementary basis of observation. The next step in this direction is to recognize that biological information-processing systems themselves are like prediction machines that adapt to the environment via self-organization. Flack (2017) calls it “endogenous coarse-graining” and argues that downward causation can occur there from simple feedback when microscopic components tune behavior in response to estimates of collectively computed macroscopic properties.

Similarly, Ellis (2012) has proposed five different classes of downward causation: algorithmic downward causation; downward causation via non-adaptive information control, downward causation via adaptive selection, downward causation via adaptive information control, and intelligent downward causation. All of them are discussed in the context of a reason by involving structural constraints, feedback control, and circular causation. Clearly, the last class is explicitly suggested as the most advanced form of downward causation to account for mental causation or free will. Downward causation requires strong emergence while mental causation entails the mind-brain dualism. Historically, Descartes had postulated his mind-body dualism without explaining how consciousness might have causal power over its physical (neuronal) substrate. Probably, Descartes would be happy with downward causation to fill this gap in his dualism. This could be possible only on the condition that information produced by the brain has its own causal power.

Today many dominant theories of consciousness in neuroscience such as Integrated Information theory (IIT), Global Workspace Theory (GWT), and Predictive Processing theory (PPT) explicitly or implicitly assume that information can have causal power in the brain via downward causation that takes form of mental causation (free will). They all start with the general idea that consciousness is a large-scale emergent phenomenon of neural activity. Its particular state is some amount of information just processed by the brain at a given moment of time. However, this neuroscientific alternative to a nonmaterial soul of Cartesian philosophy of mind does not solve another crucial question. Theoretically, consciousness can be either active or passive (epiphenomenal). If consciousness is active, how can information govern the brain whose dynamics go causally over all relevant scales in parallel according to the CEP?

To account for mental causation IIT simply postulates a maximally irreducible cause-effect structure derived from integrated information which, in turn, is attributed to consciousness (Tononi 2008; Oizumi et al. 2014). In GWT, consciousness emerges from the global workspace and becomes active by broadcasting information from this global workspace downward to different local executive modules (Dehaene and Naccache 2001; Mashour et al. 2020). In PPT, active (Bayesian) inference takes the causal role of consciousness in the brain to minimize the surprisal/free energy of a system or to maximize mutual information between priors and posteriors (Friston 2008; Williford et al. 2018). Thus, the old problem of free will in philosophy of mind turns into the problem of downward causation in modern neuroscience. The latter only reformulates mind-body dualism by admitting downward causation into dualist ontology of causation and information.

4. Causation and Information

The claim that strongly emergent phenomena in biological systems require downward causation is implicitly based on the two ideas: (i) biological systems are information-processing, and (ii) information can produce causation. Indeed, biological systems send and receive physical signals that are carried out causally. In what sense can it then be said that the signals themselves carry causal information? Such statements are either something trivial or they require dualist ontology. In the first case, according to the CEP, there is a misinterpretation since the causal linear chains carrying that information simply produce new causal chains at the same scale, and they all go on their own by physical laws. Here “information” is involved by implicitly referring to an observer-dependent description of these causal chains.

Otherwise, in the case of dualist ontology, information acquires the status of a new (complementary) kind of causation (Varley and Hoel 2022). This leads us to the concept of downward (or top-down) causation debated over decades in philosophy of science (Campbell 1974; Ellis 2012; Ellis and Kopel 2019; Davies 2012; Noble 2012; Noble et al. 2019). On a closer examination, the concept of downward causation is entirely based on the assumption that biological information-processing systems bring a new force into play – information that is responsible for the emergence of self-organization or “cooperation” in evolutionary biology.

Let us consider it in detail. In Shannon theory, anything is a source of information if it has a number of possible states where one variable contains information about another provided that their states are statistically correlated. A signal carries more information about a source if its state is a better predictor of the source, and it carries less information otherwise. Shannon information is time-independent, i.e., symmetrical: both income and outcome carry information about each other in the sense that an effect can predict its own cause as well as the cause predicts effect, e.g., via mutual information. Of course, there can be used time-delayed mutual information; however, its use is already conditioned on both the arrow of time and causal order. Other statistical methods such as effective information, extracted from Pearl’s counterfactual interventionist modeling, Granger causality, or Transfer entropy go further and make use of *inferential* nature of information to refer to genuine causal chains.

Recall these genuine causal chains are linear, i.e., both scale- and time-dependent but observer-independent as going on their own by laws of nature. In contrast, information is observer-dependent just like the common notion of cause-effect relationships, being both extracted from observations of some events (Figure 1). The principled difference between information and genuine causation we are mainly interested here is that the former preserves neither scale- nor time-dependence. We apply then information measures to everything around us in a way as it is naturally presented to us, i.e., to our conscious experience, which is also self-evident to all of us. However, by doing it, we abandon the inferential (statistical) sense of Shannon information and make use of information in the anthropomorphic sense inherent to us and, probably, to all other conscious beings. The term “information” becomes controversial even in the context of biological systems where causal processes within cells and the whole-organism are treated in terms of information and executive programs

stored in the genes (Godfrey-Smith 2007; Skyrms 2010; Levy 2011). Perhaps, consciousness and information are equally mysterious notions that must not be applicable to any system alone but only from an observer-dependent perspective.

Indeed, unlike causation, information and consciousness are intrinsically linked. Many theories of consciousness argue that conscious experience is exactly information just causally processed by the brain at a particular moment of time (see the next section). In this context, saying that information can produce causation is equivalently to saying that consciousness has causal power over the brain. Clearly, it is sufficient to assume that information can produce causation in order to come eventually to postulating mental causation (free will) advocated by many prominent scientists (e.g., Pauli, Bohm, Eccles, Sperry, Kauffman, Ellis). Hence, denying the latter entails denying the former, including downward causation. Thus, “downward causation” is not merely a technical term. It requires mysterious metaphysics such as Pauli-Jung’s psyche-physics complementarity or Eccles-Popper’s interactionist dualism (Figure 5).

5. Upward and Downward Causation

The CEP not only rules out downward causation, upward causation is forbidden too. While the latter is the controversial hypothesis, the former is thought to be scientifically legitimate. Typically, upward (bottom-up) causation is taken to schematize the multilevel organization of sciences, based upon reductionism, where each higher level is subordinated to the lower level. In this classification, physics underlies chemistry, which underlies cell biology, which in turn determines physiology, and so on over life sciences up to cosmology. A common rule of the organization is this: as the lower level dynamics proceed, the corresponding coarse-grained higher level variables will change as a consequence of the lower level change.

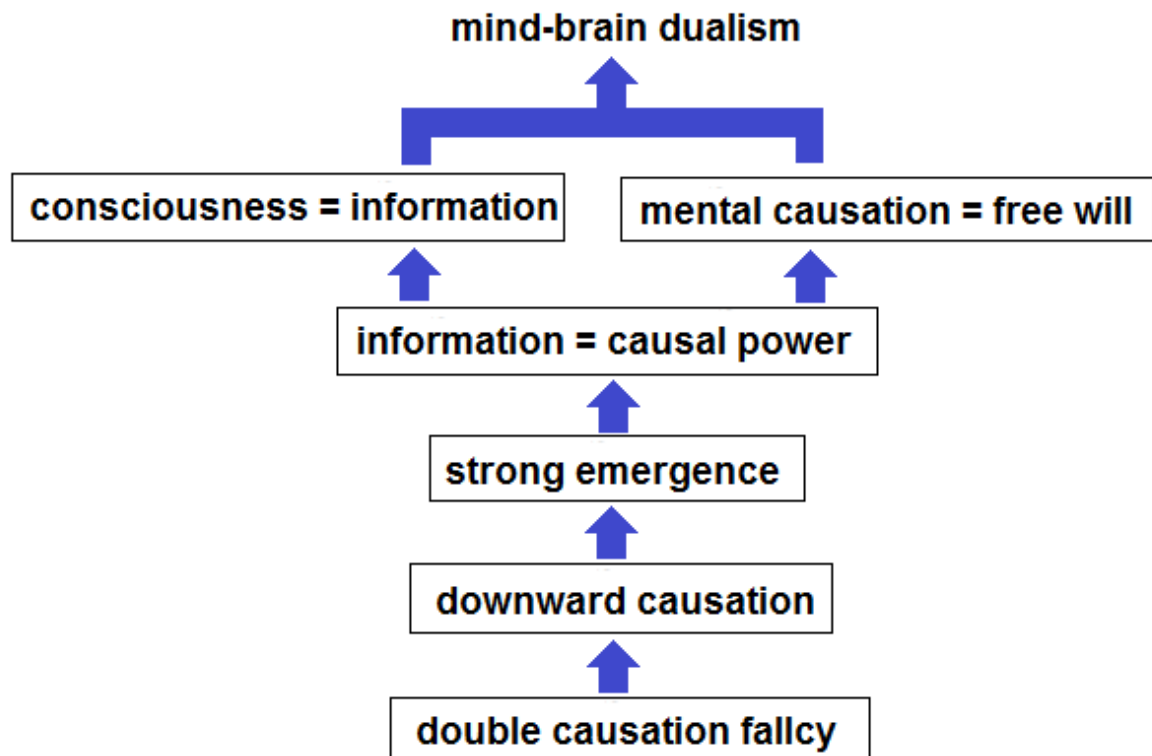


Figure 5. Dualist ontology. The diagram shows as the concepts – though different at first sight – are intrinsically linked.

This scheme can be laid on the hierarchy of emergent scale-dependent self-organization (Figure 3B). At each scale new more complex systems appear, governed by the same physical laws applied to linear causal chains at an appropriate scale of analysis. However, this hierarchy of self-organizing systems does not imply upward causation. Recall the CEP prohibits linear causal chains to go across scales up or down alike. In particular, this shows that assuming upward causation should instantly make its counterpart, downward causation, legitimate too. Here, the CEP agrees with the principle of Biological Relativity (Noble 2012), which argues that the both forms of causation should act simultaneously, thereby making upward causation alone inadequate in multiscale biological systems.

Biological relativity is the principle that there is a priori no privileged level of causation. Although being very similar to the CEP, biological relativity admits both upward and downward causation. Overall, their feasibility in multiscale systems is based on how spatial (and temporal) scales are stipulated by Noble et al. (2019). The difference between scales is presented in the form of “system-environment” division. Indeed, by the very meaning of these words, an environment is always spatially more than a system placed inside it. Their relation is naturally presented in biological systems over many scales, where, for example, cells are the environment for proteins, tissues or neural modules are the environment for cells or neurons, which in turn are placed within organs, and so on. Yet, this ‘mereological’ principle underlies all in mathematical foundations, e.g., in set theory, formal logic, topology, or graph theory.

But the principle is then somehow turned into an idea that while the mathematical equations (whatever those are) represent the dynamics of the components of the system, the initial and boundary conditions represent the historical and contextual factors without which no specific solutions to the equations would be possible (Noble et al. 2019). These conditions are commonly referred to as environmental. The feedback mechanisms of homeostasis are then proposed to account for both upward and downward causation. Upward causation is defined as the set of processes by which the elements at a lower scale interact and produce changes at higher scales. Downward causation is the set of (environmental) constraints imposed by the higher scales on the dynamics of a system through determining many of its initial and boundary conditions. Noble et al. draw then on genetic-epigenetic or evo-devo mechanisms like circular feedback mechanisms: while the genome exerts upward causation, it itself is influenced by the organism and its life-style experiences through extensive epigenetic control.

Remarkably, social scientists and philosophers often use the same form of “system-environment” division to explain downward causation by the aggregated high-level (macroscopic) variables of social systems, e.g., price, which may have an effect on individuals, associated with lower-level (microscopic) variables in those systems. The latter is, perhaps, the best example of how causation can be confused with information. Price is not a material body to produce physical events; it is only information individuals have received in their social environments. If one would carefully trace the ultimate physical ways the information was delivered to the individuals (via perceptual systems of their brain), one could find only linear causal chains there.

Of course, one might then speculate that these perceptual systems are involved in predictive (Bayesian) processing to minimize error (Clark 2013), coupled then with active inference (Friston 2008; Hohwy 2013). One might also refer to brain dynamics, which explore their attractor space to ignite the global workspace (Dehaene and Naccache 2001; Mashour et al. 2020) or to produce integrated information (Tononi 2008; Oizumi et al. 2014) with irreducible cause-effect power to account for mental (downward) causation. All these theories can be called “theories of strong emergence.” Their proponents argue that high-level variables can do main (if not all) causal work within a system (Hoel et al. 2013). This implies that stronger causal chains may exist at the macroscale rather than at the microscale. The idea that “macro beats micro” is obviously incompatible with

the CEP. All the scales within the span of a system of interest – from its elementary basis over micro-, meso-, and macro-scales to the environment – are ontologically equivalent and equally legitimate. They yield the same dynamics of the system. The only distinction between them is epistemic as expressed in the scope of detail.

At first sight, the principle of biological relativity is more moderate than the above theories as it argues for a balance between upward and downward causation. Their justification is based on the idea that biological systems are information-processing, yet adaptive systems unlike ordinary physical systems which have neither function nor purpose. However, this conceptual framework requires information to be causally effective in contrast to the position advocated here. It makes biological relativity yet another (though moderate) theory of strong emergence. Instead, the CET tells us that allowing information (which itself is carried causally) to produce complementary causation is tantamount to the double causation fallacy. A principled difference between weak and strong versions of emergence can thus depend on how these relate information to causation.

From this perspective, the argument for strong emergence can be the following. If information was causally ineffective, what advantages could biological (information-processing) systems obtain over ordinary non-living systems? Should there be any reason for evolving life? Clearly, it is a crucial argument that weak emergence must meet. The answer can be that not downward causation but quantum randomness plus classical (nonlinear) amplification is a core mechanism evolution had utilized to promote life and consciousness through “leaky” deterministic causal chains as phenomena of weak emergence (Yurchenko 2022a, 2022b).

Instead, we will focus on the (weakly) emergent patterns of autonomy and self-organization that are inherent to living systems but lacking in non-living physical systems. First, autonomy and self-organization imply function and purpose. There is no need to self-organize for ordinary non-functional and purposeless systems. Second, living systems are naturally conditioned on the “system-environment” division where a system as a whole can manifest its autonomy and self-organization within the environment while there is no autonomy for its parts within the system itself. A canonical example is the murmuration of starlings, each placed in the elementary basis of observation. As the behavior of each bird at the microscale depends on the behaviors of its neighbors in this elementary basis, there is no autonomy for them there. The trajectory of any single member would show only chaotic behavior. Autonomy and self-organization emerge spontaneously from their collective dynamics at the macroscale as if having a “life of its own” (Barnett and Seth 2021).

In this case, biological relativity or, more generally, all theories of strong emergence should argue that upward causation originates from the given elementary basis, whereas downward causation comes from the environment, or even that the flock’s self-organized “life of its own” can have causal power over any single starling. Is it the case? No. This form of strong emergence occurs only in the eyes of an observer where information generates an illusion of causal power acting on the flock. Varley, for example, calls this form of (weakly) emergent autonomy and self-organization, balancing on the edge between the flock and its environment, “flickering” (Varley 2023). It is enough for all the starlings to suddenly scatter in separate directions, each flying on its own (perhaps in response to a swooping hawk), and the flock’s autonomy and self-organization observable at the macroscale will vanish together with spurious upward/downward causation by merely breaking down to the elementary basis.

Of course, one can argue that the flock itself is a temporally unstable system, whereas every bird as an organism is temporally stable so that its autonomy and self-organization cannot be flickering. For example, in terms of biological relativity of Noble et al. (2019) one might consider the scenario where cancerous cells exert upward causation on the whole organism (causing eventually its death), while an unfavorable environment provides a macroscale variable to account for downward causation. On a closer examination, however, one would find that the unfavorable environment, e.g., radiation or chemical agents, acted on the cancerous cells via linear causal chains of the same scale. Likewise, these cells did affect the organism via the same linear chains at the cellular scale. Only by changing the scale of observation, one might then consider interpersonal relations of the cancer patient with their relatives and colleagues to track down the cause of their disease.

According to the CEP, all scales within the spatial span of a system of interest are ontologically valid, without, however, mixing them. A cell does not affect an organism, and the large-scale (ecological) environment of an organism does not affect its cells. A dangerous or favorable agent acts always at the same scale of causation.

All of this brings us to the question how autonomy and self-organization emerge in nature. In the literature, these both are usually related to a hierarchical organization of biological systems, without specifying, however, what exactly the hierarchical organization means. Most typically, the hierarchy is presented by a conceptual multilayer structure, with each layer conditioned on a corresponding scale. In other words, the hierarchical organization is merely referred to as a multiscale system. The complexity of its internal structure is primitively presented or even ignored as something implicitly obvious. Meanwhile, the main source of confusion with information and downward causation is just the internal structure of hierarchy.

6. Two Types of Hierarchy

The notion of hierarchy is often mentioned in the context of discussing complexity, emergence, and downward causation. 60 years ago, Herbert Simon (1962) argued in his essay “The architecture of complexity” that most complex systems, social, biological and physical, are hierarchically organized. He called them “nearly-decomposable” systems, where elements are densely connected into modules while preserving sparse inter-modular connectivity with nodes in other modules (Newman 2006). Hierarchy seems to be a universal property of the organization of biological systems. The examples span from ecosystems and social interactions to brain organization and cell structures. The main difficulty with the definition of hierarchy is that its conceptualization includes many descriptors such as order, level, branching, modularity, inclusion, overlapping, subordination, and self-similarity (scale-freeness), none of which alone captures either its complexity or the problem of its measure and origins (Arenas et al. 2008; Corominas-Murtra et al. 2013; Kivela et al. 2014).

After introducing a mathematical (set-theoretical) definition of hierarchy, we will restrict our analysis to the brain. The brain is thought to be one of the most complex physical systems in nature. Yet, unlike other complex systems, e.g., in biology where the applicability of “information” to molecular structures is debated, the neural networks are apparently information-processing systems. On the other hand, unlike social systems such as an ant colony or a human community, which are also information-processing, the brain has compact well-defined physical boundaries. All of these makes the brain perhaps most appropriate for studying a hierarchical organization of complex systems.

The brain can be initially defined as a set N of all its neurons (nodes) i . At this stage, there are no additional assumptions behind the idea that the brain is nothing but a set of neurons placed into the elementary basis of our research. When endowed with the physically necessary edges (links) between nodes, the N becomes a graph $G = (N, E)$. This provides a starting point for introducing many network measures (Rubinov and Sporns 2010; Lynn and Bassett 2019). Nevertheless, the graph does not capture the multiscale organization of networks to introduce a topological axis over spatial scales the brain occupies.

Now we define hierarchy as a mathematical ideal that captures many (if not all) descriptors mentioned above ((Yurchenko 2017) but also provides the multiscale model of the brain. The ideal Δ over N is by definition a structure closed under the unions (the upper bounds) and taking subsets of N . More formally, a structure Δ is an ideal if all its elements X and Y satisfy the requirements:

- (i) $X, Y \in \Delta \Rightarrow X \cup Y \in \Delta;$
 - (ii) $X \in \Delta \text{ \& } Y \subset X \Rightarrow Y \in \Delta.$
- (4)

Remarkably, an ideal emerges trivially if one will consider the powerset $\mathcal{P}(N)$ over any arbitrary set N of elements i , combined into all possible subsets X , $X \subseteq N$. What is of great importance is that a powerset $\mathcal{P}(N)$ can be mapped onto an upper semilattice $\mathcal{L} = (N, \leq)$ by closing all subsets X as nodes $\{X\}$, with no interior content at a given scale of description,

$$X \subseteq Y \rightarrow \{X\} \leq \{Y\}. \quad (5)$$

A multilayer network appears spontaneously there. By definition, each layer is an equivalence class by modulus $|X| = n$ ($n = 1, 2, \dots, N$). All subsets having the same number of elements (neurons) i constitute a corresponding layer l . First, the order is evidently transitive: $l < m \ \& \ m < k \Rightarrow l < k$. Second, the partition is modular in the sense that any set X of neurons i forms a module (by definition), and the upper module arises by adding a new element j , such that $\forall i (i \neq j)$. In network science, modularity refers to the property of groups of nodes to be densely connected within modules but sparsely connected with nodes in other modules. In the hierarchy, modules overlap but no two of them are identical. These both order and modularity allow to decompose Δ by a number of modular \subset -chains, which are nested one within another. All the modular \subset -chains are multiscale, each going from a particular neuron i in the elementary basis across scales up to the whole brain network N .

The property of different \subset -chains to reach the same module and, ultimately, to converge on the top of Δ is especially noticeable. This gives support to what is often called multiple realizability: the same macrostate can be realized from many different microstates. While all the microstates are produced by linear causal chains in the elementary basis, information carried by them can flow through modular \subset -chains across scales to a single macrostate, associated in the brain with that information. What we have on a whole is that the ideal Δ naturally displays the self-similar (scale-free or fractal) architecture as one of the fundamental hierarchical features.

The above condition (i) in Eq. (4) determines the closeness of hierarchy from above. This boundary between a system and its environment is especially important in the case of biological systems. The boundary is a place where all the exchanges of energy and matter with the environment occur. The closeness of hierarchy from above that arises over its elementary basis though modular organization allows to link autonomy to its organizational closure and account for its causal independence (Farnsworth 2018). In biological systems, modularity has been associated with robustness, the ability of a system to withstand perturbations and retain its functionality (Aldana et al. 2007). Modules are clusters of coupled elements that work under certain constraints to provide some function of a system. Accordingly, organisms can be viewed as super-modules made up of many modules that adapt as a whole to their environment (Alcalá-Corona et al. 2021).

Given the causal set, pervading the whole spacetime (Figure 2), and the definition of module, the organism is a system within which causal chains are densely connected, compared to causal chains between the system and the environment. Thus, the closeness of hierarchy from above provides both the relatively high causal density of its elementary basis and the organism's structural closure over its spatial span as prerequisites for its causal autonomy. In this way, autonomy is intrinsically linked to hierarchical self-organization that makes the organism more than the sum of its parts in the elementary basis. In particular, the structural closure resonates with what Rosen (1991) defined as “closure to effective causation” (in Aristotelian classification), which corresponds rather to “reason” that proposes a causal explanation of why something exists or happens regularly under controlled interventions (in Pearlian account).

At this stage we must strongly distinguish between a flat multilayer hierarchy as a semilattice $\mathcal{L} = (N, \leq)$, where all layers are of the same scale (Figure 6A), and a modular multiscale hierarchy Δ as a powerset $\mathcal{P}(N) = \{X | X \subseteq N\}$, where each layer corresponds to a different scale in a way as the scales naturally unfold from the elementary basis chosen for a given system of interest (Figure 6B). Accordingly, within the spatial span of a

system, the layers are roughly divided into a micro-, a meso-, and a macro-scale. While the scales below the elementary basis are ignored as noisy, the scales above the span are commonly referred to as environmental.

The flat and modular hierarchies are fundamentally different. The flat hierarchy is typically schematized by a multilayer neural network (Figure 6C). The “computing units” are arranged in a hierarchical bottom-up manner, with hidden layers placed between the input and output layers. Signal flows feedforward in the sense that a given layer exerts causal influences only on the next highest layer. The networks can also be equipped with feedback connections. However, all the computing units are of same scale, and, hence, their connections are all provided by linear causal (temporally unclosed) chains.

In contrast, a modular hierarchy is composed of units of different scales, e.g., starting from a neuron to a neural network to the whole brain (Figure 6D). Dynamics within a spatial span of the brain require different mathematical models, depending on the scale of interest. For example, the behavior of single neurons is typically described by Hodgkin–Huxley model. However, most (if not all) functional units involved in perception, cognition, conscious experience, and action depend on the collective behavior of many neurons in large-scale systems of the brain. Importantly, the descriptions of the systems’ dynamics use dimensionality reduction based on various large-scale approximations, which all are legitimate according to the CEP.

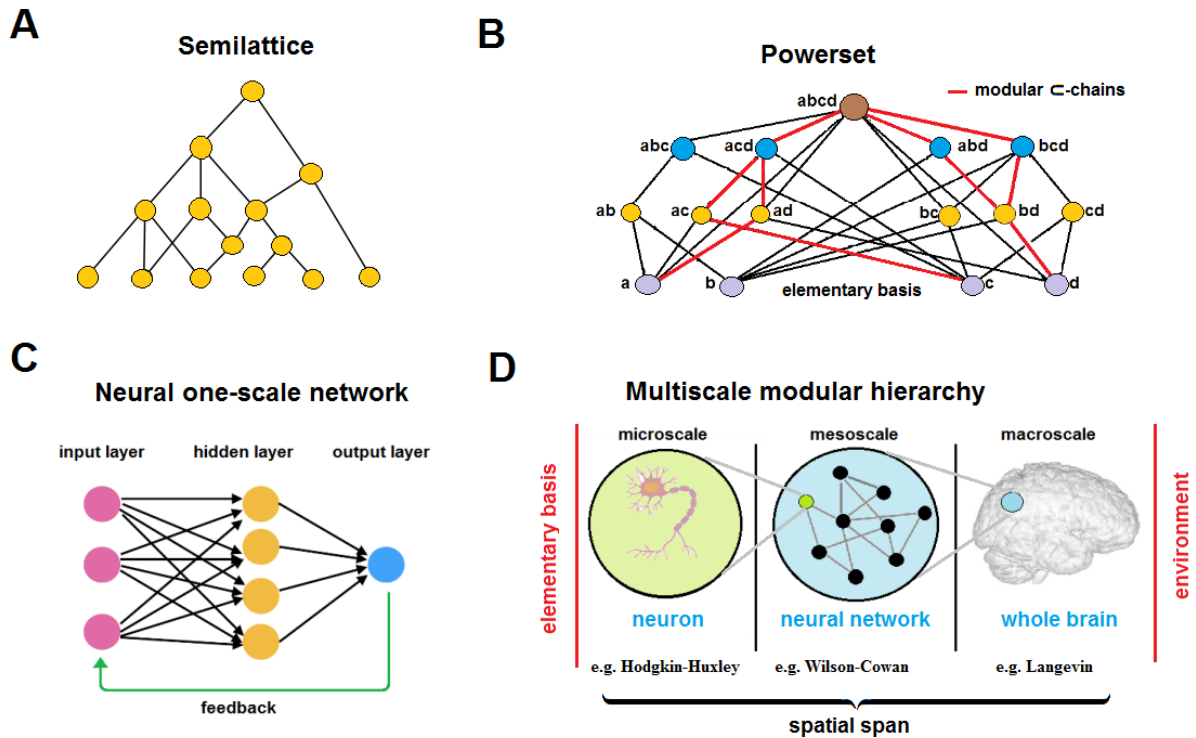


Figure 6. Two types of hierarchy. (A) Flat hierarchy exemplifies a classical (upper) semilattice where elements are arranged by layers. (B) In contrast, multiscale hierarchy is presented by a power set unfolded schematically over the elementary basis (of four elements with some links omitted for simplicity). The hierarchy consists of modular \subset -chains (colored in red) which converge on the top of the hierarchy and provide multiple realizability of the same macrostate starting from different microstates. (C) A canonical neural network of a flat hierarchy is completely linear. (D) A schematic presentation of modular \subset -chains in the brain, starting from single neurons over neural networks to the whole brain (its global workspace).

The spatial span of a system is divided into three scales of description placed between the elementary basis and the environment.

These include neural mass models, e.g., Wilson–Cowan model, or mean-field approximations and coupled oscillators models, capable of reflecting metastability and critical dynamics, e.g., Kuramoto model or Langevin formalism (for review see Breakspear 2017; Cofré et al. 2020; Vohryzek et al. 2022). It is also worth noticing that systems poised on the edge of criticality reveal dynamical properties that reflect many principled features of the modular hierarchy such as self-organization (closeness from above), or scale-freeness (topological self-similarity) showing power-law scaling (Chialvo 2010, Beggs and Timme 2012; Deco and Jirsa 2012).

The difference between flat hierarchy and modular hierarchy can best be captured by realizing how these are decomposable by chains. A canonical example of flat multilayer hierarchy is a power hierarchy where the order is defined by subordination, accompanied with the branching degree. They form a semilattice with the supremum on the top to which all members are subordinated across layers. The flat hierarchy consists exclusively of one-scale chains over which authority and control (information) can be carried out via linear causal chains (Figure 7A). In contrast, the modular multiscale hierarchy originates from elementary basis where all members reside. Instead of the supremum, the global workspace takes place on the top of hierarchy. While modules unfold across scales as functional units, their functionality in the brain is provided by synaptic neuron-to-neuron communications. If even these functional units are information-processing, it still is provided exclusively by linear causal chains. Meanwhile, information can flow across \subset -chains where modules are nested one within another like the “matryoshka” dolls (Figure 7B).

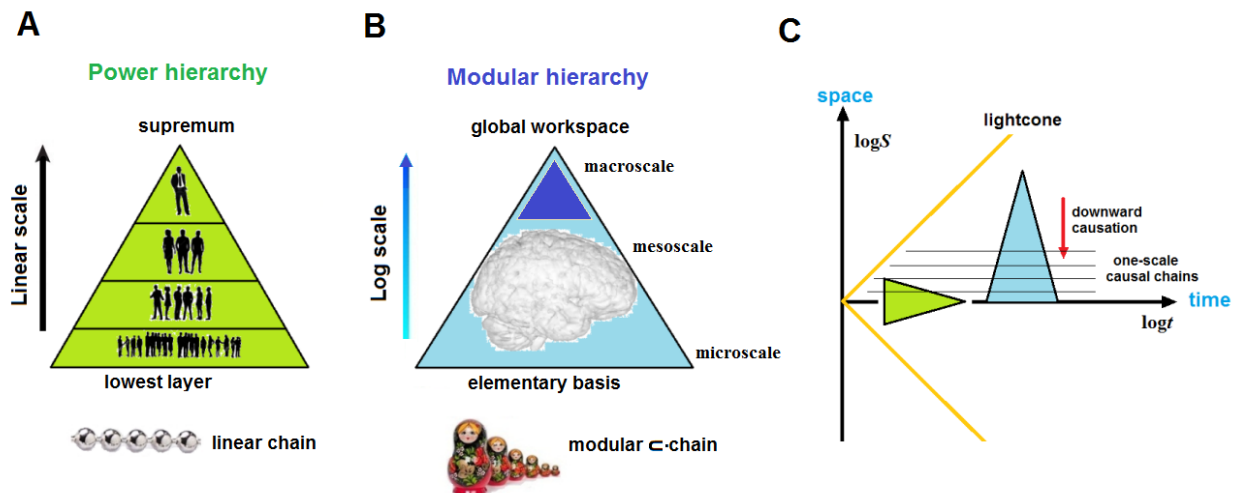


Figure 7. Comparison of hierarchies. (A) Flat hierarchy is depicted as a power hierarchy of subordinated layers. All interpersonal communications are of the same scale. (B) Modular hierarchy of the brain is divided into micro-, meso-, and macro-scales. Information carried by linear causal chains can flow over scales to and be aggregated by the global workspace. (C). The flat and modular hierarchies are topologically orthogonal to each other at log scale of spacetime in the sense that the former occupies only one scale while the latter unfold over many scales. Accordingly, downward causation is irrelevant to a flat hierarchy but might supposedly go from the top to the bottom in a modular hierarchy. Here it is depicted by a vertical line with respect to the scales. It does not mean that downward causation should be spacelike (instantaneous), i.e., faster than the speed of light. Assuming the speed of its action could be timelike (depicted by a diagonal line), its measurement would still be physically problematic or even impossible. It is easy to localize two different parts of a system as point-like entities via coarse-graining. But how might the whole system,

presented by a macroscopic variable (cause), and its own part, presented by a microscopic variable (effect), be separated in spacetime to calculate the speed of causal influence? In any case, the CET rejects this as the double causation fallacy.

In fact, these two types of hierarchy are topologically orthogonal to each other (Figure 7C). Although, as stated, a multiscale hierarchy (a powerset) can be mapped into a multilayer hierarchy (a semilattice) by closing all modules X as nodes $\{X\}$, thereby turning the global workspace into the supremum, this kind of mapping is illegitimate as it mixes all variables within the spatial span of a system reduced to a single scale of description. Dimensionality reduction allows one to turn the global workspace into a single macroscopic variable by eliminating all meso- and microscopic variables. In this case, the global workspace will represent the behavior of the whole hierarchy Δ in the large-scale environment, e.g., the murmuration of starlings. This (weakly) emergent phenomenon of spontaneous self-organization cannot be inferred from observations at the microscale where the behavior of each starling is constrained by its neighbors in the elementary basis.

7. Discussion

7.1. *Evolution vs. reductionism*

The CEP is not a reductionist principle. It starts from the fact that the notion of causation originates from our observation of phenomenal events as these occur in temporal dynamics of physical systems governed by fundamental laws. Because our notion of event is scale-independent, the CEP argues that all spatiotemporal scales of description are causally legitimate and ontologically valid. Researchers are free to choose the most appropriate scale for their causal analysis. However, the CEP forbids to mix different scales at risk to come to the double causation fallacy such as upward and downward causation. Both upward and downward causation are just observational artefacts: a false appearance resulting from failing to comply with the need to keep scale-dependent views separate. Importantly, assuming any of these two entails the legitimacy of the other. It is just the way adopted by many defenders of downward causation, starting with the assumption that upward causation is completely legitimate as enabled by information flow over scales (Campbell 1974; Ellis 2012; Ellis and Kopel 2019; Davies 2012; Noble 2012; Noble et al. 2019). This allows them to argue that physics cannot account for biological information-processing systems and, more generally, for the “hard problem” of life (Walker and Davies 2016).

The CEP rules out both upward and downward causation, yet prohibiting information to produce causation. The “flickering effect” of the murmuration of starlings (Varley 2023) is probably the best example showing that information does not produce causation. The murmuration results from synergistic information (see Williams and Beer 2010) that emerges from the spontaneous self-organization produced by the relationships among the birds. It is known that the information carried jointly by two variables in Gaussian systems is greater than the sum of information carried by each variable individually (Barrett 2015). More broadly, this synergistic information is contextually scale-dependent as a generalization of the fact that variables that are pairwise independent at the microscale can be jointly self-organized at the macroscale. An obvious conclusion here is that synergistic information can vanish and appear in the eyes of an observer while having nothing done to causation.

On the other hand, in the case of biological systems, which are multiscale modular hierarchies showing stable autonomy and self-organization without flickering, synergy is more than an observer’s impression. As stated, biological systems with all their properties emerge only at larger scales. Does it mean that life endows information with causal power? The answer must take into account the evolutionary perspective. Overall, the CEP is well compatible with the idea that the major evolutionary transitions in the history of life on Earth

involved changes in the way information is stored and transmitted (Szathmáry and Maynard Smith, 1995). Yet, the CEP argues that these changes were provided exclusively by the transitions in the hierarchical complexity of biological systems. There could not be another way than one that promoted their causal architecture (“hardware”) under the natural selection pressure for the benefit of effectively storing and transmitting information across scales in the absence of any executive program (“software”) or external guidance (and intelligent design). However, the CEP rules out dualist ontology for information that could gain direct and context-dependent causal efficacy over the matter in a downward fashion.

7.2. Information-theoretic approach to downward causation

Unlike the descriptive approaches of Ellis (2012), Noble (2012), and Walker and Davies (20013) to downward causation, Hoel et al. (2013) and Rosas et al. (2020) suggest a purely mathematical account for its justification, based on information measures. They both claim to suggest a rigorous theory of causal emergence though the very notion “causal emergence” can be misleading (Dewhurst 2021).

Hoel et al. formulate it as effective information, which assesses the causal influence of one subset of a system on another. This information-theoretic account of causal emergence is motivated by Pearl’s interventionist approach. In short, effective information measures how much a macroscale intervention (computed via the maximum entropy distribution) is predictive of the future state of a system, such that a highly organized system will have higher effective information than a chaotic system.

Effective information can amount to finding optimal coarse-graining of nodes of a network N into modules (clusters). It can then be presented by two quantities as

$$EI = H(\langle W_i^{out} \rangle) - \langle H(W_i^{out}) \rangle. \quad (6)$$

Here W_i^{out} is the out-weight vector of each node v_i , $i = 1, 2, \dots, N$, consisting of weighted edges w_{ij} between v_i and its neighbors v_j , and $\langle \cdot \rangle$ means the average integration. Assuming $\sum_j w_{ij} = 1$ means that each w_{ij} can be interpreted as the probability p_{ij} that a random walker (such as a signal) will go from v_i to v_j in the next timestep (Klein and Hoel 2020). Overall, a random walker on a high-degree node is more uncertain about which node it will visit next compared with a random walker on a low-degree node. A lower $H(\langle W_i^{out} \rangle)$ means that information is distributed only over a small number of nodes. A high $H(\langle W_i^{out} \rangle)$ signifies that information is dispersed throughout the network. The conceptualization of causal emergence is conditioned on the difference between determinism and degeneracy (Hoel 2017):

$$effective\ information = determinism - degeneracy. \quad (7)$$

These both are defined as

$$determinism = \log_2 N - \langle H(W_i^{out}) \rangle,$$

with $\langle H(W_i^{out}) \rangle \rightarrow 0$, and

$$degeneracy = \log_2 N - H(\langle W_i^{out} \rangle),$$

with $H(\langle W_i^{out} \rangle) \rightarrow 0$.

Strictly speaking, the Hoel et al.’s use of the word “determinism” is ambiguous as contraposed against statistical randomness in noisy data. First of all, statistical randomness is epistemic as depending on the state

of an observer’s knowledge about the behavior of a system of interest, whereas the behavior of the system can still be completely deterministic as it is coined in terms such as *deterministic chaos*. In physics, it is commonly assumed that all classical systems are completely deterministic, and genuine indeterminism takes place only at the quantum scale. This makes noise a context-dependent notion, placed below the elementary basis of a system of interest. For example, molecular biology treats quantum fluctuations as noise, cognitive neuroscience takes the very molecular level for noise, whereas social psychology, interested mainly in interpersonal interactions, ignores these both.

The Hoel’s conceptualization can be reformulated in terms of both the CEP, based on the spacetime compression, and modular hierarchy without even mentioning “determinism” as this:

$$\text{effective information} = \text{multiple realizability} + \text{dimensionality reduction}. \quad (8)$$

Or, in terms of Hoel, the temporal evolution of a hierarchically organized system looks more “deterministic” at the macroscale than at the microscale with many causal linear chains that can lead via modular \subset -chains to the same macrostate of the system. Thus, from the perspective of an external observer (ignorant about their internal meaning in system processing), these statistically redundant chains can account for “degeneracy” in Hoel et al.’s conceptualization. Accordingly, dimensionality reduction acts as a deterministic constraint on multiple realizability allowing for redundancy at the microscale of hierarchical systems (Figure 6B).

By using this effective information, the external observer can then show that the causal power of neuronal modules grows exclusively due to dimensionality reduction by coarse-graining many microscopic variables into a single macroscopic variable, thereby eliminating multiple modular \subset -chains that would lead to the global workspace in Δ (Figure 4B). Coarse-graining allows “seeing” what could not be seen at lesser scales. In other words, coarse-graining can gain information (a map) about weakly emergent self-organization that originates from the elementary basis (a territory). This condition holds for complex systems that are indeed highly self-organized.

Probably, a main merit of the Hoel et al.’s approach, besides its computation use in data analysis (Klein et al. 2021; Ravi et al. 2022; Zhang and Liu 2023), is that it sheds light on the nature of biological systems from the evolutionary perspective, not merely from the perspective of an external observer, analyzing data. In the case of biological systems, which are themselves information-processing, this scale-dependent effective information can demonstrate how a system that is memoryless at the microscale can accumulate information at the macroscale. This makes information a function of scale (Klein and Hoel, 2020) as a prerequisite for scale-dependent emergence of cognition and consciousness in neural networks.

More generally, the transition from non-living to living matter may therefore be identified with information that is stored in the hierarchical organization of a system, thereby providing causal efficacy of larger (modular) scales in processing information, viewed sometimes as spurious downward causation. Instead, one might speculate that the thermodynamic arrow of time is scale-dependent so that there is no time and causal order at the Planck scale. The classical deterministic world emerges from the quantum world via coarse-graining quantum redundancy, with time arising somehow from an informational space compression. In fact, this is a core idea of quantum Darwinism, where the large-scale environment mediates the emergence of a classical world out of a quantum world by acting as a “witness” (Zurek 2009; Riedel et al. 2016). In particular, this explains why physics spans two extreme scales – quantum and cosmological but is blind to living systems that take place at the intermediate scales (Figure 2B). These scales do not capture scale-dependent self-organization of complex biological systems within their characteristic spatiotemporal span. The quantum scale is too fine-grained, whereas the cosmological scale is too coarse-grained to capture emergent phenomena of the self-organized behavior in living (and conscious) multiscale hierarchical systems.

Rosas et al. (2020) have recently presented an information-theoretic account of causal emergence in multivariate systems that is similar to that of Hoel (2017), albeit based on Granger rather than Pearl causality. Note that Granger causality (or, equivalently, transfer entropy) is even a “weaker” descriptor of actual causal chains than Pearl’s interventionist approach (Lizier and Prokopenko 2010). Rosas et al. make use of a partial information decomposition (Williams and Beer 2010) which allows to discriminate between unique, redundant, and synergistic information that two ‘source’ variables Y and Z hold about a third ‘target’ variable X :

$$I(X; Y, Z) = Unq(X; Y|Z) + Unq(X; Z|Y) + Red(X; Y, Z) + Syn(X; Y, Z). \quad (9)$$

They define downward causation as a (strongly) emergent feature that has unique predictive power over the evolution of specific subsets of the whole system as if the whole has an effect on the parts that cannot be reduced to interactions at a lower scale (Rosas et al. 2020; Mediano et al. 2022). Causal emergence is then conceptualized as

$$causal\ emergence = causal\ decoupling + downward\ causation. \quad (10)$$

It is noticeable that the formalization of downward causation in terms of predictive power is implicitly based on the assumption that information can have causal power. In fact, many information-theoretic approaches aimed to justify downward causation are prone to this. Like the above case with (causally) effective information of Hoel (2017), this conceptualization can be explained in the context of both the CEP and hierarchical framework without involving spurious downward causation:

$$causal\ emergence = reductionism + self-organization. \quad (11)$$

Complex dynamical systems exhibiting spontaneous self-organization give rise to emergent phenomena with various examples in nature like that of the (flickering) murmuration of starlings. Thus, global observables, associated with some supervenient macro-variable, can indeed provide synergistic information that cannot be obtained from local observables. The murmuration is a typical pattern of weak emergence. Meanwhile, each starling as an organism is a much more stable system whose self-organization is based on its hierarchical structure. Its elementary basis resides at the molecular scale where emergent self-organization originates ultimately from Brownian motion. Only on the assumption that information flow within these biological hierarchies had causal power, weak (epistemic) emergence, based exclusively on predictive power, could be turned into its strong (ontological) counterpart to account for downward causation (Schmickl 2022; Farnsworth 2022).

7.3. Information and entropy

In this way, one might draw attention to the relationship between Shannon information and Boltzmann entropy and argue for entropic forces as fundamentally responsible for macroscopic emergent phenomena resulting from Brownian motion by the second law of thermodynamics (Verlinde 2011; Visser 2011; Wissner-Gross and Freer 2013). Here one could then seek for a loophole to somehow endow information with causal power to resist the entropy growth and even ask new physics (Schrödinger 1944). Or, on the contrary, one could argue that entropy growth is favorable to life organized in hierarchically modular systems (Annala and Kuusmanen 2009; Jeffery et al. 2019). Going further, one might also speculate on the mind-body problem or, more generally, on the “hard problem” of life from a holist-panpsychist perspective (Irwin et al. 2020). These issues will not be discussed here, with the only remark that the second law of thermodynamics should ultimately

act as a natural destroyer of any multiscale biological hierarchy, and, hence, as a natural annihilator of information the hierarchy had memorized over time.

In the CEP's specification, reductionism is the requirement that linear causal chains at one scale of description must be consistent with linear causal chains at any other scale of description, with respect to the smallest scale of causal analysis. This completely excludes the puzzling idea that causation at one scale can be stronger than at another scale in the same sense as the Lorentz transformation does not change the causal order in all inertial frames of reference, while making simultaneity relative. On the other hand, the CEP allows information to be transmitted over scales without mixing these causal chains. This is well compatible with the major transitions in evolution (Szathmáry and Maynard Smith, 1995), namely that biological systems should have evolved in causal ways that preserved and advanced the adaptive success of their scale-dependent self-organization in information processing, thereby making information (and, hence, consciousness and cognition) a function of scale (Klein and Hoel 2020). Downward causation "emerges" only in the eyes of an observer (Barnett and Seth 2021).

8. Conclusion

What about the main question of this paper: Is information the other face of causation? The answer depends on how these two are conceptualized. If information is epistemologically derived from causation in terms of predictive power, and they both are observer-dependent, the answer can be, yes. Moreover, there is now information theory based on the Shannon's measure of uncertainty that allows to make exact mathematical predictions, but there is no theory of causation. In this sense, information as an epistemic map is better than causation as an ontic territory (Hoel 2017). In contrast, if information is assumed to have its own causal power or even underlies physical reality ("It from bit"), the answer is negative by a reason just mentioned above: a map and a territory are not the same. Moreover, since information is scale-dependent, its amount could be minimum at the quantum level.

The holist dictum "The whole is greater than the sum of its parts" is observer-dependent and can be rephrased as "The view from above is better than the view from below." It is impossible to "see" a multiscale modular hierarchy by looking at its elementary basis. Thus, the view from above indeed provides information gain, not affecting linear causal chains at any scale of description. On the other hand, the information gained at a macroscale is more than epistemic since it captures genuine (weakly emergent) properties of self-organization of complex systems, which cannot be inferred exclusively from microscale descriptions.

These emergent properties depend on the multiscale modular hierarchy which appears spontaneously in ways governed by very simple rules like those in the famous Game of Life. This makes the hierarchical organization of complex systems pervasive and universal in nature. On the one hand, the pervasiveness is provided by the principled features of the modular hierarchy such as self-organization (closeness from above) and scale-freeness or power-law scaling (topological self-similarity) as necessary prerequisites of critical dynamics that are thought to be crucial to the origin and maintenance of life on the edge of chaos. On the other hand, the universality follows from the fact that the hierarchy as the powerset $\mathcal{P}(N) = \{X | X \subseteq N\}$ over any set N of arbitrary entities, placed in the elementary basis of a system, determines its state space $S(N) = 2^N$ where each entity can be in a state contextually denoted as 0 or 1.

The "algorithm of life" is this. First of all, there must be a sufficient large number of elementary entities. Now connect these entities somehow (but not uniformly or totally) with one another, and a multiscale hierarchy, related mathematically to Shannon information and Boltzmann entropy, will spontaneously emerge there. On the evolutionary timescale, the hierarchy will grow by incorporating external resources into new entities and connections like the drawbridge of biological complexification (Igamberdiev 2021). The mysterious emergence of living systems in evolutionary processes becomes a matter of the availability of elementary entities and time

that is necessary for connecting these entities by causal chains to effectively compute and transmit information over scales.

Ultimately, weak emergence amounts to triviality with respect to the fact that the universe itself we live in and observe is a hierarchically organized emergent phenomenon. There are no complex systems at the fundamental Planck scale. All things, minds, and events emerge at larger scales.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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