

To appear in: Hundertmark F., Ferreria Ruiz M., Kaiser M. I., Suárez J. (2025): Causal Bases of Potentialities in the Life Sciences. Extrinsicity, Multi-levelness, and Processuality, in: Hüttemann A., Schurz G. (eds.): Inductive Metaphysics: Insights, Challenges and Prospects. Routledge.

# Causal Bases of Potentialities in the Life Sciences

## Extrinsicity, Multi-levelness, and Processuality

### **Fabian Hundertmark**

Abteilung Philosophie  
Universität Bielefeld  
Postfach 10 01 31  
33501 Bielefeld  
Germany

E-mail: [fhundertmark@uni-bielefeld.de](mailto:fhundertmark@uni-bielefeld.de)

ORCID ID: 0000-0001-8784-9290

### **María Ferreria Ruiz**

Abteilung Philosophie  
Universität Bielefeld  
Postfach 10 01 31  
33501 Bielefeld  
Germany

E-mail: [mariaferreiraruiz@gmail.com](mailto:mariaferreiraruiz@gmail.com)

ORCID ID: 0000-0002-6931-1072

### **Marie I. Kaiser**

Abteilung Philosophie  
Universität Bielefeld  
Postfach 10 01 31  
33501 Bielefeld  
Germany

E-mail: [kaiser.m@uni-bielefeld.de](mailto:kaiser.m@uni-bielefeld.de)

ORCID ID: 0000-0002-9307-7474

### **Javier Suárez**

BIOETHICS Research Group  
University of Oviedo, Department of Philosophy  
C/ Amparo Pedregal s/n, Office 0215  
El Milán – Campus de Humanidades  
33011 Oviedo, Spain

E-mail: [javier.suarez@uniovi.es](mailto:javier.suarez@uniovi.es)

ORCID ID: 0000-0001-5851-2277

**Abstract:** Potentialities are properties that can manifest. Many potentialities are instantiated in virtue of having causal bases that are possible causes for their manifestations. This paper shows that a closer look at potentialities and their causal bases studied in the life sciences (from cell biology to psychiatry) gives us good reasons to revise our philosophical assumptions about causal bases. The investigation reveals that causal bases are often more dynamic and dependent on interactions with the environment than previously assumed. In particular, it argues that causal bases in the life sciences are regularly extrinsic and processual and that potentialities in the life sciences have causal bases at different levels.

## 1. Introduction

This paper analyzes potentialities studied in cell biology, ecology, psychology, and psychiatry. These cases provide strong reasons for revising our assumptions of causal bases, which have been based primarily on a narrow sample of cases. Specifically, we argue that causal bases in the life sciences are often extrinsic and processual and that potentialities in the life sciences have causal bases at different levels.

In philosophy, many classes of properties that are plausibly potentialities have been discussed. Here is a non-systematic – and surely non-exhaustive – list: dispositions (e.g., fragility; e.g., Lewis 1997, 148), abilities, capacities, and skills (e.g., the ability to ride a bike), tendencies (e.g., tasting good to someone; Dinges & Zakkou 2021), options (e.g., the option to go swimming; Maier, 2022), potentials (e.g., the potential to become a famous pianist; McKittrick 2018, sec. 4.4), virtues (e.g., generosity; e.g., Roberts and Wood 2007), vices (e.g., gluttony) and susceptibilities (e.g., the susceptibility to develop cancer). This paper focuses on the broad category of potentiality (see, e.g., Vetter 2015, sec. 1.5) rather than on a specific subclass (such as dispositions) for two reasons. On the one hand, it allows us to consider various examples from the life sciences. On the other hand, it allows us to sidestep disputes about the relations and peculiarities of the different subclasses of potentialities. In general, potentialities can be characterized as properties that are instantiated in part because their bearers *can* exhibit specific manifestations (more or less easily) (e.g., Vetter 2024).

Most potentialities - except for fundamental ones such as spin or electric charge (e.g., McKittrick 2018, chap. 7) - are instantiated in virtue of their bearers instantiating other properties that play a causal role in bringing about the manifestation of the potentialities. We will refer to these other properties as “causal bases.” Even though causal bases are rarely explicitly identified, the idea that potentialities have causal bases plays a crucial role in the philosophical literature. For example, causal bases have been used to justify the causal inertness of dispositions (Prior, Pargetter, and Jackson 1982) and to analyze dispositional statements (Lewis 1997). Here are some typical examples: The water-solubility of a sugar cube is causally based on the sugar cube being composed of weakly bonded sucrose molecules (Prior, Pargetter, and Jackson 1982, 251); the fragility of a wine glass is causally based on the microstructural properties of the wine glass, namely, on the fact that the glass is made of a relatively thin silicon oxide lattice (Prior, Pargetter, and Jackson 1982, 251; Lewis 1997, p. 149, McKittrick 2018, p. 132); the elasticity of a rubber band is causally based on the fact that it is made of entangled polymers (Prior, Pargetter, and Jackson 1982, 252); the soporificity (for humans) of a sleeping pill is causally based on the chemical properties of the drug, e.g., on the fact that it contains 15 mg of flurazepam (McKittrick 2018, p. 132).

These classic examples have several elements in common: they are commonplace potentialities causally based on microstructural properties; furthermore, they are such that the potentiality of some object is causally based on the fact that it consists of molecules of a particular type, which may be arranged in a certain way. Based on these homogeneous examples, it was often (implicitly) assumed that instances of potentialities have a singular causal basis, which is constituted of stable, intrinsic, and microstructural properties. In this paper, we show that this picture of causal bases of potentialities needs to be revised by taking a closer look at potentialities in the life sciences. We proceed as follows. In Section 2, we clarify what potentialities and causal bases are and give a quick overview of our case studies. After that, we use these case studies to argue for three claims:

**Extrinsicness:** Many causal bases in the life sciences are extrinsic (section 3).

**Multi-levelness:** Potentialities in the life sciences have causal bases extending across levels or multiple causal bases at different levels (section 4).

**Processuality:** Many causal bases in the life sciences are constantly changing or actively maintained (section 5).

## 2. Potentialities, Causal Bases and Case Studies

In this section, we introduce the concepts of potentiality (Section 2.1) and causal bases (Section 2.2). We also provide a brief overview of our examples (Section 2.3), which will serve as the basis for our arguments in the following sections.

### 2.1 Potentialities

This paper is about potentialities. The term “potentiality” was introduced by Barbara Vetter (2015, sec. 1.5) to provide a superordinate category for dispositions, abilities, potentials, and similar modal properties that are instantiated partly due to the fact that they *can* manifest (Vetter 2024). Here are some examples: a wine glass is breakable partly because it can break; Pietro has the ability to ride a bike partly due to the fact that he can ride a bike; Natsuki has the option to go swimming partly because he can swim; a cake tastes good to Bongani partly because it can cause him gustatory pleasure.

Even though Vetter does not provide an explicit definition, we believe that potentialities can be characterized as follows: A property is a potentiality if and only if there is a non-empty set of manifestations such that the following two conditions are fulfilled: (1) The property in question can only be instantiated if this object can show these manifestations, and (2) the property in question can be instantiated if this object does not currently show these manifestations.

Note that these conditions allow for single-track potentialities, which manifest by showing a single kind of manifestation, and for multi-track potentialities, which manifest by showing different manifestations. An object’s breakability – a single-track potentiality – manifests by the object in question breaking. If an object is breakable, it can break (condition 1), but objects can be fragile without being broken or in the process of breaking (condition 2). A multi-track potentiality like hardness has multiple manifestations, like an object producing a sharp sound if struck or causing pain if we come into sharp contact with it (Ryle 1949, p. 44). An object is only hard if it can show these manifestations (condition 1), but of course, it does

not have to show any of these manifestations at a given moment in order to be hard (condition 2).

## 2.2 Causal Bases

Causal bases are properties of objects that ensure that these objects instantiate certain potentialities by being possible causes for the manifestation of these potentialities. Here are some examples: the fact that the wine glass is made of a relatively thin silicon oxide lattice ensures that it is breakable by causing it to break under certain circumstances; the fact that the sleeping pill is made of 15 mg of flurazepam ensures that it is soporific by causing the person who takes it to fall asleep.

In philosophy, causal bases are crucial for categoricalists seeking a reductive analysis of dispositions (Lewis 1997). However, the issue also arises when discussing whether or not there can be 'bare dispositions' - dispositions without a (distinct) causal bases (e.g., Mumford 2006; McKittrick 2018, chap. 7) -, and when analyzing concrete examples of potentialities (e.g., from the life sciences), as we do in this paper. Influential approaches (e.g., Prior, Pargetter, and Jackson 1982, p. 251; Lewis 1997: 157) define causal bases solely in terms of their causal role in producing manifestations in conjunction with appropriate stimuli. Another role typically ascribed to causal bases is that of an underlying factor that explains why objects have certain potentialities (e.g., McKittrick 2017, 132; Alvarez 2017, 74). In this paper, we adopt the causal-grounding account of causal bases, which requires causal bases to play both roles (Ferreira Ruiz and Hundertmark 2024): First, the instantiation of a causal basis *metaphysically grounds* the instantiation of the potentiality, and second, the instantiation of a causal basis is a possible *cause* of the potentiality's manifestation.

Metaphysical grounding is an explanatory relationship between facts whereby a more fundamental fact grounds another fact (see Bliss and Trogdon 2024). While causality links facts and events over time, grounding is non-temporal. For example, the fact that the wine glass is made of a relatively thin silicon oxide lattice is more fundamental than the fact that it is breakable, and it explains this second fact in a non-causal way. Grounding can be either partial or full. For example, the fact that the wine glass is made of a silicon oxide lattice

partially grounds its breakability, while the full ground also includes the fact that this lattice is thin. Similarly, we can distinguish between partial and full causal bases:

**Partial:**  $o$ 's potentiality  $P$  has  $R$  as a partial causal base if and only if

1.  $Ro$  is a partial ground of  $Po$ , and
2.  $Ro$  is among the factors possibly causally contributing to  $P$ 's manifestation by  $o$ .

**Full:**  $o$ 's potentiality  $P$  has  $S$  as a full causal base if and only if  $S$  is the sum of all partial causal bases of  $Po$  that belong to one full ground of  $Po$ .

Accordingly, we can find a full causal base of a potentiality instantiation (e.g., the cake tasting good to Bongani) as follows: First, we find a set of facts that fully grounds the potentiality in question. In our case, this could be the texture and taste of the cake, Bongani's taste organs, Bongani's taste preferences, and arguably the fact that the cake has already caused Bongani gustatory pleasure (Dinges & Zakkou 2020). Second, we exclude all facts that are not property instantiations by the bearer of the potentiality (Bongani's taste organs and preferences). Third, we exclude those factors from the set that do not play a causal role in the manifestation of the potentiality (e.g., the fact that the cake already caused gustatory pleasure to Bongani). The remaining property instantiations (e.g., the texture and taste of the cake) are the causal base of the potentiality instantiation in question.

## 2.3 Our Case Studies

In this paper, we demonstrate the extrinsicness, multi-levelness, and processuality of causal bases in the life sciences. Each of these features will be argued for using two case studies and a general argument for why causal bases in the life sciences exhibit these features. Thus, our arguments are not based on the specific examples alone. Instead, the argumentative weight is carried by the case studies and the more general philosophical arguments together. Due to our disciplinary backgrounds, the case studies come from only some areas of the life sciences: cell biology (stemness, PCD), ecology (ES), psychology (SC), and clinical psychology (PR, ADHD). We are convinced that the examples are diverse enough to show, together with the general arguments, that the extrinsicity, multi-levelness, and processuality of causal bases hold for many other areas of the life sciences, such as physiology, neuroscience, and

medicine. An overview of our case studies can be found in table 1.

<b>Potentiality</b>	<b>Definition</b>	<b>Causal Base(s)</b>
<b>Stemness</b>	the capacity of some cells to self-renew and differentiate	[Genetic markers of the cell involved in cell reproduction; some environmental components from the niche; population-level properties at the tissue level]
<b>Ability to undergo programmed cell death (PCD)</b>	the ability of cells to die via an internal program	[biomolecular pathways encoded in the genome]
<b>Ecological resilience (ER)</b>	the ability of ecosystems to return to their relative density-distribution state after a perturbation affects the system	[system level network properties, like the ecosystem's diversity; the host immune system]
<b>Psychological resilience (PR)</b>	the ability of individuals to maintain or recover mental health under significant adverse conditions	[social support; ...] AND LATER [internalized beliefs; ...]
<b>Attention deficit hyperactivity disorder (ADHD)</b>	the strong disposition of persons to show symptoms of inattention, hyperactivity, and impulsivity	[novelty-seeking personality trait; living in an environment with several distracting stimuli]
<b>Size constancy (SC)</b>	the ability of animals to perceive an object as having the same size regardless of its distance	[mechanisms that integrate information about the size of the retinal image with information about the object's distance] AND [brain with specific mechanisms in MT, LIP, V1, V2, and V4].

*Table 1: Our case studies with short definitions and their causal base(s). Factors in the same square brackets belong to the same causal basis.*

### 3. Extrinsicness

Many authors hold that causal bases (CBs) are always intrinsic (e.g., Lewis 1997). According to this Intrinsic Bases Thesis (IBT), CBs of potentialities are only instantiations of properties intrinsic to the bearers of the potentiality (Contessa 2012). Of course, such a view recognizes that history and environment typically play a causal role in manifesting a potentiality. However, it asserts that these factors are merely stimulus, background, and sustaining conditions that do not metaphysically ground the instantiation of the potentiality.

IBT is supported by classic examples of causal bases, which are typically microstructural properties of the object that instantiate the potentiality in question. The water-solubility of sugar is causally based on the fact that sugar cubes are made of weakly bonded sucrose molecules, and the causal base of the rubber band's elasticity is that it is made of entangled polymers. Water solubility and elasticity are intrinsic potentialities. They are instantiated by objects solely in virtue of the object having a particular microstructure.

McKittrick (2003; 2018, chap. 8) has questioned whether all potentialities are intrinsic. She discusses several potentialities (dispositions) arguably grounded in extrinsic facts. These examples include the disposition of a liquid to dissolve the contents of my pocket, a city's vulnerability, and Bill Clinton's recognizability. However, the fact that these potentialities are extrinsic does not imply that their causal bases also are (see Contessa 2012). This requires not only that a partial (or full) ground of a potentiality is extrinsic but also that it be a property instantiation by the object instantiating the potentiality and that it is a possible cause of its manifestation (see Section 2.2). Consider, for example, the potentiality of some portion of liquid to dissolve the contents in my pocket. This potentiality is grounded in the fact that the liquid consists of H<sub>2</sub>O and that my pocket contains sugar cubes. However, the fact that my pocket contains sugar cubes is not an extrinsic or relational property of the liquid and thus excludes it from being part of the CB. Moreover, it is far from clear that the fact that my pocket contains sugar cubes is causally relevant to the manifestation of the potentiality in question. In short, the case for extrinsic CBs remains to be made. This section aims to fill this gap by closely examining the causal bases of stemness and ADHD.



Uncovering the causal basis of **stemness** has constituted one of the main tasks for biologists during the past decades. During the 80s and 90s of the past century, it was thought that stem cells should bear some specific genetic markers that grounded their stemness and played a role in their capacity to reproduce as stem cells do. However, in the early 2000s, it was shown that most of the candidate genes were also present in non-stem cells, and sometimes the same pattern of gene expression that is seen in stem cells is also seen in non-stem cells (Vorotelyak et al. 2020, Suárez 2023). Thus, the hypothesis that genes constitute the causal basis of stemness and that the causal basis of stemness is, therefore, intrinsic was discarded

The current consensus is that stemness is realized in various ways (Laplane & Solary 2019). The most robust evidence proving that stemness has an intrinsic causal basis derives from some research on dedifferentiation in cancer stem cells, which has been reported in several types of cancer (Laplane 2016). In these cases, a somatic (already differentiated) cell that lacks stemness capabilities can de-differentiate and acquire the stem cell state. This phenomenon may occur due to mere stochasticity (e.g., a random mutation) or due to systemic, higher-level properties of the system (e.g., population-level properties) in which the cell is embedded. Advocates of the system view of stemness interpret many experimental results as evidence for the view that stemness arises from population-level properties or situations of feedback control from within the system (Lander 2009). According to this approach, the system relations that a cell experiences at any given time determine its state, including whether the cell needs to differentiate or dedifferentiate, whether it needs to reproduce asymmetrically, or whether it needs to be maintained in a quiescent state. According to this interpretation of stemness, a cell's stemness is grounded in its extrinsic relationship to the system, and the manifestation of the property is also extrinsic. Thus, according to this approach, stemness is a potentiality whose causal basis partly extends to the entire cell population.

**Attention-Deficit/Hyperactivity Disorder (ADHD)** is highly heritable and highly impairing (Thagaard et al. 2016). This combination of features raises the question of why ADHD has not been strongly selected against or why it is still highly prevalent (estimates vary between 2% and 18%; Rowland et al. 2002). A plausible explanation might be that ADHD is a so-called “evolutionary mismatch” (Swanepoel et al. 2018; Le Cunff 2024). Explaining a maladaptive trait as an evolutionary mismatch requires that an adaptive trait in a particular environment be

effective in an environment detrimental to its adaptivity. ADHD is strongly associated with the ‘novelty-seeking’ personality trait (Perroud et al. 2016). The novelty-seeking personality trait is roughly the intrinsic disposition to explore novel or risky situations or objects (Arenas & Manzanedo 2016). There are different theories of how this personality trait has been adaptive in evolutionary history. Hartmann (1999), for example, speculates that individuals with a high level of this trait may have been better hunters, while others claim that it has been beneficial in rapidly changing, threatening environments with scarce food resources (see Thagaard et al. 2016 for an overview). Despite its adaptiveness, all persons with the novelty-seeking personality trait will plausibly have the disposition to show at least *some* disadvantageous ADHD symptoms (e.g., avoidance, disliking, and reluctance to engage in tasks that require sustained mental effort).

Persons with the novelty-seeking personality trait will show the clustering of maladaptive symptoms characteristic of ADHD only in certain kinds of environments. For example, the disposition to have difficulty sustaining attention in tasks or play activities will depend on two factors. On the one hand, different tasks and play activities are relevant in different societies. In modern Western societies, it will, for example, be essential to sustain attention during lectures, lengthy readings, and on similar occasions. In mobile forager societies, sustaining attention during hunting or fruit gathering will have been more critical. While people with the novelty-seeking personality trait have no problem focusing on the second kind of task, the first kind of task requires skills they lack. Furthermore, for people with the novelty-seeking personality trait, the disposition to sustain attention crucially depends on the number of distracting stimuli (Cassuto et. al. 2013). Consequently, an environment with fewer stimuli will also significantly reduce the disposition of people with this trait to show ADHD symptoms. If these hypotheses are correct, it follows that ADHD is an extrinsic potentiality. As seen above, this does not imply that ADHD has an extrinsic causal base. So, let us look at both candidates for extrinsic causal bases.

At first glance, the disposition of a person with the novelty-seeking personality trait to sustain attention in tasks and play activities is parallel to the disposition of some portion of liquid to dissolve the contents of my pockets. In both cases, the disposition that is denoted is dependent on context. However, there are different kinds of context dependency. The fact that I have sugar cubes in my pocket is no property instantiation of the liquid. In contrast, the fact that a

person with the novelty-seeking personality trait lives in an environment where they are required to attend lectures and read lengthy texts is a relational property of that person. So, this fact is, at least, a candidate for a causal base of ADHD. However, whether this fact is a potential causal factor for the manifestation of ADHD is a difficult question and depends on the specific theory of causality adopted.

However, the matter is straightforward concerning the stimuli in a person's environment with the novelty-seeking personality trait. The existence of these stimuli is a relational fact about a person with ADHD. These stimuli can obviously be causally relevant to the manifestation of ADHD symptoms. Furthermore, according to the mismatch hypothesis, they metaphysically ground ADHD rather than being stimulus conditions or mere background conditions. After all, this hypothesis requires a distinction between the adaptive novelty-seeking *personality trait* instantiated by our evolutionary ancestors and the maladaptive ADHD instantiated by people with this trait in modern-day Western culture. If these arguments are correct, the causal basis of some people's ADHD is extrinsic since it incorporates the fact that this person lives in an environment with several distracting stimuli (and maybe also in an environment where attending lectures and reading lengthy texts is essential).

Please note that we have only argued that some cases of ADHD have extrinsic causal bases. This thesis is perfectly compatible with the position that only some cases of ADHD are evolutionary mismatches, while others may have intrinsic causal bases. Our argument is also compatible with skeptical positions on ADHD. After all, these positions do not question that people with ADHD have the potential to show specific symptoms. Instead, they question whether these potentials are rightly pathologized – e.g., precisely because they are evolutionary mismatches and not intrinsic dysfunctions.

In this section, we argued that (at least some) instantiations of stemness and ADHD have extrinsic causal bases. This argument shows that some potentialities have extrinsic causal bases and that the Intrinsic Bases Thesis (IBT) is false. However, there is also a more general lesson to draw. Large parts of life sciences are concerned with the interactions of particular systems with their actual or natural environment. This applies at least to parts of cell biology, ecology, behavioral sciences, psychology, and psychiatry. Since these sciences investigate how their objects of interest interact with their environments, it is less critical to ascribe

properties based only on what is happening inside the system's boundaries. Consequently, we expect the potentialities investigated in these sciences to allow for extrinsic causal bases. If this reasoning is correct, extrinsic causal bases are likely found in other sciences, primarily investigating interactions in actual or natural environments, such as the social sciences, geoscience, or even astronomy.

## 4. Multi-levelness

The causal bases of standard examples – such as the existence of silicon oxide lattices, entangled polymers, or flurazepam – are typically relatively simple properties involving only one level of organization. Moreover, in the philosophical literature, people speak of “*the CB*” of some potentiality instantiation (e.g., Prior, Pargetter, and Jackson 1982, 251; Lewis 1997, 155; Choi & Fara 2021), implying that every such instantiation has a single causal base. In this section, we will argue that causal bases of potentialities in the life sciences are found at different levels. This claim can be specified in two different ways: On the one hand, it means that a full causal basis involves instantiations of properties at different levels; on the other hand, it means that the instantiation of a potentiality has multiple causal bases in the sense of having multiple full grounds, which can cause the potentiality to manifest.

Let us start by considering the case of **ecological resilience**. Ecological resilience refers to the ability of the ecosystem to return to its relative density-distribution state after a perturbation.<sup>1</sup> A perturbation is understood as an alteration in the relative density of one or some of the species in the ecosystem due to causes different from the system dynamics – for example, due to the introduction of novel taxa, the sudden death of several members of one or more species due to an infectious disease, or other factors. Ecological resilience refers to the magnitude of the disturbance an ecosystem can cope with while keeping the relative distribution of the species (Dakos & Kefi 2022). In this vein, a system is considered ecologically resilient when it can absorb a significant disturbance without losing its own state or dynamics.

---

<sup>1</sup> In ecology, states are known to be dynamic states. That is, an ecosystem can be in a specific state even if its relative species density distribution oscillates (as common in prey-predator systems) (Otto & Day 2011).

The microbiome – microorganisms inhabiting a specific environment, especially an animal or plant host – is an ecologically resilient ecosystem (Fassarella et al. 2021). An essential part of microbiome research explains resilience in terms of network interactions between the species composing the microbiome (Coyte et al. 2015; Xiao et al. 2020; Díez & Suárez 2023). The underlying idea is that resilience depends on the number of interacting species and their initial density distribution. These network properties are involved in the recovery capacity of a microbiome once it is perturbed. For example, Xiao et al. (2020) have shown that species diversity plays a crucial role in human microbiome stability and, in turn, in its ability to recover from specific pathologies. Focusing on the case of persistent *Clostridium difficile* infection, Xiao et al. (2020) show that having a diverse microbiome prevents disease recurrence because the microbiome maintains the pathogen's relative density under the pathogen's threshold of pathogenicity. According to Xiao et al. (2020), this phenomenon occurs because a highly diverse microbiome instantiates a network structure that can absorb several perturbations, and the network structure of the microbiome would ground its resilience while simultaneously playing a causal role in the system recovery after it is perturbed.<sup>2</sup>

At the same time, the host immune system plays a crucial role in maintaining microbiome resilience (Foster et al. 2017; Fassarella et al. 2021). This control can be exercised in different ways: by determining nutrient or oxygen availability, via immunotolerance, or by direct host-microbiome interactions. In this case, the host would exert regulatory control over the microbiome and, in doing so, would ground its resilience. At the same time, these activities of the host would play a causal role in driving the system back to a stable, resilient equilibrium when it is perturbed. Thus, the host would be the microbiome's resilience causal basis.

This example illustrates that ecological resilience may have a causal base that spans different levels: the level of the system as a whole, where the network structure of the ecological relationships is the causal basis of the microbiome, and the host level, where the host exerts regulatory control over the microbiome via different specific mechanisms.

---

<sup>2</sup> Note that, as analyzed in Suárez & Deulofeu (under Review), this causal role would depend on the system's teleological nature, rather than on the causal efficacy of the microbiome.

Let us analyze **size constancy** as a second example. Humans and other animals possess different kinds of visual perceptual constancies. These can be characterized as abilities “to perceive objects as having the same physical properties despite changes in retinal input” (Sperandio & Chouinard 2015). Examples are color constancy, lightness constancy, shape constancy, and motion constancy. While all of these different examples are potentialities with multiple levels of causal bases, we will focus on size constancy: The ability to perceive objects as having the same size despite different distances.

Organisms with size constancy have mechanisms that ensure that the representation of objects' objective size depends on the stimulus's size on the retina and cues, such as horizontal disparity and vergence angle, that indicate the object's distance (Schulte 2021). These mechanisms are a plausible causal basis for size constancy. On the one hand, organisms have size constancy because they have these mechanisms. On the other hand, these mechanisms are certainly causally involved when an organism exercises its ability to have size constancy.

While this high-level description may apply to all organisms with size constancy, it is realized differently in different organisms. According to Qian and Yazdanbakhsh (2015), human size constancy is realized by different visual processing areas in the brain along the ventral visual pathway. For example, depth cues are integrated in MT (middle temporal area) and LIP (lateral intraparietal area). At the same time, the retinal signal arrives in the primary visual cortex (V1) and is further processed in V2 and V4. Other parts of V1, V2, and V4 integrate retinal image size and distance information. These parts of MT, LIP, V1, V2, and V4 are plausible causal bases of human size constancy. First, if Qian and Yazdanbakhsh are correct, humans would not have size constancy without these specific brain regions. Thus, they are grounding size constancy. Second, these mechanisms also play a causal role in the manifestation of size constancy. In short, one causal basis for size constancy in humans is that they have mechanisms that integrate retinal image size with cues to object distance; another causal basis is that their brains contain certain specific mechanisms in MT, LIP, V1, V2, and V4.

In this section, we have argued that instantiations of ecological resilience have a causal base that spans different levels and that size constancy has multiple causal bases at different levels.

The second observation could have been made for all of our examples. In general, instantiations of potentialities have multiple causal bases (1) as long as these instantiations have multiple full grounds and (2) as long as these full grounds are also possible causal factors for manifesting these potentialities. Condition (1) is trivially fulfilled for potentialities in the life sciences because these are higher-level sciences, and the grounding relation is transitive (e.g., Correia 2010). So, usually<sup>3</sup>, if some set of property instantiations *A* grounds another set of property instantiations *B*, and *B* grounds a potentiality instantiation *C*, then *A* grounds *C*. Concerning condition (2), it is also plausible that a full ground of a causal factor for some manifestation is also a causal factor for this manifestation – assuming interlevel causation (see, e.g., Craver and Bechtel 2013). Consequently, it is to be expected that every higher-level potentiality has multiple causal bases.

This result applies not only to potentialities in the life sciences but also to most other sciences - with the possible exception of some areas of physics - and even to our standard examples (water-solubility, fragility, elasticity, and soporificity). The reason why this has not been noticed before lies in two facts. First, under the name "categorical bases," causal bases were a crucial tool for Humean metaphysicians arguing for the thesis that the world can be fully characterized in terms of instantiations of categorical properties (Lewis 1986, p. 162). In this context, it is of no importance to discover different causal bases at different levels but only to find the *one categorical* causal base for each instantiation of a potentiality. Our project, however, is different. In the spirit of naturalistic/scientific metaphysics (e.g., Ladyman et al. 2007; Chakravartty 2017) and inductive metaphysics (e.g., Engelhard et al. 2021), we explore the implications of science for our metaphysics of potentialities. Second, philosophers of the life sciences are inclined to pay attention to different levels and their relations. The New Mechanists, for example, explicitly emphasize that the components of mechanisms are often mechanisms themselves (e.g., Craver 2007, 3.3).

---

<sup>3</sup> Even though there are reasons to believe that grounding transitivity does not hold in every single case (e.g., Bliss and Priest 2018), we believe that it holds for standard cases.

## 5. Processuality

In the debate on dispositions, some authors argue that the commonly assumed static “stimulus-response” schema is unsuited for biological dispositions due to the processual nature of manifestation events (e.g., Anjum & Mumford 2018; Hüttemann & Kaiser 2018). In this section, we will argue that this line of reasoning can be extended to CBs of potentialities in the life sciences.

Even though most philosophers are aware of the fact that different instantiations of the same potentiality can have different causal bases (since dispositions are multi-realizable), they tacitly assume that CBs are static in three ways. First, they assume that the causal base remains the same during the instantiation of a potentiality. Second, they assume that the metaphysical ground of the causal base remains the same throughout its instantiation. Third, they assume that no activity is required to keep the causal base instantiated. These assumptions can be traced back to the one-sided diet of examples typically given for causal bases (see sect. 1). For example, the fact that the wine glass is breakable is causally based on the fact that it is made of a relatively thin silicon oxide lattice. It is assumed that this lattice remains the same as long as the glass remains breakable, that it has a constant metaphysical ground, and that no activity is required to keep this causal base instantiated. Similar considerations apply to the CBs of our other stock examples: that the sugar cube is composed of weakly bonded sucrose molecules, that the rubber band is made of entangled polymers, and that a sleeping pill contains 15 mg of flurazepam are all static property instantiations. In this section, we will show that CBs in the life sciences challenge this orthodoxy.

Take the example of the **ability of cells to undergo programmed cell death (PCD)**. A cell has this ability only if it has the correct internal program that can cause it to die in the appropriate way. Dying through PCD requires the activation of sophisticated biomolecular pathways, which are primarily encoded in the genome and need to be transcribed through the action of the ribosomes. For a cell to retain its PCD ability over time, it must maintain its CB. Maintaining the CB for PCD requires substantial turnover: the genome needs to be maintained, copied, and repaired to avoid potential mutations; the ribosomes need to be replaced by new ribosomes as they lose their function over time, and so on. These processes need to occur in the correct way so that the cell retains its PCD ability. An inadequate



genomic mutation at some point may turn a cell that possesses PCD into a cancerous cell that has lost the PCD ability. The same would happen if the cell gets deprived of its ribosomes or does not replace them adequately so that they can still act as genomic transcriptors. Even though particular changes are required, PCD is retained during such processes, and we contend it is retained *because of* such changes. A cell that does not maintain, copy, and repair its genome, or one in which ribosomes are not replaced after a certain time lapse, would eventually malfunction. The transcription would not be possible anymore. Genomic transcription is essential for the ability to undergo PCD because PCD depends on the cell's ability to activate an internal program, leading to its destruction from within, and the cell would lose the ability to undergo PCD. The ability to undergo PCD is non-static insofar as it needs to be actively maintained.

Second, consider **psychological resilience**, the ability to maintain or recover mental health under significant adverse conditions. In some cases, the causal base of psychological resilience changes during one person's lifetime while resilience remains constant.

For discussion, we will concentrate on psychological resilience to social anxiety disorder (SAD). People with social anxiety disorder will, on the one hand, experience anxiety in certain social situations and, on the other hand, avoid these situations. According to Rapee and Heimberg's (1997) model, people with social anxiety show distortions and biases in processing and judging social information. They will have an increased fear of corresponding situations. Which in turn leads to avoidance of social situations. This avoidance, in turn, prevents these biases and distortions from being corrected. A person who possesses a high degree of psychological resilience to social anxiety will be such that fear of social situations does not cause avoidance, which in turn allows the correction of biases. A weak causal link between fear and avoidance is plausibly already a causal base for resilience to SAD.

This link, however, will further be grounded in other factors. A lot of resilience research is concerned with so-called "protective factors" and "risk factors" for specific mental disorders. Take the following example, where a systematic review concerning modifiable risk and protective factors for anxiety disorders came to the following conclusion:

Results of the present study found that risk factors with some support for one or more anxiety disorders included alcohol use, cigarette smoking, cannabis use, avoidance behavior, occupational factors, and negative appraisals of stressful life events.

Protective factors that yielded some support included physical activity, sports participation, social support, and coping skills. (Zimmerman et. al. 2020, 112705)

First and foremost, these factors correlate with the likelihood of developing SAD. So, not all such factors will be causal bases of the psychological resilience of some people. In order to establish this, we would need to show that these factors can also play a positive or negative causal role in this person's development of anxiety disorders and that these factors *ground* this person's resilience rather than merely being manifestation conditions (like oxygen for a match's flammability) or factors that causally influence the real causal base (like water exposure alters the ignition head in such a way that it loses its flammability).

Some protective factors, however, will undeniably be causal bases of some persons' psychological resilience. To see this, we must consider a particular instantiation of psychological resilience to SAD. This step to the token level is necessary because resilience is realized in multiple ways, making it difficult to make general statements about its causal basis. However, this multiple realization is one of the reasons for the assumption that the causal basis of resilience does not remain constant. So, take a closer look at the fictional girl Natcha. Natcha has strong resilience against SAD. In Natcha's case, fear of social situations will not cause her to avoid them. This is because Natcha's family helps her deal with her fears and encourages her to face them. Plausibly, the fact that Natcha has ample social support will be a causal base of her psychological resilience. Without this factor, Natcha would more easily fall into the vicious circle of SAD. Consequently, social support grounds her resilience, and it does so precisely because this social support plays a causal role in her avoiding withdrawal in response to adverse events.

However, while Natcha's resilience to SAD may remain constant during her lifetime, its causal base will likely change. It has been shown that for children, having a functioning and supportive family is an essential protective factor against anxiety (Masten 2001; Huang et al. 2012; Wu et al. 2016). This factor, however, seems to be much less important for adults (Weisenhorn et al. 2017). This suggests that the causal base of psychological resilience can change over time. Let us, for example, consider Natcha at thirty and her resilience to SAD. Natcha is still psychologically resilient against SAD, and this is still due to the fact that Natcha does not avoid social situations despite anxiety, which, in turn, helps to correct her biases. However, while Natcha, as a child, needed external encouragement from her family,

the exact role is now played by an internalized belief that it is good to face one's fears. This belief, instead of external support from her family, is now part of the causal base of Natcha's resilience against SAD. While the causal base has changed, this does not make a difference for the instantiation of the potentiality itself.

The cell's ability to PCD and Natcha's psychological resilience to SAD show that the CBs of potentialities in the life sciences are more complex than in the examples that take center stage in metaphysical debates on potentialities. The reason is that objects investigated in the life sciences are typically dynamic. By contrast, the standard examples of potentialities and their CBs are non-living objects such as wine glasses, sugar cubes, rubber bands, and sleeping pills.

## 6. Conclusion

A detailed look at examples of potentialities and their causal bases from the life sciences shows that fundamental assumptions about causal bases must be revised. In this paper, we argued that many causal bases in the life sciences are extrinsic, multi-level, and processual. Since biology and psychology often investigate complex potentialities that entities have only because they are intimately intertwined with their environment, it is not surprising that their causal bases are often extrinsic. The hierarchical and interlevel structure of biological and psychological phenomena makes it evident that potentialities often have causal bases spanning different levels or multiple full causal bases at several lower levels. Finally, the non-static nature of living organisms ensures that the causal bases of potentialities are also non-static.

## Acknowledgments

We would like to thank the participants of the workshop "Dispositions in Life Sciences. Contemporary and Historical Perspectives" in Trier (2022), the participants of the Research Seminar Theoretical Philosophy, and the PhilBio team in Bielefeld - especially Ozan Altinok, Daniel Friedrich, Robert Frühstückl, Lara Keuck, Anton Killin and Katie H. Morrow – for valuable comments on earlier versions of this paper. Special thanks are due to Patrick Hoare for finalizing the references and proofreading.

## Funding information

Research for this paper was funded by the German Research Foundation (Deutsche Forschungsgemeinschaft, DFG), project on Complex Biological Dispositions: A Case Study in the Metaphysics of Biological Practice (Nr. 288923097) in the research group Inductive Metaphysics (FOR 2495/2). Javier Suárez also profited from a grant from the Ministry of Science, Innovativeness and Universities (project PID2022-137993NA-I00, “An Exploration of the Philosophy of Lineages in the Light of Contemporary Biology”).

## References

- Alvarez, María. 2017. “Are Character Traits Dispositions?”, *Royal Institute of Philosophy Supplement* 80: 69–86. <https://doi.org/10.1017/S1358246117000029>
- Anjum, Rani Lill and Stephen Mumford. 2018. *Causation in Science and the Methods of Scientific Discovery*. Oxford University Press.  
<http://dx.doi.org/10.1093/oso/9780198733669.001.0001>
- Arenas, Minerva C. and Carmen Manzanedo. 2016. “Novelty Seeking”, in *Encyclopedia of Personality and Individual Differences*, edited by Zeigler-Hill, Virgil and Todd K. Shackelford Springer, Cham. [https://doi.org/10.1007/978-3-319-28099-8\\_1095-1](https://doi.org/10.1007/978-3-319-28099-8_1095-1)
- Berger, Itai and Hanoach Cassuto. 2014. “The effect of environmental distractors incorporation into a CPT on sustained attention and ADHD diagnosis among adolescents,” *Journal of Neuroscience Methods* **222**, 62–68. <https://doi.org/10.1016/j.jneumeth.2013.10.012>
- Bliss, R., & Kelly Trogon. 2024. “Metaphysical Grounding,” *The Stanford Encyclopedia of Philosophy* (Summer 2024 Edition), Edward N. Zalta & Uri Nodelman (eds.), URL = <<https://plato.stanford.edu/archives/sum2024/entries/grounding/>>.
- Bliss, Ricki, and Graham Priest, eds. (2018) *Reality and Its Structure: Essays in Fundamentality*, Oxford University Press.  
<http://dx.doi.org/10.1093/oso/9780198755630.001.0001>
- Cassuto, Hanoach, Ben-Simon, Anat, & Berger, Itai. 2013. “Using environmental distractors in the diagnosis of ADHD”. *Frontiers in Human Neuroscience*, 7, Article 805.  
<https://doi.org/10.3389/fnhum.2013.00805>
- Chakravartty, Anjan. 2017. *Scientific Ontology: Integrating Naturalized Metaphysics and Voluntarist Epistemology*. Oxford University Press.
- Choi, Sungho and Michael Fara. 2021. “Dispositions”, *The Stanford Encyclopedia of Philosophy* (Spring 2021 Edition), Edward N. Zalta (ed.), URL = <<https://plato.stanford.edu/archives/spr2021/entries/dispositions/>>.
- Contessa, Gabriele. 2012. “Do Extrinsic Dispositions Need Extrinsic Causal Bases?”, *Philosophy and Phenomenological Research* **84**(3), 622–638.  
<http://dx.doi.org/10.1111/j.1933-1592.2010.00435.x>
- Correia, Fabrice. 2010. “Grounding and Truth-Functions,” *Logique et Analyse* **53**(211), 251–279.

- Coyte, Katherine Z., Jonas Schluter and Kevin R. Foster. 2015. “The ecology of the microbiome: Networks, competition, and stability,” *Science* **350**(6261), 663–666. <http://dx.doi.org/10.1126/science.aad2602>
- Craver, Carl F. 2007. *Explaining the brain: mechanisms and the mosaic unity of neuroscience*. Oxford University Press. <http://dx.doi.org/10.1093/acprof:oso/9780199299317.001.0001>
- Craver, Carl F., Bechtel, William. 2013. Interlevel Causation. In: Dubitzky, W., Wolkenhauer, O., Cho, KH., Yokota, H. (eds) *Encyclopedia of Systems Biology*. Springer, New York, NY. [https://doi.org/10.1007/978-1-4419-9863-7\\_69](https://doi.org/10.1007/978-1-4419-9863-7_69)
- Dakos, Vasilis and Sonia Kéfi. 2022. “Ecological resilience: what to measure and how”, *Environmental Research Letters* **17**(4). <https://doi.org/10.1088/1748-9326/ac5767>
- Dinges, Alexander and Julia Zakkou. 2021. “Taste, traits, and tendencies”, *Philosophical Studies* **178**(4), 1183–1206. <http://dx.doi.org/10.1007/s11098-020-01470-7>
- Díez, José and Javier Suárez. 2023. “How do networks explain? A neo-hempelian approach to network explanations of the ecology of the microbiome”, *European Journal for Philosophy of Science* **13**, 44. <https://doi.org/10.1007/s13194-023-00549-2>
- Engelhard, Kristina, Feldbacher-Escamilla, Christian J., Gebharter, Alexander, & Seide, Ansgar. 2021. “Inductive metaphysics: Editors' introduction”, *Grazer Philosophische Studien* **98**(1), 1–26. <http://dx.doi.org/10.1163/18756735-00000129>
- Fassarella, Marina, Ellen E. Blaak, John Penders, Arjen Nauta, Hauke Smidt, and Erwin G. Zoetendal. 2021. “Gut Microbiome Stability and Resilience: Elucidating the Response to Perturbations in Order to Modulate Gut Health.” *Gut* **70** (3): 595–605. <https://doi.org/10.1136/gutjnl-2020-321747>.
- Ferreira Ruiz, María, and Fabian Hundertmark. 2024. “What Are the Causal Bases of Dispositions?” *Australasian Journal of Philosophy*. <https://doi.org/10.1080/00048402.2024.2408689>.
- Foster, Kevin R., Jonas Schluter, Katharine Z. Coyte, and Seth Rakoff-Nahoum. 2017. “The Evolution of the Host Microbiome as an Ecosystem on a Leash.” *Nature* **548** (7665): 43–51. <https://doi.org/10.1038/nature23292>.
- Hartmann, Thom. 1999. *Attention Deficit Disorder: A Different Perception*. Newleaf.
- Huang, Keng-Yen, Sabrina Cheng, Esther Calzada, et al. 2012. “Symptoms of Anxiety and Associated Risk and Protective Factors in Young Asian American Children”, *Child Psychiatry & Human Development* **43**, 761–774. <https://doi.org/10.1007/s10578-012-0295-0>
- Hüttemann, Andreas and Marie I. Kaiser. 2018. “Potentiality in Biology”, in Engelhardt, Kristina and Michael Quante, eds., *Handbook of Potentiality*, 401–428. Springer, Cham. [http://dx.doi.org/10.1007/978-94-024-1287-1\\_16](http://dx.doi.org/10.1007/978-94-024-1287-1_16)
- Ladyman, James, Don Ross, and David Spurrett with John Collier. 2007. *Every Thing Must Go: Metaphysics Naturalized*. Oxford University Press. <http://dx.doi.org/10.1093/acprof:oso/9780199276196.001.0001>
- Laplaine Laplane 2016. *Cancer stem cells. Philosophy and therapies*. Harvard University Press, Cambridge MA
- Laplaine Laplane, Solary Eric. 2019. Philosophy of biology: towards a classification of stem cells. *Elife* <https://doi.org/10.7554/eLife.46563>
- Le Cunff, Anne-Laure. 2024. “Distractibility and Impulsivity in ADHD as an Evolutionary Mismatch of High Trait Curiosity”. *Evolutionary Psychological Science*, August. <https://doi.org/10.1007/s40806-024-00400-8>.

- Lewis, David K. 1997. “Finkish Dispositions”, *The Philosophical Quarterly* **47**(187), 143–158. <https://doi.org/10.1111/1467-9213.00052>
- Maier, John T. 2022. *Options and Agency*. Palgrave Macmillan Cham. <http://dx.doi.org/10.1007/978-3-031-10243-1>
- Masten, Ann S. 2001. “Ordinary magic: Resilience processes in development”, *American Psychologist*, **56**(3), 227–238. <https://doi.org/10.1037/0003-066X.56.3.227>
- McKittrick, Jennifer. 2018. *Dispositional Pluralism*. Oxford University Press.
- McKittrick, Jennifer. 2003. “A Case for Extrinsic Dispositions”, *Australasian Journal of Philosophy* **81**(2), 155–174. <http://dx.doi.org/10.1080/713659629>
- Mumford, Stephen. 2006. “The ungrounded argument”, *Synthese* **149**(3), 471–489. <https://doi.org/10.1007/s11229-005-0570-8>
- Otto, Sarah P. and Troy Day. 2011. *A biologist’s guide to mathematical modeling in ecology and evolution*. Princeton University Press. <https://doi.org/10.2307/j.ctvc4hnd>
- Perroud, Nader, Roland Hasler, Nicolas Golay, Julien Zimmermann, Paco Prada, Rosetta Nicasro, Jean-Michel Aubry, et al. 2016. “Personality Profiles in Adults with Attention Deficit Hyperactivity Disorder (ADHD).” *BMC Psychiatry* **16** (1): 199. <https://doi.org/10.1186/s12888-016-0906-6>.
- Prior, Elisabeth W., Robert Pargetter, and Frank Jackson. 1982. “Three Theses about Dispositions”, *American Philosophical Quarterly* **19**(3), 251–257.
- Qian, Jiehui and Arash Yazdanbakhsh. 2015. “A Neural Model of Distance-Dependent Percept of Object Size Constancy”, *PLOS ONE*, **10**(7), e0129377. <https://doi.org/10.1371/journal.pone.0129377>
- Rapee, Ronald M. and Richard G. Heimberg. 1997. “A cognitive-behavioral model of anxiety in social phobia”, *Behaviour Research and Therapy* **35**(8), 741–756. [http://dx.doi.org/10.1016/S0005-7967\(97\)00022-3](http://dx.doi.org/10.1016/S0005-7967(97)00022-3)
- Roberts, Robert C. and Jay W. Wood. 2007. *Intellectual Virtues: An Essay in Regulative Epistemology*. Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780199283675.001.0001>
- Rowland, Andrew S., Catherine A. Lesesne and Ann J. Abramowitz. 2002. “The epidemiology of attention-deficit/hyperactivity disorder (ADHD): a public health view”, *Mental retardation and developmental disabilities research reviews*, **8**(3), 162–170. <https://doi.org/10.1002/mrdd.10036>
- Ryle, Gilbert. 1949. *The concept of mind*. London: Hutchinson & Co.
- Schulte, Peter. 2021. “The nature of perceptual constancies”, *Philosophy and Phenomenological Research*, **103**(1), 3–20. <https://doi.org/10.1111/phpr.12693>
- Sperandio, Irene and Philippe A. Chouinard. 2015. “The Mechanisms of Size Constancy”. *Multisensory research* **28**(3-4), 253–283. <https://doi.org/10.1163/22134808-00002483>
- Suárez, Javier. 2023. “What is the nature of stem cells? A unified dispositional framework”, *Biology & Philosophy* **38**, 43. <https://doi.org/10.1007/s10539-023-09930-0>
- Suárez, Javier and Deulofeu, Roger. Under review. *A teleological approach to microbiome causation*. In J. A. Pérez-Escobar and D. Sarikaya (eds.), *Mathematical Tools in the Life Sciences - Describing, Explaining, Understanding, and Operating*. Springer.
- Swanepoel, Annie, Graham Music, John Launer, and Michael J. Reiss. 2017. “How evolutionary thinking can help us to understand ADHD”. *BJPsych Advances* **23**(6), 410–418. <http://dx.doi.org/10.1192/apt.bp.116.016659>

- Thagaard, Marthe S., Stephen V. Faraone, Edmund J. Sonuga-Barke, and Søren D. Østergaard. 2016. “Empirical Tests of Natural Selection-Based Evolutionary Accounts of ADHD: A Systematic Review.” *Acta Neuropsychiatrica* 28 (5): 249–56. <https://doi.org/10.1017/neu.2016.14>.
- Vetter, Barbara. 2015. *Potentiality: From Dispositions to Modality*. Oxford University Press. <http://dx.doi.org/10.1093/acprof:oso/9780198714316.001.0001>
- Vetter, Barbara. 2024. “Properties, potentialities and modality”, in A. R. J. Fisher & Anna-Sofia Maurin (eds.), *The Routledge Handbook of Properties*. London: Routledge. pp. 315-324.
- Vorotelyak Ekaterina, Andrey Vasiliev and Vasiliy Terskikh. 2020. “The problem of stem cell definition”, *Cell and Tissue Biology* 14, 169–177. <https://doi.org/10.1134/S1990519X20030086>
- Weisenhorn, David A., Laura M. Frey, Jason D. Hans, and Julie Cerel. 2017. “Suicide Ideation, Anxiety, and Depression: Are Children a Protective Factor for Male Veterans?” *Journal of Family Social Work* 20 (1): 41–51. <https://doi.org/10.1080/10522158.2017.1286278>.
- Williams, Neil E. 2010. “Dispositions and the Argument from Science.” *Australasian Journal of Philosophy* 89 (1): 71–90. <https://doi.org/10.1080/00048400903527766>
- Wu, Yi-Le, Xue Zhao, Yi-Feng Li, Xiu-Xiu Ding, Hui-Yun Yang, Peng Bi, and Ye-Huan Sun. 2016. “The Risk and Protective Factors in the Development of Childhood Social Anxiety Symptoms among Chinese Children.” *Psychiatry Research* 240 (June):103–9. <https://doi.org/10.1016/j.psychres.2015.08.046>.
- Xiao, Yandong, Marco Tulio Angulo, Songyang Lao, Scott T. Weiss, and Yang-Yu Liu. 2020. “An Ecological Framework to Understand the Efficacy of Fecal Microbiota Transplantation.” *Nature Communications* 11 (1): 3329. <https://doi.org/10.1038/s41467-020-17180-x>.
- Zimmermann, Martha, Adrienne K. Chong, Catalina Vechiu, and Anthony Papa. 2020. “Modifiable risk and protective factors for anxiety disorders among adults: A systematic review”, *Psychiatry Research* 285 (March):112705. <https://doi.org/10.1016/j.psychres.2019.112705>.