Including or Excluding Free Will

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1 Introduction: Why being caused doesn't imply being fixed in advance

For much of modern thought, the following principle, or something close to it, has been widely thought to have empirical support:

The laws of physics (or nature)—which include laws concerning causes—together with the past, determine or fix the present and future.

Historically, supposed evidence for the truth and complete generalizability of Newtonian or Classical mechanics was thought to serve as evidence for the truth of this principle—let's call it the *principle of determinism*. Consequently, this principle has been formative in shaping how many scientific and philosophical communities think about causation. So much so that *being caused* has had a long history of being thought of as implying *being determined or fixed in advance,* or, in some sense, necessitated [1, 2]. This is forcefully seen in Laplace's famous view that:

> We ought to regard the present state of the universe as the effect of its antecedent state and as the cause of the state that is to follow. An intelligence knowing all the forces acting in nature at a given instant, as well as the momentary positions of all things in the universe, would be able to comprehend in one single formula the motions of the largest bodies as well as the lightest atoms in the world, provided that its intellect were sufficiently powerful to subject all data to analysis; to it nothing would be uncertain, the future as well as the past would be present to its eyes. The perfection that the human mind has been able to give to astronomy affords but a feeble outline of such an intelligence [3].

As it relates to our conduct, Robert Sapolsky has recently expressed the same general notion about causation in this way:

> When you behave in a particular way, which is to say when your brain has generated a particular behaviour, it is because of the determinism that came just before, which was caused by the determinism just before that, and before that, all the way down [4].

According to such ways of thinking: If change is caused, it is fixed in advance.

Over the past 100 years or so, in part precipitated by discoveries about quantum motion, Classical pictures of the universe have become antiquated. And, while this has undermined the notion that the principle of determinism has empirical support (cf. [5]), this hasn't always trickled down to notions about causation. It, thus, isn't uncommon to still come across the idea that:

To the extent change is caused, it is fixed in advance.

What this implies is that:

To the extent change isn't fixed in advance, it isn't caused.

And such notions about causation continue to underlie claims to the effect that science is revealing that 'we are nothing more or less than the cumulative biological and environmental luck, over which we had no control, that has brought us to any present moment' [4].[1]

The problem, however, is that this way of thinking about causation is nothing more than an artefact of a bygone era. Though the modern tendency to think that being caused implies being fixed in advance took hold due to the popularity of attempts to use Classical mechanics to explain all physical change, this tendency hasn't waned along with the waning of its basis. Thus, though this implicature was once thought to reflect sound inductive reasoning from observations about the universe, it doesn't. Rather, it is perhaps clearer now than ever that the belief that being caused implies being fixed in advance is mistaken [2].

To provide an illustration for how change can clearly be caused without being fixed in advance:

A radioactivity detector (e.g., a Geiger counter)—which moves with the release of energy by radioactive material—might be set up such that it stops a mechanism in a machine that repeatedly stamps 'Coca Cola' onto pieces of tin moving on a conveyor belt below the stamp. The mechanism that does this stamping might have parts that are continuously making a circular motion, moving the stamp either up or down. And this machine might be engineered such that a 'tiny bit' of radioactive material is imbedded in it,[2] and positioned within or against the detector, such that, simultaneous to the release of energy by this material, the detector shifts into place, stopping this circular motion. This release and shifting could take place similar to how putting a dent into the hood of a car occurs simultaneous to one sitting on it [8, 9].

In this example, the machine causes the continuous circular motion of the parts moving the stamp up or down. And not just to some extent. This is true even though whether this motion will occur at any moment isn't fixed in advance. The reason is the release of energy by the radioactive material imbedded in the machine will stop this motion, and whether this will occur at any moment isn't fixed in advance.

This example illustrates something which—especially with Classical pictures of the universe being obsolete—shouldn't be controversial. Namely, that change—in the example here, the circular motion of machine parts—might be completely caused *without* its occurrence being fixed in advance.

In fact, for at least most physical systems that contain causes, it is at least logically possible that something outside that system might, at any given moment, intervene to stop causal processes occurring in it [2, 9, 10]. This, along with the principle of determinism lacking empirical support, raises the very real possibility that many caused changes aren't fixed in advance. And yet this possibility, by itself, doesn't bring into question whether these changes are *completely* caused.

So, then what is caused might very well not be determined or fixed in advance.

Moreover, our state-of-the-science models for the behaviour of a great number of physical systems are statistical or stochastic. They are probabilistic. There is, thus, some margin of error between what is predicted using these models and what happens. And this is true of our models for many phenomena studied across every domain of science including physics.[3] Thus—as made famous by the Bohr-Einstein debate in quantum mechanics [48, 49]—at least one of two things must be true about these models:

[1] For a more philosophically sophisticated articulation of this sort of view see [6].

[2] This would be similar to the amount Schrödinger [7] has us imagine in his box, such that there are equal probabilities that one atom will decay or not decay over the course of an hour.

[3] For example, changes in molecules in liquid state [11]; motion of molecules in liquids and gases [12–16]; thermodynamic processes [17–20]; chemical processes, including biochemical processes in living things such as the regulation of gene expression [21–31]; growth of bacterial populations [32]; development and growth of plants [33–35]; physiological processes [36, 37]; neural processes in functioning nervous systems [38–40]; psychological processes and animal and human behavior [41]; changes within financial markets or, more generally, social structures [42–47].

- at least one cause is missing; and/or
- what is being modelled isn't, at every moment, fixed.

The implication is that the extent to which our best scientific models are stochastic are what we should expect given many everyday changes which are caused aren't fixed in advance. For example, the wearing out of a spark plug over years of use, which eventually causes a car engine to stall. Or changes in cold and hot air moving up and down a rocky mountain slope over the course of a day and night, and any effects this change has. The only reason I used the above example involving radioactive decay—which isn't an everyday example—is because it has been clear for some time that any deterministic assumptions about this decay lack empirical warrant. And so, it permits of a relatively quick and easy example of where a cause might cause change without it being fixed in advance that it will do so.

But what does this have to do with free will? After all, free will—and not, say, radioactive decay—is our focus here. Having seen that we shouldn't think that it is out of the ordinary for change to be caused without being fixed in advance, let's turn to this question.

2 Stochastic neural models and the inclusion of free will

Our best neuroscientific models for the overall behaviour—or, that is, for all of the changes in states and activities—of a conscious animal's nervous system are stochastic [38–40]. So:

When only considering *the variables—including causes—of interest in neuroscience*, the overall behaviour of a conscious animal's nervous system can only be *probabilistically predicted*.

This is a pivotal observation since—to apply the lesson learned from the Bohr-Einstein debate—at least one of two things must be true where the best models for physical change are stochastic:

- at least one cause is missing; and/or
- what is being modelled isn't, at every moment, fixed.

Thus, since—as we just saw—we shouldn't think there is anything out of the ordinary about change being caused without being fixed in advance, the fact our best neuroscientific models for the overall behaviour of a conscious animal's nervous system are stochastic might, at least in part, result from not including what the conscious animal, *as a distinct whole*,[4] causes *when* they think or act. In short, they might be a missing cause from our models.

What a conscious animal thinks and/or does is not included as a *distinct* variable in our neural models for the behaviour of their nervous system.[5] And yet, when we consider that we are, after all, modelling the behaviour of *a conscious animal's* nervous system, we must have other competing ideas to dismiss the idea outright that some of what *they* think and/or do might have *unique* explanatory value in explaining and predicting certain changes in *their* neural states and activities.[6] We must have other competing ideas to not consider the possibility that, when the animal thinks and/or acts, they cause changes in the states and/or activities of their nervous system;[7] and sometimes

[4] By this, I mean a distinct physical thing, substance, or 'system' with its own distinct characteristics which, under certain conditions, operates as a distinct cause [50–53]. For my purposes here, I am not defining any further what it is to be a conscious animal. I am simply identifying it as a distinct physical thing that might be a cause irreducible to and unanalysable in terms of any other physical thing that might also be a cause.
[5] Distinct in the sense of not being reducible to or analyzable in terms of some other variables.
[6] To have unique explanatory value is to add to the precision and accuracy of explanations and predictions in a way that isn't duplicated by any other variable or variables.
[7] I will say more about these competing ideas in the next section.

cause changes not caused by any other cause.[8]

This initial observation is important to the conversation about free will because:

Including some of what one thinks and/or does as a distinct variable within models for certain behaviour of one's nervous system—and thus also within models for certain bodily changes—would be a prerequisite for *including one's expression of free will as a distinct variable*.

Here, by 'free will', I mean the ability to do as one chooses without undue constraint; for example, due to being blackmailed or, in some relevant way, manipulated. This ability is expressed—whether actively or passively—whenever:

- one is aware of genuine alternative ways one might behave on a given occasion;
- these ways are not limited, and one is not being compromised, in unusual ways; and
- one intentionally behaves in one of these ways.

And if what one thinks and/or does as an expression of free will is a distinct variable, which provides *unique explanatory value* in explaining and predicting certain changes (such as certain neural changes), then one at least sometimes causes changes not caused by any other cause when one expresses free will in active ways.

Let's consider a basic example: A person making a choice in a forced-choice task.

In these tasks, one agrees to comply with instructions by choosing to behave in one of two ways without forethought or reason. In other words, one agrees to choose spontaneously between two options, thereby intentionally acting in one of two ways. One agrees to make a choice on an urge or impulse. In a common version of the task, one chooses to press a button with either one's left or right hand.

Now, if any of what we naively think of as our chosen behaviour is fixed in advance, behaviour in such tasks certainly would be. Despite this, predictive neural models about what one will do on isolated trials based on changes in neural states and activities in the seconds leading up to one's action only have around 59% accuracy [54, 55]. They are wrong around 41% of the time. That is, up until milliseconds before one acts, when they are still wrong around 17% of the time [56].

To add to this, the probability motor-related neural activities will unfold, and thus that one will act, in certain ways on any given trial, is influenced by:

- what one has previously done and experienced in similar situations [57–59] and
- stochastic neural fluctuations that occur while one is conscious and at rest [60–62].

Moreover, various disposing factors can shape the probability one will act in a certain way, even when one is unaware of it.[9]

But, over and above all this, on any given trial—including those where the prediction turns out to be accurate—one could change one's mind about what to do, and even about whether to continue participating, *after* predictive motor-

[8] For example, the simultaneous occurrence of certain coordinated neural states and/or activities in various areas or pathways of the brain might, in part, be caused by the animal when they think or act in certain ways in interaction with their environment. In this case, without taking into account what the animal does or is doing, this occurrence would appear to occur to some extent randomly or by chance.
[9] In addition to neural factors, these include affordances, biases, and various situational cues and factors [63–67].

related activities commence. One could, in fact, change one's mind up to around the occurrence of primary motor cortical activity leading to muscle contraction and extension when one acts [68, 69]. It's just that variables related to the likelihood of this happening, while relevant, aren't included in predictive models for what one will do in these studies since one has *already agreed to comply* and has, in fact, been complying, making this likelihood very low.

The overall point, here, is that given state-of-the-art neuroscientific observations:

The functioning nervous systems of conscious animals, including people, are not only *stochastic systems*, but *many variables extending over time—including those at least related to what they have already done and experienced—* provide unique explanatory value in explaining and predicting these system's behaviour as it relates to the animal thinking or doing something.

What's more, under many conditions:

There will be error in our predictive neural models for the relevant behaviour of a person's nervous system leading *up to when they think or do something*.

And:

The further away we are from a person thinking or doing something, *the more opportunity they have to think and/or do other things leading up to it*, and *the greater the error* in our predictive neural models.

This includes predictive neural models for changes in motor-related states and/or activities which lead to the relevant muscle contractions and extensions when they act. And, as we've seen, this is even true in cases where a person is set on acting upon impulse and thereby choosing between two previously agreed upon options.

The implication is that, under many conditions, what a person ends up doing—not to mention how they come to do it (e.g., what they have agreed to, considered or haven't considered, already done)—provides an explanation for why certain relevant changes occur when we have no other empirically warranted explanation for their occurrence. For, if functioning nervous systems are truly stochastic systems in the sense that we have evidence they are, without using what the person does, and how they come to do it, to explain or predict the occurrence of these changes, we are left with the conclusion that they occur randomly or by chance (a topic we will come back to). We are left without an explanation as to why they occur. And this wouldn't be due to a lack of knowledge about the underlying neural states and activities.

What this means is that, as things stand:

Including what a person thinks and/or does as expressions of free will in models for certain behaviour of their nervous system has unique explanatory value.

In this case, our neuroscientific findings align with the idea that when a person—as a conscious animal—does something as simple as spontaneously press a button with their left hand instead of their right, they cause certain changes in their neural states and/or activities. And sometimes, when they do such things, they cause changes not caused by any other cause.

3 Stochastic neural models and the exclusion of free will

Earlier, I mentioned that competing ideas might lead us to *exclude what one thinks and/or does* from stochastic models for relevant neural changes. Considering what we've just seen, the biggest threat to this inclusion would seem to come from accepting what is called *the causal closure principle*.

In general terms, this principle speaks to the sufficiency of the causes studied in physics—or at least some set of sciences deemed the 'hard sciences'[10]—in accounting for all physical effects. And the truth of this principle, as supported by science, would seem to imply that how a person expresses free will *doesn't offer unique explanatory value* in explaining any physical change. In essence, it may seem that our empirical findings support the truth of the causal closure principle to the exclusion of free will from the relevant models.

There isn't, however, one accepted causal closure principle. This principle has been stated in many non-equivalent ways [70]. Some versions have relied on the principle of determinism being true. And, thus, the lack of evidence for the truth of this principle means there is a lack of evidence for these versions.

What is, then, needed is a causal closure principle that doesn't rely on the principle of determinism which:

- implies how a person expresses free will doesn't offer unique explanatory value in models for any physical change; and
- has empirical warrant.

The problem is there is no such causal closure principle. Causal closure principles with the required implicature lack empirical warrant.

To see this, let's look at an example:

'Every physical effect has its chance fully determined by physical events alone' [71].[11]

Given this principle, *at every moment,* to whatever extent *the chance* of *all* physical effects is fixed, this chance is fixed by *the causes studied in the hard sciences alone*. So, at every moment, these causes are all it takes to set the chance of all physical effects to whatever extent this chance is set. Thus, the truth of this principle would entail that how a person expresses free will doesn't offer unique explanatory value in predictive models for any physical change.

Here, it bears noting that principles like the one stated above are, to some extent, holdovers from when Classical pictures of the universe were popular and the principle of determinism was widely endorsed as having empirical support.

Traditional causal closure principles state or imply that *all physical effects themselves* are, at every moment, fixed by physical causes. However, due to a lack of evidence for the truth of the principle of determinism, causal closure principles have been tweaked to state or imply that it is *the chance of all physical effects* (rather than the effects) that are, at every moment, fixed by sufficient physical causes. The problem is, just like their predecessors, these tweaked versions of the causal closure principle lack empirical warrant.

As we've already seen, our state-of-the-science models for many kinds of physical change that have a cause—including neural changes relevant to the expression of free will—are stochastic. There is error in these models. This is true across every domain of science, including the hard sciences.[12] And, to once more apply the lesson learned from the Bohr-Einstein debate:

Within every domain of science including the hard sciences, the extent to which our best models for physical change

[10] Typically, physics, chemistry, biology, astronomy, and geoscience, and their subfields.
[11] D. Papineau provides a similar example: 'The chance of all physical effects are always fixed by sufficient physical causes' [72]. Here, I should note that 'sufficient physical causes' is used as a term of art referring to causes that, when present, *always* cause their effect. This use of sufficient is, thus, different from its common use to refer to being enough, adequate, or all it takes. For discussions of other types of causal closure principles, including those that don't exclude the expression of free will see [70].
[12] See Footnote 3.

are stochastic is consistent with their being causes missing from these models.

In fact, the extent to which our best models are stochastic is what we should expect given there are causes other than those considered in these models which, under certain conditions, have a physical effect.

The upshot is that what we observe is what we should expect if causal closure principles that imply the exclusion of free will—like the one presented above— are false. There, thus, isn't empirical support for causal closure principles which imply that how a person expresses free will doesn't offer unique explanatory value in predictive models for certain physical change.

To put this in more colloquial terms, the conclusion that if we 'put all the scientific results together, from all the relevant scientific disciplines... there's no room for free will' [4] is false.

Having said this, it might be argued that we should exclude the expression of free will from our models for the relevant changes due to the lack of evidence for 'fundamental mental forces' [73]. Here, however, it is important to recognize that we don't directly see any force—or power, ability, or disposition for that matter [74]. These are abstract concepts that derive their meaning from more concrete ones [2, 75]. And these more concrete concepts are specific to the models to which they belong and/or what is being modelled [75, 76]. When it comes to scientific models, it is this specificity that ensures precision of meaning.

Furthermore—often with technological help—what we observe are specific substances or entities (e.g., magnets, electromagnetic waves/particles, ions, objects of mass), and/or the manifestation of specific forces or abilities under certain conditions. To illustrate, we see a magnet, and we can see it exert a magnetic force by, for example, moving metal objects toward or away from it when they are located at different positions in relation to it. We can't, however, directly see a magnetic force.

Likewise, if a conscious animal were to cause certain physical changes by thinking or behaving in certain ways, we shouldn't expect to see a mental or psychological ability. Rather, we should see the manifestation of this ability. And this would, then, be exactly what we see under ordinary circumstances when we, for example, see a person choosing to press a button with either their left or right hand.

Nevertheless, it has been suggested that if a conscious animal were to manifest a psychological ability, and thereby cause change not caused by any other case, we should somehow find this ability operating somewhere within their nervous system (cf. [68]). We should find it in play alongside the relevant physiological and biochemical forces within nervous systems. It should be cooperating and/or competing with these forces.

The problem with this suggestion is that, if we were to somehow discover *the operation* of a psychological ability *in discrete places* in nervous systems—alongside physiological and biochemical forces—this would not be evidence of the conscious animal manifesting a psychological ability. It would be evidence of something *within* the animal manifesting a force or ability, like a homunculus. It would be evidence of something other or less than the animal doing something.

Rather, if a conscious animal were to manifest a psychological ability, and thereby cause change not caused by any other cause, what we should find is that the best models for some of the relevant behaviour of their nervous system are *stochastic as long as* we don't take into account their manifestation of this ability. We should also find that taking into account this manifestation increases the precision and explanatory power of these models. In other words—as outlined earlier—we should find exactly what we are finding through neuroscience.

Thus, despite claims to the contrary [4, 72, 77, 78], our scientific findings don't provide evidence for the exclusion of what one thinks and/or does from models for the relevant changes in one's neural states and/or activities. They don't, then, provide evidence for the exclusion of free will.

There is, on the other hand, a problem that proponents of those who wish to exclude the expression of free will from stochastic models for certain neural changes must face.

If:

- under certain conditions, including what a person chooses or intends to do on a given occasion provides unique explanatory value in explaining and predicting how the behaviour of their nervous system unfolds, as it seems to; and
- the best neural models for this behaviour are stochastic,

then those who wish to exclude the expression of free will from these models are left to conclude that neural changes pivotal to the generation of the most complex behaviour of the most evolutionarily advanced animals we are aware of are, to some extent, random or left to chance. These changes are to some extent uncaused. And it seems implausible that this would be adaptive over ways of integrating experience and/or knowledge to come to a determinate way of behaving. This seems even more implausible where the person can give reasons for why they behaved as they did. It seems rather more plausible that—through evolutionary processes—the potential for a wide range of *distinct abilities*, including psychological abilities such as awareness and choice, becomes actualized and manifested.

But including the expression of free will brings up its own questions. These include questions about:

- the neural substrates of awareness;
- how neural states and activities might change as one becomes aware of more alternatives to choose from; and
- the sequence of coordinated neural and bodily changes that might occur as one chooses one alterative over others in the awareness of what one is doing and of various alternatives open to one.

And, here, we need to be careful not to confuse states of awareness with attention. Awareness ordinarily occurs, and changes, throughout the events and activities of daily life and can be manifested by paying attention or by simply behaving in certain ways. So, for example, one may manifest awareness of a chair by simply walking around it without paying attention to it, thinking about it, or otherwise directing one's mental activities toward it. And the same goes for one's awareness of one's behaviour, such as one's awareness of walking around the chair and what else one might be doing in that moment.

Regardless, including the expression of free will in our models for the relevant neural changes brings up questions concerning the neural substates of awareness and choice.

4 Conclusion: Implications for AI and everyday life

The observation that some of what one thinks and does might at least potentially have unique explanatory value in explaining and predicting some of the behaviour of one's nervous system holds implications both for the development of AI and, more expansively, our daily lives.

First, if we wish to develop and explore AI processes that simulate or model thought and behaviour in all its variety, we need to explore the development of AI which involves simultaneous coordinated stochastic processes, where changes are caused without these changes being fixed in advance; and where these processes can interact and change as a result of previous processes. We also need to keep in mind that the extent to which these processes are *not* the processes of actual brain matter is the extent to which they are replicas. It is the extent to which they aren't the real thing. This is true however close we might be able to get their behaviour to simulate the behaviour of the functioning nervous systems of conscious animals.

To illustrate, we might build a contraption that exactly replicates the motion of metal moving toward and/or away from a magnet. But, unless this motion is caused by an actual magnet, it is just a replica or model, involving different processes.

The point is generalizations to the effect that, in every substantial way, AI processes are the same as neural processes require some empirical warrant. That is, if such generalizations are to be the result of sound inference—or sound inductive reasoning from observation. Specific to our purposes here, we would thus need some evidence for the conclusion that all the characteristics of actual nervous systems that are different from computer hardware do not substantiate different conditions relevant to being aware or making a choice, or to manifesting any other psychological ability.

Second, if the expression of free will has unique explanatory value in predicting some of what unfolds, then some of what we think and do determines, or fixes, some of what happens in our lives, communities, and in the world.

Now, this doesn't mean we cause anything out of nothing or that we experience unbridled possibilities. Rather, there are limits to what we can fix. Other things also provide unique explanatory value in predicting some of what unfolds. This includes, but isn't limited to, what we and others have previously done.

Furthermore, we must be given quite a lot, without having done anything, for us—and our nervous systems—to get to where we can make choices or behave intentionally. For starters, we must have a certain genetic inheritance, and certain of our physical and communal/relational needs must be met throughout infancy into childhood [79, 80].

But, if some of what we do has unique explanatory value, then when the required conditions are met—amid other disposing factors making it more or less likely we will behave in various ways—we knowingly do things and thereby fix outcomes that haven't already been fixed. This includes when we explore and learn more about the likely outcomes of behaving in various ways, which can change our state and, in turn, the likelihood we will behave in these or other ways. And, in this case, we can knowingly affect the likelihood certain things will happen, including that we will be disposed to behave in certain ways in the future. In this way, we can come to give ourselves, and our abilities, over to different processes or ways of life and their outcomes. Some of which promote growth. Others of which don't.

These observations tie what we are learning through neuroscience and psychological science—and can learn—to longstanding religious and philosophical ideas concerning the importance of practicing self-awareness and mindfulness, as well as other-awareness, for discerning the various factors disposing us to behave, and respond, in certain ways under certain conditions, and the outcomes of behaving in these ways. What many of these traditions hold in common is that, through such practices, we become more aware of influences on us, and opened to new possibilities for freely and intentionally behaving in ways that promote growth, forgoing ways that don't.

This sort of awareness seems particularly important today given how rapidly new technologies are being introduced into society, and the power they have to substantially alter the way we live and interact with one another and our environment. However, for all the time and money spent developing these technologies, a small fraction of that is spent discovering how their use influences us. And, often, this is only *after* their integration into our daily lives. As a result, we are collectively giving ourselves over to processes with little awareness of their influence, and the negative outcomes of this approach are becoming evident.

To illustrate, in the mid-2000's—before the widespread use of GPS—it was observed that the size and connectivity of parts of London taxi drivers' hippocampi was associated with years of experience, and was greater than that of matched controls [81]. The hippocampus is required for the use and development of spatial memory abilities, which have been associated with more extensive persistent memories over time and other abilities [82]. Additionally, increased spatial memory is a strong negative predictor for developing dementia [83, 84]. The problem, then, is that habitual use of GPS has recently been shown to negatively impact spatial memory [85]; and this is just one example of how relying on forms of AI in place of our own abilities can interfere with our growth and life in ways we haven't fully anticipated.

To offer another poignant illustration, the integration of social media into daily life has coincided with sharp increases in depression, anxiety, and self-harm, particularly among emerging adults [86–91]. However, despite the fact social media use has been widespread for some time, there still remains insufficient research on how its various forms and uses influences our collective growth.

What these examples illustrate is that, with the development of more sophisticated forms of AI, there is a growing need to prioritize exploring the foundational question as to whether such technologies can be introduced, and used, in ways that promote collective growth. And this needs to be done *before their full-scale adoption*. That is, at least, if we are to *be able* to intentionally use them in ways that promote collective growth, forgoing ways that don't. Furthermore, ultimately, it seems that whether this prioritization occurs will, in part, be uniquely explained by whether we give ourselves, and our abilities, to practices that promote discernment of how various factors influence us.

References

1. Hume, D. (2007). A*n enquiry concerning human understanding: and other writings,* edited by S. Buckle, CUP, 50.
2. Anscombe, G. E. M. (1993). Causality and determination. In E. Sosa & M. Tooley (eds.), *Causation*. OUP, ch. 5.
3. Laplace, P. S. (1820/1951) 'Essai Philosophique sur les Probabilités'; forming the introduction to his *Théorie Analytique des Probabilités*, Paris: V Courcier; repr. F. W. Truscott and F. L. Emory (trans.), *A Philosophical Essay on Probabilities*, Dover, 4.
4. Sapolsky, R.M. (2023). *Determined: A science of life without freewill*, pp.3–5. Penguin Press.
5. Loewer, B. (2008). Determinism. In *The Routledge companion to philosophy of science*, pp. 355–364. Routledge.
6. Strawson, G. (1994). The impossibility of moral responsibility. *Philosophical Studies: An International Journal for Philosophy in the Analytic Tradition, 75*, 5–24.
7. Schrödinger, E. (1935). Die gegenwärtige Situation in der Quantenmechanik. *Naturwissenschaften,* 844–849.
8. Kant, I. *Critique of Pure Reason*, translated/edited by P. Guyer & A. Wood, CUP, 1787/1997, A203/B248.
9. S. Mumford, S., & Anjum, R.L. (2011). *Getting causes from powers.* OUP.
10. Bird, A. (2007). *Nature's metaphysics: Laws and properties*. OUP.
11. VanGunsteren, W.F., Berendsen, H.J.C., & Rullmann, J.A.C. (1981). Stochastic dynamics for molecules with constraints: Brownian dynamics of n-alkanes. *Molecular Physics, 44*(1), 69–95.
12. Brown, R. (1828). XXVII. A brief account of microscopical observations made in the months of June, July and August 1827, on the particles contained in the pollen of plants; and on the general existence of active molecules in organic and inorganic bodies. *The Philosophical Magazine, 4*(21), 161–173.
13. Kac, M. (1947). Random walk and the theory of Brownian motion. *The American Mathematical Monthly, 54*(7P1), 369–391.
14. Feynman, R.P. (1963). *The Feynman lectures on physics,1*,46.
15. Brush, S. G. (1974). The development of the kinetic theory of gases: VIII. Randomness and irreversibility. *Archive for History of Exact Sciences,* 1–88.
16. Jenny, P. M. N., & Heinz, S. (2010). A solution algorithm for the fluid dynamic equations based on a stochastic model for molecular motion. *Journal of computational physics, 229*(4), 1077–1098.
17. Seifert, U. (2012). Stochastic thermodynamics, fluctuation theorems and molecular machines. *Reports on Progress in Physics, 75*(12), 126001.
18. Seifert, U. (2018). Stochastic thermodynamics: From principles to the cost of precision. *Physica A: Statistical Mechanics and its Applications, 504*, 176–191.
19. Ciliberto, S. (2017). Experiments in stochastic thermodynamics: Short history and perspectives. *Physical Review X, 7*(2), 021051.
20. Jarzynski, C. (2017). Stochastic and macroscopic thermodynamics of strongly coupled systems. *Physical Review X, 7*(1), 011008.
21. Kramers, H.A. (1940). Brownian motion in a field of force and the diffusion model of chemical reactions. *Physica, 7*(4), 284–304.
22. Bartholomay, A.F. (1957). *A stochastic approach to chemical reaction kinetics*. Diss.
23. Bartholomay, A. F. (1962). A stochastic approach to statistical kinetics with application to enzyme kinetics. *Biochemistry, 1*(2), 223–230.
24. McQuarrie, D. A. (1967). Stochastic approach to chemical kinetics. *Journal of Applied Probability, 4*(3), 413–478.
25. Kerker, M. (1974). Brownian movement and molecular reality prior to 1900. *Journal of Chemical Education, 51*(12), 764.
26. Fedoroff, N., & Fontana, W. (2002). Small numbers of big molecules. *Science, 297*(5584), 1129–1131.
27. Qian, H., & Elson, E. L. (2002). Single-molecule enzymology: Stochastic Michaelis-Menten kinetics. *Biophysical Chemistry, 101*, 565–576.
28. Blake, W. J., Kaern, M., Cantor, C. R., & Collins, J. J. (2003). Noise in eukaryotic gene expression. *Nature, 422*(6932), 633–637.
29. Schnell, S., & Turner, T.E.(2004). Reaction kinetics in intracellular environments with macromolecular crowding: Simulations and rate laws. *Progress in Biophysics and Molecular Biology, 85*(2–3), 235–260.
30. Turner, T. E., Schnell, S., & Burrage, K. (2004). Stochastic approaches for modelling in vivo reactions. *Computational Biology and Chemistry, 28*(3), 165–178.
31. Samoilov, M. S., Price, G., & Arkin. A. P. (2006). From fluctuations to phenotypes: The physiology of noise. *Science's STKE* (366), re17.
32. Kuthan, H. (2001).Self-organisation and orderly processes by individual protein complexes in the bacterial cell. *Progress in Biophysics and Molecular Biology, 75*(1–2), 1–17.

33. DeReffye, P. (1979). *Modélisation de l'architecture des arbres par des processus stochastiques. Simulation spatiale des modèles tropicaux sous l'effet de la pesanteur. Application au Coffea robusta*. Diss.
34. DeReffye, P., Edelin, C., Francon, J., Jaeger, M., & Puech,C. (1998). Plant models faithful to botanical structure and development. *ACM Siggraph Computer Graphics, 22*(4), 151–158.
35. Kang, M.Z., Cournède, P.H., DeReffye, P., Auclair,D., & Hu, B.G.(2008). Analytical study of a stochastic plant growth model: Application to the GreenLab model. *Mathematics and Computers in Simulation, 78*(1), 57–75.
36. Glass, L. (2001). Synchronization and rhythmic processes in physiology. *Nature, 410*(6825), 277–284.
37. Talattof, A., & Amidon, G. L. (2018). Pulse packet stochastic model for gastric emptying in the fasted state: A physiological approach. *Molecular Pharmaceutics, 15*(6), 2107–2115.
38. Deco, G., Jirsa, V. K., Robinson, P. A., Breakspear, M., & Friston, K. (2008). The dynamic brain: From spiking neurons to neural masses and cortical fields. *PLoS Computational Biology, 4*(8), e1000092.
39. Rolls, E.T., & Deco, G. (2010). *The noisy brain: Stochastic dynamics as a principle of brain function*. OUP.
40. Breakspear, M. (2017). Dynamic models of large-scale brain activity. *Nature Neuroscience, 20*(3), 340–352.
41. Townsend J. T., & Ashby, F. G. (1983). *Stochastic modeling of elementary psychological processes*. Cambridge University Press Archive.
42. Wasserman, S. (1980). Analyzing social networks as stochastic processes. *Journal of the American Statistical Association, 75*(370), 280–294.
43. Hayek, F.A. (1989).The pretence of knowledge (Nobel Lecture). *American Economic Review, 79*(6), 3–7.
44. Obstfeld, M., & Rogoff, K. (2000). New directions for stochastic open economy models. *Journal of International Economics, 50*(1), 117–153.
45. Smets, F., & Wouters, R. (2003). An estimated dynamic stochastic general equilibrium model of the euro area. *Journal of the European Economic Association, 1*(5), 1123–1175.
46. Snijders, T.A.B. (2011). Statistical models for social networks. *Annual Review of Sociology, 37*, 131–153.
47. Walsh, C.E. (2017). *Monetary theory and policy*. MIT Press.
48. Bohr, N. (1996). Discussion with Einstein on epistemological problems in atomic physics. In *Niels Bohr Collected Works* (Vol. 7, pp. 339–381). Elsevier.
49. Born, M., Einstein, A., Born, H., & Russell, B. (1969). *Albert Einstein, Hedwig und Max Born: Briefwechsel* 1916–1955; geleitwort von Bertrand Russell. Nymphenburger.
50. Strawson, P.F. (2002). *Individuals*. Routledge.
51. Bennett, M. R., & Hacker, P. M. S. (2003). *Philosophical foundations of neuroscience*. Blackwell, esp. 81–107, 111–119.
52. Lowe, E. J. (2000). *An introduction to the philosophy of mind*. CUP, 15–21.
53. Steward, H. (2012). *A metaphysics for freedom.* OUP, ch. 8.
54. Soon, C. S., Brass, M., Heinze, H. J., & Haynes, J. D. (2008). Unconscious determinants of free decisions in the human brain. *Nature Neuroscience, 11*(5), 543–545.
55. Haynes, J.D. (2011). Decoding and predicting intentions. *Annals of the New York Academy of Sciences, 1224*(1), 9–21.
56. Maoz, U.,Ye, S., Ross, I., Mamelak, A., & Koch, C. (2012). Predicting action content on-line and in real time before action onset–an intracranial human study. In F. Pereira, C. J. C. Burges, L. Bottou, & K. Q. Weinberger (Eds.), *Advances in neural information processing systems, 25* (pp. 872–870). MIT Press.
57. McCombe Waller, S., Forrester, L., Villagra, F., & Whitall, J. (2008). Intracortical inhibition and facilitation with unilateral dominant, unilateral nondominant and bilateral movement tasks in left-and right-handed adults. *Journal of the Neurological Sciences, 269*(1–2), 96–104.
58. Kumru, K., Soto,O., Casanova, J., &Valls-Sole, J.(2008). Motor cortex excitability changes during imagery of simple reaction time. *Experimental Brain Research, 189*(3), 373–378.
59. Kumru, H., Albu, S., Pelayo, R., Rothwell, J., Opisso, E., Leon, D.... & Tormos, J.M. (2016). Motor cortex plasticity during unilateral finger movement with mirror visual feedback. *Neural Plasticity*.
60. Schurger, A.,Sitt, J.D., & Dehaene, S. (2012). An accumulator model for spontaneous neural activity prior to self-initiated movement. *Proceedings of the National Academy of Sciences, 109*(42), E2904–E2913.
61. Schurger, A., & Uithol, S. (2015). Nowhere and everywhere: The causal origin of voluntary action. *Review of Philosophy and Psychology, 6*, 761–778.
62. Schurger, A., Mylopoulos, M., & Rosenthal, D. (2016). Neural antecedents of spontaneous voluntary movement: A new perspective. *Trends in Cognitive Sciences, 20*(2), 77–79.
63. Bargh, J. A., Gollwitzer, P. M., Lee-Chai, A., Barndollar, K., & Trötschel, R. (2001). The automated will: Nonconscious activation and pursuit of behavioral goals. *Journal of Personality and Social Psychology, 81*(6), 1014–1027.
64. Bargh, J. A., & Ferguson, M. J. (2000). Beyond behaviorism: On the automaticity of higher mental processes. *Psychological Bulletin, 126*(6), 925–945.
65. Rietveld, E. (2008). Situated normativity: The normative aspect of embodied cognition in unreflective action. *Mind, 117*(468), 973–1001.
66. Bruineberg, J., & Rietveld, E. (2014). Self-organization, free energy minimization, and optimal grip on a field of affordances. *Frontiers in Human Neuroscience, 8*, 599.
67. Ackerman, J.M., Nocera, C.C., & Bargh, J.A. (2010). Incidental haptic sensations influence social judgments and decisions. *Science, 328*(5986), 1712–1715.
68. Groß, J., Tass, P. A., Salenius, S., Hari, R., Freund, H. J., & Schnitzler, A. (2000). Cortico-muscular synchronization during isometric muscle contraction in humans as revealed by magnetoencephalography. *The Journal of Physiology, 527*(Pt 3), 623–631.
69. Schultze-Kraft, M., Birman, D., Rusconi, M., Allefeld, C., Görgen, K., Dähne.,.... & Haynes, J. D. (2016). The point of no return in vetoing self-initiated movements. *Proceedings of the National Academy of Sciences, 113*(4), 1080–1085.
70. Lowe, E. J. (2008). *Personal agency: The metaphysics of mind and action*. OUP, ch. 2.
71. Noordhof, P. (1999). The overdetermination argument versus the cause-and-essence principle. *Mind, 108*(430), 367–376.
72. Papineau, D. (1998). Mind the gap. *Philosophical Perspectives, 12*,386.
73. Montero, B. G., & Papineau, D. (2016). Naturalism and physicalism. *The Blackwell Companion to Naturalism,* 182–195.
74. Mumford, S. (2003). *Dispositions*. Clarendon Press, esp.ch.1.

75. Cartwright, N. (1999). *The dappled world: A study of the boundaries of science*. CUP.
76. Cartwright, N. (1989). *Nature's Capacities and their Measurement*. Clarendon Press.
77. Papineau, D. (2013). The rise of physicalism. *Proper Ambition of Science*, pp.182–216. Routledge.
78. Vicente, A. (2006). On the causal completeness of physics. *International Studies in the Philosophy of Science, 20*(2), 157–162.
79. Fromkin, V., Krashen, S., Curtiss, S., Rigler, D., & Rigler, M. (1974). The development of language in Genie: A case of language acquisition beyond the "critical period." *Brain and Language, 1*(1), 81–107.
80. Kuhl, P.K., Conboy, B.T., Padden, D., Nelson, T., & Pruitt, J. (2005). Early speech perception and later language development: Implications for the "critical period." *Language Learning and Development, 1*(3–4), 237–264.
81. Maguire, E. A., Woollett, K., & Spiers, H. J. (2006). London taxi drivers and bus drivers: A structural MRI and neuropsychological analysis. *Hippocampus, 16*(12), 1091–1101.
82. Peters, M., Munoz-Lopez, M., & Morris, R. G. (2015). Spatial memory and hippocampal enhancement. *Current Opinion in Behavioral Sciences, 4*, 81–91.
83. Wood, R. A., Moodley, K. K., Lever, C., Minati, L., & Chan, D. (2016). Allocentric spatial memory testing predicts conversion from mild cognitive impairment to dementia: An initial proof-of-concept study. *Frontiers in Neurology, 7*, 229237.
84. Tangen, G. G., Nilsson, M. H., Stomrud, E., Palmqvist, S., & Hansson, O. (2022). Spatial navigation and its association with biomarkers and future dementia in memory clinic patients without dementia. *Neurology, 99*(19), e2081–e2091.
85. Dahmani, L., & Bohbot, V.D. (2020). Habitual use of GPS negatively impacts spatial memory during self-guided navigation. *Scientific Reports, 10*(1), 6310.
86. Lin, L. Y., Sidani, J. E., Shensa, A., Radovic, A., Miller, E., Colditz, J. B., ... & Primack, B. A.(2016). Association between social media use and depression among US young adults. *Depression and Anxiety, 33*(4), 323–331.
87. Woods, H.C., & Scott, H. (2016). #Sleepyteens: Social media use in adolescence is associated with poor sleep quality, anxiety, depression and low self-esteem. *Journal of Adolescence, 51*, 41–49.
88. Vannucci, A.,Flannery, K.M., & Ohannessian, C.M. (2017). Social media use and anxiety in emerging adults. *Journal of Affective Disorders, 207*, 163–166.
89. Vidal, C., Lhaksampa, T., Miller, L., & Platt, R. (2020). Social media use and depression in adolescents: A scoping review. *International Review of Psychiatry, 32*(3), 235–253.
90. Boursier, V., Gioia, F., Musetti, A., & Schimmenti, A. (2020). Facing loneliness and anxiety during the COVID-19 isolation: The role of excessive social media use in a sample of Italian adults. *Frontiers in Psychiatry, 11*, 586222.
91. Boer, M., Stevens, G. V., Finkenauer, C., de Looze, M. E., & van den Eijnden, R. J. (2021). Social media use intensity, social media use problems, and mental health among adolescents: Investigating directionality and mediating processes. *Computers in Human Behavior, 116*, 106645.