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Getting it Wrong: Biological Mistake-Making as a Cross-System, Cross-Scale Phenomenon

David S. Oderberg [a], Jonathan Hill[b], Ingo Bojak[b], Jonathan M. Gibbins[c], Christopher Austin[a] and François Cinotti[c]

[a]Department of Philosophy, University of Reading, Reading, UK; [b]School of Psychology and Clinical Language Sciences, University of Reading, Reading, UK; [c]School of Biological Sciences, University of Reading, Reading, UK

ABSTRACT

The making of mistakes by organisms and living systems generally is an underexplored way of conceptualising biology and organising experimental research. We set out an informal account of biological mistakes and why they should be taken seriously in biological investigation. We then give an indirect defence of their importance by applying the concept of mistake-making to three kinds of activity: timing, calculation, and communication. We give a range of examples to show that mistakes in these kinds of behaviour can be found across a diversity of scales and systems. We also suggest ideas for empirical research that naturally arise from these cases. The reality and potential for mistake-making across such a wide range of biological entities shows that it is not a purely human phenomenon. Getting it wrong seems to be central to biology as a whole, and to be a potentially productive organising principle for generating novel research questions and experimental hypotheses.

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1. Introduction

Living systems make mistakes. The property of having the potential for mistake-making, and actually making mistakes, seems ubiquitous across scale as well as system. Whether we are examining the tiniest of organism such as a unicellular eukaryote or prokaryote, organisms at the macro scale, sub-systems, or cellular parts, we see biological entities *getting things wrong*.

It is crucial to appreciate that getting something wrong does not require the mistake-maker to be self-conscious or capable of reflecting on what it is doing. Humans can reflect on their mistakes, no doubt other animals; but not cells, or bacteria, or antibodies, or insects. It may be that (i) not even minimal consciousness is required for mistake-making or (ii) only consciousness of the barest kind is required. Mistake-making does

CONTACT David S. Oderberg d.s.oderberg@reading.ac.uk Department of Philosophy, University of Reading, Reading, RG6 6EL, UK

not require free will, or responsibility—let alone moral responsibility. Nor does it require that the mistake be avoidable. A person may have an ineliminable 'blindspot' that causes them to make erroneous judgments in certain situations, no matter how much conditioning away from that liability they may be subject to (by themselves or others). The same can be true for other organisms—individuals and species. Most animals will never recognise themselves in the mirror and many instead mistakenly think they see a rival, butting repeatedly into the mirror. They can never unlearn that erroneous behaviour.

We need, then, to disentangle the general phenomenon of mistake-making from the properties contingently associated with it albeit essentially associated with more specific instantiations of that phenomenon. Once we make this move, we have access to something that appears true across biology. Given its wholly general nature, mistakes offer a framework, both conceptual and experimental, for interrogating living systems.

The purpose of this paper is to give what might be called an indirect defence of mistake theory by illustrating its application to a range of diverse systems. It is by highlighting the phenomenon of mistake-making in this way that its theoretical plausibility is given some weight by its empirical usefulness. More technical details of the theory, and arguments in favour, have been provided in Oderberg et al. (2023) and see also Hill et al. (2022). Here, we begin with a more succinct and partly informal statement of the theory, followed by applications that suggest pathways for developing new and testable hypotheses.

2. Mistakes and Well-being

The potential for mistakes gives us a route into understanding the current capacities of an organism but also hypothesising about those it may acquire or modify, for example through learning. (From now on, unless specified otherwise, when we speak of organisms in a general mistakes context we mean to include parts and sub-systems.) Focusing on mistakes will help us deepen our understanding of how an organism interacts with its environment, whether external or internal. Mistake-making should stimulate us to ask questions about the mechanisms that make it possible: organisms do not just get things wrong by coincidence, whatever the role of accident and happenstance in their transactions with their environment. There will be a reason why a mistake is made that is at least partly to do with the organism's constitution—especially if the organism's mistakes have a pattern or type of regularity. In this way, asking questions about mistake potential, and identifying actual mistakes, will lead experimental biologists to hypothesise about possible mechanisms and processes responsible for their occurrence. We see this empirical productivity as central to the motivation for taking biological mistake-making as a plausible conceptual framework for empirical investigation.

It is with this context in mind that we proceed to investigate a range of phenomena that speak to the ubiquity of mistake-making across biology. We will apply the general framework of (Oderberg et al. 2023; see also Hill et al. 2022) for conceptualising biological mistakes as well as interrogating systems for their mistake-making both actual and potential. Applying these ideas to a range of systems uncovers a distinctive approach available to experimental biologists, but one that we intend to *complement* rather than supplant existing productive theories and frameworks for research. That said, mistake theory is thoroughly *normative* in its conceptual content, in contrast to most other perspectives in the philosophy of biology. By normativity we do not mean a feature to do

essentially with *values* or value *judgments* or preferences. Some creatures—notably ourselves—do (for the most part) value the things that are normatively beneficial to us, such as life, nutrition, health, community, and so on. This is not, however, a truth across biology, and is moreover true only of ourselves and—perhaps—organisms sufficiently like us. Normativity, according to mistake theory, is a far broader phenomenon not tied essentially to valuing as an activity. To this extent, perspectives that exclude—whether by fiat or by argument—biological normativity as a real phenomenon will be inconsistent with our approach. We suggest that this might be true of certain accounts of biological function, e.g. that of (Garson 2019). Or, to put the point more concessively, it would be inconsistent with our approach to reject biological normativity as, at the very least, heuristically and methodologically necessary for much fruitful biological research. Further, one might adopt a more pluralist perspective, where non-normative accounts of trait function in the technical sense are supplemented by broader normative accounts of how organisms behave and operate (for more on this, see Oderberg et al. 2024). Although we are ontologically non-reductionist in our approach to biological normativity, the approach is relatively congenial to a methodological non-reductionist who is more concerned with the generation of testable hypotheses than with the underlying metaphysics. Extended discussion of the issue raised here requires a separate treatment (See Hill et al. forthcoming a).

We informally define a biological mistake as follows: an organism (part, sub-system, group) makes a mistake just in case its behaviour threatens either its effective action in its environment, or the effective action of some whole that it subserves, or the effective action of some part or sub-system of the organism. By 'effective action' we mean 'function' in a sense that is broad, essentially normative, and distinct from (though potentially informed by) more technical senses of 'function' in the longstanding functions debate. (Again, see Oderberg et al. 2024.) This concerns the ability of an organism, as it were, to 'make a living' in its environment—to 'get on', in other words to protect, promote, and enhance its welfare or well-being, which usually but may not (think sterile hybrids) include its reproductive success. The concept, then, is essentially normative: can the organism act *well* in its environment? (Again, mutatis mutandis for parts, subsystems, and collectives.)

To say that a mistake 'threatens' effective action/broad normative function means either that the mistake is itself an act that departs from effectiveness, or it *would* do so if left unmitigated in some way. To depart from effectiveness is for the act to cause a state of affairs inconsistent with effectiveness. Mitigation means the breaking of the causal link between that mistaken act and a state of affairs inconsistent with effectiveness. So a mistake is *itself* an act—mistakes do not just happen, they are *made*—that threatens further *effective* action by the organism in its environment. By 'environment' we are quite liberal in meaning: it is the spatio-temporal region in which the organism finds itself and within which it can both act and be acted upon. Moreover, the environment can be external or internal. Drinking poison is a mistake relative to an organism's internal environment (causing internal damage), albeit with repercussions for action in the external environment. Walking into a lamp post is a mistake relative to an organism's external environment, albeit with repercussions for action in its internal environment.

It is distinctive of our theory that mistakes can be made by members of species, parts of members of species, and species themselves, along with sub-systems within members

of species—in fact any entity capable of acting in such a way as to threaten further effective action. We will say more about this later. It is also important that the concept of mistake is essentially *relational*, albeit not *relative*. Mistakes are objective realities, but the actions constituting them are assessed relative to an environment. Hence the same action can be a mistake relative to one environment but not relative to another. A wolf might misstep and fall down a ravine, threatening its bodily integrity. Yet the fall might put it in the presence of some delicious prey. Its misstep was a mistake relative to the ravine and its walking terrain, but not relative to a larger area within which it can hunt. Environments can be nested, or overlap, or be disjoint. In all cases, there is rarely—unless there is a unique environment—such a thing as a *mistake no matter what*, or an *absolute* mistake. We take this simply to be an unsurprising feature of a world full of change and instability. Does this relativity have implications for effective action itself? Yes: what the wolf is *better off* doing is exercising the right trade-off of risk and reward, calculating, predicting, assessing its environment and its options, and acting so as to *maximise* the prospect of effective action. Some organisms have a rich repertoire of cognitive abilities to enable just that; think of us humans, or higher mammals. Others are less well equipped. But then, again unsurprisingly, the ones less well equipped are also less likely to be subjected to a rich potential for mistake-making in a diverse array of environments. Perhaps the notorious panda is an exception.

Finally, at least for the present purpose of giving flesh to a theory containing a fair amount of details and distinctions, and so that we can see how it is applied to empirical investigation as discussed below, it is important to emphasise that the concept of mistake lives within a network of related but distinct concepts, the most prominent being *malfunction* and *failure*. One might think that since mistakes are defined in terms of effective action, and that we do not reject a very broad, normative sense of 'function' as equivalent to it, we already have malfunction covered. The reply is: yes and no. Yes, inasmuch as when an organism makes a mistake, it fails to act effectively (let us put *threat* aside for now to keep things simple), and this is a kind of malfunction in a normative sense. No, inasmuch as by 'malfunction' as a concept distinct from mistake, we mean something more specific—what is usually thought of as a kind of *systemic breakdown*, an internal disintegration, the destruction or damage of parts, disease processes, and so on. When someone says the toaster has malfunctioned, they do not mean that someone forgot to plug it in; they mean that there is something wrong with it internally, e.g. a short circuit (Garson 2019, 126). The same when the doctor tells me my kidneys are malfunctioning: it might be *caused* by not drinking enough water (or it might not), but the malfunction is the disease process within the organ.

With this in place, we assert that a mistake is *not* the same as a malfunction, though it might involve one, e.g. cause one or be caused by one. A simple example is the proverbial domestic hen that habitually tries to hatch a golf ball. This is a mistake: whatever your favourite idea of fitness, or welfare, whether you think chickens evolved or were designed by God or farmers, chickens are not *supposed* to hatch golf balls. They are supposed to hatch chicken eggs. A hen that tries to hatch a golf ball threatens its effective action—to reproduce according to its kind—albeit in an admittedly remote and attenuated way. If all hens habitually did it they might well make themselves extinct. For the farmer, it is a boon—broody hens can be managed with golf balls, plastic egg-looking objects, and the like. But it is interesting and important that one

organism's mistake can be another one's windfall. In any case, such a hen is *not* malfunctioning. Of course it *might*, say if it was blind or had a brain disease. But golf-ball-hatching hens are not typically like this. They are perfectly healthy; they just do not have the cognitive resources for distinguishing eggs from golf balls. The same applies to fish and bait. Neither the hen, nor the fish that takes the bait, is subject to any kind of systemic breakdown. They just make mistakes—but they do not malfunction in this specific sense.

Again, a mistake is not what we call a 'mere failure'. As said earlier, mistakes are *made*: they require some kind of agency, however minimal. (For more on this, see Oderberg et al. 2023.) Mistakes need not require consciousness, or self-awareness, or free will, language, responsibility, flexible behavioural repertoires, and so on. But they do require the ability to act in at least some minimal way. As such, something bad that *merely happens* to an organism is not a mistake. Being hit by lightning out of the blue, struck by a deadly parasite, or born with a defective gene is a mere failure, even though mistakes may be made as a result of such misfortune or leading up to it. It is interesting and useful to study mere failures, as it also is to study malfunctions. Our concern, however, is with things that organisms *do* that depart from a standard of correctness for the kind to which they belong, such that their welfare is threatened or undermined in some way.

In addition, we leave aside for present purposes the question of whether individual or species well-being is the ultimate aim of individual organismic behaviour. As for 'selfish gene' and other approaches that do not put the organism centre stage as the ultimate or near-ultimate locus of well-being, as in (Dawkins 1989), these we also put to one side. Well-being includes such states and characteristics as (naturally) survival, mental and physical integrity, safety and security, overall health, reproductive success (in non-hybrids), and community (in social organisms). These are not perspectival phenomena; they are real ways in which organisms live, for good or ill depending on the state they are in, the threats they face, and so on. For mistake theory, the question 'Can this organism act effectively in its environment?' is the test of well-being. Effective action means simply behaving in accordance with the demands and capacities of its nature, such that it is in sufficiently harmonious interaction with its environment for the judgment that it is *doing well* to be correct.

At this point it might be objected that talk of effectiveness should not be conflated with the more robust, normative idea of biological well-being appealed to here and encapsulated in a broad conception of function in terms of 'making a living' or 'getting on well' in the environment. (We are grateful to an anonymous referee for encouraging us to clarify this issue.) After all, an organism can act effectively simply by pursuing certain goals in a successful way, such as survival or reproduction. This does not imply anything normative about the goals themselves. All it suggests is that there is *instrumental* normativity in living systems, whereby an organism (or part, group, and so on) either achieves a goal successfully or not. We can build into the concept of instrumental success such features as efficiency, balanced trade-offs, precision, and perhaps all of the normative features we will go on to consider when examining particular systems. So although there is room for a distinction between correctness and incorrectness, or between success and failure, when examining instrumental behaviour, we should say nothing further that imports a more full-blooded, broadly Aristotelian notion of normativity into biological behaviour.

Several remarks in response are apposite. First, on a certain conception of biology—not one we share—the objection can be conceded. Even if there is only instrumental normativity—if there is no such thing as organismic well-being defined in terms of ends pursued by certain means—this still leaves plenty of room for a theory of instrumental biological mistakes, opening up a rich vein of observation and hypothesis generation in its own right. It might be a relatively slimmed-down version of the kind of theory we defend, but it would still involve a framework for classifying, analysing, and empirically testing the purely instrumental mistakes to which all living systems are subject.

Secondly, on our view there would however be something problematic about this narrow conception of how organisms operate. Perhaps the main problem is that the 'mere instrumental normativity' conception is of doubtful coherence. For how would instrumental success and failure be determined if not relative to an ultimate goal whose achievement or non-achievement was itself correct or incorrect? For example, if a criterion of instrumental success involved balanced trade-offs—say, between the time taken to find prey and the time a predator is itself left exposed to predation by an organism higher in the food chain—how would the success of the trade-off itself be assessed? If, let us suppose, the organism did find prey but immediately after its meal was itself devoured by a predator, should we count it as having been instrumentally successful nonetheless? To do so seems bizarre, since its act of predation led to its own death. Presumably we ought to say that a trade-off is only instrumentally successful (or correct) in such a case if the organism can find nutrition and itself evade danger, living for another day. In which case any success (or failure) in the instrumental behaviour derives from whether the further goal—in this case survival—is *itself* successfully achieved. The organism that lingers too long in the search for food can end up being food itself—and this would be a mistake *even if* the lingering led to food in the first instance. In other words, it is difficult conceptually, if not impossible, to disentangle instrumental success from success in achieving the goals for which the means are used.

Thirdly, the holistic nature of an individual's life suggests we cannot take each goal in isolation. Imagine, to continue the hypothetical case, our organism is successful at obtaining food *and* avoiding predation. It is so good, in fact, that it becomes obese and lethargic, thereby unappealing to a mate. So its survival goal will suffer. Again, it would be positively misleading to assess its food-obtaining and predator-avoiding behaviours in isolation from its mate-finding behaviour. There would, as a matter of objective biological fact—so mistake theory claims—be something mistaken about the first two behaviours when taken together with the third. There can be relatively narrow successes in one area of behaviour that lead to mistakes in another, so that the former are therefore themselves mistaken *relative* to another goal—in this case reproduction. It is not our purpose here to analyse the holistic evaluation of biological mistakes; that is a large project awaiting another occasion. All we point out is that a narrowly instrumental conception of biological normativity is all but impossible to immunise from a broader and richer conception of the normative along the lines we suggest. Hence, for mistake theory as we understand it, 'effective action' needs to be taken in a robust, broadly Aristotelian sense that includes not just every means open to an organism but every goal whose achievement forms part of its overall welfare.

A fourth, but perhaps more modest and eirenic point to be made in response to the objection concerning the role of goals in effective action, is that we do not need our underlying

metaphysics to be more robust that necessary for the theory at hand. In particular, it is no part of mistake theory that it *requires* a strictly essentialist view of organisms, whether Aristotelian or otherwise. It certainly does not require any kind of extrinsic goal-directedness, whether by the guiding hand of an intelligent designer or the 'invisible hand' of natural selection (to borrow philosopher of economics Adam Smith's term). The metaphysical demands of mistake theory are to this extent relatively modest. Organisms have natures: they are 'built' to a certain biological blueprint (however we interpret this), such that whatever they do either promotes their well-being, undermines it, or at least does not harm it. There is, in other words, a conception of flourishing that is objectively applicable to biological systems (unlike in physics and chemistry). This leaves plenty of room for debate as to what constitutes flourishing for a particular species, the role of reproduction, the work of underlying selectionist forces, and so on.

The tantalising hypothesis at the heart of mistake theory is that mistake-making is universal across living systems, at least those in which there is an *active* or *agential* element. In mistake theory, we call the concept of agency necessarily in play *minimal*. The Minimal Biological Agent is one that can produce, destroy, or change another entity, whether that other entity is living or non-living. (For the formal definition see Oderberg et al. 2023.) Enzymes, for example, would not at first blush be thought of as agents. And yet they are paradigmatic biological agents, at least at the sub-organismic scale: they destroy the substrate in the catalysis of a chemical reaction. The behaviour of polymerases in DNA proofreading and repair also highlights the active nature of enzymes. When thinking in general terms about mistake-making, we should not be biased towards the larger scales any more than we should assume that what a macaque does when it is attracted correctly (or incorrectly) to possible food is just what an enzyme does when it binds to a correct (or incorrect) substrate. Correct and incorrect performance at its most generic—where correctness favours organismal (or species) welfare and incorrectness threatens it—does not require consciousness or mental representation at any arbitrary scale. What is more important is to investigate possible similarities across systems and scales and seek to uncover any common or analogous processes or mechanisms that may be present.

In what follows we focus on three general kinds of mistake-making: timing; calculation; and communication. We consider systems from the small scale to the large (relatively speaking). Our aim is not to demonstrate that each of the three kinds of mistake occur (actually or potentially) in every system we examine; only that each is found in more than one. The resulting network of mistake types across systems will be sufficient to show that mistakes are a general phenomenon—possibly ubiquitous—stimulating, in ways suggested by our conceptual framework, new ways of testing biological capacities across living systems.

Note that we are *not* claiming, for any broad kind of mistake, that *it* should be expected across all living systems. Our most general claim is that the *potential* for mistake-making *in general* should be found right across organisms, collectives, and at least some parts or sub-systems of every species (Figure 1). In this we fully agree with Georges Canguilhem (1991, 22): 'life is what is capable of error.' It is not something we can prove; rather, we propose it as a regulatory principle to inform more specific investigations. As will be seen below, however, mistake-making of specific kinds can be found, and we predict will be found, across ranges of species and higher taxa.

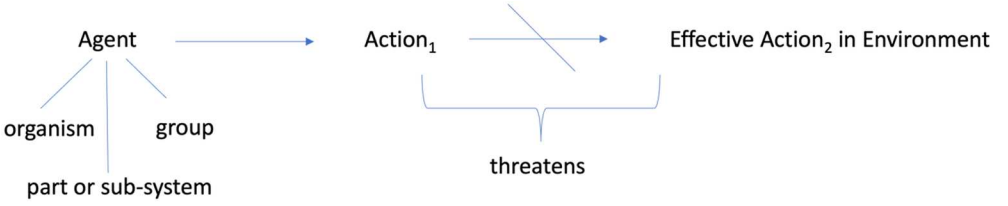

Figure 1. Agent makes mistake when it acts in such a way as to undermine or threaten its further effective action in its environment (its well-being, flourishing). Agent may be an organism (e.g. bird or bacterium), a part or sub-system (cell, immune system), or a group (flock of birds, school of fish).

3. Timing

Aristotle famously held in his *Nicomachean Ethics* that to be virtuous is to act for 'the right reason', at 'the right time', in 'the right way', and towards 'the right things' (See e.g. III.7, 1115b17-18; Crisp 2004, 50). One could also add in 'the right place', to 'the right amount', similarly across all the key Aristotelian categories of being. Consider 'the right time'. The predator that arrives too early or too late misses the prey. The organism that takes too long to make a decision may leave itself open to predation, or without shelter, or without a mate. Animals are also known to stalk their prey, or court a prospective mate, for what may seem like an interminably long time to humans and also takes up an objectively high proportion of the animal's own daily activity in a given season. The organism must take enough and only enough time to get the job done, but in a way that does not overly compromise its other necessary activities. Hence there will often be trade-offs, e.g. between speed and accuracy (Chittka, Skorupski, and Raine 2009).

At the scale of macro-organisms such as birds, bees, and ants, the importance of timing and trade-offs is well known albeit difficult to understand in terms of processes and mechanisms. Ant colonies choose a nest more quickly in harsh conditions than in benign ones (Franks et al. 2003). Bumblebees sacrifice speed for accuracy when mistakes are penalised (Chittka et al. 2003). Songbirds time intervals between songs and song cycles so that they and conspecifics know that a performance is completed—say, in order to attract or choose a mate, or in laboratory situations to initiate a shock avoidance response (MacDonald and Meck 2003; Naguib and Riebel 2014; Rodríguez-Saltos, Duque, and Clarke 2022). There is the potential for mistake-making in all such scenarios, for instance as regards what is known as scalar timing, where time-keeping accuracy decreases as intervals between episodes increase (Rodríguez-Saltos, Duque, and Clarke 2022).

The circadian system is of course one of the principal time-keeping processes within virtually all living things (Albrecht 2010; Sehgal 2015). It is unknown whether there are circadian clocks in organisms isolated from the day-night cycle, though there is evidence they are present in blind cavefish (Idda et al. 2012). Molecular circadian clocks are even found in individual body cells such as mammalian peripheral body cells, all entrained by a master circadian clock in the brain's hypothalamus, itself entrained by the twenty-four-hour day-night cycle (Kornmann et al. 2007). Phase shift experiments by chronobiologists are used to investigate circadian misalignment, e.g. in cases of human jet lag (Serkh and Forger 2014) and to find methods of efficient re-entrainment of the systemic clock to match the demands made on the organism by the new cycle. Misalignment can

lower cognitive alertness, cause depression and other symptoms, which from the normative perspective undermine the welfare of the organism. Misalignment is a kind of mistake in the broad sense of a state of the system that threatens its effective action in its given environment. What about mistake-making? What part of the system *mistimes* the cycle? It can be a mistake for a person to undertake activity with a high cognitive load when in a state of circadian misalignment, but does the misalignment itself result from mismeasurement activity within the system? In mammals, the hypothalamic suprachiasmatic nucleus is thought to be the circadian pacemaker, synchronising multifarious cell-specific and organ-specific clocks operating through the organism; but the synchronisation process is not well understood (Schibler and Sassone-Corsi 2002). Moreover, the circadian system in plants has no analogue of a central pacemaker but consists instead of multiple integrated negative feedback loop networks whose co-ordination is not centralised and is also not well understood (Somers 1999). From the perspective of mistake theory as an organising framework for hypothesis generation, probing diverse systems for what might be underlying, highly general time-keeping mechanisms could yield important results. Moreover, interrogating systems for general time-keeping properties should give insight into whether there are mechanisms for *protection* against mistakes, or *correcting* them once they have occurred. Why, for instance, does a circadian system not undergo a phase shift just by exposure to a flash of lightning? To what extent is measurement of the *duration* of the exposure a factor?

Time measurement in living systems is, however, an important phenomenon going beyond specific chronobiological processes such as the circadian rhythm (Figure 2). The haemostatic system, for example, is not one where timing would be expected to loom large and yet it is of the essence when it comes to correct thrombus (clot) formation. If formation of a clot occurs too late—when the blood flow is too great to be stemmed, or when the organism is on the verge of death—then the system may have behaved mistakenly. Perhaps the platelets have not responded quickly enough, or some other component has mistimed its action. Similarly, if the clotting process begins too early, say before enough collagen has been exposed, then the mistiming could lead to damage—such as an exaggerated response relative to the scale of the injury. If the process continues beyond the point at which the damage is sufficiently controlled—the bleeding stopped to the point of not threatening the system—then the excessive clotting will itself pose a danger to the system, potentially causing blockage of blood flow through blood vessels. The timing has to be right—to some as-yet-unknown degree of precision—for the clotting process to be effective.

Note that mistiming could be caused by other mistakes. Suppose—as needs to be investigated—platelet collagen receptors may bind to other proteins that share some of the same features as collagen (amino acid residue sequence or three-dimensional structure or shape); then it will be misidentification of the normal target that causes mistimed haemostasis. Whether these mistakes occur has not been considered by biologists, although in principle the capacity for such mistakes has been established through the use of artificial, collagen-like proteins or peptides (Asselin et al. 1997). Furthermore, some bacteria have been found to produce collagen-like proteins (Yu et al. 2014) and we note that bacterial infection is sometimes the cause of mistimed and certainly deleterious clotting.

Timing mistakes will often not be generated by pure time-keeping processes such as the circadian clock. Rather, a number of mechanisms will interact in such a way that mistakes can arise. In other words, mistakes involving 'when?' will almost always be

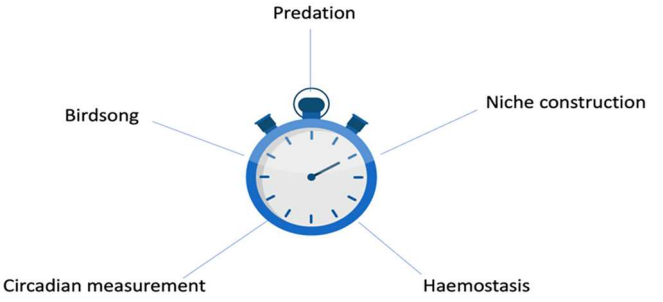

Figure 2. Examples of mistake-making potential in time-keeping.

intertwined with mistakes involving, say, 'why?', 'how?, or 'what?' If I am late for an appointment—a timing mistake—it might be the result of a pure timing error such as misreading the clock. But it is more likely to be caused by other kinds of mistake, such as being distracted by my phone or forgetting I had an appointment until the last moment. If a cat mistimes its pounce on a rodent, without further investigation it is as likely to be due to some other mistake, such as miscalculating distance, as to a pure error of timing. Mistake theory encourages research into the distinction between what we can call 'pure' timing errors (or pure errors of any kind) and 'mixed' errors involving more than one kind of mistake.

To continue with haemostasis, we might hypothesise that clot formation continues beyond the optimal stopping time because the *quality* of the clot has been mistakenly evaluated. The mass of platelets and fibrin that forms a thrombus has to have sufficient size, strength and consistency effectively to stop blood loss. The follow-up question—additional to the one concerning why and how the timing error occurred—would arise as to whether platelets, or other parts of the haemostatic system, or perhaps the system as a whole, or even the *whole organism*, has the capacity for evaluating clot quality. Might it be through co-ordinated platelet signalling, or some kind of top-down monitoring? For the mistake theorist, there must be a way in which the organism as a whole, or the haemostatic system or its parts, distinguishes good from bad clots. If a clot has not fully formed to the point of being able to stop blood loss, yet the aggregation process ends, then it will have ended too early but possibly due to *other* mistakes, such as the misrecognition of an inadequate clot. Human collagen itself may be present inside blood vessels in the absence of injury, most notably in the fibrous cap of atherosclerotic lesions (fatty lesions present for example in coronary arteries): this case would not be one of misidentification but a correct response to a stimulus that should not be there (inappropriate context)—causing a condition often referred to as atherothrombosis (Ruggeri 2002).

4. Calculation

Humans need to calculate correctly. Mistakes in calculation threaten effective action, whether at work, at play, or in vital settings such as a hospital or war zone. Yet numerical competences—divorced from linguistic ability—are increasingly being found in other species, such as newborn chicks (Rugani et al. 2015) and pigeons (Scarf, Hayne, and Colombo 2011), leaving aside the less surprising case of the primates (Brannon and

Terrace 1998). Recent research has shown that both chicks and rhesus monkeys are able to select particular items in an array (even the middle, in the former case) though the numerical and spatial cues diverge (Rugani et al. 2022, 2007). A surprising mistake discovered by researchers is that for primates presented with edible items in quantity discrimination tasks, there is often low performance due to stimulus salience. So even if, for example, chimpanzees are tasked with pointing to the smaller quantity of food to obtain the larger quantity as reward, they are unable to inhibit their impulse to reach for the larger amount. They performed much better when the food stimuli were replaced with numeric symbols (Boysen and Bernston 1995). Mistake theory would treat this as a numerical mistake—failing to pick the smaller quantity. One might also suggest that it was caused by a deeper mistake—one of misidentification, since the chimpanzee misidentified which quantity of food would lead to the desired reward. Mistakes are causally intertwined, as we know from the human case: one mistake can cause another, and the more mistakes that are made in a given task, the further the agent is likely to depart from what we call the 'standard of correctness' for performance in that context. In the quantity discrimination task, more recent research suggests that stimulus salience is only one problem for optimal performance by primates. Another might be mistakes in representation, i.e. the inability to keep separate the monkey's representation of food as a stimulus and food as a reward (Schmitt and Fischer 2011).

Calculation is a good example of how mistake-making might be common to quite diverse systems. It is striking that biologists attribute the ability to *E. coli* bacteria (Bray 2009, 94–5) as well as to the sperm of sea urchins (Alvarez et al. 2012). More specifically, in these two cases it is argued that *receptors* or receptor *clusters* perform calculus by computing a time derivative. In *E. coli*, the MCP (methyl-accepting chemotaxis protein) receptor for a given attractant initiates phosphorylation of the Chemotaxis Y protein, which binds to the bacterial flagellum, modulating bacterial movement in the gradient of that attractant. Enzymes in the receptor cluster are continuously adding and removing methyl groups from the receptors, which tracks the concentration of attractants in the recent past. This then acts as a measure of the rate of change of concentration in the gradient, which means the bacterium can track the increase or decrease in the quantity of food molecules in a given region. In sea urchin spermatozoa, researchers speculate that motion control (steering, speed, direction) is mediated by differential Ca^{2+} binding to CaM (calmodulin) receptors on parts of the sperm cell's flagellum. At a steady state (static CA^{2+} input level), the output (path curvature of the sperm cell) does not change, even if the attractant concentration is high; but the output varies consistently with the rate of change of input. This indicates the presence of a chemical differentiator, which enables the sperm to 'maintain their responsiveness … during their sojourn [sic] to the egg' (Alvarez et al. 2012, 660).

In both cases, then—small-scale and arguably even sub-systemic—it looks as though mathematical operations are performed without any of the mental equipment required by a human being or, perhaps, some other macro-scale organism such as an ape or a bird. Although one should not expect identical processes of differentiation across systems and scales, it would be reasonable to look for overlaps—common processes whether universal or shared by clusters of systems at similar scales (For an account of evolutionary origins for basic numerical competences across living systems, see Deheane 2011.). If there are such processes, then there will be equally common ways in which

systems can make calculation mistakes, for example by not tracking a rate of change with consistency or reliability, or by not judging the correct absolute or relative quantity of a given item. On the other hand, even if certain competences were highly system-specific or scale-specific, all this would entail is that the correlative mistake potential was also highly specific. Suppose that there was nothing biologically significant in common between, say, a cellular differentiator and a human differentiator. Then the way the former could make a mistake—say by a deviation in the addition and removal of methyl groups from a receptor—would have no analogue in the human case. A human mistake in differentiation would only result from something like lack of knowledge or attention, or short-term memory failure, with perhaps some neuronal explanation but without the involvement of a chemical differentiation process or anything similar.

5. Communication, Signalling, Interpretation

Communication is a hallmark of animal life, and with it the vast potential for mistake-making: misunderstanding, communicating in a less than timely fashion, confusion due to noise accompanying the signal, and so on. It is most commonly intraspecific, but can also be interspecific: human communication with domestic animals is a commonplace, but even in the wild there is inter-species communication (For examples, see Fichtel 2004.). At smaller scales, however—descending even to the sub-systemic—talk of 'communication' tends to give way to talk of 'signalling', even when the signalling goes both ways—to and from cells, organisms, and so on. This is perhaps because of the temptation to associate communication with consciousness, or language, or self-reflection, or social convention.

Yet just as mistake-making must, as a general organic phenomenon, be divorced from particularly human or higher-animal characteristics, so should the activities and behaviour that make mistakes possible. Communication, at its most abstract, is the transfer of information for some goal or purpose. Information, of course, is notoriously hard to define (for discussion see Floridi 2011), but a fair approximation is that information is an abstract entity that is a potential stimulus for responsive behaviour by some living thing in furtherance of its goals (Figure 3). But the ability to register, process, and act upon something abstract need not require the ability to *reason* with abstract *concepts*. A fly that settles on a ripe banana is informed, in some way we do not understand, of the existence of food. It would take a lot of experimentation to show that the fly had the *concept* of food, or *deliberated* about settling on the banana in order to have a nice meal. Although we should not want to say that the banana *conveys* information about itself to the fly, it is plausible to hold that it *contains* information about itself, in Fred Dretske's sense of *natural* information (Dretske 1981) (For recent discussion and critique, see Baker 2021.) The fly acts on quite selective information about the fruit's nutritive potential (which we call a 'marker'; see Hill et al. forthcoming b), even if the fly also has some sense of, say, the banana's shape. In order to be able to act on something abstract without performing any intellectual *abstraction*, perhaps all we need to say is that the fly has a hard-wired suite of instincts for reacting consistently to the presence of certain food cues in its environment. There may be highly general instincts that are honed by experience with respect to specific kinds or instances of food, or the instincts might be quite specific in the first place. All of this is for empirical investigation relative to

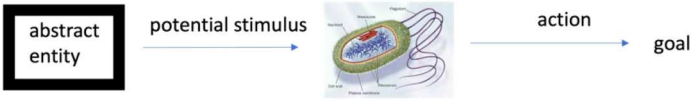

Figure 3. A definition of information.

species. The most we need say here is that the cues are informative and so the fly is informed—but with only the minimal cognitive apparatus necessary for performing in this way.

Flies make mistakes. They are attracted by light sources that are part of traps and so *misinterpret* the information contained in the signal. They land on inedible things, perhaps in exploration for food in their environment. It is part of mistake theory that getting things wrong can be exploited for the mistake-maker's benefit. Exploration leads to dead ends but it also informs organisms of their surroundings, and if they can learn they will learn. Indeed for organisms that can learn, mistakes are largely what they learn from. Yet others can never learn—like fish returning to the same bait again and again. Whether bacteria can learn is moot (Tagkopoulos et al. 2008) but we do know that they communicate, as the phenomenon of quorum sensing demonstrates. Bacteria are able to detect their population density so as to control their behaviour above or below a certain threshold. The co-ordination of their behaviour is via the exchange and detection of signalling molecules; in other words, they share information—they communicate—about their density in a given environment (For an excellent overview, see Bassler and Losick 2006.) Moreover, some bacteria, such as *Pseudomonas aeruginosa*, send 'mixed messages': it sends its signal molecules in membrane vesicles, which when received by its conspecifics enables advantageous group behaviour such as increasing virulence (Miranda et al. 2022). But the same vesicles contain antibiotics that kill other bacterial species (Bassler and Losick 2006, 239). The interpretation is irresistible: both communication and protection (competitor elimination) are taking place, with the respective goals being evident on their face.

Since communication takes place across systems and scales, so—according to mistake theory—does the potential for miscommunication. At the macro scale, we know that vervet monkeys use alarm calls for communication about predators. Different sounds are associated with different predators. Younger monkeys *learn* the calls from older monkeys, and in the earlier stages they make mistakes—such as attributing the alarm for an eagle to the presence of any large bird, however harmless (Tomecek 2009, 79).

At the sub-systemic level, dopamine neurons have been proposed to perform an *evaluative* function with respect to birdsong, in particular that of male zebra finches (Duffy et al. 2022 and personal correspondence with the lead author; see Figure 4). Dopamine spikes correlate significantly with fluctuations in a combination of measures such as song pitch, frequency, and Wiener entropy (a measure of where the sound stands between a pure tone and white noise), such that higher spikes are associated with closeness of the song to the one it learned from its father and lower spikes with greater fluctuations away from that template. Dopamine seems to act like a mistake detection and correction system, maintaining the song's relative fidelity to what the finch learned (see also Gadagkar et al. 2016). From this we infer not only that the bird is itself capable of mistake-making, but that the dopamine neurons *themselves* might be capable of performing

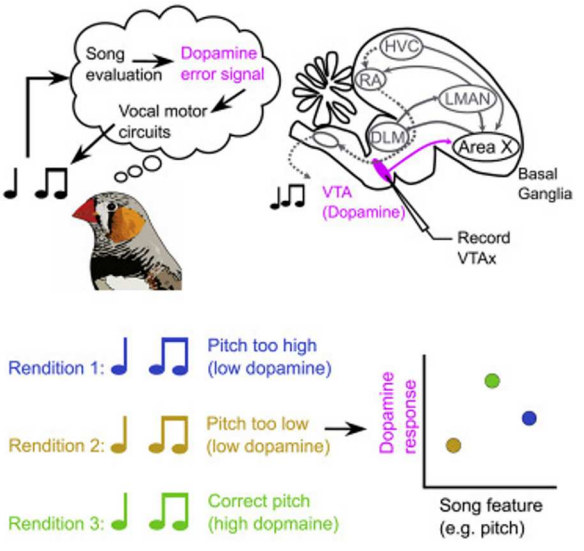

Figure 4. Role of dopamine in zebra finch song evaluation. From Duffy et al. (2022).

their evaluative function mistakenly. This latter possibility will need further investigation: does the dompaninergic system monitor or regulate the song evaluative function of dopamine neurons, and if so how? Further, how is the 'standard of correctness' for song production represented by the zebra finch, especially since each one has a unique correct song that it is taught? Given that birdsong is a form of communication, mistake theory focuses on whether and how a bird may *get it wrong* in, for instance, attracting a mate through song. Interestingly, the dopaminergic error signalling that operates when the bird practises alone is turned off in the presence of a potential mate and retuned to feedback from the other bird (Roeser et al. 2023). It would be informative to investigate whether this retuning was itself mistake-prone.

Consider again the haemostatic system. The timely and effective formation of blood clots in response to tissue damage involves a complex and intricate system of cellular signalling and activation (Stalker et al. 2012; see Figure 5). Signalling between platelets co-ordinates activation on exposure to collagen, rate and size of thrombus growth, and platelet adhesion, among many other actions (Van der Meijden and Heemskerk 2019). At its most general, the signalling between bacteria and between platelets is the same: the transfer of information for the purpose of effective, co-ordinated behaviour. For the bacteria, the purpose is their own survival and proliferation. For the platelets, the purpose is the health and integrity of the organism they subserve. For the mistake theorist, the comparison raises many questions for exploration. For example, one might wonder in what ways the mistake potential of bacteria parallels that of platelets. Are either or both capable of *mis*-communication? In other words, one might wonder whether bacteria and platelets are both capable of sending the *wrong* signal. One might investigate to what extent quorum-sensing bacteria are capable of signalling the wrong population density and so mistakenly initiating unnecessary or even harmful co-ordinated behaviour.

For a possible case of signalling mistakes by platelets, we now know that platelets communicate through gap junctions formed by connexin proteins (Vaiyapuri et al. 2012). We

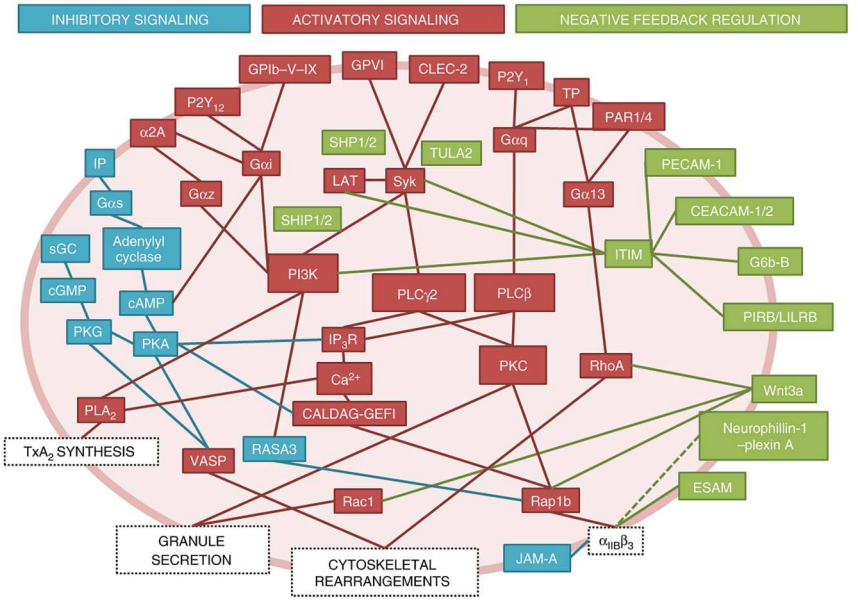

Figure 5. Platelet signalling pathways. From Bye, Unsworth, and Gibbins (2016).

also know that platelets interact with immune cells—and there is some evidence that they communicate with immune cells (Ribeiro, Branco, and Franklin 2019). This might explain how chronic inflammation could trigger inflammatory thrombosis, namely via the stimulation of immune responses by platelets (thromboinflammation). The mistake theorist might hypothesise that the platelets are miscommunicating with the immune cells due to misidentification of them as other platelets. Further research is needed to determine whether such a hypothesis is supported by evidence concerning the exact nature of the platelet-immune cell interaction, or whether for example such behaviour is not mistaken but part of correct platelet operation, even if we do not yet know what the underlying purpose might be.

6. A Framework for Investigation

We have looked briefly at timing, calculation, and communication—behaviour found across systems and scales, and all crucial for effective action by organisms in their environments. At its most general, mistake theory begins with a question concerning which environmental cues elicit responses from organisms. Every organism is selective in what it responds to: without selectivity, an organism will perish either through being overwhelmed by stimuli or completely uninfluenced by stimuli. In either case, it would either act *excessively or deficiently*. This selectivity involves what the mistake theorist calls a 'marker', as noted above. Perhaps we can think of selectivity in response to the environment as the master regulator of organismic behaviour: selective response, i.e. response to markers, is the meta-effective action that makes effective action possible at lower and more specific levels.

Once we have a good idea of at least some of the responses an organism makes to its environment, we can ask particular questions about *kinds* of potential for mistake-making.

For example, does the organism, in order to act effectively, have to *time* its behaviour in certain ways? If so, we can ask whether the organism can make mistakes of timing—and what mechanisms underlie timing, which would enable identification of where precisely a mistake can enter the system. The same can be asked of both calculation and communication, as suggested above. We can also investigate whether an organism has to *discriminate* between different environmental cues in order to act effectively, and if so, to what mistakes of identification it is subject. The same goes for location, for measurement, and for other common behaviour that enables organisms to navigate their environments, maintaining their own well-being and that of their conspecifics. At a more general level, the mistake theorist will also be guided by the distinction between pure and impure mistakes outlined above. In each specific case the research pathway would be laid out: if, for example, communication mistakes are possible for a given organism, are there both pure and impure kinds or only one kind?

Note that mistake theory is both explanatory and predictive. An example of the former is *Bacillus subtilis*, which forms a spore in a nutrient-limited environment, thereby converting into a relatively inert, metabolically inactive cell. Timing, however, is crucial. If the bacterium sporulates in an environment that fluctuates, so that nutrient limitation is only transient, its premature behaviour puts it at a competitive disadvantage to cells that do not sporulate in such an environment and are able to continue proliferating. *B. subtilis* has a mistake-protection mechanism to guard against such behaviour. In the nutrient-limited environment, about half the bacterial population begins sporulation and half do not. The half that does also produces a toxin that kills non-sporulating siblings. Their death leads to the release of nutrients that delay or reverse sporulation in the cells in which this has already begun. When the nutrients are used up and cannibalism is no longer possible, and no other nutrients enter the environment, only then does spore formation become irreversible. In other words, the bacterial population does not fully commit to sporulation and is able to feed on itself for some time, allowing for enough to delay to irreversible behaviour that would be disadvantageous were environmental conditions to improve (Bassler and Losick 2006, 242–3). By explaining this strange cannibalistic behaviour on the part of *B. subtilis* as a mistake-protection mechanism—preventing untimely commitment to sporulation—we render intelligible what is otherwise mystifying. An interesting side question concerns what we might call 'meta-mistakes': does *B. subtilis* sometimes fail to implement its first-order mistake-protection mechanism? If so, under what conditions? Further research would be needed to answer these questions.

An example of predictiveness can be found in bird nest building. Birds that build nests for hatching young must produce nests that are good enough for the purpose, which includes the nest's having the right temperature (Healy, Tello-Ramos, and Hébert 2023). Birds will modify their behaviour in response to varying physical conditions but only where those conditions threaten the goal of a good nest, fit for egg hatching. These modifications may vary considerably, while always serving the same goal of constructing a good nest. Experimentally, it is possible to challenge birds to restore the right thermal conditions for embryo development in response to heating their nests during building. Mistake theory predicts that the way they do this may vary, but the goal will remain the same. As mentioned earlier, actions in biological systems occur, not in response to all of the detail of an environment, but to key *markers* with implications for action. This is seen in clotting, where platelets are activated not by the injury itself but by a reliable marker for injury,

namely exposure to a subset of amino acids on collagen. We predict that this will also be the case for responses to physical variations in nest building. Applied to nest construction, we predict that where the number of building materials is restricted experimentally, birds will take compensatory action based not on the number of missing items but on one or other marker of nest quality which is impacted by the reduced number.

Mistake theory is therefore forward-looking: we should not rest content with explaining existing processes and behaviour but interrogate a system for its *potential* to make future mistakes, including quite unfamiliar ones. If an organism relies on timing, or location, or measurement, to maintain its health, integrity, and survival, to what mistakes in these areas might it be prone? Not only do we need to test its mistake potential, but also whether it can learn or be conditioned out of a particular kind of mistake. If not—because the mistake is an unavoidable facet of the organism's limited behavioural repertoire—then we can test conditions for minimising the mistake's potentially deleterious consequences for the organism. This is done to a certain extent by farmers in the management of broody hens. As mentioned above, domestic hens lack the perceptual capacity to distinguish eggs from vague simulacra such as golf balls, and will try to hatch the latter. Farmers use this unavoidable mistake-proneness to manage broody hens, thereby settling them down when they don't have their own eggs. In addition, researchers can test for ways in which mistakes—in particular unavoidable ones such as the behaviour of hens just mentioned—can be exploited either by the mistake-maker, a conspecific, or another species. Just as errors in DNA transcription and translation can give rise to novel mutations that drive species development, so mistakes at organism and species level can be exploited—as typically happens when the mistake of a prey means food for the predator.

In this context, it is important to grasp what is and is not offered by a theory of biological mistakes. The theory itself is philosophical and conceptual in nature: we take normative concepts such as mistakes and correctness, attributing them in general terms to living things as necessary for explaining otherwise unintelligible behaviour. However, the further *application* of the theory to specific systems, be it birds, insects, mammals, or some species or other, requires dialogue with experimentalists about how they do and could interrogate their target systems. The theory itself, at its most general, is philosophical in content. At its most specific—in application to living systems—it then acquires properly biological content, by seeking to uncover the mechanisms, processes, activities, and markers of mistake-making, correction, prevention, minimisation, and so on. Similarly, when comparing and contrasting across kinds, the theory acquires biological content concerning what kinds of mistake are common to different species, or distinctive of a given species, and similarly for higher taxa. In this way, we take it to be a virtue of mistake theory that it straddles the boundary between disciplines that are all too often only nominally in contact with each other.

What is distinctive about mistake theory is not that experimental biologists never have mistakes in mind when investigating their target systems. It is that the theory provides a philosophical framework for investigation at the outset. Without contradicting anything we already know from our best biological theory and data, its aim is to give biologists a path into a system by testing for the ways in which it may get things wrong. Getting things wrong ties directly into individual and species welfare, since it is a lens through which we can examine the goal-directedness of an organism's behaviour. Just as disease is a window into health (and vice versa), so mistakes are a window into successful

performance. Without understanding achievement—what an organism is trying to do, whether consciously or not, as part of its very nature—we cannot understand life itself. Making mistakes, which is a part of all life, takes us to the same purposive phenomena but from a different point of departure.

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ORCID

David S. Oderberg http://orcid.org/0000-0001-9585-0515

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