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Emergent properties and the context objection to reduction

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Abstract. Reductionism is a central issue in the philosophy of biology. One common objection to reduction is that molecular explanation requires reference to higher-level properties, which I refer to as the *context objection*. I respond to this objection by arguing that a well-articulated notion of a mechanism and what I term *mechanism extension* enables one to accommodate the context-dependence of biological processes within a reductive explanation. The existence of emergent features in the context could be raised as an objection to the possibility of reduction via this strategy. I argue that this objection can be overcome by showing that there is no tenable argument for the existence of emergent properties that are not susceptible to a reductive explanation.

Introduction

The central issue in debates about reduction involves the direction of explanation. Can we provide a full explanation of phenomena at higher levels of biological organization using only lower-level components and their interactions? One of the claims made by anti-reductionists is that explanation at one level of biology either does not depend on or cannot be achieved by a description of events at a lower level. Furthermore, the anti-reductionist claims, the opposite is often true: causal processes at higher levels are sometimes required for an adequate explanation of events at the lower level making reduction an in principle impossibility. Schaffner traces this "structural argument" back to Mill and through to its more recent defenders such as Mayr and Polanyi (Schaffner 1993, pp. 415–419). Modern developmental biology has given renewed vigor to such arguments, providing tantalizing examples such as morphogenetic fields where explanation seems to derive from the higher-level phenomena.[1]

I call the argument that the context or reference system is often an ineliminable part of an explanation and that this context requires reference to

[1]See, for instance, Laubichler and Wagner (2001), and Kitcher (1999). Sterelny and Griffiths (1999, pp. 145–147) provide an overview of such work and the arguments it supports. Brandon (1984) argues for this position in the context of evolutionary biology.

higher-level entities the *context objection to reduction* . The obvious reply that the reductionist can make to this to this objection is to claim that the context itself can be reduced, i.e. that the relevant higher-level context (organelles, cells, tissues, etc.) and its causal influence can be accounted for on the basis of a description in purely lower level terms. In response, the anti-reductionist may argue along one of two lines. She may contend (1) that such a strategy does not provide a reduction since the context is (or may sometimes be) multiply realized, or (2) that the context involves emergent properties which cannot be given a description in lower-level terms. If the context objection is solely a matter of multiply-realized higher-level contextual properties, then, as will be discussed in the next section, the result will be a stand-off between the two sides whose final resolution appears to depend on whether one takes a stricter (e.g. the traditional "layer-cake" model described by Oppenheim and Putnam (1958)) or more moderate view of what reduction consists of. This matter has been extensively debated in the literature (cf. Fodor 1975; Putnam 1975; Rosenberg 1985, 2001; Kincaid 1990; Waters 1990; Sober 1999; Frost-Arnold 2004; Steel 2004) and this paper does not aim to add to the arguments already made on either side. If, on the other hand, the context objection is about emergent higher-level properties – as it is often made out to be (e.g. Laubichler and Wagner 2001) – the implications are quite different. Resolution of this question in favor of the reductionist would support "token–token" or mereological reductionism[2] but leave open the question of whether multiple realizability creates additional difficulties for reductionism (from this point onwards, I will use the term reduction to mean token–token reduction unless otherwise specified). This paper will focus on how this second line of argument about emergent aspects of context might be resolved. I suggest that mechanisms provide a useful way of approaching this issue and propose a strategy of *mechanism extension* to resolve this aspect of the context objection.

The debate over reduction is often carried out in terms of the mechanisms that are supposed to provide explanations of biological processes. This is a natural way of speaking, as biologists themselves customarily talk about seeking and describing mechanisms for various phenomena. It is also, I will argue, a productive way of understanding token–token reduction and of resolving disputes over whether or not certain types of phenomena are reducible. To accomplish this, it will be necessary to examine more closely what mechanisms are and how they explain. For while the term is ubiquitous, it is often given only a very vague definition or left entirely undefined. I argue that once we have a satisfactory analysis of mechanisms, the concept of *extending* a mechanism can serve as the basis for a defense of token–token reductionism from the context objection. Extension of a mechanism allows incorporation of the required structure or context into the explanation of a given phenomenon.

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[2]I use the term token–token reductionism in Jerry Fodor's sense that in any particular case, the properties of a higher-level phenomenon can be explained by the properties of its parts and their interactions.

Briefly, if we take elucidation of a mechanism to provide an explanation and if we can expand the boundary of the lower level mechanism in question to incorporate the relevant components of the context, described in lower-level terms,[3] then the context is not irreducible. This strategy will fail only if the context, or some part thereof, is emergent, and if emergence can be understood in such a way that it supports an argument against token–token reduction. I will argue that it is unlikely that such an account of emergence can be defended.

My project has four parts. First, I describe more fully what I have referred to as the context objection to reduction. Second, I examine what mechanisms are and how they explain. Toward this end, I discuss some recent work that has attempted to provide a more thorough analysis of mechanisms. Third, I develop an account of how we can extend a mechanism to incorporate the required higher-level context into a lower-level explanation.[4] Fourth, I consider the possibility that mechanism extension may fail to incorporate the context into a reductive explanation if the context includes features that are emergent and not susceptible to reduction. This will involve consideration of various accounts of emergence to assess whether they are able to support a cogent argument against reduction. I will conclude that it is unlikely that there can be an account of emergence that is both useful and in opposition to reduction.

The context objection to reduction

The most common argument for anti-reductionism appeals to the multiple-realizability of biological kinds (cf. Fodor 1975; Putnam 1975; Kitcher 1984, 1999; Kincaid 1990). The explanation of a multiply-realized property is unified at a higher level but involves many disparate lower level explanations. So if biological properties are often multiply-realized, then higher level explanations will often be more unified and hence superior. This argument has been criticized on the basis that unified explanations are not always superior; for example, in many cases the lower level details provide information that is important for explaining exceptions to higher level generalizations (cf. Waters 1990; Sober 1999). This suggests that reduction in such cases is successful in virtue of this explanatory success of the lower level account and that unification is not a necessary condition of a successful reduction. While this response involves abandoning the traditional inter-theoretic model of reduction in which the higher level generalizations are deduced from a small set of more funda-

[3]That mechanism extension requires not only incorporating aspects of the higher level context, but also requires their re-description in lower-level terms is essential. Interlevel mechanisms as discussed by Craver (2001) do not require this re-description and so cannot be used to resolve the context objection.

[4]I am explicitly not concerned with genetic reduction, but with reduction of higher level "functional" biology to molecular biology. I take the base level to be biological macromolecules (including DNA, RNA, proteins, signaling molecules, etc.) and their interactions.

mental laws, this is not necessarily a bad consequence. The view that more moderate versions of reduction (including but not limited to token–token reduction) are both philosophically interesting and can legitimately count as reduction has found considerable support (Wimsatt 2000; Frost-Arnold 2004; Steel 2004). Thus, the question of whether or not multiple realizability really does create insuperable difficulties for reduction seems to be a stalemate in which the positions taken depend upon what one understands reduction to be. The question of whether properties of the biological context are emergent will not affect the status of this dispute. Even in the case that the reductionist comes out the clear winner in with respect to emergent properties, the question of multiple realization will remain.

In contrast to traditional reduction, token–token reduction requires only that for every higher-level phenomenon (object, property, process, or event) there exist an explanation composed entirely of lower-level objects, properties, and processes and the rules applying to them that provides a complete causal explanation of each instance of the higher-level phenomena. Based on the notion of mechanisms that will be discussed later, token–token reduction requires existence of a mechanism composed of lower-level entities and activities that explains the higher-level phenomena. This sense of reduction has been defended by Wimsatt (2000), and Bechtel and Richardson (1993). The explanation need not be a unified one; there might be different mechanisms that generate instantiations of a particular higher-level phenomenon in different species, for instance. If the context objection were found to collapse into multiple realizability, therefore, it might be used as a challenge to a more stringent form of reduction but would not be an effective argument against token–token reduction. If, however, some features of the context are emergent and cannot be explained in terms of the parts and their properties, then token–token reduction will fail.

A straightforward statement of what I am referring to as the context objection to reduction is made by Laubichler and Wagner:

> One of our central arguments will be that the relevant context or reference system for all explanations in developmental biology is either the cell or the spatial, regulatory, and dynamical properties of developing systems, rather than the physico-chemical properties of the involved molecules. (200l, p. 55)

While their statement refers exclusively to developmental biology, the same argument has been presented in more general terms elsewhere by Kincaid (1990) and is an essential part of the debate surrounding the concept of the gene and reduction in genetics.[5] As is made clear in the above quotation, the issue is whether or not the essential aspects of the higher-level context presupposed by the alleged reduction can be satisfactorily accounted for in lower-

[5]See Chapters 6 & 7 of Sterelny and Griffiths (1999), Schaffner (1993, 1996), Waters (1994), Neuman-Held (1998), Oyama (1995), and Beurton et al. (2000).

level terms. Kincaid (1990) as well as Laubichler and Wagner (2001) argue that the context cannot be described in this way and, consequently, that reduction fails. Kincaid is more concerned with theory reduction and links the context objection to multiple realizability, which is not a problem for token–token reduction. The challenge for token–token reductionism is Laubichler and Wagner's (2001, p. 61) argument for how context fails to be adequately accommodated in a reductive explanation. They claim that the reason that explanations of the context in terms of lower-level objects fail is that emergent properties of the higher-level objects prohibit explanation in terms of only lower-level properties .[6] For this to hold, they require that higher-level entities have causal powers that are not present in the lower-level entities. This, they argue, is true in virtue of the fact that higher-level entities consist of lower-level entities that are organized and structured both spatially and temporally (2001, p. 58). Because this organization or structure cannot be re-described in lower-level terms, features of higher-level entities which depend on this structure can be said to be emergent and to possess novel causal powers.

In order to argue against the context objection, it is necessary to identify in what sense context (or certain components of it) can be claimed to be emergent in a way that is incompatible with the interpretation of reduction used here. The understanding of reduction presented above relies on the notion of higher-level phenomena being mechanistically explicable in terms of lower-level phenomena. Thus, in order to pursue my argument, something needs to be said about what mechanisms are and how they figure into explanations.

What are mechanisms and how do they explain?

The term "mechanism" is ubiquitous in both science and in the philosophy of science literature. While "mechanism" is virtually never analyzed or defined within science, it is generally clear to the scientists within a field both what form a mechanism must take and when a satisfactory version has been achieved. Essentially, a mechanism[7] must trace how a phenomenon is caused using the objects and activities appropriate to the field and must account for each step in this process, leaving no gaps unaccounted for (these gaps, while often present, are frequently represented by question marks in scientists'

[6]The argument is that the higher-level explanation contains causal information that *cannot* be contained within the lower-level explanation and is thus more complete. This must be distinguished from the argument that explanations in higher-level terms are often superior in terms of cognitive manageability. In the latter case, there is no claim that the higher-level explanation is more complete. I agree that explanations or mechanisms involving higher-level entities (or inter-level mechanisms incorporating both higher- and lower-level entities) are sometimes better suited to a particular explanatory goal. My concern is only with the argument that it is sometimes impossible for explanations in terms of higher-level entities to be fully represented in lower-level terms

[7]"Mechanism" and "pathway" are often used interchangeably. I consider them to be identical and will treat them as such in this paper.

representations of mechanisms and suggest questions remaining to be answered). This understanding of the notion of mechanism is essentially that elaborated by Machamer, Darden, and Craver (hereafter MDC), to be discussed shortly.

As a central but unanalyzed term in science, we might expect mechanisms to be of interest to philosophers of science. Indeed, it does appear with some regularity, particularly in discussions of explanation and reduction. However, the term has often been taken as possessing a self-evident meaning and left undefined or as impossible to capture in a single definition.[8] Nevertheless, the concept of a mechanism is in heavy use, particularly in the reductionism debate where reductionists and anti-reductionists alike call on mechanisms to serve their respective purposes.

Recently, however, there have been several attempts by philosophers to gain a better understanding of mechanisms in biology. One way in which an analysis of "mechanism" has been attempted is in terms of parts of systems and their activities. Such an analysis has recently been attempted by MDC. According to them:

> Mechanisms are entities and activities organized such that they are productive of regular changes from start or set-up to finish or tennination conditions. (2000, p. 3)

This definition of "mechanism" shares many features with other recent accounts provided by Bechtel and Richardson (1993) as well as Glennan (1996). MDC's emphasis on "entities and activities" rather than the "parts and interactions" of these other accounts is, in their view, an essential difference. The distinction does not make a difference for the use I will make of mechanisms, however, so I will not address this issue. But since I will adopt the MDC account of mechanisms, I will briefly discuss the features that are relevant to my project.

One of these features is that activities are not only activities undertaken by an entity in isolation, but with respect to other entities. Without the inclusion of activities of entities at the lower level into our explanatory toolbox, we would quickly discover that mechanisms are unable to provide explanations of higher-level phenomena. The situation is analogous to the debate between methodological individualist and holist explanations in social science.[9] If the individualist is permitted only individuals, explanations at higher levels often appear to be irreducible to the individual level, but if the relations between individuals are allowed, social phenomena can be argued to be reducible. Similarly, without allowing relational properties of biological entities to belong to the entities themselves, all biological molecules in isolation will be largely causally inert and all biological phenomena irreducible. Thus, part of this

[8]See Brandon (1984), Schaffner (1993), Rosenberg (1997), and Laubich1er and Wagner (2001).

[9]See Petit (1993), Kincaid (1994, 1996 Chapter 5). Note also that Laubichler and Wagner make reference to this issue (2001, pp. 65–66).

discussion will depend on providing an account of activities that a lower-level entity does not always engage in, but engages in only in response to or in conjunction with specific other lower-level entities. Again, mechanisms can help elucidate this aspect of the problem.

The idea that a mechanism involves a regular set of changes that proceed from a beginning stage to an ending one is also important since it explicitly attributes a directionality to mechanisms. As we shall be concerned with the direction of causation and of explanation, this is a crucial feature. The regularity of the mechanism is essential and is provided by the "productive continuity" between steps of the mechanism (*ibid*., 3). In their use of this term, MDC are clearly making a claim about causation.

Importantly, MDC do not limit satisfactory mechanisms to linearly organized sequences. Branching or converging paths as well as cycles are perfectly acceptable (MDC, p. 12). Very few mechanisms, in fact, are likely to be linear. Most biological mechanisms are regulated to a greater or lesser degree, either by internal feedback loops or external (to the linear mechanism) regulators. The spatial or temporal domain in which this regulation acts may be broad or narrow. For a mechanism to be linear, the spatial or temporal domain that is of interest for a particular field or a particular investigation must be smaller than that at which the regulation is acting. Even so, descriptions of mechanisms in biology are often multi-level with lower-level entities, properties, and activities as components in mechanisms that produce higher-level phenomena (MDC 2000, p. 13; Craver 2001).

If it is left unspecified in most of the literature on reduction what exactly a mechanism is, it is clear what they are supposed to do for us: they provide *causal explanations* of the phenomenon of interest. This shows why understanding mechanisms can help in the debate about reduction in biology. Moreover, mechanisms are directional and the key point on which the context objection turns is the *directionality* of explanation. The context-dependency of biological processes has been argued to require that some higher-level entities (comprising part of the context needed to explain a phenomenon) cause effects at lower levels, making reduction impossible in principle. The argument is that reduction requires connections between levels but if these are context-dependent, and to describe the context requires reference to higher-level entities, we will never be able to establish a one-way description of the connection. A natural response to this objection is to maintain that the context itself may be amenable to reduction.

Extension of mechanisms

Mechanisms are defined relative to what are often a specific set of concerns; when we want to talk about causation and reduction in more general terms, it will often be necessary to define the boundaries of a mechanism differently. The boundary of a mechanism can be understood as having both a spatial and a

temporal dimension and as separating components of interest from those that are considered part of the background. Redefinition of the boundary may be spatial, temporal, or both. It will incorporate into the mechanism itself some of what was previously counted as background.

The strategy I suggest consists in trying to *extend* the mechanism to incorporate the context required for the explanation. If in doing so the sequence of steps in the mechanism (the productive continuity as represented by arrows in the diagrams used to represent mechanisms) always goes from lower-level to higher, then the direction of explanation is also always from lower to higher and context can be incorporated into a lower-level explanation. Before elaborating what it means to extend a mechanism, four points should be made:

(1) Physicalism, the idea that biological systems are composed of nothing but physical stuff or matter, is not a contested issue. At issue is whether some version of *non-reductive* physicalism is coherent. It is uncontroversial to claim that a description of a higher-level biological entity or process *can* be given in terms of biological molecules and macromolecules. The dispute concerns whether such descriptions can be explanatory.
(2) Mechanisms, while necessarily directional, have no logically defined start or finish conditions. They are always defined relative to pragmatic concerns, or to what is considered significant in a field or experimental system. Nothing prohibits changing the boundaries of a mechanism. If we want to explain, as relevant parts of the context, some higher level entity (X) which is already (necessarily) present at the start (time *t)* of a mechanism (M), we are permitted to extend the mechanism backwards to incorporate the molecular events (add mechanism Y to M to produce mechanism M^1) which resulted in X being present at *t*.
(3) Mechanisms may also be extended in a more synchronous manner. The relevant context may change concurrently with the operation of the mechanism of interest. In this case we must broaden the scope of the mechanism to include more entities and activities that become relevant at some point in the mechanism. This may include adding branches or cycles to what had been a simple linear mechanism.
(4) Mechanisms are not necessarily modular. This follows from the second point above. Whether we extend a mechanism by integration of a single new variable or of entire mechanisms that have been described independently, it is not always the case that the causal influences can be combined in an additive manner.

To extend the mechanism is to integrate into the primary mechanism of interest one or several other mechanisms, each of which contributes to the total causal explanation. It refers to incorporating more of what might be considered the background or context when looking at an isolated mechanism defined in terms of a particular inquiry. Essentially, it requires integrating into the primary mechanism those other mechanisms that generate, regulate or otherwise provide the required context. The resulting mechanisms will often resemble

complex networks or webs of interactions. This is most evident when we are trying to assign a causal role not just to a gene or a protein, but to a larger entity such as a signal transduction pathway.

Extension of a mechanism is represented schematically below. Figure 1 shows that what gets included in a mechanism and what is counted as context or background is relative to the interests and goals of the scientist and how we can push back what are considered the background conditions. The mechanism in panel (a) shows a mechanism with start point W and termination point Z. A is a necessary part of the background conditions for operation of the mechanism, but it is not included as part of the mechanism itself. The arrow used to connect A and X is intended to convey this. W, X, Y, and Z are intended to represent specific macromolecular entities (e.g. components of a second-messenger signal transduction pathway); the arrows between them represent the bottom-out activities required for productive continuity (e.g. binding of one protein to another via their respective SH2/SH3 domains or phosphorylation of a transcription factor by a kinase). A could be a specific macromolecule or it might be represented at a higher level of abstraction, for instance as class of entities sharing some function (e.g. one of a set of receptors which, when bound by their ligand, activate a particular second messenger pathway). Panel (b) shows extension of the mechanism to include the entities and processes involved in producing A. B, C, D, the arrows between them, and the arrow from D to A represent the same types of entities as processes as described for

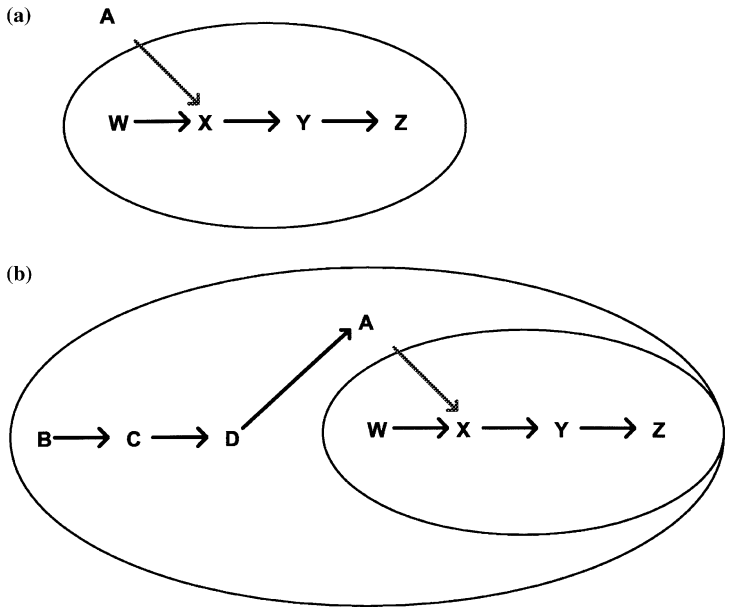

Figure 1. Mechanism extension.

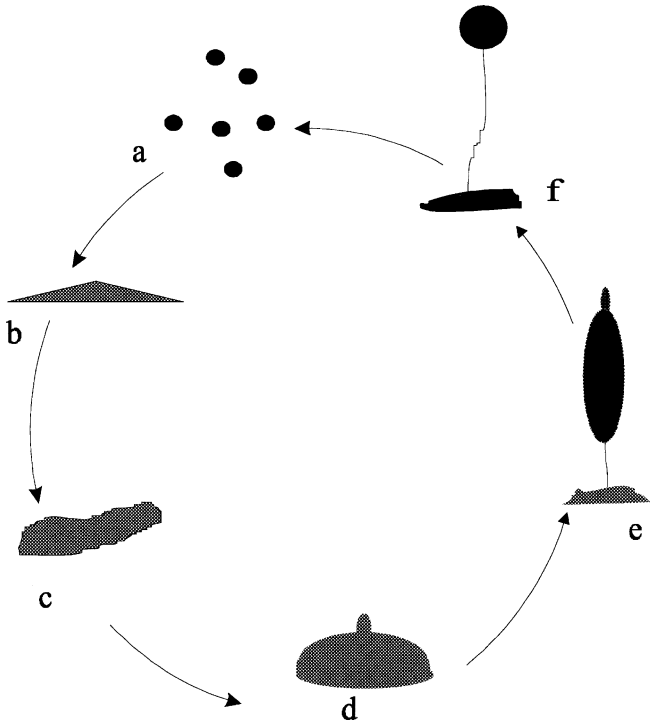

Figure 2. Life cycle of *D. discoideum* . The stages represented are: a, amoebae; b, aggregate; c, slug; d, tipped aggregate; e, early culminant; and f, fruiting body.

W, X, Y, and Z. This extension of the mechanism might, for instance, consist in describing the mechanism by which cell surface expression of a particular receptor is regulated.

As an example, consider the eukaryotic slime mold *Dictyostelium discoideum*. *D. discoideum* lives as single-celled amoebae feeding on bacteria in the soil. When this food source is depleted, the amoebae respond by initiating a cAMP (cyclic adenosine monophosphate) signaling cascade and moving up a gradient of cAMP concentration to produce aggregates of about 10^5 cells which then undergo a co-ordinated path of differentiation and morphogenesis (see Figure 2) to form a fruiting body made up of a spore head suspended on a stalk. I will examine aggregation in this organism more closely in the next section, but for now I will limit the discussion to the role of protein kinase A (PKA).[10] PKA plays a critical role in aggregation as well as in all later stages of development (Kessin 2001, p. 94). During the growth to development transition, activation of PKA is required for chemotaxis and aggregation. During early culmination,

[10]I will not attempt to portray the full mechanism. The reader interested in more detail is referred to Kessin (2001).

activation of PKA induces prestalk cells. During spore formation, activation of PKA leads to encapsulation of the spores. Clearly, what PKA does is highly dependent on the cellular, or even organismal (in the case of post-aggregative stages) context. It seems impossible to say what the role of PKA is without at least specifying the type of cell (amoeba, prespore, prestalk). But if we turn to the mechanism by which PKA is activated and exerts its effect, it is actually fairly easy to extend the mechanism to incorporate the elements of the cellular context that are needed to explain the divergent roles of PKA. The most important element of the context is the type of cell surface cAMP receptor (cARl, 2, 3, or 4), which is present during the various stages. [11] Which receptor is expressed at a given time can be described in biochemical terms as a mid-point in a large interconnected mechanism which consists of a cascade of signal transduction pathways being activated or inhibited resulting in different transcription factors being activated and different patterns of gene expression (including the *car* genes) being found at different stages of development. The essential point is that a mechanism in terms of protein interactions and genetic regulation can be provided to account for the expression of different receptors at various times in development.

What this example illustrates is how mechanism extension can show the context to be molecularly explicable, and hence reducible in the sense of token–token reduction. The question is whether this strategy will always provide a satisfactory response to the context objection. Unless the context involves some feature that is emergent in a sense that opposes reduction, it seems that mechanism extension will succeed. But is there a coherent and tenable sense of emergence that stands in opposition to causal reduction?

Emergence

There is a long history associated with emergence and, accordingly, many modifications to the concept that have been proposed. The classic British emergentists such as C. D. Broad and S. Alexander intended emergence to provide a middle ground between reduction (or mechanism) and vitalism. In their view, everything is constituted by matter – there are no other mysterious forces or entities – but some properties of complex systems are not explainable by, predictable from, or reducible to the properties of their constituent parts. Thus, what is often termed strong emergence is a form of non-reductive materialism and explicitly contrasts with reduction. More recent proponents of an emergence that falls into this class are many analytic philosophers, partic-

[11] This is a simplified picture as other signaling components in the cell, particularly the α subunits of the heterotrimeric G-protein which binds to the cAMP receptors, are also developmentally induced and play a role in the activation of PKA. For the sake of brevity and clarity, I am restricting my discussion to the cAMP receptors. The mechanism can be extended to incorporate the other relevant components, but to do so here would be unwieldy.

ularly those interested in philosophy of mind[12] as well as many who support the view that different levels of organization are ontologically distinct (Emmeche et al. 1997). This version of emergence also seems intrinsic to the idea that complexity of biological systems is irreducible[13] (Mitchell 2002). The questions that need to be asked of strong emergence are, first, whether or not it can be given a clear enough interpretation to support an argument against token–token reduction, and, second, whether there actually exist any properties that are emergent in this sense.

A second type of emergence that is currently discussed is a weaker sense that does not contrast with token–token reduction. Weak emergence refers to the idea that complex systems can possess some properties that are not possessed by its component parts in isolation (i.e. in the absence of the structure or organization of the complex system). Wimsatt (2000) is a notable defender of this version of emergence; others who have supported it include Rueger (2000a, b). Because its adherents explicitly admit that it is consistent with reduction, I will not consider this view further.

Strong emergence

Strong emergence is commonly defended with the claim that features of some systems are not explicable in terms of the non-relational properties of arts and that the relational properties are at a higher level of organization so that phenomena which require an explanation in terms of the non-relational properties are not reducible to the lower level. Since I am not concerned here with theory reduction, and specifically not with whether everything can ultimately be reduced to physics, I will not address the question of how relational properties should be understood with respect to the most fundamental entities of physics. I will instead restrict my attention to how they relate to token–token reduction within biology.

The most extreme form of an argument for strong emergence based on relational properties is that any feature of a system which results from *any* relational property of its parts counts as strongly emergent. If the token–token reductionist is not permitted any relational properties of molecules at all, then the anti-reductionist will be the clear winner since structure, organization, and interaction with other macromolecules are crucial to even the most basic biological phenomena. Even a simple molecular event such as one protein binding to another would be non-reducible. Is there any principled reason why the reductionist should be allowed to include relational properties in her

[12]See Shoemaker (2002), Klee (l984), and Welshon (2002). Kim (1999) has argued against the position.

[13]This view is not unanimous. For example, Ricard (1999) claims that only weak emergence applies to complex systems.

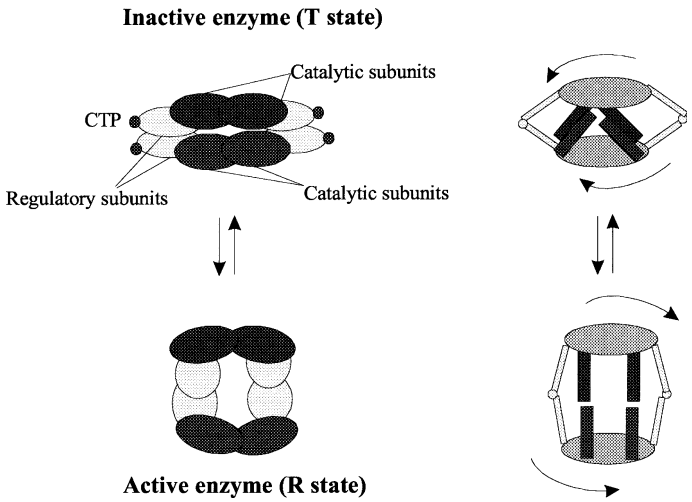

Figure 3. The allosteric transition of aspartate carbamoylase. The binding of the appropriate substrate (CTP) to the regulatory subunits of the enzyme causes the two domains to move relative to each other. The motion of the rigid J3-sheets of these subunits acts like a lever to rotate the catalytic subunits and pull them together into the T state. When this occurs, specific amino acid residues in the catalytic subunits are brought in close enough proximity to form hydrogen bonds. This results in a widening of the cleft that forms the active site of each catalytic subunit, thereby destroying the binding sites for their substrates, carbamoylphasphate and aspartate. A similar account can be given for the reverse transition to the R state. (This figure is adapted from Figures 5–9 on p. 201 of Alberts et al. 1994).

explanatory toolkit? To see that she should, consider what the central claim of the token–token reductionist is: that properties of molecules and the physical laws applicable to them can explain, on a case-by-case basis, higher-level biological properties and events. The reductionist, then, should be allowed to work with the physical or chemical laws (or generalizations) in question and the properties to which these laws make reference. And some of these properties are relational, such as those concerning electrostatic attraction and repulsion that are essential to molecular binding.

Moreover, there are many relational properties that do seem to be able to be given a straightforward explanation in tenns of their parts.[14] Thus it would be unreasonable to claim that inclusion of relational properties any kind is not allowed in reduction. For example, the allosteric transition from the relaxed (R) to the tense (T) conformation in the enzyme aspartate transcarbamoylase can now be explained in atomic detail as a result of biochemical and X-ray crystallographic studies (see Figure 3). If this sort of interaction is not permsitted to count as having a reductive explanation then nothing in biology

[14]Shoemaker's account (2002) of micro-latent and micro-manifest properties accounts well for just the kind of relational properties we find in biology.

can be. While this might be claimed to be a victory for the anti-reductionist, it is a rather uninteresting victory that does little to shed illuminate the broader philosophical issues in play and, furthermore, is one which no anti-reductionist seems eager to claim.

This much is conceded by Frost-Arnold (2004), but he goes on to suggest that it may be specifically spatio–temporal relations that pose a challenge to the reductionist in the case of context. He argues that while the reductionist should be allowed spatio–temporal relations at the molecular scale (e.g. molecule A is between molecules B and C), relations that require reference to some super-molecular part of the cell (e.g. molecule A is in the top half of the cell) should not (pp. 85–86). He then argues that there are cases such as formation of the anterior–posterior axis in the fertilized chicken egg that provide a possible challenge to the reductionist on the basis that their explanation (specifically, explanation of the fact that lighter molecules are on one end – the top – of the blastodisc and heavier ones at the other end) seems to require reference to the spatial orientation of the (whole) egg with respect to gravity. The claim is that we cannot explain the differential distribution of these molecules without referring to the top and bottom of the egg. The difficulty with this position, however, is that we do not in fact need to refer to anything other than the mass, density, and spatial position relative to other molecules in the cytoplasm of the egg (not even relational properties) together with the existence of gravitational force to explain gradient formation. The gradient forms as molecules of different mass and density move differentially through the cytoplasm which is essentially a molecular sieve. It is true that we need to make some reference to the existence of membranes (the cell membrane which ultimately places a limit on how far gravity can take these molecules as well as membrane-bound organelles which may stand in the direct gravitational path of the molecules), but we need to refer only to molecular-level facts about them and their spatial position (i.e. that they are impermeable to the migrating molecules). It seems then, that a notion of strong emergence cannot be based simply on the need to take relational – or spatial – properties into account.

An example of strong emergence?

The final possibility I will consider is whether it is possible to give a sound argument for strongly emergent properties by examination of another concrete example. The process of aggregation and development in *D. discoideum* is often cited as a case of emergence. Solé and Goodwin (2000, p. 21) identify this phenomenon as "one of the clearest illustrations of both the value and the limitations of reductionism in biology" and a case where "the distinctive emergent forms of the living realm cannot be understood simply in terms of particular cell properties"(pp. 26–27). Several other authors have also used this as an example of complexity and emergence (Nicolis and Prigogine 1989; Camazine et al. 2001; Mitchell 2002). That this is a case of emergence is gen-

erally taken to be evident and so is not argued for; a brief description of the phenomenon is taken to be sufficient. The key features which are identified are the appearance of organization where previously there were only independently living cells (i.e. self-organization) (Camazine et al. 2001), the non-linearity which characterizes the interactions of the individual cells during the process of aggregation (Nicolis and Prigogine 1989), the dependence of the behavior of the system on one critical variable – food, and the applicability of new rules to the organism once aggregation takes place (Mitchell 2002). What I will do here is to look more closely at the molecular details of aggregation in *Dictyostelium*. In doing so, I hope to clarify whether a strong notion of emergence is applicable to this case, and which features of aggregation might provide the basis for this emergence. If in looking at the mechanism by which this process occurs we can locate emergence, then there may also exist cases where mechanism extension fails to provide a reductive explanation of the role of context.

The life cycle of *D. discoideum* was previously outlined, so I will not review it. Here, I will primarily be concerned with the crucial feature of aggregation – the ability of cells to synthesize, release, detect, and degrade cAMP. These are capacities that are induced by starvation; they are not present in growing cells. That the cells seem to gain new abilities as well as form a more complex structure does seem to hold some promise for a non-reductive sense of emergence. I argued previously against the idea that the relevance of context to defining the functional role of a molecule can provide the argument for emergence that we need. But what I have not yet considered is whether there are either certain features within the mechanism of aggregation that should not be counted as being genuinely explanatory. If this is the case, these steps might be argued to generate emergence. But are there any such steps? As in any area of study, there are some steps in the mechanism of aggregation that are not yet known. In MDC's (2000) terms, then, we are dealing with a mechanism schemata rather than a fully articulated mechanism. It is always possible, then, that we might fail to identify emergence due to this incompleteness. I consider this to be unlikely, however, since the missing elements are intermediates in a chain of similar types of entities and activities and so are not likely to provide a fundamentally different sort of interaction. Let us then turn to the mechanism of aggregation.

It can be shown that aggregation can be explained in molecular terms and that none of the steps in the mechanism is individually productive of strong emergence. Aggregation is a complex process involving a large number of components and many phases of gene induction. I will again provide a simplified account of the mechanism here.[15] The first point to note is that amoebae do not sense starvation simply in terms of the number of bacteria or the concentration of some bacterial metabolite. Rather, they have two mechanisms for sensing the density of *amoebae* . The first of these is mediated by a molecule called prestarvation factor (PSF) and acts both to inhibit the cell cycle (thus

[15]For a full account of the details to the extent they are now known, see Kessin (2001).

terminating the growth phase of the life cycle) and to control induction of a group of genes expressed very early in the transition from growth to development. The other mechanism is mediated by another molecule called conditioned medium factor (CMF) and acts slightly later, during aggregation. I will focus on PSF as it is responsible for producing some initial changes that are required for the CMF pathway to become active. PSF is synthesized and secreted by the amoebae during growth and accumulates in the local environment according to the cell density. Starvation can be sensed in terms of PSF because its effect of inducing the set of early genes is inhibited by the presence of bacteria. The PSF starvation-sensing mechanism, then, actually detects the ratio of amoebae to bacteria. The role of PSF in initiating the transition from growth to development proceeds via a signaling pathway whose key compo-

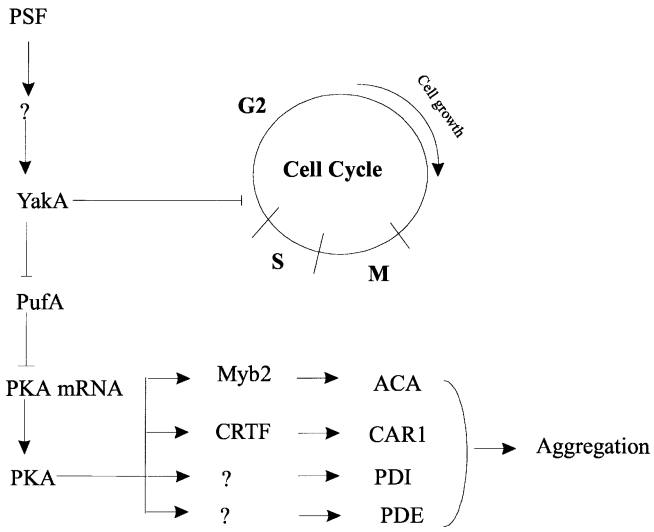

Figure 4. Mechanism of the transition from growth to development. PSF induces expression of YakA, a kinase which inhibits the cell cycle as well as repressing the transcription of the pufA gene. PufA is a transcriptional regulator which inhibits translation of the PKA mRNA by binding to specific sites on the mRNA. Thus, the expression of YakA releases PKA from repression by PufA. The resulting translation of PKA has several effects, most importantly the activation of a number of transcription factors (Myb2, CRTF, as well as others) which activate transcription of a set of genes required for establishment of the cAMP relay, chemotaxis, and aggregation (ACA, or adenylyl cyclase is required for cells to produce cAMP; cARl is the major cAMP receptor in early development; PDE and PDt are, respectively, a phosphodiesterase and a phosphodiesterase inhibitor which are required to establish the pulsatile nature of the cAMP signal). Initiation of this relay requires,in brief, the interaction of a heterotrimeric G protein (not shown) with cARl which transfers information on the occupancy of the receptors (determined by PDE and PDt as well as concentration of cAMP) to signal transduction pathways that regulate both the cytoskeleton (required for motility and chemotaxis) and direct the synthesis of more cAMP (Figure modified from Kessin, 2001).

nents are YakA, PufA, and PKA (see Figure 4). The result of activation of this pathway by PSF is initiation of the cAMP signaling that drives chemotaxis and aggregation. The essential point is that all of these events can be explained in molecular terms. There are many more molecular events that are essential to aggregation, but I will only consider further the two aspects of this process that seem to provide the best potential for emergence. These are the existence of the initiator cells (the cells which initiate the formation of an aggregation center) and the establishment of the cAMP relay.

The fact that a few cells begin to secrete cAMP before the others has been of interest not only to scientists but also to philosophers.[16] However, the explanation of why this is so turns out to be random variation in micro-environmental conditions and metabolic state of cells. There is no difference genetic or otherwise between the cells which form an aggregation center and those which do not (Kessin, 2001). There is, however, some variability in the micro-environment of each cell as well as in the metabolic state of individual amoebae. It is only these factors that determine which cells will be the first to initiate signaling. Probability cannot be the basis of a strong notion of emergence, so this phenomenon does not help us.

The production of the cAMP relay may be more promising in that it seems to consist of just the sort of spatial and temporal influence that might be impossible to explain in terms of properties of individual cells or their component parts. However, the spatial and temporal aspects of the relay can be explained in molecular terms. In essence, these features are simply due to the fact that the relevant molecular interactions are fast and depend on physical contact between the various components. Even in the case of diffusible molecules, this increases the likelihood of downstream events occurring close to rather than far from the previous step in the mechanism and so produces and maintains a gradient (see Figure 5). Again, it is essentially a question of probability – where in the cell certain interactions are most likely to occur. Thus, aggregation and, specifically, the aspects of aggregation that are sometimes referred to as generating emergence, can be provided with what seems to be an unproblematic reductive explanation. Each step in the mechanism outlined above can be explained in molecular terms using interactions that are the basis of all biological phenomena. This example can support a claim of strong emergence only if we deny the reductionist all relational properties – a position that was rejected earlier. Unless there is some way that emergence can be a feature of a set of non-emergent steps, aggregation in *Dictyostelium* is not an instance of strong emergence. Trying to accomplish this in terms of the mathematical description of the series of steps relative to the parts does not seem to be a promising strategy since Rueger (2000a, b) fails to achieve any more than weak emergence in this way. It seems that we are forced to conclude that this case does not allow us to formulate a notion of emergence that opposes reduction. Clearly, there may be other examples that could be argued

[16]See Fox-Keller (1999).

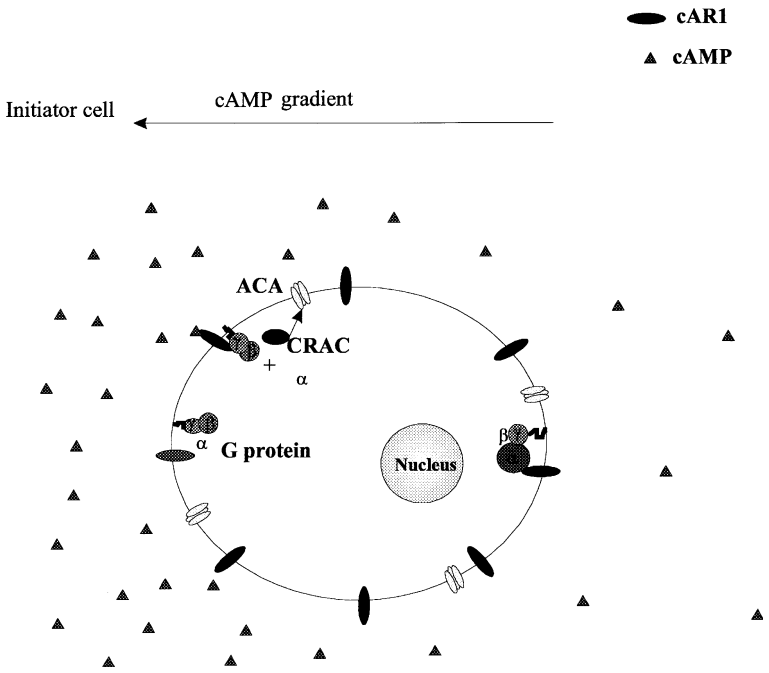

Figure 5. Maintenance and amplification of gradient. Activation of the chemotactic response has a spatial aspect because cAMP receptors at the end of the cell with the highest cAMP concentration will exhibit a higher occupancy rate. The G-proteins linked to these receptors, therefore, will be activated at a higher rate. Cells can respond to a difference in receptor occupancy, from one end of the cell to the other, of as little as 2% (Kessin, 2001, p. 128). Signal transduction via G-proteins involves both a components which remains bound to the receptor (Gα) and one which dissociates (G$\beta\gamma$). Signal transduction through each is spatially restricted in that each subunit is more likely to encounter nearby downstream signalling components. One of these components is CRAC (cytosolic regulator of adenylate cyclase). The G$\beta\gamma$ subunit binds to and activates CRAC which, in turn, binds to and activates adenylate cyclase (ACA). ACA then produces more cAMP which is secreted proximally to that ACA molecule, thus steepening the cAMP gradient.

to be cases of emergence and which display features different from the one just examined that could be claimed to be the basis of this emergence. However, from the discussion here of what intuitively seems to be a case of emergence, I think that it is unlikely that such examples exist.

Conclusion

I have argued that an understanding of explanation based on mechanisms and, in particular, a strategy of mechanism extension can serve as the basis for a defense of token–token reduction in the face of the context objection. Explanatory generalizations at higher levels are, without question, informative. However, there is no plausible argument that they have novel causal powers

that are inexplicable in terms of the underlying mechanisms producing them. The approach of extending mechanisms provides us with an explanation for *why* certain patterns of constraint by higher-level entities are seen. Looking at patterns of interaction between all the lower-level components provides an explanation of, for instance, structural constraints on a phenomenon. Because the activities engaged in by molecular entities do not act at a distance, spatial and temporal aspects of biological context can be explained in molecular terms. Emergent aspects of context do not undermine this strategy since this objection either devolves into the multiple realizability argument which does not pose a challenge to token–token reduction or refers only to weak emergence which is not in opposition to reduction.

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