11 The Paradox of Population Thinking: First Order Causes and Higher Order Effects

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Darwin's discovery of descent with modification was inaugurated by a shift in perspective that Ernst Mayr (1975) has dubbed "population thinking." Darwin realized that the explanation of the fit and diversity of organic form should be approached as a question about the constitution of populations. Rather than ask how individual organisms come to acquire their remarkable features—their complexity, their functional integration, their exquisite adaptedness to their conditions of existence—we should ask how populations come to comprise such individuals. Reframing the question in this way, and deploying a rudimentary grasp of the principles of population change, Darwin was able to demonstrate that the daily activities of organisms' lives (plus some heritable variation) suffice to account for the array of "endless forms most beautiful and most wonderful." There is no need to invoke nonsubstantival forms, entelechies, vital forces, or providential design, as prior theories of fit and diversity of form were wont to do (Ariew 2008). Though this is a simple conceptual maneuver, it ushered a seismic change in the project of explaining biological form that Mayr (1982) is quite right to acclaim as both revelatory and revolutionary.

The Modern Synthesis version of population thinking is an advance on Darwin's. It involves a radical reconceptualization of the object of evolutionary study. In place of assemblages of individual organisms, the Modern Synthesis casts biological populations as ensembles of abstract types, commonly gene types (Fisher 1930 [2000]; Morrison 2002). The study of evolution is the study of the kinematics of these ensembles. The principal virtue of this version of population thinking lies in its capacity to account for evolutionary change *without* having to advert to the complex, multitudinous properties of individuals. Evolutionary explanations in this mode cite only the ensemble-level properties. By way of illustration, Fisher draws an analogy between the study of evolution and the "Theory of Gases" in which:

> … it is possible to make the most varied assumptions as to the accidental circumstances, even the essential nature of the individual molecules, and yet develop the general laws as to the behaviour of gases. (Fisher 1930 [2000], 36)

This rarefied form of population thinking permits biologists to explain, predict, and quantify evolutionary change in a way that would have been impossible without the ascent to this ontology of abstract types.

The orthodox interpretation of Modern Synthesis population thinking is that it articulates the causes of evolution that operate exclusively at the level of populations.[1] Natural selection, for example, is that processes that occurs to a population when and only when there is predictable variation in the fitnesses of the trait types (Sober 2013). It is taken to be a discrete population-level cause, and the only process that introduces a systematic adaptive bias to evolution: "allele frequency change caused by natural selection is the only credible process underlying the evolution of adaptive organismal traits" (Charlesworth, Charlesworth, and Barton 2017, 10). The orthodox interpretation has the implication that if the causes of evolutionary change can be exhaustively described without adverting to the properties and activities of individual organisms, then organisms would appear to be expendable as parts of the apparatus of evolutionary theory. Modern Synthesis population thinking appears to imply that the lives and deaths of individual organisms play no (or at best a peripheral) role in the explanation of evolutionary population change.[2]

> The population is an entity, subject to its own forces, and obeying its own laws. The details concerning the individuals who are parts of this whole are pretty much irrelevant. Describing the single individual is as theoretically peripheral to a populationist as describing the motion of a single molecule is to the kinetic theory of gases. In this important sense population thinking involves *ignoring individuals*. (Sober 1980, 344 [emphasis added])

And yet, the cardinal lesson to be drawn from the startling empirical advances of twenty-first-century biology is exactly the opposite. Evo devo, Eco-evo devo, extended inheritance, niche construction, epigenetics—these fields unanimously point to the significance of individual organisms as difference makers in evolution. The organismal-level processes of development, ecology, behavior, learning, immunity, epigenetic imprinting, alternative splicing, genome engineering, and ecosystem engineering (to name a mere few) influence the tempo, mode, and direction of evolutionary change. These are among the processes whose marginalization from Modern Synthesis evolutionary thinking has evidently been licensed by population thinking.[3]

So, it seems that our best current biology is embroiled in something resembling a paradox: the causes of individual living, dying, and reproducing both are and are not indispensable parts of the explanation of evolutionary change. More simply, current evolutionary thinking is lumbered with a "Paradox of Population Thinking" (PPT):

> PPT: (i) the processes occurring within and between organisms in their day-to-day lives are (among) the causes of evolution.
> (ii) these processes are precluded from the explanation of evolution.

The tension highlighted by the PPT is clearly evident in the most fervid dispute in current evolutionary biology, the so-called Extended Evolutionary Synthesis (EES) debate.

In essence, the debate concerns whether the Modern Synthesis, as standardly interpreted, can assimilate the insights of twenty-first-century biology. Those who call for a wholesale revision (Shapiro 2011; Laland et al. 2015; Noble 2017) argue that ignoring the various activities of individual organisms renders an impoverished understanding of evolution. Those who defend the Modern Synthesis against these challenges (Wray et al. 2014; Charlesworth et al. 2017) maintain that natural selection operating on genetic variation is wholly adequate to the explanation of evolutionary change. Proponents of the EES press considerations in support of (i), while supporters of the status quo argue from (ii). There seems to be no resolution in sight.

But, if the PPT really is at the heart of this debate, then it is no real dispute at all. It is possible to reconcile Modern Synthesis population thinking with a substantive causal/explanatory role for organisms in evolution. Properly understood, population thinking is in no way inimical to according a substantive explanatory role to individual organismal processes in evolution, *even if* in some important sense it "involves ignoring individuals": (ii) is false (at least on one reasonable reading). The fault lies not so much with population thinking itself but in the orthodox interpretation. The objective of this chapter is to take a step toward the reconciliation between Modern Synthesis population thinking with a substantive causal/explanatory role for organismal processes as the causes of evolution. The place to start is back at the *Origin*.

1. Darwin's Answer

Early in chapter 3 of the *Origin*, Darwin poses the question around which the entire work is structured: "How have all those exquisite adaptations of one part of the organization to another part, and to the conditions of life, and of one distinct organic being to another being, been perfected?" (1859 [1968], 114). We might be excused for supposing that his answer is "natural selection." Natural selection, after all, is the process that (alongside Wallace) Darwin discovered, the one most intimately associated with his name. But that is not the answer that Darwin gives. The very same paragraph ends: "All these results … follow inevitably from the struggle for life" (Darwin 1859 [1968], 115). Struggling for life is not natural selection. Natural selection happens to populations, whereas struggling is something that organisms do. Moreover, Darwin says these population changes "follow inevitably" from what organisms do. Two important features of Darwin's answer—*following inevitably* and the *struggle for life*—point to the reconciliation between Modern Synthesis population thinking and a substantive role of organisms in evolutionary explanation.

When a population undergoes evolutionary change, typically three things happen to it: (1) It changes in its lineage structure. That is to say, as organisms are born, survive, reproduce and die, some ancestor/descendent lineages become more prevalent than others. (2) It changes in its adaptedness; the population comes to comprise individuals generally

better suited to their conditions of existence. (3) It changes in its trait structure; some trait types increase in their relative frequency at the expense of others.[4] Darwin's theory is obviously primarily an account of the causes of the first kind of population change. Yet, he is explicit that, in certain circumstances (specifically, that the trait types that confer greater survival and reproduction are heritable), changes of the second and third kind happen as consequences of the first. Richard Lewontin, for example, has repeatedly argued that Darwin's theory of evolution requires heritable variations in individual fitness. The general idea is that when individual fitnesses and the traits that confer them are reasonably heritable, then as a population changes in its lineage structure, two further changes will happen: the fitness conferring trait types will increase in their relative frequency, and the population will tend to comprise better adapted individuals. So, we may see Darwin's version of population thinking as offering two things. First, it is an account of changes in the adaptedness of form (type 2 change), and changes in population trait structure (type 3), as *concomitants* of changes in lineage structure. Second, it is a detailed account of the individual-level causes of changes in lineage structure. "All these results … follow … from the struggle for life."

The Modern Synthesis theory of evolution has a different principal objective. Like Darwin's theory, it explains changes of the third kind—change in the trait structure—in a population.[5] It does so by appeal to population-level processes, selection, and drift in a way that dispenses with the causes of type 1 change—those that impinge on the lives of individual organisms (Walsh, Lewens, and Ariew 2002; Walsh, Ariew, and Matthen 2017). It plots changes in trait structure as a function of variation in the growth rates (fitnesses) of the trait *types*. The varying growth rates of the trait types at one time predict and explain the trait frequencies at a later time.

As mentioned, the orthodox interpretation supposes that in identifying population-level processes that explain change in relative trait frequencies, the Modern Synthesis version of evolution has articulated the causal structure of evolution; selection and drift explain population change because they are population-level causes of evolution.[6] Maybe so; but a naïve interpretation of this metaphysical picture threatens to obscure Darwin's great insight. "Following inevitably" suggests that change in trait structure is an *analytic consequence* of the struggle. Moreover, it is a *higher order effect*. These two concepts, *analytic consequence* and a *higher order effect*, are crucial to an understanding of the relation between individual-level organismal processes and population-level change. They will need some explication.

1.1 Analytic Consequence

Imagine two particles in a container, p_1 and p_2, moving away from one another at constant velocity, such that their center of mass, c, remains fixed.[7] Suppose now that p_1 contacts the wall of the container and rebounds back, while p_2's motion continues as it was. Immediately, the location of c changes. It is tempting to say that the change in c is caused by

the change in p_1, or by the change in the relative motions of p_1 and p_2. I have no desire to legislate the use of these terms, but if this is causation, it is no run-of-the-mill causal relation. It couldn't be the case, for example, that some causal signal is propagated from p_1 to c.[8] The change in c is instantaneous. Such a signal would have to travel faster than the speed of light, and we're told that no causal signal can. Rather, the change in c is *entailed* by the change in p_1. The location of c, at any time, is a function of the straightline distance between p_1 and p_2 and the product of their masses. The motion of c is simply *constituted by* the relative motions of p_1 and p_2. The change in c, I shall say, is an *analytic consequence* of the locations and masses of p_1 and p_2.

Similar considerations apply to changes in trait structure. Given that a biological population—even an ensemble of trait types—is constituted of individual organisms, there are basically two ways that it can change in its constitution: arrival and departure. Organisms arrive by immigrating or being born (hatched, budded, germinated, etc.). They depart by emigrating or dying.[9] As they enter or leave the population they bring or take their trait tokens with them. As a consequence of all this arrival and departure, the trait types that those tokens belong to may become more or less numerous.

The arrival and departure of individual organisms doesn't *cause* the frequency of the trait types in a population to decrease or increase (in any conventional sense), any more than the movement of the particles, p_1 and p_2, causes the change in the center of mass, c. The change in trait structure *just is*, or is *entailed* by, the aggregate of individual arrivals and departures. Equivalently, the disproportionate deaths of white morph (*Biston betularia*) individuals, for example, doesn't *cause* the increase in relative frequency of the black morph (type), it just *is*, or *entails*, the increase in relative frequency of the black morph. If the objective of Modern Synthesis population dynamics is to quantify, predict, and explain these changes in trait structure, then it is a theory of the analytic consequences of arrival and departure.

1.2 Higher Order Effect

Change in trait structure is, moreover, a special kind of analytic consequence; it is a *higher order effect*. A higher order effect is the effect on an ensemble of the aggregated effects of the causes affecting the components severally. That's a bit of a mouthful, but the idea is simple. Consider the changing pattern of colored balls in a billiard game. The first player breaks. If the cue ball contacts a red ball, which scatters the other reds, one of which in turn hits the brown, and then ricochets off to bump the green, which nudges the yellow, the forces acting on the balls (severally) cause each one to move. As a result, the pattern of balls on the table also changes: perhaps the average distance between balls has increased, perhaps the reds are more evenly distributed across the table. The change in pattern is a higher order effect of the forces acting on each the billiard balls severally.

Higher order effects are familiar to us and highly significant. Passive diffusion is a helpful example. If we put a drop of concentrated potassium permanganate into a beaker

of water, it diffuses. The highly localized deep-purple droplet becomes an evenly distributed light pink. Erwin Schrödinger (1944) explains diffusion in the following way. Suppose we were to place a membrane into the solution some time before it has reached equilibrium. Even if the motions of the permanganate molecules are random (i.e., none has any greater propensity to move in one direction over another), we would find that there are more collisions with the membrane on the high-density side than on the low-density side. That is to say that there are more molecules traveling from high density to low than the other way around. And this will continue until the solution has reached an equilibrium distribution of permanganate molecules.

Diffusion is thus a—highly significant, non-trivial—higher order effect: the effect on an ensemble of the aggregate of causes acting on the individual components. It is a regular, projectable occurrence that is discernible only at the level of ensembles; it is a population-level phenomenon. We must ascend to the level of population structure to explain it, or even to see it. But Schrödinger's account demonstrates that we do not need to posit a population-level force or cause in order to explain it. All the causes in this system are those that move the individual molecules around. To posit a further, population-level, *diffusive* cause or force would be to overpopulate the world with causes.

So too with Darwin's account of evolution. According to Darwin, the changes in a population's structure—both trait structure and adaptedness—are also higher order effects. Each is the effect on the structure of a population of the complete suite of causes of individual birth, survival, death, and migration, in the "struggle for life." The answer that Darwin gives to his central question, then, is that—to paraphrase—evolutionary change in the trait structure of a population is both a higher order effect and an analytic consequence, of the individual-level causes of the arrival and departure of organisms.

2. Higher Order Effect Explanations

Where there are individual-level causes and higher order effects, we can distinguish two kinds of explanations of ensemble change. In one, we cite the way the causes acting on individual components of an ensemble affect those components. I'll call these "first order cause" explanations. In the other, we cite the way the distribution of these effects affects the structure of the ensemble over time. I'll call these "higher order effect" explanations. These two forms of explanation have different characters and tell us different things about the change in the structure of an ensemble.

The first order cause explanation of diffusion demonstrates that the final arrangement of the molecules (their respective locations and momenta) is a result of whatever caused each molecule to move in the way it did. The final arrangement is sensitively dependent upon the properties of individual-level molecules, where they started out, and what external forces acted on them. This explanation (causally) entails that were we to have this

exact combination of initial conditions and individual-level causes again, the final arrangement of locations and momenta would be exactly the same. The explanation is highly specific and detailed. But there is a lot about the dynamics of this system that it doesn't explain. For example, it tells us that diffusion, a macroproperty of the system, is dependent upon its microproperties—the arrangement of individual causes—but it says nothing about how sensitive diffusion is in general to variations in these microproperties. It fails to answer a version of the question that Woodward (2003) takes to be the mark of a good explanation: "What if things had been different (in relevant ways)?" As such, it doesn't tell us much about what this particular instance of diffusion has in common with any other. The first order cause explanation lacks the generality and projectability that we usually want from an explanation of diffusion.

The higher order effect explanation fills in these lacunae. It demonstrates that while diffusion is causally dependent upon the arrangement of molecules and their velocities, it is largely insensitive to the differences, and rather more sensitive to the *distribution* of velocities. In doing so, the higher order effect explanation represents diffusion as a robust and general phenomenon, realizable in a huge array of systems, given virtually any of a range of arrangements. The higher order effect explanation of diffusion has particular virtues; while it does not have much causal detail, it is robust and generalizble.

The first order cause and the higher order effect explanations demonstrate that the phenomenon to be explained—change in population structure—exhibits a modal dependence on two different kinds of properties. First order cause explanations demonstrate that the phenomenon depends upon the behavior of individuals. Higher order effect explanations demonstrate that the phenomenon to be explained counterfactually depends upon a property of ensembles.

The relation between higher order effect explanations and first order cause explanations has a significant pedigree. It is implicit, for example, in Maxwell and Boltzmann's development of statistical mechanics. Maxwell and Boltzmann demonstrated that the macroscopic behavior of gases are higher order effects of the aggregated motions of individual molecules. Boltzmann discerned that a gas of a given kind at a given temperature would have an equilibrium distribution of molecular velocities. This distribution explained the gases' typical behavior.

> The molecules are likewise just so many individuals having the most varied states of motion, and it is only because the number of them that have, on the average, a particular state of motion that is constant, that the properties of the gas remain unchanged. (Boltzmann 1872; cited in Uffink 2017, translation by Uffink)

Moreover, each microstate of the molecules (i.e., the arrangement of individual momenta) realizes a particular macrostate (i.e., a distribution of energies). Those microstates that instantiate high entropy macrostates occupy a much greater volume of the state space than those that instantiate low entropy macrostates (Frigg 2011). It is thus overwhelmingly

more likely that a volume of gas will move from lower to higher entropy macrostates than the other way around. Classical mechanics delivers a first order cause explanation of the change or stasis in the structure of a gas. Statistical mechanics offers a higher order effect explanation of the same phenomena. In implicitly exploiting the distinction between first order cause explanations (of classical mechanics) and the higher order effect explanations (of statistical mechanics) Maxwell and Boltzmann were able to ground the thermodynamics of ensembles in the Newtonian physics of forces impinging on individuals. They were able to demonstrate that thermodynamic phenomena are simply the consequences (higher order effects) of first order causes.

The distinction between first order cause explanations and higher order effect explanations is equally applicable to evolutionary biology. Suppose we wish to explain the change in trait structure (i.e., the relative frequencies of trait types) of a particular population. We can do so *either* by citing the first order causes *or* the higher order effects. The first order cause explanation describes the ways in which biological, ecological processes impinged on the lives, deaths, and reproductions of individual organisms, and how the aggregate of these in turn was realized changes in trait structure. We could say, for example, that the visibility of white morph moths on soot-blackened trees caused disproportionately heavy predation on black moths. The aggregate effect of these predations is registered at the population level as a decrease in relative frequency of the white morph individuals.

This explanation has its limitations.[10] It does not tell us, for example, how sensitive the change in trait structure is to variations in individual causes of living and dying. For that we need to offer the sort of higher order effect explanation we find in Modern Synthesis population thinking. In order to do so, we first reconfigure the population as an ensemble abstract entities (trait types) as Fisher did. Then we assign a parameter (trait fitness) to the abstract trait types that measures their relative growth rates. Assigning differential fitnesses to the trait types will allow us to explain and predict their change in relative frequency (trait structure), irrespective of the details of the individual-level causes in operation. These explanations are highly robust, abstracted, projectable, and largely substrate neutral (Matthen and Ariew 2002; Walsh et al. 2017).

The first order cause explanations and the higher order effect explanations explain the *same phenomena*, change in population trait structure. But they do so in different ways. First order cause explanations represent population change as a consequence of those processes that impinge on individual organisms. Higher order effect explanations represent the change in trait structure as the consequence of differential rates of change in the relative frequencies of abstract trait types. First order cause and higher order effect explanations are each complete in the sense that neither augments the other. Once we know that rate of change of the various trait types, we can give a higher order effect explanation. Any further information about which causes impinge on which individuals is strictly redundant. Furthermore, once we know the individual-level causes of evolution, and which traits each individual has, the growth rates of the abstract trait types are explanatorily redundant

(though they can be deduced). Nevertheless, the explanations are complementary and non-competing. Each tells us something different about the dynamics of the population; neither replaces the other (Walsh et al. 2017).

3. The Two-Force Model

It has not been customary to represent the relation between the individual-level causes of evolution and the population-level phenomena of change in trait frequency in this way. Under the sway of the orthodox interpretation of Modern Synthesis population thinking, population-level processes (selection and drift), and individual-level processes are conceived as distinct causes of evolutionary change. I call this the "two-force model" of evolution (Walsh 2003, 2015). It is the two-force model, I contend, rather than population thinking itself, that has perpetuated the apparent conflict between the Modern Synthesis approach to explaining population change, and the explanatory role of individual-level biological processes in evolution, of the sort promoted by advocates of the Extended Evolutionary Synthesis. The two-force picture makes two fundamental kinds of error. It is wrong on empirical grounds, and it is wrong on conceptual grounds.

The two-force model treats populations as concrete particulars and selection and drift as forces acting on them. This metaphysical maneuver promoted the marginalization of organisms that progressively infiltrated twentieth-century biology.[11] Organismal development provides a striking example. Viktor Hamburger (1980) notes that development was "blackboxed" in Modern Synthesis evolutionary biology. There didn't seem to be much that its details could offer one way or another to the account of how populations change. Development is simply treated as a conduit that delivers traits to the arena of selection; whereupon the population-level forces have at them. Bruce Wallace's suggestion that "problems concerned with the orderly development of the individual are unrelated to those of the evolution of organisms through time" (Wallace 1986, 149) seems to have been reasonably representative of the attitude to the relevance of organisms to evolution that prevailed at the time. As John Maynard Smith declared, "it is possible to understand genetics, and hence evolution, without understanding development" (Maynard Smith 1982, 6).

One traditional way to implicate development in evolution was to cast it as a constraint, a particular kind of "bias in form" (Maynard Smith et al. 1985). If selection is the sole force that imparts an adaptive bias to evolution, then development will show up only as an impediment to that force. Even among those who advocated the importance of development to evolution, it was generally seen as a constraint on the adaptation promoting power of selection.

> The nature of the existing developmental system somehow constrains or channels acceptable change [of form in evolution], so that selection is limited in what it can achieve given some starting anatomy. (Raff 1996, 294–295)

> For instance developmental constraints frustrate selection by restricting the phenotypic variation selection has to act upon. Adaptations would be able to evolve only to optima within the constrained space of variability. (Wagner and Altenberg 1996, 973)

The processes occurring in the lives of individual organisms are explanatorily relevant, then, only when selectional explanations cannot fully account for the distribution of biological form.

> This has led to a 'dichotomous approach' in which constraint is conceptually divorced from natural selection and pitted against it in a kind of evolutionary battle for dominance over the phenotype … much of the constraint literature over the last 25 years has explicitly sought to explain evolutionary outcomes as either the result of selection or constraint. (Schwenk and Wagner 2004, 392)

The picture generalizes to all individual-level processes, inheritance, mutation, migration, niche construction, plasticity, epigenetic marking. The list of such processing is long and expanding. Selection and the individual-level causes of organismal form are both represented as "forces" that move a population around its state space (or impede its motion). Moreover, individual-level processes and population-level processes are distinct, and autonomous. One force promotes adaptation, while the others either oppose adaptive change, or are adaptively neutral.[12] As they typically have different effects on population structure, the supposition goes, they must be different kinds of causes.

The empirical error of the two-force model has been comprehensively exposed over recent years. Evo devo, eco-evo devo, Developmental Systems Theory, multiple inheritance systems, epigenetics, ecosystem engineering, genome engineering, niche construction: these fields of study have all brought to light the various ways in which the processes that occur within and to individuals make a substantive positive contribution to evolution. The responses of organisms to their circumstances introduce stable evolutionary novelties into a population (West-Eberhard 2003). They structure the inheritance of characters (Uller and Helanterä 2017). They affect the kind and degree of variation in a population (Sultan 2003). They introduce heritable, adaptive biases into evolution (Moczek et al. 2011; Laland et al. 2013; Herman et al. 2016).

The volume and variety of empirical corroboration is mounting daily. Organisms are no longer "irrelevant." Nor are organismal processes a mere check on those processes that promote adaptation. They are *causes* of adaptive evolutionary change. One cannot understand evolution unless one understands the ways that individual-level processes systematically drive bias and constitute evolution. I take it to be the single-most significant achievement of twenty-first-century evolutionary biology that it has established the contribution made to the adaptiveness of evolution by the processes that occur within and between individual organisms.

The traditional two-force picture could be amended to accommodate the great empirical advances of recent evolutionary biology, without being dismantled altogether. We could

acknowledge, for instance, that individual-level forces work alongside, or in conjunction with, the population-level processes of selection and drift. They both promote adaptive evolution; they are both reflected as changes in trait structure.[13] The complete account of the causes of evolution, then, must incorporate *both* individual-level and population-level processes. We might even find ourselves seeking a division of explanatory labor, apportioning a certain amount of causal responsibility to individual-level processes and a distinct portion to the population-level processes. Indeed, many of those who advocate for an expansion or extension of the Modern Synthesis work with this modified two-force model (implicitly or explicitly) in mind. Strategy might provide a welcome corrective to the marginalization of individual-level processes from evolutionary explanations. It might further offer a partial solution to the empirical error of the two-force model.[14]

But reconfiguring the relation between individual-level causes of evolution and population-level processes in this way would perpetuate the conceptual error of the two-force model. It is the conceptual error that generates the Paradox of Population Thinking. It is also responsible for the marginalization of organisms that progressively took hold throughout twentieth-century Modern Synthesis biology. The revised two-force picture encourages us to ask: "how much of evolutionary change is due to individual-level processes and how much to selection?" But one cannot partition causal responsibility between the first order causes of population change and their higher order effects in this way. Consider the analogue in the case of diffusion; we might ask, "How much of the change in distribution of the molecules is due to individual-level forces and how much is due to diffusion?" Similarly, in the case of the behavior of a gas we might ask, "How much of the change in his gas is due to the forces acting on individual molecules, and how much to entropy?" These questions make no sense. The incoherence results treating the causes of molecular motion on the one hand and (say) entropy on the other as discrete, interacting causes, as one might treat, say, gravitational and electromagnetic forces. But they are not; they are effects of a suite of common causes. Both the change in entropy and motions of molecules are effects of the same cause, of the forces acting on individual molecules. Change in entropy is the higher order effect; the motion of molecules is the first order effect. Analogously, if change in population trait structure—selection and drift—is a higher order effect of individual-level processes, then it is strictly illegitimate to ask how much of the change is due to selection and drift (on one hand) and how much is due to individual-level processes (on the other). Selection and drift, are higher order effects, and organismal living, dying, reproducing, are first order effects *of the same causes*. All these causes operate at the level of individual organisms.

The error of the two-force model lies in construing individual-level causes and ensemble-level processes as somehow on an ontological par, as interacting causes of ensemble change. This generates the competition for explanatory relevance that is the mark of the two-force picture. But this is a category error. A new picture is needed.

4. The Two-Level Model

My suggestion is that we should replace the "two-force model" with the "two-level model." According to the two-level model, there is one level of causation; *all the causes of evolution are the causes of arrival and departure* (the "struggle for life"). Yet, there are two discernible levels of effect. There are effects on individual organisms (first order effects), and there are effects on the distribution of abstract trait types in a population (higher order effects). Consequently, there are two wholly distinct kinds of evolutionary explanation. First order cause explanations tell us how the processes that impinge on individuals *cause* evolutionary change in population structure. Higher order effect explanations tell us how changes in trait structure at one time *depend* upon the distribution of growth rates of the abstract trait types at an earlier time. So, one and the same phenomenon—evolutionary change in trait structure—can be explained in two different ways: by citing first order causes or higher order effects. They each have their uses. These two kinds of explanation tell us very different things about the evolution of a population. First order cause explanations identify the causes of population change but are typically less good at quantifying and predicting rates of population change. Higher order effect (natural selection) explanations measure and project rates of change in trait structure. But they are silent on which individual-level causes impinge on individuals, and on how changes in trait structure depend upon individual-level causes.

First order cause explanations and higher order effect explanations can both either be specific, applying to a single population at a time, or highly general, applying widely across biological populations and times. But they generalize in different ways.[15] First order cause explanations can be generalized to show, for example, how *in general*, individual-level processes—the plasticity of development, epigenetic inheritance, the construction of one's niche, social learning, the interaction with one's microbiome, the engineering of one's own genome—can impact the dynamics of evolution. These processes can cause the origin and maintenance of novelties, or the resemblance of offspring to parent. They may account for the resistance to perturbing effects of mutations or environmental disturbances. They explain the origin of adaptively biased novelties, the capacity of gene regulatory networks to search adaptive space. These are exactly the sorts of contributions to evolution that those who advocate the extension or revision of the Modern Synthesis highlight. Higher order effect (selection and drift) explanations, for their part, generalize in another way. The principles they appeal to (distribution of trait fitness, population size) apply to *any* population in which the abstract trait types can be assigned growth rates, no matter what the causes are or who they affect. These two modes of biological explanation are autonomous, complementary, and wholly noncompeting kinds of explanations. Evolutionary biology needs both kinds of explanations.

The take-home message of the two-level model, in a slogan, is *one level of causation; two levels of effect*. All the causes of evolution are the individual causes of arrival and

departure. The effects of these causes can be registered as the differential growth of lineages (first order causes), which has as an analytic consequence change in trait structure. Alternatively, the effects of individual-level causes can be measured as the differential growth of abstract trait types (higher order effects). Alongside some conceptual clarity, the two-level model offers a resolution of the paradox of population thinking. Recall that the PPT says:

(i): the processes occurring within and between organisms in their day-to-day lives are (among) the causes evolution.

(ii): these processes are precluded from the explanation of evolution.

The two-level model endorses (i) but shows (ii) to be ambiguous. The problem with (ii) is that "the explanation of evolution" can mean either of two things: "first order cause explanation" or "higher order effect explanation." Only one of these kinds of explanation ignores individual processes. These are the higher order effect explanations (those that invoke selection and drift). They are indispensable to evolution. But they are not the only sorts of evolutionary explanations we might want. First order cause explanations, however, do not ignore individuals; they cite the causes of individual arrival and departure. These too are indispensable to evolutionary biology. On one reading of (ii), the PPT is true but wholly unparadoxical, on the other, the PPT is paradoxical, perhaps, but nevertheless false.

5. Proximate and Ultimate

The misunderstanding of population thinking encapsulated in the PPT pervades much of the discourse in current evolutionary biology and its philosophy.[16] A case in point is the recently renewed debate over the propriety, or otherwise, of Mayr's (1961, 1982) proximate/ultimate distinction.

Mayr (1961) distinguished between two kinds of questions we might pose in evolutionary biology, the "how" and the "why." We may ask how an individual came to acquire a particular trait, or we may ask why a particular trait is prevalent in a population. Mayr correctly perceives that these questions call for different kinds of explanation. The former is an individual-level explanation, the latter is a population-level explanation. He proposes that these different kinds of explanations correspond to different kinds of *causes*—proximate and ultimate causes respectively (Scholl and Pigliucci 2015). The proximate causes are "biological" processes—processes that occur within individual's lifetimes. The ultimate causes are "evolutionary processes," those that happen to populations over evolutionary time (specifically selection and drift). The upshot of the proximate/ultimate distinction seems to be that "proximate causes," those individual-level processes that cause organisms severally to have the traits they have, do not appear in evolutionary explanations.

Understandably, those who seek to extend or expand the Modern Synthesis, by incorporating these very processes into the explanations of evolutionary change, resist the proximate/ultimate distinction: "The proximate ultimate distinction has given rise to a new confusion, namely, a belief that proximate causes of phenotypic variation have nothing to do with ultimate, evolutionary explanation" (West-Eberhard 2003, 6). They do so on two eminently reasonable grounds. First, they point to the fact that (as per thesis (i) of the PPT) individual-level processes most certainly do contribute to evolution (see Odling-Smee, Laland, and Feldman 2000; Laland et al. 2011; Laland et al. 2015).

> Proximate mechanisms both shape and respond to selection, allowing developmental processes to feature in both proximate and ultimate explanations. (Laland et al. 2011, 1512)

One prime example of this contribution is "developmental bias," the capacity of organismal development to introduce adaptive bias.

> Developmental bias is potentially widespread in nature and can contribute to evolutionary stasis … or promote evolutionary adaptation. … If the proximate biology of a lineage makes some variants more likely to arise than others, these proximate mechanisms help construct evolutionary pathways. (Laland et al. 2011, 1513)

As a consequence, the contributive role of organismal processes to evolution, like development, has been undersold.

> … too much causal significance is afforded to genes and selection, and not enough to the developmental processes that create novel variants, contribute to heredity, generate adaptive fit, and thereby direct the course of evolution. (Laland et al. 2015, 6)

Opponents of the proximate/ultimate distinction further point out that, given the nature of causation in evolution, we cannot partition the causes neatly into the "evolutionary" and the "biological":

> ... causation is reciprocal. This means that "ultimate explanations" must include an account of the sources of selection (what caused the selective environment?) when they are modified by the evolutionary process itself. (Laland et al. 2013, 725)

There should be little wonder, then, that there is widespread resistance to the proximate/ultimate distinction.

Those who wish to preserve the distinction argue that there is explanatory value to be had by distinguishing the population-level explanations of selection and drift from individual-level biological explanations of the causes of living and dying (Wray et al. 2014; Dickins and Rahman 2013; Dickins and Barton 2012).

> To answer how an individual operates within a particular environment is to give a proximate account. To answer why that individual operates in that way is to give an ultimate explanation. In this way ultimate accounts address the reasons why a trait evolved, typically in terms of selection, but

also potentially including traits that are by-products of selection or selectively neutral traits spread by genetic drift. (Dickins and Barton 2012, 749)

These authors argue that the prevalence of proximate mechanisms that contribute to evolution are themselves the consequences of evolutionary processes. They should be explained in the way that the prevalence of any adaptive trait is explained. Take, for example, developmental bias. "Developmental bias, if real, is the result of proximate mechanisms that themselves have ultimate explanations" (Dickins and Barton 2012, 753). The quite reasonable point is that the individual-level processes that contribute to evolution have evolved. To cast them merely as causes of evolutionary change, and not as consequences of evolution, is to risk overlooking the fact that they too have evolutionary "why" explanations. The Dickins and Barton point is a platitude of course. It is agreed on all sides that prominent traits have evolutionary explanations, and that biologists quite legitimately distinguish between "how" and "why" questions.[17] As I understand it, their point is that this platitude alone speaks strongly in favor of the proximate/ultimate distinction.

We seem to have reached an impasse. On the one hand, preserving the proximate/ultimate distinction seems to debar us from allowing merely "biological" processes from participating in evolution. This is not only implausible, it threatens to undo the great empirical advances of twenty-first-century biology. On the other hand, abandoning the distinction seems to debar us from distinguishing genuine evolutionary explanations of the distribution of trait types in populations ("why" explanations) from "biological" explanations of the occurrence of trait tokens in individuals ("how" explanations). But this distinction too is undeniable. It looks like evolutionary biology needs but cannot have a proximate/ultimate distinction.

The dispute is spurious, of course. In fact, the proximate/ultimate debate is just a rehash of the PPT. Clearly, those who argue against the proximate/ultimate distinction, and those who defend it, construe "evolutionary explanation" differently. The former (implicitly) interpret it as "first order cause" explanations, and the latter as "higher order effect" explanation. Those who oppose the distinction argue (i) that individual-level processes are among the causes of evolution, and should figure in evolutionary explanations. Those who wish to preserve the distinction do so from the perspective of a particular reading of clause (ii). They argue, correctly, that (a certain kind of) evolutionary explanation ignores individual-level causes. But once the equivocation on "evolutionary explanation" becomes clear, we see that these positions are consistent.

Consider the arguments on each side of the debate. Those who propose to abandon the distinction do so on causal grounds.[18] Those who wish to preserve the proximate/ultimate distinction cite explanatory considerations. They appeal to the different kinds of "accounts" we give of individual-level and population-level phenomena. So, revisionists argue that there is no defensible *causal* proximate/ultimate distinction, while retentionists argue that there is a genuine *explanatory* distinction. They are both right.

Just as the two-level model resolves the PPT, it defuses the proximate/ultimate debate. The revisionists are right in insisting that there is no such thing as an ultimate cause as distinct from a proximate cause of evolution. All the causes of evolution are causes of individual living and dying. It is "proximate" causes all the way down. Nevertheless, there is a workable distinction between proximate and ultimate *explanations*.[19] The proximate explanations are the first order cause explanations that trace the way that causal processes affecting individual organisms bring about change in population trait structure. The ultimate explanations are the higher order effect explanations that trace the way that the distribution of growth rates of the abstract trait types in a population results in change in trait structure.

Those who insist that the ultimate explanations of selection and drift somehow preclude, or compete with, the proximate explanations have simply misunderstood the relation between individual-level (first order) causes and population-level (higher order) effects. I detect the influence of the two-force model at work here. The two-level model demonstrates that evolution needs both levels of explanation, but it has no use for two levels of causation.

6. Conclusion

For far too long evolutionary biology has been gudgeoned by an unparadoxical "paradox":

PPT: (i): the processes occurring within and between organisms in their day-to-day lives are (among) the causes evolution.
(ii): these processes are precluded from the explanation of evolution.

The apparent paradox is the result of an equivocation on "evolutionary explanation," which is aided and abetted by the two-force model of evolution. That model makes the erroneous supposition that the processes that occur within individual lives constitute one set of causes, and the processes that occur to populations (e.g., selection, drift) constitute another, wholly distinct set of causes.

The resolution of the paradox lies in giving due weight to Darwin's discovery that all the causes of evolution are to be found in the "the struggle for life"—*inter alia* in the living, dying, and reproducing of individuals—and that evolutionary changes "follow inevitably." The population change we are interested in as evolutionists is change in trait structure. It has two kinds of explanation. One kind adverts to the activities of individuals: organisms live, they die, they migrate, they mate, they give birth, they produce adaptive novelties, they change their environments, they regulate their genome structure and function. These in turn bring about changes in trait structure *as an analytic consequence*. The explanation of these changes invokes the causes that impinge on individual organisms; they are first order cause explanations. The other kind of explanation adverts to the rates of change of the abstract trait types. Some trait types increase in relative frequency with

respect to others. A selection (higher order effect) explanation accounts for change in trait structure exclusively in terms of the variation in the growth rates of abstract trait types. The first order cause explanations are "proximate"; the "higher order effect explanations are ultimate."

There is no Paradox of Population Thinking. Thesis (i) is true; thesis (ii) is ambiguous. There are two kinds of explanation of population change, only one of which involves "ignoring individuals." Population thinking is in no way inimical to according individual organisms an indispensable place in evolutionary explanation. This is a lesson we should have learned from the answer that Darwin gave to the central question that structures the *Origin of Species*: "All these things follow inevitably from the struggle for life."

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Notes

1. The *locus classicus* is Sober (1984), but there are many advocates of this view, for example Millstein (2006), Brandon (2006).

2. It was not always supposed to be that way. Many of those who forged the Modern Synthesis, like Ernst Mayr and (early) Theodosius Dobzhansky, stressed the significance of the activities of individual organisms to evolution. Gould (1983) calls the progressive exclusion of organisms from evolutionary theory the "hardening of the synthesis."

3. There are many such appeals. See West-Eberhard (2003), Pigliucci (2009), Pigliucci and Müller (2010), Shapiro (2011), Laland et al. (2013), Laland et al. (2015), Noble (2017). See Huneman and Walsh (2017) and essays therein.

4. By "trait structure" here I simply mean the pattern of relative frequencies of the abstract trait types. If the black morph of *Biston betularia* becomes more frequent than the white morph, this is a change in trait structure.

5. It sometimes further assumes (or attempts to demonstrate; see Grafen 2014) that the other two kinds of population change are concomitants of change in trait structure.

6. See, for example, Sober (1984), Okasha (2006), Shapiro and Sober (2007), and Pence and Ramsey (2015).

7. This example originates with Matthen and Ariew (2009).

8. On one prominent approach to understanding causation (Salmon 1984), a causal process is one that transmits a signal (or alternatively, a conserved quantity; Dowe 1992). According to Salmon, the motion of *c* is a pseudoprocess.

9. I am leaving aside for now the increasingly important process of organisms changing their evolutionarily significant characters. I'm grateful to Earnshaw-Whyte (2012) for this discussion.

10. I discuss below the ways that first order cause explanations can be made general.

11. Hence the significance of the quote from Sober (above) that begins: "The population is an entity, subject to its own forces, and obeying its own laws," and ends: "… population thinking is about ignoring individual" (1984, 344).

12. Inheritance and mutation are neutral in the sense that the former is strictly conservative and the latter is random.

13. As Pocheville (this volume) argues, they may be "entangled" or "intertwined" to varying degrees.

14. I thank Kevin Laland for pressing this point.

15. I thank Tobias Uller for pressing this issue.

16. I suggest it is the principal motivation behind the "levels of selection debate," though that debate could, in some etiolated form, survive the demise of the two-force model (see Walsh 2003).

17. I thank Kevin Laland and Tobias Uller for help here.

18. In effect, they are arguing from thesis (i) of the PPT.

19. Others including Ariew (2003), Calcott (2013), Scholl and Pigliucci (2015), and Gardner (2013) have argued that the distinction should be seen as primarily an explanatory one. My own view is an extension of Ariew's.

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