Biological Theory (2019) 14:254–266
https://doi.org/10.1007/s13752-019-00329-3

ORIGINAL ARTICLE

Evolution is About Populations, But Its Causes are About Individuals

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Received: 9 April 2019 / Accepted: 26 August 2019 / Published online: 12 October 2019

Abstract
There is a tension between, on the one hand, the view that natural selection refers to individual-level causes, and on the other hand, the view that it refers to a population-level cause. In this article, I make the case for the individual-level cause view. I respond to recent claims made by McLoone that the individual-level cause view is inconsistent. I show that if one were to follow his arguments, any causal claim in any context would have to be regarded as vindicating a form of population-level cause view. I show why this is implausible and how a consistent individual-level cause position can be held within the interventionist account of causation. Finally, I argue that there is one sense in which natural selection might be said to refer to population-level causes of evolutionary change. The upshot is that, as noted by others, natural selection can be regarded as referring to a population-level cause in the context of frequency-dependent selection and other situations of fitness-altering interactions between the individuals of a population. But whether this statement is true will depend on the empirical case investigated, not some a priori conceptual distinction. Thus, even though situations of frequency dependence might be ubiquitous, it is orthogonal to the conceptual question of whether frequency-independent natural selection—McLoone's target—refers to individual- or population-level causes.

Keywords Causation · Drift · Individual-level causes · Modularity · Natural selection · Population-level causes

Let me be that I am and seek not to alter me.
William Shakespeare,
Much Ado About Nothing

Introduction

Over the past 20 years or so, the question of whether the relationship between natural selection and evolutionary change is a statistical or a causal one has been highly debated within the philosophy of biology. Much ink has been spilled on this question between, on the one hand, the statisticalists, and on the other hand, the causalists. The statisticalists argue that causation does not play a role in evolutionary explanations—they are purely statistical explanations—while the causalists argue that it does. I will not reiterate the points made by each camp here; the relevant discussions can be found in a number of publications.[1] All I will say is that a causalist position can be defended quite effectively against the statisticalists, and that although the statisticalists make some good points, I side with the causalist camp in the view that natural selection is a causal process. Worthy of note is that Otsuka (2016b) provides an excellent defense of the causalist position, yet to be rebutted by the statisticalists.

Although important, the debate between the statisticalists and the causalists has had the consequence of leaving other questions about the nature of natural selection and other evolutionary processes (i.e., drift, mutation, migration) in the background. In this article, I explore one of those questions. It emerges from an internal tension within the causalist camp when they have defended themselves against the statisticalists. This question is whether natural selection (and by extension drift and other evolutionary causes) refers

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[1] For instance see Matthen and Ariew (2002), Matthen and Ariew (2009), Walsh (2000, 2010), Walsh et al. (2002), Walsh et al. (2017), Bouchard and Rosenberg (2004), Rosenberg and Bouchard (2005), Millstein (2006), Millstein (2013), Reisman and Forber (2005), Shapiro and Sober (2007), Huneman (2012), Stephens (2004), Krimbas (2004), Hitchcock and Velasco (2014), Otsuka (2016b), Bourrat (2018).

to individual-level[2] or population-level causes of evolutionary change. The view that natural selection and drift refer to individual-level causes, hereafter the "individual-level cause view," has been defended by several authors including Bouchard and Rosenberg (2004), Glennan (2009), and Otsuka (2016b), while the view that natural selection and drift refer to population-level causes, hereafter the "population-level cause view," has been defended by, among others, Reisman and Forber (2005), Millstein (2006, 2013), and Stephens (2004).[3]

In a recent paper, McLoone (2018) defends the population-level cause view. He anchors his arguments in two publications from Otsuka (2016a, b), who, using causal modeling within the interventionist account of causation, provides a causal foundation for evolutionary genetics (Otsuka 2016a) and vindicates the causal view against the statisticalists (Otsuka 2016b). Following the interventionist account, a variable X is a cause of Y, if there exists at least one ideal intervention on X that would lead to a difference in the value of Y. An ideal intervention on X is understood as a change in the value of X which at the same time produces no other change in any other variable (Spirtes et al. 2000; Woodward 2003, 2010; Pearl 2009). Otsuka defends the individual-level cause view on the grounds that, in the standard models of evolutionary genetics he presents, the variables associated with natural selection upon which one can intervene are individual-level, not population-level variables. In response to that move, McLoone argues that if such a view is adopted, one has to accept not only that natural selection can occur on a single individual, not a population, but also that it can occur in the absence of variation in a population. This is because, he argues, natural selection requires *variation* in individual-level properties. And since variation is a population-level property this, according to him, warrants the population-level cause view.

In this article, I show that the arguments presented by McLoone are flawed and that they do not characterize the individual-level cause view adequately. I defend Otsuka's position, with one caveat. Finally, I show that there is one way in which the population-level causal view can be grounded. It relies on whether *physical* as opposed to *ideal* interventions on individual-level properties in a given population are *modular* or not. A non-modular intervention in a system alters its causal structure. When this happens, it becomes difficult to ascribe causation to individual-level properties in the sense that intervening on the system at that level changes the values of the properties of other individuals in the population. In such situations, of which frequency-dependent selection is a case, I argue that a population-level cause view can legitimately be defended. The upshot is that whether natural selection and other evolutionary processes refer to individual- or population-level causes of evolutionary change is not an a priori conceptual question but rather an empirical one.

The article will run as follows. After having presented McLoone's position I show why it is untenable. I then present Otsuka's causal model in broad strokes, followed by his argument for the individual-level cause view. At that point I depart slightly from Otsuka's proposal, one aspect of which is problematic. From there, I make my own proposal for the individual-level cause view. Finally, I show how modularity can make the notion of population-level causation a compelling and empirically grounded notion in the context of evolutionary genetics. I conclude by highlighting the potential significance of the distinction between modular and non-modular intervention in the context of the controversies surrounding the units of selection question, and the interpretation of heritability estimates.

The Exclusive Appeal to Individual-Level Property

McLoone's argument against Otsuka's individual-level causal position rests on the premise that the individual-level cause view ought not to mention any population-level property as part of the explanation. He writes, for instance, that, "Otsuka explicitly says he can show that natural selection can cause evolution *while only referencing properties that belong to individuals*" (McLoone 2018, p. 11; my emphasis). If true, it would make Otsuka an individual-level causalist with the view that individual-level causes are never contingent on population-level properties. There are a few things to say about McLoone's statement.

First and foremost, this is not what Otsuka claims, which rather is the following: "[i]t should be noted that *the causes* of evolutionary changes as shown above all belong to the level of individuals, in the sense that *these variables*, including environmental factors, *denote properties of individuals*" (Otsuka 2016a, p. 577; my emphases). There is a clear difference between claiming, on the one hand, that one can show that natural selection can cause evolution by referencing *only individuals' properties*, and on the other hand, that the *variables* of a model refer only to individuals' properties, so that in that sense they cause evolutionary change. The distinction between a property and a variable is important here. Given a causal model, a variable is an object's property that varies. Yet, there might be properties of individuals or

[2] By "individual" I will refer to any entity that is part of a Darwinian population after Godfrey-Smith (2009). To be clear, I do not necessarily refer to a biological organism when I use this word.

[3] In Bourrat (2018), I show that the individual-level cause view can be defended against some of the arguments made by Reisman and Forber (2005) for the population-level cause view with a focus on drift rather than natural selection.

the population that do not vary in the model, and they might be, in some sense, causally involved in the production of an effect. However, they do not represent causes following the causal modeling framework. These properties are classically referred to as "parameters." One charitable interpretation of Otsuka's point is that given certain values of parameters (which might refer to individual- or population-level properties), one can change the strength of natural selection by intervening only on the values of individual-level variables in this model, which thus vindicates the individual-level cause view. This is different from claiming that Otsuka only requires reference to individual-level properties for his view to hold, as claimed by McLoone. In fact, the parameters of the model might be population-level parameters, without this harming the soundness of the individual-level cause view, since the only causes in the model are individual-level variables.

Second, I believe that the proposal that the individual-level cause view ought to cite only individual-level properties is not a position held by anyone in the literature. To my knowledge, nobody has ever argued that the properties of a population[4] will be irrelevant when it comes to predicting or explaining its evolutionary dynamics. I think Bouchard and Rosenberg (2004) hold a sound and representative position of what the individual-level cause view is. Their position has furthermore been explicitly referred to by Otsuka (2016b, p. 477) in his defense against population-level views of natural selection (not necessarily causal ones for that matter), and has also fueled a response from Millstein (2006) who defends the population-level cause view. For that reason, I will assume that contesting the individual-level causalist position amounts to disagreeing with Bouchard and Rosenberg (2004, p. 710) when they write that: "selection [is] a contingent causal process in which individual fitnesses are the causes and subsequent population differences are the effects."[5]

From the quote above, to disagree with Bouchard and Rosenberg, a population-level causalist would have to deny that natural selection is tracked causally by the fitness of the individuals in the population, not that these causes are not contingent to a given population. Bouchard and Rosenberg are not explicit about what they mean by "contingent," but I assume that they have in mind the contingency for an individual to be in a particular population and environment, to have a particular evolutionary history, and so on. In fact, Bouchard and Rosenberg want to establish a principle of natural selection, in which natural selection is a contingent truth. The notion of contingent truth is classically opposed to the notion of necessary truth, the latter of which could never be false, while the former could be. Note furthermore that there is nothing in Bouchard and Rosenberg's characterization of natural selection preventing population-level properties in the background to be relevant for characterizing the link between natural selection and evolutionary change.

The idea of contingency has close connections with Woodward's notion of stability—sometimes confusingly called "invariance"[6] (Woodward 2003, 2010; Pocheville et al. 2017). Stability measures whether, and to what extent, a causal relationship holds as changes in the background are performed. In other words, stability refers to both the presence or absence of an effect under changes in the background, but also to the magnitude of the effect under background changes.[7] If X is a cause of Y and B is a variable that represents the background of the relationship (for instance a population of different size), there is at least one ideal intervention on X that leads to a change in Y for at least one value of B. From there, the more this relationship remains unchanged as the values of B change, the more this causal relationship is stable.

Perhaps with the exception of laws of nature, because they are not limited to any spatiotemporal interval, there are no absolutely stable causal relationships following the interventionist account of causation. There are certainly no absolutely stable causal relationships in the special sciences. Thus, any causal relationship mentioned in evolutionary theory is to some extent unstable because it would be different in some subsets of different spatiotemporal possible backgrounds. To use a nonbiological example of a very unstable relationship, take the case of sand grains added one by one to a heap until it collapses. The causal relationship between adding a sand grain and the heap collapsing is a very unstable one since whether the collapse occurs and with what

[4] Note that by distinguishing individual- and population-level properties, I do not mean "population-level" in a sense that would invalidate the supervenience of population-level properties on individual-level properties. I simply mean that our knowledge about a particular evolutionary dynamics could come from properties that we are only able to characterize at the population level.

[5] Note that by "fitness" Bouchard and Rosenberg have in mind an individual-level property that has some effect on reproductive output. Thus, importantly, fitness (which they characterize as "ecological fitness") is different from reproductive output in their view. For more on the distinction between fitness and reproductive output see Bourrat (2015a, 2017, 2018, 2019a).

[6] As pointed out by Pocheville et al. (2017), there is some ambiguity in the literature surrounding the notion of invariance. The term invariance has been used to refer to, on the one hand, whether, and if so to what extent, a relationship holds as the value of the causal variable is intervened upon; and on the other hand, whether, and if so to what extent, the relationship holds as diverse variables in the background of the relationship are changed. Following Pocheville et al., by "invariance" I mean the first and by "stability" the second.

[7] For a precise information-theoretic measure of stability see Pocheville et al. (2017). Information theory is best suited for categorical variables. To my knowledge, a quantitative measure of stability for quantitative variables does not exist in the literature.

magnitude (e.g., small or large collapse) will depend on very particular configurations of the heap (the background of the relationship). In spite of the relationship being very unstable, there is no question that adding a sand grain is a cause of the heap collapsing when that occurs. Adding a sand grain to a heap is just a contingent cause of the heap collapsing. We will come back to this example briefly.

Based on the distinction between contingent and non-contingent individual-level cause, it is important to see that one need not regard population-level properties as irrelevant for the individual-level cause view to hold—quite the contrary. To see why, suppose now the following biological example. Imagine that the same point mutation occurs on a given individual in two populations, one made of 10 individuals while the other is made of 100 individuals. The evolutionary change resulting from this mutation in the population of 10 individuals, assuming all the individuals considered produce the same number of offspring, and that reproduction is asexual, perfect (no mutation during the reproductive phase), and occurs in discrete synchronous generations, is different from that of 100 individuals. In fact, in the population of 10 individuals, the change in the population composition is ten times as large as the change in the population made of 100 individuals when this change is assessed relative to the population. There seems thus to be a difference when the same intervention on the same variable is performed in different population backgrounds (9 versus 99 individuals).[8] Yet, despite the outcome being contingent upon population size, because the intervention is carried out on an individual-level variable and it leads to a difference in the population composition, there is no reason to call this a "population-level cause." It is simply a highly contingent individual-level cause because the causal relationship it is involved in scores low on causal stability.

I have proposed so far that natural selection is a population-level process that constitutes one component of evolutionary change, but for which the causes are individual variables. At that point one line of attack against this view might be that since the effect variable (evolutionary change in mean character) is a population-level variable (average change in character), one ought to keep the same level of description throughout the causal explanation and thus any relevant causal variable in an evolutionary explanation should refer to population-level variables. Although this might at first seem appealing, this *petitio principii* is regularly violated in both everyday and scientific examples. Take the example of adding a sand grain to a heap mentioned above. Adding a sand grain is an individual-level cause, yet the heap collapsing is a population-level effect since a heap refers to a "population" of sand grains. A given individual sand grain either causes or does not cause the collapse of the heap. This causal explanation is perfectly valid in spite of having causal relata at different levels.[9] To take a second example, the presence of a virus in a population, which is an individual-level event, might be said to cause an epidemy, which is a population-level phenomenon.[10] Coming back to evolutionary genetics, take now the mutation example used earlier. When a point mutation occurs, it changes the state of one allele from one value to another. This affects the allelic composition of the population, or in other words produces some evolutionary change. Nobody, I assume, would claim that such a mutation is a population-level cause of change, even though an evolutionary process of mutation at the population level constantly occurs and brings new variation in a population.[11]

These examples, I believe, are enough to cast some doubt on the thesis that a causal explanation requires the same level of description for all variables of the explanation. Once again, descriptions of variables at any level are consistent with the interventionist account of causation. This includes

[8] Note also that other individual-level properties in the background of the relationship might be relevant for the stability of the relationship, such as whether the cell in which the mutation occurs is somatic or germinal. In the former case, this mutation would be associated with no evolutionary change, demonstrating another way in which the relationship is unstable.

[9] Note that one *could* describe a heap of sand collapsing as a sum of individual grains moving, but that would still make the explanation given of the change refer to all the grains of sand constituting the heap at once. Consequently, if one is interested in the fate of all the grains at once, describing the collapse from the perspective of individual grains would still mean that the effect variable refers to the population of sand grains. Note also that one might be interested in providing a causal explanation about one single sand grain moving in the heap following the addition of another grain to the heap. In this explanation, the other grains of the heap would only be considered as the background of the causal relationship, not as part of the effect variable. In consequence, the resulting explanation would not be an explanation of the heap collapsing, but of a sand grain moving during the collapse. Analogously, one might be interested in a given individual-level event (e.g., the fate of a particular organism) caused by an individual-level variable during an evolutionary process. Although this would be a perfectly valid causal explanation, it would not be an evolutionary one.

[10] Again, I am not claiming that to understand a virus outbreak one need not take into account population-level parameters, just that the level at which the causal relata are described can be different.

[11] Note that even though a given *token* mutation is an instance of a *type* of mutation and that some might regard this difference as vindicating two different sorts of causation, I follow Woodward (2003, p. 40) in his view that "a claim such as '*X* is causally relevant to *Y*' is a claim to the effect that changing the value of *X* instantiated in particular, spatio-temporally located individuals will change the value of *Y* located in particular individuals." In other words, type-causal claims are generalizations of token-causal claims but both refer to the same hierarchical level.

situations when different causal relata are defined at different levels of description.

The Causal and the Constitutive View of Natural Selection

Having presented the individual-level cause view in the previous section, one might argue that this does not refute the population-level cause view since natural selection and evolutionary change necessarily refer to a population. In fact, natural selection and evolutionary change do not occur in a single individual, only in populations. Although this argument seems intuitive, I think it misrepresents not only what the individual-level cause view is about, but also what the process of natural selection is. In this section, I clarify what is at stake with respect to the individual-causal view, and why the claim that natural selection is a population-level phenomenon cannot be a good argument against the individual-level cause view.

First, it is uncontroversial that both natural selection and other evolutionary processes are population-level phenomena. But it should be clear that these processes *combine* together to produce evolutionary change, rather than *cause* evolutionary change. This means that if natural selection in and of itself is not evolution as famously written by Fisher (1930, p. vii), one way to interpret this statement is that natural selection represents one part of evolutionary change, namely the part that would remain once the influence of other evolutionary processes are eliminated (Bourrat 2019a). Thus, these processes (drift and mutation, assuming an isolated population) together with natural selection constitute evolutionary change. Note that this constitution is a spatiotemporal or diachronic one, rather than purely synchronic.

To see that more concretely, following standard formalism in evolutionary theory such as the Price equation (Price 1970; see also the next section, where I present the equation more formally), I define the total evolutionary change of a character z in a population between two times, typically generations, as the mean change in the value of this character ($\Delta\bar{z}$). Following Frank (2014), this total evolutionary change can be separated into different components. Depending on what one is interested in, the number of components might vary. Frank separates $\Delta\bar{z}$ into two components, one he calls "selection" ($\Delta\bar{z}_s$), the other he calls "transmission" ($\Delta\bar{z}_t$), with transmission subsuming all evolutionary processes different from selection (such as mutation and drift). We thus have:

$$\Delta\bar{z} = \Delta\bar{z}_s + \Delta\bar{z}_t. \quad (1)$$

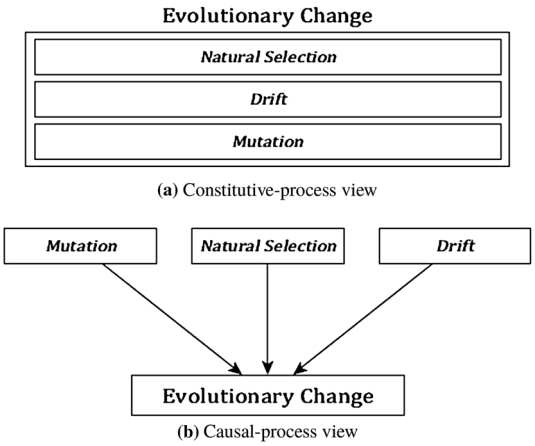

Fig. 1 Illustration of the contrast between **a** the constitutive-process view, in which natural selection and other evolutionary processes (e.g, drift and mutation) *constitute* evolutionary change, and **b** the causal-process view, in which natural selection and other evolutionary processes *cause* evolutionary change. The causal-process view is incorrect because selection, drift, and mutation are population-level processes that conjointly occur to produce evolutionary change. Yet that does not imply that the causes they refer to are population-level causes

From Eq. (1), one can see in what sense natural selection constitutes rather than strictly causes evolutionary change.[12] In fact, the two terms on the right-hand side are terms that refer to a mean change in character value between two times. They are just components of $\Delta\bar{z}$. I call this view the "constitutive-process view." By contrast, were natural selection a cause—following the interventionist account of cause—of evolutionary change, it would refer to a variable at one point in time, to which a change in value affects the change in mean character at a later point in time (the next generation). One might refer to this view as the "causal-process view." (The two views are depicted in Fig. 1). I regard the causal-process view as flawed. This is because evolutionary processes can only be manifested in populations and over time. When they occur, this implies *at the same time*, ceteris paribus, an evolutionary change at that level. In other words, there is no sense in which natural selection occurring does not refer to a population-level change.

From there one can distinguish two dimensions over which the individual-level and population-level cause views might depart. First, individual-level and population-level causalists might disagree about whether natural selection and other evolutionary processes can be accurately described by referring to causes at the individual level or the population level, where causes are variables upon which

[12] Frank does say that selection and transmission are "causes," but he uses this word in a vernacular sense.

interventions produce evolutionary change. Second, the individual-level and population-level causalists might disagree about whether natural selection can be considered as a cause *tout court* at the population level or at the individual level.

I take it that what opposes the individual-level causalists to the population-level causalists concerns the first dimension only, since if the constitutive-process view is correct then, by definition, natural selection and other evolutionary processes are population-level processes, which strictly speaking are not causes and can only be considered as referring to population. In other words the second dimension over which the two views might depart is a red herring. We will see, however, that McLoone seems to consider that the individual-level causalists argue for the causal-process view as depicted in Fig. 1b in which natural selection would be characterized at the individual level. It is thus important to be clear that, applying the principle of charity, it cannot be so. What is at stake in this debate is whether the causal variables relevant for producing evolutionary change refer to individual-level properties or to population-level properties.

Variation in Fitness as a Population-Level Cause?

To sum up so far, I have shown that (1) by "causes" Otsuka only refers to those properties that are variables,[13] where these variables are contingent causes in a causal model; (2) that the causal relata within the interventionist account can be described at different levels; and (3) that the individual-level and population-level causalists ought to be disagreeing about whether natural selection and other evolutionary processes can be described accurately by referring to causes at the individual and at the population level respectively, not about whether natural selection is an individual- or a population-level causal process.

Some will consider that these points are sufficient to regard the individual-level cause view as a sound one. Yet this might not convince McLoone, who contends that to be consistent with the standard interpretation of natural selection, one cannot solely refer to fitness but needs to refer to its variation. Since variation is a population-level property, his argument goes, the locus of causation for describing natural selection is not at the individual level but at the population level. In this section, I show why this argument, which crops up at different places in McLoone's manuscript, is flawed.[14] For instance, he writes:

> Imagine a population of individuals in a homogeneous environment and that each individual has precisely the same phenotype and fitness—that is, there is no trait variation and no fitness variation. These clones will of course possess some trait (z) that causally affects fitness (w)—for instance, their (identical) hearts. So it is true of this population that [...] there will still be a causal relationship between z and w even if there is but one individual in this population. According to the standard account, there is no natural selection in this example, since there is no trait or fitness variation in the population. (McLoone 2018, p. 5; notation for variables slightly altered)

Or again:

> any attempt to describe natural selection by referencing exclusively the properties of individuals will fail. This is because trait or fitness variation is a population-level property. Variation is like frequency in this regard. The frequency of red marbles in a set of marbles is a property of the set, not a property of any particular marble. This is why it makes no sense to speak of "the frequency of red marbles of that red marble."(McLoone 2018, p. 11)

Recall that Otsuka argues that since the variables he refers to in his models are about the individuals of the population, this is enough to warrant an individual-level causal position. The quoted response provided by McLoone is that what does the causal work is the *variation* on these variables—which is a population-level property—not the values of the variables themselves. This claim is problematic for two reasons. First, it seems to take the individual-level cause view to be a version of the causal-process view in which natural selection is an individual-level cause as depicted in Fig. 1b. As argued in the previous section, charitably interpreted this is not what the individual-level cause view is about. Second, the claim that variation in some variable leads us towards the population-level cause view is particularly surprising. In fact, following the interventionist account of causation, variation on a variable is *always* necessary for establishing a causal relationship. Without variation (whether actual or potential) there is no causation within this account.[15] If it is correct that variation in a population is necessary for natural selection to occur, it does not follow that the lack of variation in this population means that the causes of natural selection

[13] We will see in the next section, however, that there is some ambiguity surrounding the notion of "variable" when Otsuka makes his move against the population-level cause view.

[14] Note that Sober (2013) makes a similar point.

[15] Russo (2009) provides a defense of causation as variation, especially in a scientific context, against the view that regularities are enough to establish causation.

refer to the population level. McLoone confuses the requirement of variation for establishing causation with the necessary condition of variation for natural selection to occur in a population. Insisting that variation on a property, rather than the property itself, is what causes a phenomenon is simply not in line with the notion of causation defined within the interventionist account and beyond, and consequently cannot be used as an argument against the individual-level cause view. And in fact, if one were to follow McLoone's argument, they would have to conclude, for instance, that performing a linear regression analysis and establishing that a plant's leaf area causally influences its growth, as part of a randomized experiment, is not locating causation at the level of the individual plant because one will need to refer to *variation* in leaf area—a population-level property—to establish causation. McLoone's argument, a slippery slope, thus demonstrates that the presence of variation cannot be considered as a way to adjudicate the debate between the individual- and population-level cause views.

It seems furthermore that McLoone did not see the relevance of an important distinction made within the interventionist account, namely between an *actual* and a *potential* difference maker after Waters (2007).[16] This distinction is precisely designed to demarcate cases in which one could intervene on a property to show that it is causally involved in an outcome, yet the value the variable would take after the intervention is performed is never actually exhibited in the population of events considered, from cases in which this value is exhibited in this population and the cause is observed. In the former cases the cause is a potential difference maker, while in the latter cases, it is an actual difference maker.

More precisely, for X to be an actual difference maker with respect to Y, at least two conditions should be verified, according to Waters. First, X should be a cause of Y, that is, following the interventionist account there should be at least one possible intervention on X that leads to a difference in Y. Second, X should be defined over an actual population which exhibits a range of variation, and there should be at least some interventions on X using values from this range that make a difference in Y. By contrast, for X to be a potential difference maker only the first of the two conditions needs to be satisfied.[17] This implies that all actual difference makers are also potential difference makers, while the converse is not true.

As a way of illustrating the distinction, Waters uses the example of two genes causing eye color in *Drosophila*. One gene exhibits no variation in the population, but a possible ideal intervention would produce a difference in eye color. This gene is a potential difference maker. The second gene exhibits some variation in the population, and this leads individuals to have different eye colors in this population. This gene is an actual difference maker in the population under investigation. We see here again that the presence of a varying population, whether potential or actual—which might be defined over a single object varying temporally or a collection of objects— is necessary for establishing causation.

Following McLoone's argument, any actual difference maker in a population of objects ought to be considered as a population-level cause, effectively rendering the notion of "individual-level cause" vacuous and going against the classical interpretation that actual difference makers have causal powers at the level they are defined.

The Individual-Level Causalist Position Interpreted Correctly

In the previous sections, I showed that McLoone missed the mark in characterizing the individual-level cause view. I suspect that this mischaracterization might have originated from a misunderstanding of the role of a regression coefficient within the interventionist account which itself might have been fueled by one problematic move made by Otsuka in his defense of the individual-level cause view. To be completely fair, this problematic move is identified by McLoone in his footnote 4, but quickly dismissed.

Before presenting Otsuka's move and showing why it is problematic, I must briefly provide a simplified version of the model used by Otsuka. Following standard evolutionary theory (see, for instance, Lande and Arnold 1983), the reproductive output of a given individual i in a population[18] can be characterized as follows:

$$w_i = \beta z_i + \epsilon_i, \tag{2}$$

where w_i and z_i are the absolute reproductive output and the character of this individual respectively, β is the linear regression coefficient of w on z, and ϵ_i is the residual (typically conceived of as an environmental deviation).

Given this model, Eq. (2) can be inserted in the Price equation, presented informally above, which is a very abstract way to describe evolutionary change between two generations, or more generally between two times (Price

[16] I believe that the distinction made by Glennan (2009), to defend the individual-level cause view, between productive and relevant cause is on a similar track.

[17] Recently, Griffiths et al. (2015) have implemented this distinction using a causal variant of mutual information.

[18] Note that strictly speaking this is not the fitness of this individual since it is the outcomes of all factors influencing reproductive output, including those that have nothing to do with natural selection.

1970; Frank 1998; Rice 2004; Okasha 2006; Luque 2017). The classical form of the Price equation for z, following the hypotheses of asexual and perfect reproduction in discrete synchronous generations, in which drift is neglected and mutations are absent (in other words the transmission term in Eq. (1) is nil), is the following:

$$\Delta\bar{z} = \frac{1}{\bar{w}}\mathrm{Cov}(w_i, z_i), \tag{3}$$

where, as previously, $\Delta\bar{z}$ represents the average change in character between the parental population z and offspring population z', that is $\Delta\bar{z} = \bar{z'} - \bar{z}$, and $\bar{w}$ represents the mean reproductive output in the population. $\frac{1}{\bar{w}}\mathrm{Cov}(w_i, z_i)$ corresponds to the selection term of Eq. (1). Under our assumptions, the average population-level change depends solely on the covariance between the trait of individuals and their reproductive output (assuming correlation implies here causation), that is natural selection.

Once w_i in Eq. (3) is replaced by the linear regression model provided in Eq. (2) we get:

$$\Delta\bar{z} = \frac{1}{\bar{w}}\mathrm{Cov}(\beta z_i + \epsilon_i, z_i). \tag{4}$$

Using the distributive properties of variance, and the property that a covariance between a variable and itself is the variance of this variable, this becomes:

$$\Delta\bar{z} = \frac{1}{\bar{w}}\beta\mathrm{Var}(z_i) + \frac{1}{\bar{w}}\mathrm{Cov}(\epsilon_i, z_i). \tag{5}$$

Since following the method of least squares, which underpins linear regression analyses, there is by definition no covariance between z and the residuals ϵ, so we have $\mathrm{Cov}(\epsilon_i, z_i) = 0$. Consequently Eq. (5) can be simplified into:

$$\Delta\bar{z} = \frac{1}{\bar{w}}\beta\mathrm{Var}(z_i). \tag{6}$$

Equation (6) tells us that the total evolutionary change depends on the variance in z ($\mathrm{Var}(z_i)$), and the strength of relationship between z and w, or in other words the influence of z on w (measured by β).

Based on this model, one of Otsuka's characterizations of selection is the following:

> Selection, as discussed above, is a causal influence of the trait on fitness, whose linear magnitude is measured by coefficient β. [...] This parameter, in turn, should depend on selective environments including biotic (for example, prey abundance) as well as abiotic (for example, temperature) factors. [...] Intervening on the selection-as-process thus amounts to a modification of these fitness-related factors controlling β. (Otsuka 2016a, p. 265)

But there is a slight problem with this move. First, after having explained his reasoning, Otsuka intervenes on β itself. This is surprising since, as rightly noted by McLoone, and as mentioned earlier: "One potential objection to Otsuka's discussion of an intervention on β is that interventions are typically understood to be carried out on variables, not parameters. I will not consider that objection here" (McLoone 2018, p. 4, fn 4). I will go one step further than McLoone. I claim that if an intervention is carried on β, it makes β a variable of the model, not a parameter. If β is a variable then it needs to vary and to be connected to other variables. Using hierarchical linear modeling (Snijders and Bosker 1999; Bourrat 2016), one can consider that the slope of a linear regression is itself a dependent variable explained by some independent variables different from the one of the main regression. But in the model considered by Otsuka, β is a parameter of the model (which we might only be able to *estimate* from real data, but that should not concern us here) and consequently it belongs purely to the structure of the model. Being a parameter prevents it from being subjected to ideal interventions.[19] As written by Steel (2006, p. 450): "an ideal intervention, by definition, does not alter causal influences emanating from the variable it targets."[20] As was just discussed, β precisely measures the strength of the influence between the causal and the effect variable, that is between z and w. And in fact, for the causal interpretation of a structural equation to hold true as argued by Pearl (2009, pp. 159–162) and Woodward (2003, p. 322), β should remain invariant when interventions are performed on an independent variable. Thus, in the causal models discussed by Otsuka, only variables such as z and w can be intervened upon.

In all fairness, Otsuka (2016a, pp. 263–265) discusses different ways to understand the individual-level cause view as interventions on w_i or z_i, something that McLoone does not acknowledge. However, Otsuka claims that performing "hard" interventions on z or w, i.e., interventions that break all the incoming arrows to the variable intervened upon in the causal graph, will not lead to changes in composition at the next generation because "[a hard intervention] effectively interrupts all the treks from w to z so that the Price covariance becomes zero, that is, there is no evolutionary response"(p. 264).[21] He goes on to explain that this is valid

[19] As was suggested to me by Jun Otsuka, an alternative way to deal with β being a parameter and not a variable is to think of β as a "constant variable" b with all the probability mass at one value and zero elsewhere (aka Dirac delta distribution). Then the causal graph is $b \rightarrow w \leftarrow z$, with the structural equation $w = bz$. This will effectively make the linear parameter a manipulable variable.

[20] More on the significance of this statement in the next section.

[21] Hard interventions are opposed to "soft" interventions, the latter of which do not break incoming arrows in causal graphs but change the probability distribution of the variable intervened upon.

for any z which is in line with Weismann's doctrine that acquired characters are not transmitted to the next generation. Contrary to Otsuka, I believe that it is only valid for some interventions, namely those that do not produce heritable changes. But z might be considered, for instance, as the number of alleles of a given type an individual has in the germline, as is often done in some versions of the Price equation. In such a case an intervention on this character would lead to a change in the composition of the population at the next generation and consequently to evolutionary change.[22]

Taking all these remarks together, how should we interpret the individual-level cause view? I propose that one way to do so is the following. Because only the variables of a model can be intervened upon, and the individual characters (z) of the individuals are the independent variables of this model, they are the causes in this model. Yet, since each individual of the population does not have the same effect on the change in the population at the next generation, the *mean* influence is reported. It corresponds to $\beta \mathrm{Var}(z)$.[23] But in principle one could take each individual and see how it contributes to the mean change in the population. In fact, an ideal intervention on the value of z within the range of observed values (since we are referring here to actual difference makers) in the population would make a difference in the average value of the character at the next generation. As a matter of fact, the effect of a single individual on the population dynamics is what evolutionary biologists sometimes aim at uncovering (see, for instance, Coulson et al. 2006).[24]

To see this, we can define the squared deviation from the mean character z of an individual i as y so that $y_i = (z_i - \overline{z})^2$. y is here an individual-level variable even if it is defined in reference to a population-level variable; any standardized variable is. Assuming n is the number of individuals in the population, from this definition and the definition of the variance for a variable X as $\mathrm{Var}(X) = \frac{1}{n}\sum_{i=1}^{n}(X_i - \overline{X})^2$ we can rewrite Eq. (6) as:

$$\Delta\overline{z} = \frac{1}{\overline{w}}\beta\frac{1}{n}\sum_{i=1}^{n}(z_i - \overline{z})^2 = \frac{\beta}{\overline{w}n}\sum_{i=1}^{n} y_i. \tag{7}$$

One can see clearly in Eq. (7) that changing the value of an individual's y by an ideal intervention—for instance decreasing it—would lead to a change in $\Delta\overline{z}$, assuming that the intervention on y produces a heritable change. If this assumption is respected, then following the interventionist account of causation, y—an individual-level variable associated with natural selection—causes some evolutionary change. This leads to an individual-level causal interpretation of natural selection as the average change made by heritable fitness affecting individual characters on evolutionary change.[25]

When is Natural Selection a Population-Level Cause of Change?

In the previous section, I have established that in one of the simplest possible settings, insofar as one can intervene on the character of an individual and produce a change in the character of the population at the next generation, then the individual-level cause view holds since the change produced would be associated with natural selection. Does this mean that the notion of population-level causation in reference to natural selection should always be rejected? I do not think so. Although in any situation an ideal intervention on individual-level properties would, in the model discussed, lead to evolutionary change, two types of situations that matter empirically should be distinguished. Recall that β belongs to the structure of the causal model and will not be altered by an ideal intervention allowed by the model. Yet a *physical* intervention or manipulation that is a change in the value of a variable an agent might carry (or nature itself) might or might not alter β. Note crucially that this means that ideal interventions can do things that would be impossible in the real world.[26] With the distinction between an ideal and a

[22] Note also that heritability might be considered as positive for intragenerational changes in line with the point made in Bourrat (2015b).

[23] Note that leaving aside β which is supposed constant and thus only modulates uniformly what each individual contributes, this is almost literally what a variance is. In fact, in words, the variance of a variable is the expected squared difference between an individual value and the mean value in the population (for a formal definition see the main text). This difference is squared in order to obtain the magnitude of this difference, since some deviations will be positive, others negative, and on average will cancel out each other. But rather than the squared difference one might decide to take the standard deviation, which is the square root of the variance, to talk about the average effect of an intervention. Standard deviation is often considered a better measure of variation since it has the same unit as the variable to which it refers.

[24] I thank Peter Takacs for this point.

[25] Note that if y is not perfectly heritable one will have to compute its heritability h^2, which, like β, is a parameter of the model. Following our hypotheses, we assumed here that reproduction is perfect, so that ($h^2 = 1$).

[26] This is why the notion of ideal intervention is often associated with that of a miracle. For instance, Woodward (2003, p. 135) considers an ideal intervention to be synonymous with a "localized miracle," borrowing the notion of miracle from David Lewis. Pearl (2009) talks about "surgical procedure." He is very clear that ideal interventions can do things that might not be possible physically when he writes "[s]ymbolically, one can surely change one equation without altering others and proceed to define quantities that rest on such 'atomic' changes. Whether the quantities defined in this manner correspond to changes that can be physically realized is a totally different question that can only be addressed once we have a formal description of the interventions available to us" (p. 365). For more on

physical intervention made, I argue that whether a physical intervention would alter the value of β can ground a distinction between individual-level and the population-level causation.

To see how, following the literature on this topic, let us call a physical intervention on a variable (such as z) that does not alter the value of the structure of the model (such as β) a "modular intervention," and an intervention that does a "structure-altering intervention" a "non-modular intervention."[27] Based on this distinction, Illari and Russo (2014, p. 105) characterize a modular causal relationship as follows:

> For manipulationists, in the model describing the system, causal relations are modular if the causal structure of the underlying system isn't altered when one makes interventions on the putative cause. So for example, a system that satisfies this is: the causal relationship between smoking and lung cancer isn't altered by intervening to reduce smoking. We can alter cancer rates by intervening on smoking behavior, precisely because the relation between smoking and cancer still holds.

Note importantly that the word "intervention" in this quote refers to physical interventions, since they are interventions performed by agents.[28]

To see in what sense modularity can ground the distinction between the individual-level and population-level cause view in relation to natural selection (but the application is much broader), take a population in which, as previously, the individuals reproduce asexually, perfectly and in discrete synchronous generations. Suppose now that there is a linear deterministic causal relationship between the height of an individual (z) and the number of offspring it produces (w). To make things simple, suppose that height goes from 1 to 10, and that the number of offspring goes deterministically from 1 to 10, with a height of 1 leading to the production of 1 offspring, a height of 2 to 2 offspring, and so forth. In such a situation, if one were to physically intervene on the character of one individual by adding one unit of height—we could imagine that scientists are able to manipulate a gene responsible for height—one would also increase the number of offspring by one unit of this individual and as a result β would remain unchanged. In any causal linear situation as just described β will remain the same. This is a situation of modular causal relationship between z and w.

Suppose now the same type of setting, but one in which physically intervening on the character of an individual also changes β. A situation satisfying this phenomenon is frequency-dependent selection.[29] In cases of frequency-dependent selection, changing the character value of one individual has some effect on the fitness (i.e., reproductive output here) of at least another individual. In such a case, the causal relationship between z and w will be non-modular. How should one interpret causally this non-modular situation? The lack of modular causal relationship between z and w can be taken as vindicating the population-level cause view since if one considers an individual to have some influence on the evolutionary dynamics of the population, the nature of this influence would change if one were to physically change the character of some other individual(s) in the population. The locus of causation is thus not "cleanly" located in the individuals forming the population, but rather distributed among them. By contrast, in cases where modular physical interventions are possible, the locus of causation leading to evolutionary change is without any doubt the individual on which the intervention is performed.

To be clear, non-modular interventions are not enough to vindicate the population-level cause view. In fact, there might be non-modular interventions on an individual which affect parameters other than β without affecting other individuals of the population. For instance, we could suppose that the fitness of a bird depends both on its weight and its height. One could devise a linear-regression model with two predictors for fitness: height and weight. Since it is impossible to physically intervene on the height of an individual without at the same time intervening on its weight, the causal relationships between height and fitness, on the one hand, and weight and fitness, on the other, are non-modular.[30] Yet because all the independent variables involved here refer to a single individual, causation is purely at the individual level. Thus, it is only insofar as the relationship between z and w is non-modular in frequency-dependent situations *and* that the reason it is non-modular is that intervening on one individual's character affects the fitness of more than one individual, that a non-modular intervention on z with respect to β can be seen as vindicating the population-level cause

Footnote 26 (continued)

the view that ideal interventions need not be physically possible see Woodward (2016).

[27] Woodward (2003, pp. 329-330) distinguishes modular interventions from "level-invariant" interventions. The former refers to invariance of the parameters of different equations in a system of structural equations, while the latter refers to invariance of the parameters of the equation in which the intervention is performed. For my purpose, I will refer to these two types of interventions as "modular." Note also that modularity is intimately linked to the notions of stability and invariance presented earlier; see Footnote 6.

[28] Illari and Russo (2014, p. 105) borrow the idea of structure-altering intervention from Steel (2006, 2008).

[29] In general, any fitness-altering interaction between the individuals of a population will satisfy this phenomenon.

[30] Note that this would be a particularly bad model precisely because the two characters are correlated.

view. Sarkar (2008), although he does not use the notion of modularity, makes a similar point concerning the population-level nature of causation (or at least above the level of the individual) in cases of frequency-dependent selection (see also Millstein 2006).

Note importantly that strictly speaking, non-modular causal relationships involving β could still be considered from the individual-level cause perspective, in two ways. First, one might want to refer to an *ideal* intervention on z, not a *physical* intervention when characterizing the causal nature of evolutionary processes. Such a description will, however, come at the cost of not representing the actual mechanistic processes occurring in the population, and refer to unstable causal relationships. In fact, although one will be able to describe a situation as if each individual were contributing a certain part to the evolutionary change observed, this will not represent the way "nature" proceeds to produce the outcome.

Second, one potential response to the problem posed by situations of frequency-dependent selection to the pure individual-level cause view is to say that there exists a causal model in which different linear coefficients (βs) describe the dynamics of frequency-dependent selection in a modular way. More precisely, fitness will be considered as the function of two or more modular causal influences, each coming from a different individual. Although this reasoning is sound and would be one way to accurately describe the dynamics of a population, it does not respond fully to the problem posed by frequency-dependent selection, and fitness-altering interactions between individuals more generally. In fact, linked to the notion of individual-level cause, is not only the view that the variables intervened upon are individual-level variables, but also that an individual-level property, such as fitness in this case, does not depend too much on the properties of the other individuals of the population (for discussions of this point see Ramsey 2006, 2016; Abrams 2009; Pence and Ramsey 2013; Bourrat 2015a, 2017, 2019a, b). By "too much," I mean here that in a model these interactions could be considered as negligible. The fact that one could not predict the population-level change made by intervening on a single individual without knowing the states of (potentially all the) other individuals in the population, is, in my view, enough to warrant the use of the notion of population-level causation in this situation.

Grounding the individual-level/population-level cause view in modularity might have the further benefit of explaining why different authors have had conflicting views about the nature of selection. Some authors, when reasoning about natural selection, might immediately come up with frequency-dependent selection cases, while others might think of frequency-independent cases as more natural (even if effectively much rarer in nature). It is interesting to note that Millstein (2006) resorts to a case of frequency dependence (a rock-paper-scissors situation) when she defends the population-level cause view against one form of the individual-cause view, which she calls "sophisticated individualism." However, one must be careful and distinguish whether the population-level cause view is argued for empirical or conceptual reasons. As mentioned earlier, I agree with Millstein that frequency-dependent selection, and other situations in which the fitness of an individual depends on the property of more than one individual of the population, can be used to vindicate a population-level cause view for empirical reasons. However, I do not believe that this warrants the claim that natural selection can be regarded as being at the population level for conceptual reasons. This is, however, what she seems to argue at times using a similar rhetoric as McLoone. For instance, she writes that the "comparative nature of natural selection [...] entails that it is a population-level process" (pp. 644–645) and later claims that "[...]'variation in genotypes' is a property of the population, as is 'variation in the abilities of genotypes.' These population-level properties are the causal engine of selection. This is causality at the population level; the differing physical abilities, an attribute of the population, do the causal work of selection" (p. 645). As we have seen, causation within the interventionist account and beyond is by nature comparative—it requires variation—and thus following Millstein's reasoning, one would have to conclude that any causal relationship observed in a population is by nature a population causal process, which we have seen is not sound.

Thus, although in this section I have proposed one way in which it might be legitimate to talk about population-level causation in the case of selection, it should be clear that it is quite different from the one we started with. Whether what causes evolution should be better characterized at the individual or at the population level will depend on the situation studied and the question of interest. One might determine that for such and such evolutionary dynamics one must appeal to population-level causes because causes at the individual level fail in modularity, while in other cases a purely individual-level perspective will do the job. That said, given that frequency-dependent selection is ubiquitous (Ayala and Campbell 1974; Dieckmann and Ferrière 2004) and, more generally, fitness-affecting interactions between the individuals of a population, it might be wise to consider in the general case that there will be important practical limits to a pure individual-level cause view and rather claim that the causes of natural selection refer to population-level variables. One more time, it should be clear, however, that this interpretation of the population-level cause view is very different from that advanced by McLoone, who considered the simplest possible situation, following Otsuka, of frequency-independent selection. Furthermore, had McLoone used a frequency-dependent selection setting and argued for its

ubiquity to support the population-level cause view, I still do not believe that one would have to conclude that natural selection *necessarily* refers to population-level causes of change. Conceptually, in simple cases of frequency-independence, the causes of natural selection can be considered as referring to the individual-level, however one looks at it. Whether the type of situations described in the model are met often in nature is orthogonal to such a claim.

Conclusion

In this article, I have argued that the individual-level cause view on natural selection is largely warranted on conceptual grounds if the distinction between what I called a "contingent individual-level" cause and a "population-level" cause is made. The strategy proposed by McLoone to defend the population-level cause view primarily fails because it leads to the slippery slope that any causal relationship is a population-level causal phenomenon, rendering the notion of individual-level cause vacuous. Although I rejected the view that the population-cause view can be a priori justified, I proposed nevertheless that situations in which no modular physical intervention at the individual-level is possible could empirically ground a view in which causation is characterized at the population level.

To conclude, it is important to note that the modular/non-modular intervention distinction has very important implications for the causal interpretation of linear regressions in general. In fact, any situation in which a regression coefficient would be altered by a physical intervention should raise questions about the causal interpretation given of the phenomenon if the aim of this interpretation is to be a mechanistic one. This is significant because regression analyses are at the basis of the traditional toolkit in evolutionary biology, psychology, and social science, and mechanisms are considered as the basis of most phenomena in these disciplines. For instance, in relation to evolutionary biology, one natural domain in which the lack of modularity of regression will be an important aspect to consider is in regards to the controversy surrounding the units of selection debate and more particularly the interpretation of kin selection and group selection (Sober and Wilson 1998; Okasha 2006; Nowak et al. 2010; Okasha 2016; Birch 2017). Although the overreliance on statistics to establish causation has been criticized, perhaps most notably with the bookkeeping objection to the gene's eye view (see Okasha 2006, Chap. 5, for a review of the debate), as far as I am aware, the link between these criticisms and the possibility of modular interventions has not been drawn. The same is true about the controversy surrounding the causal interpretation of heritability estimates (Lewontin 1974; Sesardic 2005; Lynch and Bourrat 2017; Bourrat, forthcoming).

Acknowledgements I am thankful to Mathieu Charbonneau, Paul Griffiths, Jun Otsuka, Peter Takacs, and two anonymous reviewers for comments on previous versions of the manuscript. I also thank the members of the Theory and Method in Biosciences group at the University of Sydney and in particular Stefan Gawronski who proofread the final manuscript. This research was supported by a Macquarie University Research Fellowship and a Large Grant from the John Templeton Foundation (Grant ID 60811).

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