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Fitness, Mutational Load, and Eugenics

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Abstract

Eugenic arguments are not a thing of the past. In 2016, geneticist Michael Lynch published an article in *Genetics* arguing that human mental and physical performance are declining at a rate of 1% per generation. This estimate is not based on measurements of performance but on an argument from mutational load: Medical interventions are relaxing selection on the human population, which will lead to a buildup of deleterious mutations. This simple argument from mutational load is invalid. When the argument is made valid, it is not obvious that there are any significant consequences for human population health.

1. Introduction

In 2016, geneticist Michael Lynch published a perspective in *Genetics* arguing that human physical and mental performance are declining at a rate of 1% per generation (Lynch, 2016a). This estimate was not based on measurements of physical and mental performance but rather on an argument from mutational load: Because medical interventions, such as "surgical procedures, pharmaceuticals, nutritional supplements, and physical and psychiatric therapies," have mitigated the effects of selection on "bad genes," the incidence of deleterious mutations (mutational load) in the human population has risen and will continue to rise.

Lynch is not a fringe figure in the genetics community. He is the former president of the Genetics Society of America, the Society for Molecular Biology and Evolution, the Society for the Study of Evolution, and the American Genetic Association, as well as the winner of the 2022 Thomas Hunt Morgan Medal for lifetime contributions to genetics. Of his more than 250 papers, 12 have over 1000 citations, and his 2007 textbook, *Genetics and the Analysis of Quantitative Traits*, written with Bruce Walsh, has over 12,000 citations (Scholar, 2024b). With an impact factor of 4.556 at the time of publication, the journal *Genetics* is not a minor venue, either (BioxBio, 2024).

The mutational load argument is not original to Lynch. It originates with Muller (1950), who urges steps to combat this increase in load and the subsequent dark future. Muller chose not to use the word *eugenics* (Paul, 1987), but his suggestions were exactly that:

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> [I]t must be pointed out that there is after all one and just one way of avoiding the fiasco of a full fledged resumption of ordinary natural selection. That method, whether we like it or not, is purposive control over reproduction, exercised in such wise as to anticipate and forestall the need for natural selection of the usual, externally imposed type. (Muller, 1950)

Unlike Muller, Lynch stops short of advocating eugenics. In a reply to those who claim his article advocates eugenics, he says he is "simply highlight[ing] the fact that long-term, population genetic issues merit recognitions and discussions" (Lynch, 2016b). Scientists, he says, "have a responsibility to present what we believe to be the facts, and the release of information should not be biased by preconceived notions of what is good vs. bad." I take it, therefore, that Lynch thinks the problem he is addressing is a distinctively biological one, as opposed to an ethical or political one. It's hard, however, to escape the conclusion that Lynch thinks this is a serious issue that we ought to do something about. He does nothing to avoid the eugenic conclusion, stating:

> What will it take to promote serious discourse on the slowly emerging, long-term negative consequences of policies jointly promoted by political, social, and religious factors? Should such a discussion even be pursued or should the process of accelerated genetic change simply be allowed to run its course—a slow walk down the path to what Hamilton (2001) called "the great Planetary Hospital"?

In spite of this potentially eugenic conclusion, criticism has been muted. Two responses were published in *Genetics*, but neither tackled the core issue. Roth and Wakeley (2016) point out that the argument is nothing new, whereas Teicher (2018) traced the history of the term *load* to its roots in German eugenic programs. Neither reply, however, seriously challenged the validity of Lynch's argument. On the contrary, Google Scholar lists 129 unique articles citing Lynch's article—most of them uncritically (Google Scholar 2024a). Readers are left thinking that in spite of a lack of novelty and an unfortunate history, the argument itself is sound.

The aim of this article is to show that the argument from genetic load is overblown, if not simply mistaken. The simple version of this argument is invalid. More sophisticated versions may be correct, but they do not present us with a problem of great significance.

A comment on the scope of the argument is necessary before I begin. I will not be asking whether individual or population fitness is a morally significant metric, nor will I be asking the general question of what makes eugenics morally wrong. Both of these, I take it, are controversial issues requiring more attention than can be brought to bear here. That said, ethical arguments depending on these biological conclusions have been made by Powell (2015) and Gyngell et al. (2019). If the biological conclusions do not hold up to scrutiny, arguments based on them will be undermined as well.

2. Mutational load

Mutational load, briefly put, is the effect that mutation has on the fitness of a population. It is one component of genetic load, which is the difference in fitness

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between the maximum fitness genotype in a population and the mean fitness of the population (Crow, 1970).[1] Muller and Lynch's arguments, however, are better understood as concerned with the expected *number* of deleterious mutations in an individual in a population, as opposed to, in the first instance, a difference in fitness.

Muller's introduction to this concept is particularly clear. He imagines a delivery company that purchases two new trucks per year (n). The trucks persist (p) in the fleet until they wear out—3 years in the example—and are subsequently removed from the fleet. Eventually, an equilibrium is reached, and there is a constant number of trucks in the fleet (f). In this case, that number is six. Thus, the number of trucks in the fleet at equilibrium may be found by the following equation:

$$f = np.$$

The same holds for deleterious mutations. Mutations enter the population at a rate (n) proportional to the mutation rate, they persist (p) in the population for a time inversely proportional to the strength of selection, and the expected number of mutations in the population per generation (f) is determined by these values. Muller estimated, prior to the molecular revolution in genetics, that this number was eight in humans. Current estimates put the number at 12,000–13,000 single-nucleotide polymorphisms (SNPs), although, of course, their mean effect size is much smaller (Henn et al. 2016).

Given the previous formula, there are two ways to increase the mutational load of a population. The first is to increase the mutation rate. The second is to relax selection pressure.

Living, as he was, at the beginning of the atomic age, Muller was concerned with an increasing mutation rate due to atomic weapons and the proliferation of radioactive devices in medicine, industry, and the home. Crow (1997) speculates that had he been aware, Muller would also have been concerned by the spread of chemical mutagens. In this, he was undoubtedly right. An increase in radioactive and chemical mutagens is a cause for concern. It is likely to result in an increase in both somatic and germline mutations, and those mutations that affect fitness are, it is commonly assumed, overwhelmingly likely to be deleterious.[2]

Lynch is also concerned about increases in the mutation rate, although his concern is primarily with increases in the germline mutation rate due to paternal age. It is well known that because the process of spermatogenesis involves the duplication of cells many times throughout a male's lifetime, there is an increasing probability of mutations in sperm cells as males age. Thus, a trend toward older fathers increases the effective germline mutation rate.

The main target in both articles, however, is the relaxation of selection due to changes in the human environment. Lynch points out:

[1] Crow (1970) separates genetic load into load due to mutation, segregation, and heterozygosis.

[2] Both Muller and Lynch assume that the vast majority of mutations are deleterious or neutral; thus, advantageous mutations may safely be ignored. There has been pushback on this assumption (see Shaw et al. 2002, 2003), but Muller and Lynch's view is the received view among geneticists (Keightley and Lynch, 2003).

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> [T]he myriad of clinical procedures for mitigating the consequences of bad genes (e.g., surgical procedures, pharmaceuticals, nutritional supplements, and physical and psychiatric therapies) can only result in the relaxation of natural selection against a broad class of deleterious mutations. (Lynch, 2016b)

This is what makes humans "exceptional" as per the title of Lynch's paper. We shape our environment and, in so doing, reduce selective pressures on mutations in the population.[3] This, given the previous formula, increases the number of deleterious mutations in the population.

3. The simple argument from load

The simplest version of the argument is as follows:

> Relaxing selection on a population will increase the expected frequency of deleterious mutations. We are relaxing selection on human populations. Therefore, the expected frequency of deleterious mutations in human populations will increase. This argument is sound, but more is needed to get the required biological conclusion:
>
> Human populations can be expected to decline in fitness as a result of an increase in the expected frequency of deleterious mutations.

The issue here is that an increased *number* of deleterious mutations does not necessarily correspond to a reduction in fitness. Fitness depends on selective environment as well as phenotype, but by hypothesis, the selective environment has changed, and thus we cannot immediately conclude that fitness will decline.

As a concrete example, consider type 1 diabetes. Type 1 diabetes typically develops in childhood, and in the ancestral environment, this would likely be a deadly affliction, resulting in a realized fitness of 0. But in our current environment, with early detection, blood sugar monitoring, insulin, insulin pumps, and so on, individuals with type 1 diabetes are increasingly likely to survive and reproduce. The expected number of offspring for an individual with type 1 diabetes now approaches that of an individual without type 1 diabetes (Sjöberg et al., 2013). Fitness of the phenotype in the modern environment is not that of the individual in the ancestral environment, so it is false that the increased number of deleterious mutations causes a decrease in population fitness.

In fact, holding all else fixed, we can expect an *increase* in absolute fitness, or rate of growth, as a result of improved medical technologies. This is especially obvious given how common assisted reproductive technologies have become. Many of us have used or know someone who has used in vitro fertilization or other treatments for infertility. Many who would not otherwise have had children now can and do.

[3] There is a question of whether this truly makes humans exceptional. Many organisms shape their environments in ways that reduce (or increase) selection on various traits (Pascoal et al., 2023). Humans may be exceptional in the extent and rapidity of such shaping, but we are hardly unique.

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Ignoring the fitness effects of changes in the environment seems like an elementary mistake, but it is one Lynch seems to make in passages like the following:

> For the most extreme case of completely relaxed selection ($s_n thinsp; = thinsp; 0$), beneficial alleles will ultimately be lost entirely ($\hat{p}_n thinsp; = thinsp; 0$), with the rate of increase of deleterious alleles (with hidden effects) being entirely governed by the mutation rate to defective states.

This raises an obvious question: What is a deleterious allele when there is no selection? Lynch says that the effects are hidden, but the reference environment he uses is the ancestral one, not the current environment. Lynch needs to establish that the ancestral environment is, for some reason, a privileged reference environment and the one that ought to count when we consider the fitness of a population. "Hidden" fitness effects are simply not fitness effects in the current environment. As Pascoal et al. (2023) observe in a study of parental care in the common burying beetle, *Nicrophorus vespilloides*, relaxed selection "results in a greater mutation load, for which it is also a phenotypic antidote."

Roth and Wakeley (2016) point out that our current environment may be very different selectively from our past environment, with some traits that were advantageous in the ancestral environment being disadvantageous in the current environment, and vice versa. This is undoubtedly true for some traits, but Lynch replies that many others, such as psychological conditions and hereditary cancers, have "absolute effects in essentially all environments" (Lynch, 2016a). This may be correct—cancers seem unlikely to have positive fitness effects, although I am less confident about most psychological conditions—but it misses a larger point. That larger point is that environments with relaxed selection conditions like psychological disorders and hereditary cancers *by hypothesis* do not have the same fitness effects. This is not to deny that many traits are deleterious in both ancestral and relaxed environments—the claim is not that selection has been *completely* relaxed. The claim is that the selective effect of these conditions is less than it was in the ancestral environment, so they do not have the same fitness consequences.

Similarly, Lynch argues that even if selection is "soft," deleterious mutations will still have "very real costs." But it is unclear what these costs are. In hard selection, genotypes have the same fitness differences in different environments, such as a trait that reduces fertility in all environments, but in soft selection, population density makes a difference.

Wallace (1991) used the example of hibernating bears to make this clear. If there are fewer dens to hibernate in than bears, being passive is a disadvantage—an aggressive bear gets a den over a passive bear. But if the number of dens is greater than the number of bears, being passive is no disadvantage at all—every bear gets a den regardless. Whether or not being passive is a disadvantage, and to what degree, depends on the selective environment, in this case, the population density.

Muller (1950) makes a similar mistake in his argument that an increase in mutational load would be detrimental for the human population, as Wallace (1991) points out. Muller calculates that the decrease in human fertility due to load is approximately 0.2. Because of this, the 2.3 children per couple in Western societies (as of 1950) represents the survivors of about 3 zygotes per couple. Doubling the load

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would then reduce the probability of survival per zygote from about 0.8 to 0.6, leading to only 1.8 children per couple, which is not sufficient for replacement and would lead to population decline and eventual extinction. What Wallace points out is that this ignores the fact that rates of reproduction in humans (especially in societies with access to birth control) are dependent on population density and are therefore soft.

If Lynch means that these conditions have constant costs across environments, then he is simply claiming that selection is hard. But that is not true for many of the conditions we might consider. Consider again the case of type 1 diabetes. Arguably, selection is soft on this trait because it depends on the availability of medical care. If care is expensive and rare, then there will be more intense selection pressure on the trait. But if care is cheap and common, there will be less selection pressure, if any, on the trait. Obviously, the cost and availability of care vary by condition, so Lynch is right that selection is hard for some traits. But this only shows that the selection against these traits is not as relaxed as it might be. If selection is stronger than we thought, then the expected increase in mutational load is also lower. One is left wondering what Lynch means when he says that there are "very real costs." If they are fitness costs, there is less load; if the costs are something else, what are they?

4. The argument from declining baseline human performance

In order to make the argument that relaxed selection poses a problem for human populations, there needs to be a bridge from the claim that there will be an increase in the number of deleterious mutations to the claim that something bad for the population will happen. In a second version of the argument, rather than a decrease in fitness, an increase in the number of mutations is expected to result in a reduction in "baseline performance." Lynch makes this claim, saying:

> [O]ur current knowledge of the rate and likely effects of mutation in humans suggests a 1% [per generation] decline in baseline performance of physical and mental attributes in populations with the resources and inclination toward minimizing the fitness consequences of mutations with minor effects.

This claim is based on mouse studies by Uchimura et al. (2015) in which a mutator strain of mice was seen to decrease in offspring number and body weight and increase in "obvious phenotypic abnormalities." Lynch notes that mice and humans have similar genetic and genomic architectures, and thus he holds that the inference from mouse to human is valid. This is too quick, however. Even if the inference from mouse to human holds, the study by Uchimura et al. (2015) is not a study of relaxed selection; rather, it is a study of increased mutation rate. Mutator strains of mice are strains with impaired DNA-repair mechanisms and thus have a higher mutation rate than wild mice. As we've seen, relaxing selection is likely to *increase* offspring number, not decrease it, because previously less fit phenotypes now have higher numbers of offspring.

If there is a signal to be found here, it is that we will observe non-fitness differences, such as changes in body weight and "obvious phenotypic abnormalities." But in order to assume that these will affect baseline human performance, we would

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have to know what human performance metrics we're looking at and whether these are or have until recently been under selection.

So what performance metrics does Lynch consider? The primary one that Lynch considers is intelligence. He mentions studies from Crabtree (2013) and Woodley of Menie (2015) that suggest there has been a decline in intelligence in the United States and the United Kingdom and that, although controversial, these claims give us reason to think that there is a problem.[4] Lynch is right that these studies are controversial; indeed, they are extremely problematic for several reasons. Woodley of Menie (2015), for example, uses socioeconomic status as a proxy for intelligence, then points to a historical association between socioeconomic status and realized fitness, or actual offspring number, to indicate that intelligence has a fitness effect. Unpacking all that is wrong with this would take some time, but the real problem for Lynch is elsewhere.

The real problem is that these articles assume the conclusion that Lynch wishes to draw. Both of the cited articles take it as given that mutational load *is* affecting intelligence and attempt to estimate the amount of decline that has occurred. But this is exactly Lynch's argument: Selective pressure has been reduced, and therefore there has been (or will soon be) a decline in intelligence. Far from providing independent support for the claim that intelligence has decreased or will decrease, it makes the very same argument that Lynch is making.

There's also the question of what the 1% decrease is with respect to. The way the worry is phrased, it might sound like we'll see a 1% decline in hundred-meter dash times, maximum bench-press strength, or measured IQ. This isn't the case. The claim is that *fitness* will decline by 1% per generation due to the spread of deleterious mutations. That is, it's a claim about a decline in fitness from the improved baseline after intervention and not about declines in phenotypic measures of performance.

Furthermore, the mapping from fitness measures to phenotypic measures isn't at all obvious. What decline in speed, strength, or IQ would equate to a 1% decline in fitness, and in what selective environment?[5] What aspects of strength or intelligence are under selection in the first place? Much more would need to be said before we can conclude there is a significant worry here.

Now consider Lynch's claim that for traits that were under selection, we can expect to see an increase in "obvious phenotypic abnormalities." Here again, more needs to be said. Why are these "abnormalities" problematic? Eyeglasses—and more recently, laser surgery—could well be reducing selective pressure on visual acuity.[6] If that's right, we should expect an increase in the proportion of people needing glasses. But as James Crow pointed out with respect to this question, "Who worries about having to wear spectacles?" (Crow, 2000). Crow's point applies more generally. Advances such as insulin pumps and continuous blood glucose meters mean that where such care is widely and cheaply available, outcomes for type 1 diabetes continue to improve (Beck

[4] This suggested decrease in intelligence is in conflict with an observed increase in measured IQ during the 20th century, sometimes called the *Flynn effect* (Williams, 2013).

[5] There are good reasons to be skeptical that IQ correlates with anything like "intelligence," but I set aside these worries for now (Gould, 1996).

[6] The recent "nearsightedness epidemic" is likely to be due to changes in the environment, such as sunlight exposure in childhood, rather than reduced selection on vision, although this does not undermine the possibility that selection has been relaxed as well (Dolgin, 2024).

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et al., 2019). Soon we may be able to say, "Who worries about having to check their blood sugar?"

In order to make this point stick, Lynch would have to show that some important good would be threatened if these conditions were more common. But doing this would move away from the apparently value-neutral—"presenting the facts as they are"— argument Lynch seems to want and into explicitly ethical territory. By framing the question in terms of "baseline human performance," I take it that Lynch tries to stay in the biological realm while also making a covert value judgment that these performance metrics are or ought to be worrying.

5. The argument from the long term

This brings us to the third argument that mutational load is a problem in need of a (eugenic) solution. Lynch argues that solutions like eyeglasses and insulin only exacerbate the problem (Lynch, 2016b). They further relax selection, leading to a greater need for medical intervention. But there's no guarantee that medical technology will keep ahead of the curve.

This argument differs from the previous ones in an important respect. Here, the argument isn't that fitness declines with respect to the ancestral environment. The argument is that fitness will decline with respect to the improved baseline after the introduction of improved sanitation, medicine, and so on. After a sharp jump up, there will be a slow decline back to mutation-selection equilibrium—the point at which the introduction of deleterious mutations equals the rate at which they are removed by selection.

Muller makes the same claim in his 1950 article. He points out that relaxing selection makes no difference in terms of "genetic deaths." At mutation-selection equilibrium, population fitness depends only on the mutation rate. This is because the effect of a deleterious mutation on the population is the same no matter how weakly or strongly deleterious the mutation is. The only difference between a weakly deleterious mutation and a strongly deleterious one is that a weakly deleterious mutation will end up affecting many more individuals than a strongly deleterious mutation before it is removed from the population. Eventually, a new equilibrium will be reached, and natural selection will be back in full force. Our efforts to cheat natural selection through technology will have been in vain. That's why Muller concluded that the only way to avoid a return to full-force natural selection was to take the more "humane" route of artificial selection—in other words, eugenics.

Lynch builds on this, arguing that this is not merely about the introduction of *new* mutations into the population. Even if no new mutations were introduced, there would be a reduction in fitness due to preexisting deleterious mutations drifting to higher frequencies (Lynch, 2016a).

While this is all correct, it is a long-term problem and does not show that medical intervention was futile. Lynch estimates that if the selection coefficient of a mutation is reduced from 0.01 in the ancestral environment—which he estimates to be the usual size of a deleterious mutation—to 0.001 in the new environment, the halfway point will be reached in about 700 generations if no further advances in medical technology and availability are made. Until equilibrium is reached, there will be a reduction in "genetic deaths" from natural selection. Is the fact that we'll eventually

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reach equilibrium a reason that the reduction in selective force until then was "futile"? Postponing or pausing a problem is not a bad thing.

We're all familiar with Keynes's quip that "in the long run we're all dead." His point was in reference to the quantity theory of money. According to that theory, increasing the amount of money in an economy makes no difference because eventually prices will simply rise to match. Real prices will not have changed at all; therefore, injecting money into an economy is futile. But as Keynes points out, even if this is true in the long run, we may care very much about the short-term changes that occur. The same can be said for the short-term changes that occur through medical and other technological interventions.

6. Conclusions

The simple versions of the argument that mutational load poses a threat to human populations fail for three reasons. First, they equivocate on selective environment and therefore on fitness. Second, they conflate the effects of the mutation rate and reduced selection—although both increase the frequency of deleterious mutations, when these affect fitness, they do so through different mechanisms. Third, phenotypic claims about "baseline human performance" cannot be read off the claimed decline in fitness from an out-of-equilibrium state.

As for the more sophisticated long-term claim, I don't know what the human population looks like in 14,000 years—the approximate time for half of the total reduction in fitness to occur—but I *do* know that there are many more pressing issues for the human population between now and then. Lynch compares the rate of fitness decline to the rate of global warming, suggesting the two worries are similar, but this is misleading. Global warming and its attendant challenges for human health are already a serious concern for the human population; mutational load is not (Patz et al., 2014).

There are likely further reasons for rejecting Lynch's claims that mutational load is a worry for human populations. These include questions about the relevance of fitness concerns to moral and policy questions, the social impact of healthcare availability on populations, and questions about the value of human diversity. I have focused on fitness effects because that is where I understand Lynch's argument to be targeted—the worry is meant to be a distinctively evolutionary one. If that claim does not hold up, then arguments based on the supposed biological worry are undermined as well.

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