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New Barriers on the Slippery Slope?

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There is an unspoken distinction in ethical criteria in bioethics. On the one hand, there are criteria that can be used with discretion and judgement by an individual or small group of people like an IRB. On the other hand, there are rigid criteria necessary for rule-based public policy. In his very insightful article, Bryan Cwick identifies a number of important criteria to consider in human gene editing (HGE) (Cwick 2020). They all could be applied with discretion and judgement. In this paper I evaluate whether they could also function as rules in a policy context.

The best way to understand ethical rules in the HGE debate is, as Cwick notes, a sociological version of the slippery slope. (I base this commentary on my book on slippery slopes in the HGE debate (Evans 2020).) Many debates in bioethics that are input for policy are set up as slippery slopes (abortion, euthanasia, chimeric organisms, embryonic experimentation, and much more). This is not the “philosophical” version of the slippery slope, which is considered bad reasoning (Walton 1992), by an empirical slope, where taking a step on the slope increases the odds of taking the next. There are many social mechanisms that increase the odds of sliding down the slope, such as the knowledge developed by implementing an act high on the slope being used to make possible acts lower on the slope. In bioethics, the main source of grease on the slope is cultural –

getting used to the act at step A on the slope makes step B seem more reasonable.

After starting as a debate among reform eugenicists in the 1950s, by the early 1970s the slope in the HGE debate was set. At the top was somatic gene therapy and at the dystopian bottom was germline enhancement of traits like intelligence (Evans 2002). So, how does a debate slip down the slope? Each step down the slippery slope is the result of a lack of a sharp line between cases, where the cases are on a “gradient or continuum,” where “arguments or policies are connected by a series of small (perhaps infinitesimally small) steps” (Rizzo and Whitman 2009:691). There are two mechanisms for slippage. The first is “continuity vagueness,” where the terrain of the slope has a continuous measure. (Rizzo and Whitman 2009:691) For example, if we said that it is acceptable that a future baby’s IQ could be increased if they were predicted to be in the bottom 15th percentile, the limit easily slides to the 16th percentile because the numeric difference between 15 and 16 is arbitrary. The second feature of a slippery terrain is “similarity vagueness,” where “measurement is not possible, irrelevant, or when it depends, at least in part, on imprecise components” (Rizzo and Whitman 2009:691) As a demonstration, the reader is unlikely to be able to derive a moral difference between treating sickle cell and thalassemia. This distinction is hopelessly vague.

What is necessary to stop the slide down the slope from somatic gene

therapy to germline enhancement is a barrier on the slope that arrests the slide – what Buchanan and his colleagues label “moral firebreaks” (Buchanan et al. 2000:13-14). Whereas an ethical analysis for an individual or in a small group can depend upon identifying criteria that must be measured and weighed, policy in liberal democratic societies generally requires rigid limits that can literally be put to paper – a barrier.

Finally, a strong barrier separates two steps on the slope that cannot be made similar through either continuity or similarity vagueness. A barrier between 15 and 16 cannot hold because it will be easy to argue that 16 is just like 15. A barrier between sickle cell and thalassemia also cannot hold because these will be considered to be the same.

By the early 1970s the HGE debate had reached consensus on two barriers on the slope: the disease/enhancement and the somatic/germline. The former had diseases in the allowed area upslope of the barrier and enhancements in the not allowed downslope area. The disease/enhancement barrier was originally strong when the two were very distinct – when a disease was a “genetic disease” like sickle cell and the exemplar enhancement was intelligence. Those could not be made the same. However, when our knowledge of genetics expanded so that we understood genetic propensities toward disease and late onset diseases, the disease/enhancement barrier became subject to similarity vagueness. Was HGE to avoid a propensity to a non-genetic disease later in life an

enhancement? By 1997, Eric Juengst would write that “the treatment/enhancement distinction . . . often seems in danger of evaporating entirely under its conceptual critiques” (Juengst 1997:126). That barrier, while sometimes gestured to, has fallen over.

The other barrier had somatic upslope in the permissible region and germline downslope in the impermissible region. It has been very strong as it is very difficult to make the individual and the species – or a present and future person – the same. The somatic/germline barrier was put into policy and survived. But, two international summits of national science societies have called for dismantling the somatic/germline barrier, as have some prominent commission reports. It is in this context that Cwick proposes a number of distinctions, which I think can be used in small groups that allow deliberation, discretion and judgment. But, can they also be used as raw material with which to construct barriers below the somatic/germline barrier? Without constructing new barriers, we will slide as far downslope as our technical abilities will take us.

Let us start with a barrier built with Cwick’s distinction between “revising” and “correcting.” Above the barrier, and thus allowed, would be “correcting:” replacing an uncommon, typically disease-causing variant with a common variant. Below the barrier would be “revision:” to replace a common variant with an uncommon variant. In my book I create a barrier using a related distinction located on the slope at “most prevalent variant.”

Upslope and allowed would be to modify your offspring by giving them the most prevalent variant of a gene, downslope and not allowed would be inserting any variant but the most prevalent. The basic insight here is that giving someone the most prevalent variant would heal any of what are considered to be genetic diseases, but all of the advantage-producing variants are not going to be the most prevalent. Put differently, you could be made ordinary through HGE, but not special, and for people with genetic diseases, ordinary is good. In Cwick's terms, you could correct an unusual variant, which is what genetic diseases are, by placing the most prevalent. You could not revise by placing an uncommon variant, as He Jiankui tried to do with CCR5. I think that Cwick's distinction here is not only important for discretion and judgment, but it could be made into a barrier on the slippery slope for policy purposes. It would hold because it is not susceptible to the two vagueness mechanisms: there is only one "most prevalent."

Similarly, a very strong barrier could be built at Cwick's "transferring," where non-transfer modification is upslope and allowed, and transfer is downslope and not allowed. The terrain on the two sides of this slope do not suffer from continuity vagueness because the two acts are not described numerically. They do not suffer from similarity vagueness because taking from another cell cannot be the same as modifying what is in a cell. However, such a barrier would be of limited applicability – probably only useful for debates about mitochondrial DNA.

What Cwick calls “outcome” is “the total suite of possible effects on an individual’s physiology.” Extremes on this dimension would be a gene that impacts one narrow function (say, something in the eye), vs. a gene that appears to influence a wide range of bodily functions (like CCR5). This is an important insight for the application of discretion and judgement, but cannot be built into a barrier. To see why, let us pretend that we could count the distinct bodily functions impacted by a gene. A barrier built at one vs. more than one would be somewhat strong in that there is a strong cognitive difference in at least the West between one and more than one. A barrier built between three and four bodily functions would be hopelessly susceptible to continuity vagueness. Similarity vagueness would be even more of a problem, due to an inability to separate out distinct physiological effects. If CCR5 had two different impacts on the immune system and two different impacts on cognition, is that two or four impacts? In sum, it would not be possible to distinguish between acts on the two sides of the barrier. If decisions about HGE were made by a committee, this criteria might work, but it cannot be made into a barrier for policy.

Cwick’s category of “goal” (“intended changes in gene function”) is broader than the familiar version of goal we are all familiar with: the disease/enhancement distinction. Could we build barriers out of other distinctions in goals? Cwick identifies one barrier for which there would probably be widespread agreement: acts where the same physiological goal can be

achieved through established treatment would be below the barrier and not allowed.

This too would be subject to similarity vagueness. Someone will always be able to say that the HGE version produces a superior outcome because of ambiguity in measurement. Saying that the amount of symptom relief for sickle cell is the same for two modalities would be impossible. Compare this barrier to the clarity of “inherited” vs. “not inherited” in the somatic/germline barrier.

The somatic/germline barrier has served as a rule-like structure on the slippery slope for many decades with a very strong design – it is extremely hard to make the acts on the two sides the same. Many now argue that it should be taken down. For those who do not welcome the bottom of the slope, we need to identify ethical distinctions like Cwick has done, and then see if any of these can be built into barriers.

AUTHOR BIOGRAPHY

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