Commentary on A.W. Eaton's "A Sensible Antiporn Feminism"

ISHANI MAITRA
Departments of Philosophy and Women's and Gender Studies
Rutgers University, Newark/New Brunswick
430 Conklin Hall
175 University Avenue
Newark, NJ 07102
ishanim@andromeda.rutgers.edu

In "A Sensible Antiporn Feminism" (2007), A.W. Eaton develops a version of the feminist case against pornography that is designed to defend against certain misunderstandings and caricatures to which it has been subject.[1] Eaton tries to show that, properly understood, the feminist case can be a "sophisticated and reasonable" position (Eaton 2007, 675). She does this in at least two ways. First, she clarifies a central claim in the pornography debates, what she labels the *harm hypothesis* (about which more below). Second, she describes and defends a novel approach to the project of establishing the truth of that central claim, modeled on the methods of epidemiology. In so doing, Eaton successfully shows that certain objections to the feminist case against pornography are

simply wrongheaded. Additionally, she goes a long way towards spelling out the empirical commitments of this version of the feminist case.

The harm hypothesis (hereafter, *the HH*) is, for Eaton, the view that "pornography shapes the attitudes and conduct of its audience in ways that are injurious to women" (Eaton 2007, 677). A few clarifications will help explain how Eaton intends this claim. First, and very crucially, the HH is meant to be a causal thesis, in that it is concerned with the harms *caused* by pornography.[2] Second, for the purposes of this thesis, *pornography* is to include only "inegalitarian pornography", i.e., "sexually explicit representations that as a whole eroticize relations (acts, scenarios, or postures) characterized by gender equity" (Eaton 2007, 676). Finally, the HH is to focus only on "postproduction harms", i.e., those harms to women that are due to the distribution and consumption of pornography, not those that occur in and are due just to the process of producing pornography (Eaton 2007, 677).

Eaton's discussion of the HH lays the groundwork for careful empirical investigation of the effects of pornography. It makes clear how much remains to be investigated, but at the same time, opens up avenues of further exploration. But, as Eaton may well agree, there is more work to be done to fully develop the feminist case against pornography. Thus, my aim in this commentary will be to try to get clear about what Eaton has accomplished in this article, as well as what remains to be addressed.

The commentary proceeds as follows. In § I, I begin my discussion by focusing on the conception of causation that Eaton thinks can help clarify the HH. Here, I argue that, without further clarification, that conception cannot do the necessary work. In § II, I consider the place of the HH in the pornography debates. Here, I argue that at least some objections to the feminist case against pornography are left

untouched by the truth of the HH. Finally, in § III, I end by briefly considering the epidemiological model recommended by Eaton, and raising a question about how that model might be put to use in empirical investigations of the HH.

I

One of Eaton's noteworthy contributions in this article is to focus attention on the notion of causation at work in the HH. As she very rightly points out, some objections to this claim can be defused just be adopting a reasonable conception of causation.   The conception Eaton recommends is a probabilistic one, which she describes as follows:

x is a cause of *y* if and only if (i) *x* occurs earlier than *y* and (ii) the probability of the occurrence of *y* is greater, given the occurrence of *x* than the probability of the occurrence of *y* given not-*x* (Eaton 2007, 696).

On this conception, pornography can be a cause of harm to women even if not every consumer of pornography behaves in ways that harm women, as long as the consumption raises the probability of such harm. That seems like the right result.

I agree with Eaton that the probabilistic conception of causation is better than some of the competing conceptions, such as the "deterministic" conception she discusses (Eaton 2007, 695). But the probabilistic conception faces several difficulties as well. Some of these difficulties are not relevant in the current context, but others are. I mention two that are relevant below.

First, as Eaton notes, an important question in the pornography debates is whether consumption of pornography causes harm to women, as required by the HH, or whether consumption of pornography and harm to women by consumers of pornography are merely linked by some common cause (such

as the existing system of gender hierarchy). If the latter, then the HH would be false. Unfortunately, it turns out that this question is hard to formulate using the probabilistic conception given above, because that conception has trouble capturing the notion of a common cause.

To see this point, consider the following adaptation of an example of Eaton's. Suppose that smoking causes both caffeine-craving and (later in life) lung cancer, but that caffeine-craving does not cause lung cancer. Since the caffeine-craving precedes the lung cancer, and since the probability of lung cancer is greater given caffeine-craving than given lack of caffeine-craving, the probabilistic conception mentioned above says that caffeine-craving causes lung cancer. But that's wrong: *ex hypothesi*, caffeine-craving and lung cancer are merely linked by a common cause. So, this account does not capture our intuitive notion of a common cause.[3]

Second, many feminists who have been concerned about pornography think not only that pornography is harmful to women, but also that it is particularly efficacious in its harmfulness.[4] The latter claim – which I shall dub *the particular efficacy hypothesis*, or *the PEH* – plays an important role in the feminist case against pornography, in at least the following way. For those feminists who think that pornography should be subject to some form of state regulation, the PEH can help explain why that is so. The HH does not suffice here, for many things that are harmful, even severely harmful, nevertheless should not be regulated. (Consider, for example, some forms of lying.)[5]

At first glance, the need to make sense of the PEH seems to be another point in favor of the probabilistic conception. Perhaps we can say, given two causes of the same effect, that one is more efficacious than the other iff (roughly) the first raises the probability of the effect more than the second. Eaton flags the

fact that the probabilistic conception admits of degrees in this manner as another attractive feature of this conception (Eaton 2007, 697). On closer inspection, however, it turns out that, without further clarification, the conception makes counter-intuitive predictions on questions about comparative efficacy.

To see this, consider a well-worn example from the causation literature. Suppose that a match is struck, causing it to light. On the probabilistic conception, the striking of the match is a cause of its lighting, but so are certain background conditions, such as the presence of oxygen in the environment. That seems okay, but intuitively, we would like to be able to say that the striking is more efficacious than the availability of the oxygen. But it is not clear that we can get that result. At least on a fairly natural way of approaching the question, because there are ways of lighting the match without striking it, but no ways of lighting it in the absence of oxygen, the presence of oxygen makes a greater difference to the probability of the match lighting than its being struck.[6] As a result, the presence of oxygen turns out to be a more efficacious cause of the match lighting than its being struck.

For similar reasons, it may turn out, on the probabilistic conception, that certain background conditions, such as the very existence of women, or the existence of sex-drives, or even the existence of a system of gender hierarchy, are more efficacious than pornography with respect to harmfulness to women. Again, that seems counter-intuitive.

Nothing that has been said above shows the probabilistic conception to be fundamentally wrong. Rather, it merely shows that the conception, as presented above, is underspecified. At the very least, we need some way of distinguishing genuine cause-effect pairs from pairs that are merely linked by a common cause; and we need a better understanding of the notion of probability at issue here, so that – among other things – we can figure out how to use that

notion to compare the efficacy of different causes. Unless we fill in these details, reliance on this conception of causation may well lead us astray, especially in a case where the causal web is as complex and intricate as it is in the case of pornography.

II

As I've already implied, feminists have been concerned not only about the harms of pornography, but also with what, if anything, should be done to redress those harms. Some, but not all, have thought that some form of state intervention is necessary as a way of combating these harms. Eaton distinguishes several possible responses to the (alleged) harms of pornography, and asks whether the truth of the HH would be sufficient to license any of the forms of response that involve state intervention. She writes that "the answer depends *entirely* on just which sorts of harm pornography causes" (Eaton 2007, 691, emphasis added), and takes this as reason for feminists to carefully articulate which harms they take to result from pornography.

It seems to me right to suppose that whether the state should regulate pornography depends on precisely what harms it causes. If the harms are fairly trivial, for example, that is reason to think that the state should not regulate. Therefore, Eaton's work in describing a taxonomy that allows clear specification (and empirical testing) of the harms in question represents a significant contribution towards developing a plausible version of the feminist case against pornography.

At the same time, however, the thought that the question of state regulation is entirely decided by the sorts of harms caused by pornography seems wrong. In fact, a substantial part of the resistance to the feminist case against pornography rests not on the denial of the HH, or even on disagreement

about which sorts of harms are caused by pornography, but rather on disagreement about what response would be warranted by those harms.

One source of resistance to the feminist case depends on a particular conception of a principle of free speech. According to this conception, because pornography counts as speech in the relevant sense, state intervention in pornography cannot be justified merely by showing that it causes harms, even substantial ones. Rather, at the very least, we must balance any such harms against the harm that would be done to our commitment to free speech by increased regulations of speech. An extreme version of this conception might even regard pornography as political speech, and as such, deserving of the highest level of protection even compared to other categories of speech. At any rate, a defender of such a conception of free speech might well grant the HH, and even the sorts of harms at issue there, but nevertheless resist the call for state intervention.[7]

A second source of resistance to the feminist case focuses on the agency of the consumer of pornography. On this view, even if the HH is true, the responsibility for those harms – and the blame and sanction for them – should rest entirely with the consumers. A relevant analogy here might be a sort of argument that is frequently made against state regulation of gun sales, according to which such sales are admitted to be a cause of shooting deaths, but that is taken to not provide sufficient reason for further regulation. Rather, according to this line of argument, the responsibility for the deaths lies entirely with the errant gun owners, and it would be unfair to punish other gun owners for their malfeasance.

Note that I do not intend to endorse either of the lines of argument sketched in the last two paragraphs, nor to suggest that feminists have nothing to say in response. What I do want to emphasize, however, are the following two points. First,

and most importantly, the truth of the HH, and even agreement about the sorts of harms caused by pornography, are not sufficient to license any particular response to or redress for those harms. Second, though the HH is clearly controversial, there are other perhaps equally controversial issues in the pornography debates that don't turn on the truth of the HH at all. In fact, the persistence of the controversy surrounding some of these other issues gives me reason to wonder whether it is right to say that it is the HH that "lies at the center of the pornography debate" (Eaton 2007, 693).

III

Finally, I want to turn to an aspect of Eaton's discussion that I have only briefly mentioned thus far, namely, her recommendation that feminists use the methods of epidemiology to investigate the relationship between pornography and harms to women. Eaton argues that a reasonable version of the feminist case against pornography can model that relationship on those that hold between diseases and their causes. Accordingly, she suggests, the same methods that are used in epidemiology to uncover the causes of diseases can be usefully transferred to feminist research to examine the causes of harms to women, and in particular, to decide whether pornography is one of them.

This is an intriguing idea, and one that deserves further exploration. Eaton's discussion here is very suggestive, but it would be useful to spell out in greater detail precisely what the epidemiological methods are, and how they might be put to use in research into the effects of pornography. To illustrate the sort of detail I have in mind, let me end by asking a question about how epidemiological methods may be used to establish that a particular correlation is in fact causal. That is precisely the sort of thing at issue in debates over the HH.

Therefore, if epidemiology can offer some insight into how such debates can be settled, that would be genuine progress.

Eaton mentions several criteria that an epidemiologist might use to establish a causal relationship (Eaton 2007, 709-10). It seems plausible that genuine cause-effect pairs do satisfy these criteria. Unfortunately, it also seems that pairs of effects that are merely linked by a common cause can satisfy them as well. To see this, let us return to the example mentioned in § I, namely, smoking causing both caffeine-craving and (later in life) lung cancer. We can imagine that: caffeine-craving generally precedes the onset of lung cancer (temporality); there is a strong association between caffeine-craving and lung cancer (strength); increases/decreases in level, intensity, and duration of caffeine-craving are correlated with increases/decreases in the risk of lung cancer, for both are directly proportional to increases in level, intensity, etc. of smoking (quantal dose relationship and cessation data); these findings are replicable (consistency) and plausible given what is known (plausibility). That is to say, we can imagine that caffeine-craving and lung cancer satisfy all the given criteria, but that would not suffice to establish a causal relationship between the two. Then, what would supply the missing link?

Here is one idea about how to proceed. The trouble in this example is created by the fact (noted in § I) that caffeine-craving, though not a cause of lung cancer, is nevertheless probabilistically relevant to lung cancer, in the sense that the probability of lung cancer given caffeine craving is greater than its probability given lack of caffeine-craving. But, to put it very roughly, the probabilistic impact is really due to smoking: given smoking, there is no *further* probabilistic impact of caffeine-craving on lung cancer. That is to say, smoking *screens off* caffeine craving from lung cancer. And, to a first approximation, where there is such a screening factor, there is not a genuine causal relationship.[8] Both the probabilistic conception of causation and the criteria

mentioned in the previous paragraph run into trouble in part because they fail to make allowances for such a screening factor.

With this idea in mind, then, we can show that caffeine-craving and lung cancer do not constitute a genuine cause-effect pair by showing that smoking acts as a screening factor between the two. We can do this by finding a population of smokers, and showing that, *within* that population, caffeine-craving and lung cancer do not satisfy the criteria mentioned above. But – and here is the real worry – how would we transfer this idea to pornography research? Recall that the analogous question is whether pornography and harm to women are screened off from each other by some other factor, such as the existing system of gender hierarchy. To show that pornography and harm to women do constitute a genuine cause-effect pair, we would have to show that there is no troublesome screening factor. And to do that, we would need to show that there is *no population* in which pornography and harm to women fail to satisfy the criteria mentioned above. But that, to put it mildly, is a big task.[9]

References

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NOTES

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[1] By "the feminist case against pornography", I have in mind the various feminist arguments to the effect that pornography harms women. There are several such arguments, proceeding from quite distinct premises. Here, I shall say that each such argument corresponds to a version of the feminist case. The version on which Eaton focuses is a kind of *causal* argument, i.e., an argument to the effect that pornography *causes* harm to women.

[2] See note 1.

[3] One way to address this problem would be to use the idea of screening. For more on this idea, see the final paragraphs of this commentary. For an excellent overview of this and other related difficulties for the probabilistic account, see Hitchcock (2008). There is a vast literature on the issue of how to identify common causes, but see especially Glymour *et al* (1987, 1991).

[4] Eaton notes the latter claim, which she regards as a premise in a feminist argument for the HH (Eaton 2007, 683).

[5] The PEH might play another role as well. For those feminists who think that non-pornographic inegalitarian representations can also be harmful, but that pornography is more deserving of feminist concern, the PEH may be used to explain the difference.

[6] Slightly more formally, here is the idea. Let $Pr (A \mid B)$ be the probability of A given B. Let L be the match lighting, S the match being struck, and O the presence of oxygen in the vicinity. Then, to compare the efficacy of the two causes, we can compare $Pr (L \mid S \ \& \ O) - Pr (L \mid S \ \& \ not\text{-}O)$ with $Pr (L \mid O \ \& \ S) - Pr (L \mid O \ \& \ not\text{-}S)$, where the first is meant to be a way of measuring the difference the presence of oxygen makes to the probability of the lighting, and the second a way of measuring the difference the match's being struck makes to the same probability. But $Pr (L \mid S \ \& \ O) = Pr (L \mid O \ \& \ S)$, $Pr (L \mid S \ \& \ not\text{-}O) = 0$ since the match cannot light without oxygen, and $Pr (L \mid O \ \& \ not\text{-}S) > 0$ since the match can light without being struck. Thus, $Pr (L \mid S \ \& \ O) - Pr (L \mid S \ \& \ not\text{-}O)$ is greater than $Pr (L \mid O \ \& \ S) - Pr (L \mid O \ \& \ not\text{-}S)$.

Note that I am not suggesting that this is the only way of measuring the difference in efficacy between the two causes. Rather, my point is that until we have some principled explanation of what is wrong with this reasoning, we do not understand the underlying conception of probability well enough to rely upon it.

[7] A particularly striking instance of this argument was offered by Judge Frank Easterbrook, in the decision that struck down the Indianapolis anti-pornography ordinance. Judge Easterbrook accepted the claim that, for women, pornography "leads to affront and lower pay at work, insult and injury at home, battery and rape on the streets", but at the same time concluded that this "simply demonstrates the power of pornography as speech" (*American Booksellers v. Hudnut*).

[8] Slightly more formally, here is the idea. Let L be getting lung cancer, C be craving caffeine, and S be smoking. Then, to say that smoking screens off caffeine-craving from lung cancer is to say that $Pr (L \mid C \ \& \ S) = Pr (L \mid S)$. Intuitively, this just says that given smoking, caffeine-craving has no further probabilistic impact on lung cancer.

For more on this notion of screening, and further refinements on the use of this notion in a probabilistic conception of causation, see Hitchcock (2008).

[9] I would like to thank Jennifer Saul and Sally Haslanger for helpful comments on this commentary.