When considering how we need to think causally in a public health setting, the salient points involve the usefulness of the information for prediction and possibly intervention. Thus, the usefulness and value of the long, patient description ultimately derives from how well people are convinced that this information provides a basis for some kind of intervention or prediction. Although much has been written about causation, we may, as Sosa and Tooley [12] and Cartwright [1] argue, never be able to have anything but a very specific "singularist" sense of causation; i.e., a sense of causal thinking that is not capable of being generalized with rules or methods, but is insurmountably contextual [8]. Far from being an obstacle, such an approach allows for a great deal more clarity regarding the interpretation of epidemiological research.

As mentioned above, this approach appears to be in line with the work of Parascandola and Weed [5] when they point out that probabilistic models of causation are essentially more flexible than deterministic approaches. Whether discussing determinism in causation or more humble, but no less important, issues about causal tools, there is no need to worry about generalizing the discussion. At best, these tools may act as guides that may make specific research more useful for the purposes of intervention or prediction, without providing access to posited objective causal relationships. That certain contexts, such as legal definitions of what constitutes cause, may force a specific notion of cause to come into play; e.g., as demanded by a rule making body like the office of the Surgeon General, or a Judge, provides no added significance to saying that X causes Y. Clearly, certain contexts may demand a very specific use of causal language. Such technical usage of "cause" etc., perhaps in a deterministic way, as might be demanded by a legal process, will occur in a specific setting. For example, a question such as "how much of the paralysis was caused by the faulty tires?" may be unambiguously germane for the purposes of adjudicating a tort case in which a specific notion of cause is introduced and accepted by all parties. Perhaps such uses of "cause" etc. need this level of description.

In another related example, one might ask, what of the situation when undertaking a marginal structural model (MSM) analysis in which the research differentiates between casual effects and mere effects? Is this not a justified use of cause? The answer is a qualified yes, because such a use is highly defined and limited in its meaning. For the sake of MSM analysis, a causal effect is differentiated from a non-causal effect as a function of how well relevant (a judgment call as to what is relevant) confounders and indirect effects are included in the model. The more complete the more "causal" argument is in regard to alternative hypotheses, the better – i.e., more causal – the model. Crude effects, on the other hand, are those that have included minimal, if any, control of relevant confounding and inclusion of indirect effects. There is no hard-and-fast test of when a mere effect becomes a causal effect. This assessment is up to those doing the research and those who assess it. Thus, what a causal effect seems to actually stand for is a more rigorous analysis. This rigorous analysis will hopefully yield more useful information than a less rigorous analysis in regard to intervention and/or prediction. The work here is not in the naming of something as causal, but in the actual rigor of the analysis. The causal language is thus a shortcut that denotes such rigor. Any foundational causal claims are, in fact, the result of circular reasoning. The main point here is that highly contextualized technical/statistical uses of causal language are not the same as making general causal claims about, for example, smoking causing lung cancer. We are always forced back into asking "under what conditions?"

Conclusion

We have argued that saying smoking causes lung cancer is either an empty or a redundant statement from a scientific perspective; implicitly or explicitly it belongs in the realm of health policy. Epidemiologists need to be constantly aware of the limits of causal language and also of the demands of making explicit causation claims.

When attention is not sufficiently paid to properly contextualizing causal claims and loosely using causal language, there are potentially real world consequences. For example, cigarette company lawyers were often heard to say that the case has not been definitively made that smoking caused lung cancer. They said this knowing full well that in the real world, there is nothing that can be definitively claimed. Nevertheless, this should not, in any way, be an obstacle for epidemiologists in the role, for example, of expert witnesses, who put forward the strongest possible account of a given research program, such as one that links smoking to lung cancer. Indeed, the best we can hope for here is to make the most compelling case, the most persuasive account, and hope that it will be more, rather than less, convincing. This is not a nihilistic throwing of the baby out with the bathwater. Not being able to say something is definitively causal does not mean that extremely useful information is not available; it is simply not available in the way that is traditionally demanded by this specific research community.

And here we must emphasize that there really is something different about implicit and explicit causal arguments. We can easily defend the claim that a ten-fold risk was found for two pack-a-day smokers compared to those who did not smoke. We simply cite the methods and research findings. We cannot defend the additional explicit claim that this is a causal relationship in the same manner. In fact, trying to justify such a claim results in cir-