ers than nonsmokers," or "Women cigarette smokers experience an increased risk for subarachnoid hemorrhage."

These statements can be contrasted with others such as "It is also more prudent to assume that the established association between cigarette smoking and coronary disease *has causative meaning* than to suspend judgment until no uncertainty remains" (p. 13, our emphasis). The discomfort on display in the last sentence is clear: responsible prudence apparently dictates the use of explicit causal language even though the findings, on their own, i.e. "established association" can be taken to be a meaningful statement of cause (more on this below). There is, further, an implied and somewhat ambiguous assumption that complete certainty, although not available here, is theoretically achievable, yet not needed, for causal information to be conveyed. Although this latter point is one with which we can strongly agree, we nevertheless argue that worrying about complete certainty is not useful for the simple reason that this level of certainty is not available. Indeed, if the information obtained from the "established associations" allows for effective prediction and/or intervention, then it is not clear what other information or language would be needed in terms of causal argumentation. Thus, the above remark seems to be intended to justify and/or motivate health policy (not necessarily a bad thing but off-point for the purposes of this discussion) rather than to improve our causal understanding of the relationship between smoking and coronary disease.

One problem is that, as Hume described and the authors confirm [2,4], while the use of causal language can be psychologically compelling, the causal nexus will never lend itself to be empirically detected or generally proven. Particularly in regard to the last statement from the Surgeon General's report, uncertainty will always remain. This uncertainty can be thought of as supporting a more probabilistic approach towards causation [5]. Parascandola and Weed point out that probabilistic models of causation are essentially more flexible than deterministic approaches. Their argument centers on the idea that since it is impossible to ever know all the constituent elements in a deterministic causal model, why not allow for some level of probabilistic ambiguity? The need to say anything definitive about this dichotomy, however, is not in the realm of the strictly scientific, nor is their discussion of what constitutes science and what constitutes public health policy, and why different notions of science might apply to the two. Although these are different contexts, the inability to "prove" or objectively "see" causation, however, still applies to both.

More importantly, a fundamental inability to determine cause is not necessarily a serious concern for epidemiologists because causal information can still be conveyed without getting bogged down in such epistemological and metaphysical issues. Thus, the struggle to develop a causal context relating tobacco to illness in the new report displays a level of anxiety that would be unnecessary if a more pragmatic approach toward causal information were used.

A short caveat on realism in science

We are not in this essay attempting to revisit the long-standing debate between realism and pragmatism in science. (A relatively current, although ultimately unconvincing, exploration of realism in epidemiology was discussed by Renton [6].) For the sake of making our argument, we accept the natural ontological attitude (NOA) developed by Arthur Fine [7] as being closely in line with our approach toward thinking about causation. His argument is, in fact, a generalization of what has been discussed here. That is, he asks what is the efficacy of having something be considered "real" in the same manner as something being determined as "causal." He is interested in the ability to manipulate the world, to predict and intervene. Being able to determine something as real, in a metaphysically emphatic sense, something he and we doubt can ever be accomplished, is beside the point when dealing with the actual process of doing science.

An alternative approach toward causal thinking

Once a famous epidemiologist, K, stated that causation is easy, "smoking causes lung cancer," adding a sarcastic "it's obvious" shrug of his shoulders to emphasize his point. This was in answer to a naïve query regarding how certain we could be about ever saying that X causes Y. K's response was a catalyst to our interest in epidemiologists' use of causal language, both in the day-to-day workings of any particular epidemiological project and in the more extended long-range meta-discussions bearing on causal thinking in epidemiology. It is our suggestion that K's remark, while presumably intended to lend scientific weight to the findings that he had in mind, might rather have done them a disservice.

What, if anything, would underwrite an explicit causal claim, in this kind of context? We shall consider an admittedly not uncontroversial discussion of causality by G.E.M. Anscombe [8]. She identifies a claim shared by received philosophical views about causal connections as being either a kind of *necessary connection* between events, or as *instancing an exceptionless generalization* – a universal claim – saying that a certain kind of event will always be preceded by certain others: "If an effect occurs in one case and a similar effect does not occur in an apparently similar case, there must be a relevant further difference." [8] (p. 88)