

Supervenience and the Public Health Standard for Psychoactive Substances

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Abstract: This paper uses Geoffrey Rose’s theory of population health change to identify that part of lack of resolution of the harm reduction debates emerges from the philosophical question of the possibility of group-level causation. Group-level causation, otherwise known as the supervenience of higher levels onto lower levels, has been a core matter of philosophical and ethical debate. This paper argues that there, the public health standard depends crucially on the contextual environment rather than the individual product’s intrinsic properties. Interpretation of the benefits or the harms of e-cigarettes or other harm reduction products depends on the often unstated assumptions of the potential policy and cultural environments in which e-cigarettes exist, as well as the separate debate over the absolute or relative level of harm of e-cigarettes. Unfortunately, most of the research conducted will not contribute to our understanding of the potential impact of e-cigarettes until we focus on the potential changes to the social and political environment.

Keywords: public health; populations; causation; policy; e-cigarettes



Citation: Chaiton, M. Supervenience and the Public Health Standard for Psychoactive Substances. *Psychoactives* **2023**, *2*, 194–200. <https://doi.org/10.3390/psychoactives2020013>

Academic Editors: Ricardo Dinis-Oliveira and Matteo Marti

Received: 15 November 2022

Revised: 31 March 2023

Accepted: 24 May 2023

Published: 31 May 2023



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1. Introduction

In the United States, the Food and Drug Agency uses what it calls a “public health standard” to judge the overall value of tobacco harm reduction products. Instead of the traditional “safe and effective” standard used by the FDA to regulate medical products, the public health standard for tobacco products is defined as “a flexible standard that focuses on the overall goal of reducing the number of individuals who die or are harmed by tobacco products” [1]. The language is intentionally broad, focusing on protecting the health of the population as a whole, e.g., “Prioritizing Population-Level Effects, Not Individual Harms to Users”. Researchers and advocacy organizations have called on the FDA to ensure that it uses this public health standard [2–5].

This public health standard presents the compelling possibility of a scientifically driven, evidence-based answer to the harm reduction dilemma that sidesteps a number of knotty ethical difficulties. The FDA’s Public Health Standard relies on a conception of a public health wherein the characteristics of the “public” can be identified and measured separately from the behaviour of individuals. However, it may not be true that research evidence based on individuals within a particular context provides conclusive or meaningful evidence towards this standard. On the other hand, investigating the social and political processes that influence Public Health can provide knowledge that does inform the standard without relying on ethical or other assumptions.

In this paper, I argue that the public health standard does in fact offer this possibility of a scientific solution to harm reduction, but in order to accept this standard, we must grapple with a philosophical issue about the nature of causation. In order for this criterion to be meaningful, it is necessary to provide a clearer clarification of the assumptions that are involved in defining a problem as one that pertains to public health, in addition to placing a direct focus in research on investigating those assumptions themselves.

The concept of “supervenience,” which refers to the capacity of a higher level of organisation to have a causal influence on a lower one, is at the heart of this philosophical question. The concept of supervenience is primarily investigated in the philosophical literature in relation to the question of whether or not the “mind” possesses characteristics that are separate from the biological matter that makes up the body. This study’s primary focus is on determining whether group- or population-level organisations (i.e., the public) can have emergent characteristics that are distinct from those of the individuals that make up those organisations. The key messages of the paper are (a) that supervenience is a difficult issue and there is disagreement about whether it is possible (b) that public health requires the belief in supervenience, and (c) that supervenience requires a different level of evidence beyond that of individual-level risk factors that are commonly assessed in individual-level studies (including longitudinal cohorts and randomized controlled trials).

2. The Debate over Supervenience

One way of understanding supervenience is to examine its counterpart of “upward” causation. Upward causation occurs where higher level units of organization are caused by their component parts. Hence, the whole equals the sum of its components. That is, we can add up, or extrapolate from our knowledge of the parts, in our case, individuals to know and predict the sum of the whole. This mode of thought suggests that causation can operate upward from the smaller unit to the larger. This direction of causation is intuitive and obvious. A population is made of people and thus it seems clear that any population effects would be the effect of the sum of behaviour of those people. The time shown on a watch is the effect of the gearing of the watch; the phenotype of an individual is determined by their genes; the mind is composed of the neurons in the brain. This mode of upward causation is not controversial.

On the other hand, there are those that believe that the whole can be more, or different, than the sum of its parts. This mode of thought suggests that there is real group-level causation that works at the level of the group, not the individual. Forces affecting the group cause changes in the individuals, but changes in the individuals do not necessarily affect the forces affecting the group. This causal mechanism whereby the group level “supervenes” on the individual level is not intuitive to everyone, and it continues to be an item of philosophical and ethical debate. In evolutionary biology, for instance, debates regarding whether altruism is a true group-level cause (animals sacrifice themselves for the good of the group) or an individual-level one (animals sacrifice themselves because it is good for their own genes). Furthermore, phenotype is considered to supervene on genotype in that the classic examples of convergent evolution are also examples of how the environment supervenes on genes. For instance, organisms without predators become larger, organisms limited by food tend to become smaller, organisms that live in cave pools tend to lose their sight [6–8]. In each case, however, the actual genetic changes are diverse. The environment determines changes, but what exactly those changes are and how they are coded by genes is widely varied. The environment determines the “what” and the individual genes determine the “how”. This implies that we can predict what changes to the phenotype of an organism will happen if we make changes to the environment even if we do not know what the genetic changes may be. Conversely, it suggests that changes to a genotype cannot predict long-term phenotype unless we know or hold constant the environment.

3. Why Does Public Health Require the Belief in Role of Supervenient Causes?

Although there are many different definitions of public health, the pre-eminent definition and one that is consistent with the description of the FDA public health standard implies a role for supervenient causes, i.e., the “public” in public health. This conception of the importance of the public is well described by Geoffrey Rose [9]. As described by Rickles in the book *Public Health*, Rose “takes ‘public’ to refer to a population of individuals

so that public health refers to the collective health state of the population. In other words, the basic system of public health is the population rather than the individual" [10].

However, the public health definition of the public or population is considered to be more than simply a collection of individuals but a thing in itself. Rickles says the following of public health: "This system, like an individual, can have a health state that can be measured, tracked, compared to other systems, and modified". Rose, for instance, claims that the incidence rate is a feature of a population and not that of individuals [9]. Furthermore, the causes of disease in a population are distinct from the causes of individual harm—that the determinants of health within a population are different than the determinants of health between populations. Rose states that these are two separate etiological questions. In his example, the following questions are presented: why do some individuals experience hypertension? In addition, why do some populations have much hypertension, and in others it is rare? For public health, populations have their own health states with their own etiological causes. Healthiness is a characteristic of the whole population, and not simply of its individual members.

Rose's insight is that risk factors are not necessarily associated with the health of a population. He defined population health based on overall relative incidence rate of a disease between population. For instance, for hypertension, men are at higher risk of the disease than women, but men in Country X may have lower risks of hypertension than those in Country Y. The former is related to individual health, and the latter is related to population. It would not be possible to know the incidence of hypertension in Country Y by extrapolating the information from Country X. It is possible to assume at Country X has the same distribution of risk factors as Country Y. We often see that some differences between countries are due to different distributions (e.g., standardizing for age, sex, etc.), but some differences can remain, assuming a belief in the supervenient causes.

Rickles identifies Rose's position in the following way:

"Rose adopts (though doesn't argue directly) for a holistic position well opposed to methodological individualism. This, he says, provides the 'sociological basis' for his idea of a population-level prevention strategy [10]. As he notes, the view has noble ancestry that he traces to Durkheim. In the health context, it involves the thesis that "healthiness is a characteristic of the population as a whole and not simply of its individual members" [10].

A further consequence of supervenience means that It is not sufficient to regard the aggregation of individual-level results to predict changes in the health of a population. The logic of aggregate measures is based on the ability of a system to be determined solely by the health of its individual parts and the ability to use the information of the individual parts to predict the system itself. Individual-level data can provide information about the population within a particular context or environment, but it cannot provide prediction of change without understanding the forces affecting the population context.

Villanti argues that the studies of the harms to individual users—individual-level studies—are less informative to the public health standard than "representative" surveys—population-level data [1]. Villanti describes that these nationally representative surveys provide data on trends and associations that might be used to provide rigorous evidence to inform the public health standard. Villanti's example of representative surveys is used as an attempt to measure those population characteristics, but still does so by measuring the health of the individual members within a particular social context. It assumes a lack of supervenience in the population levels and implicitly suggests that the population level results are determined in some sense by the states of the individuals. By measuring individual-level phenomena and summarizing them assumes an individualistic doctrine whereby the sum of individual behavior is equal to the whole of the health of the population. As we have seen, it is insufficient to use individual-level measures to predict future changes in the public. The forces affecting population health are independent of the relationships at the individual level, and so the questions assessing the former do not inform the latter.

This distinction between individual-level and population-level effects has led to a number of results that seem like paradoxes. For instance, on the individual level, people

with lower nicotine dependence are more likely to quit smoking. It would then seem obvious that we can predict that as more people quit smoking on the population level (i.e., the prevalence of smoking falls), the level of nicotine dependence in the population will rise because those with higher nicotine dependence are left in the population. This is the extrapolation of individual effects to the population—the assumption that the change in prevalence is caused by people quitting. However, consistently, nicotine dependence remains constant or even falls in the population as prevalence declines. This apparent paradox can be explained by supervenience: the forces that are causing the decline in smoking prevalence at the population level are also causing a decline in nicotine dependence in the population. The fact that nicotine dependence is causally associated with quitting at the individual level does not inform predictions of changes at the population level.

Similarly, longitudinal studies show that e-cigarette use is associated with smoking initiation at the individual level, but changes in e-cigarette use have been associated with decreases in smoking initiation. It is not a paradox to be explained—both of these have the possibility of being true causal effects because the effects on individuals are independent of the effects on populations. We also know from randomized clinical trials that e-cigarettes can be an effective smoking cessation device, but it does not follow that the availability of e-cigarettes causes declines in smoking prevalence. The effects on individuals are independent from the effects on populations.

However, individual factors can depend explicitly on the population-level factors/context. Individual-level longitudinal cohort studies, whether or not they are controlling for demographics or population-level factors, provide individual-level outcomes, not population-level ones. For instance, in the current environment in North America, age has a strong effect on e-cigarette use—the youth are much more likely to start using e-cigarettes. In a different environment, one could imagine the reverse, where adults would be much more likely to use e-cigarettes. Identifying the effect of age on individuals in the first context does not apply to the effect on a population. A similar observation can also be made in regard to the effects of other demographic characteristics such as race, gender, or socio-economic status.

4. What Is Evidence Required for the Public Health Standard?

As Rickles wonders, how does one measure this state if not by measuring the states of the individuals and aggregating the results? [10]. If we follow a public health standard, then we must examine the context in which the product is introduced. This context is complex and multifactorial and we must directly measure this context or make assumptions about that which we cannot measure. The context is not secondary but the thing itself of interest to the public health standard: what policies we make concerning e-cigarettes and cigarettes, what are the social norms around use, etc., are more conclusive than the individual-level data.

There have been attempts to integrate context into individualistic models. For instance, Pacek et al. (2019) develops a multilevel framework of product, person, and context/situational factors where interactions between the levels results in dynamic complementarity [11]. Fundamentally, however, the role of individual-level risk factors is that these are determined by the population context. Attempting to model population health through individual risk factors is akin to modelling the orbits of planets using epicycles—adding complexity because we are missing the essential models. If we want to understand and predict the effect of population-level forces, then we need to study those forces directly. The information gained from individual-level studies is valuable—the magnitude of biological health effects for a given dose of a particular product, for instance—but the population effects are not extrapolatable from these individual data. Multi-level models can also help us interpret better the effects on individual health, but this is still limited in the ability to predict changes from one environment to another in which a novel intervention will be implemented.

The implication of downward causation directly impacts the interpretation of analyses. Each time the individual-level effects are extrapolated, the contextual or group-level effects must be assumed either explicitly or implicitly. These assumptions do vary. Some assume that the effect of e-cigarettes will result in a status quo environment (i.e., all else being equal) while others assume that the introduction of e-cigarettes will have various group-level effects (e.g., on normalization of nicotine). Some argue that this extrapolation must occur in a market-based competition, while others argue that the extrapolation depends on government interventions, and so forth.

There is the potential for e-cigarettes to facilitate continued or increased levels of smoking and there is the potential for e-cigarettes to facilitate the process of quitting smoking. Given that we assume that the mechanism of addiction to e-cigarettes is similar to that of cigarettes, e-cigarette could in practice or in theory be used to supplement continued smoking (dual use), provide a gateway to smoking through the introduction of nicotine, provide an alternative to starting smoking if people start vaping and never start smoking, and provide a means of smoking cessation. There is theoretical potential for e-cigarettes to act on individual behavior in all these ways and evidence that it does do so, at least in some populations. The impact depends very little on the product itself, and overwhelmingly on the context in which it might exist.

Unsurprisingly, then, there have been calls for more research, but we are looking for answers that will not be able to come from research on individuals within a particular context [12]. Yet, even with time and funding, the answer to the studies will not settle the debate unless the context is addressed. Every result on an individual level can be explained or explained away by auxiliary hypotheses either supporting or disputing the context: “If only there was more advertising. If only there was appropriate regulation. If only tobacco control would support more. If only. . .”.

The question then for e-cigarettes and harm reduction more generally is whether or not we can use the results from our individual-level studies to predict population-level effects in different contexts. The answer depends on whether one subscribes to a belief in supervenient group causes. We are comfortable with supervenience until it comes into conflict with traditional causal direction. It is hard not to assume that acting on the individual level will necessarily affect the population, but if the major forces are at the population level, there is no particular reason to believe that what happens to individuals can predict changes to the population level.

There is value to individual-level research. First, within a particular context, identification of risk factors is useful for clinical treatment, targeting and tailoring. Second, the absolute magnitude of individual health effects can be determined. Neither, however, is necessarily predictive of changes to population health under a different context. They are simply answering different questions than those required by the population health standard. This disconnect between individual-level causes or risk factors and population effects helps identify the issue with most of the research on harm reduction. Most current research focuses on the effect of a product on individual behaviour and individual health. While this research accurately quantifies the individual health risks of the product, it cannot be extrapolated to provide population-level estimates that can inform a utilitarian calculus of costs and benefits under a different policy context. Yet, this extrapolation occurs mostly without acknowledgement of assumptions in those extrapolations or without acknowledgement that assumptions are malleable and not dependent on the individual-level effects.

This issue of extrapolation from the ways in which effects in individuals affect changes in populations is broader than the population health standard for tobacco. Indeed, it affects our ability to ascertain the impact of policies affecting any psychoactive substance. The impact of cannabis on dependence, for instance, may be different in legalized compared to criminalized policy contexts. Research on psychedelics has noted the effect of setting, but has yet to grapple fully with the effect of significant changes to the policy environment on public health effectiveness.

To study population-level causes requires a “population of populations”. The groundbreaking International Tobacco Control Policy Evaluation Surveys (ITC), for instance, are premised partly on this conception [13]. ITC surveys use the same designs in multiple countries to evaluate the impact of interventions over time [14]. In this design, it is the between-population differences that reflect the causal effect of the intervention, not the individual differences. This design functions as a quasi-experimental design controlling for the two aspects of changes in differences in environments: the environment before the intervention compared to the environment after the intervention, and the environments between populations. For instance, ITC demonstrated that the salience of health risks increased with the implementation of pictorial health warning after the introduction of pictorial warnings in England and that this effect was larger than increases in saliences in countries where pictorial warnings were not introduced [15–17]. We can only examine the true effect of an intervention in a population if we have another population for comparison. We will only be confident about the effect of a harm reduction product if it has been implemented in multiple countries with appropriate population level controls unless we can improve our ability to understand the environmental and social context factors that act to determine the success of an intervention.

That is, we cannot be confident about the impact of a population intervention until that intervention is implemented in a population and compared to another population. We can develop strong theoretical reasons to believe that an intervention may be effective, and we can test our assumptions through simulation models [18,19], but fundamentally, until an intervention is implemented, we cannot be sure of the effect. This corresponds to the relationship of a pharmaceutical agent and individual health. To be confident of the effect of the agent, an understanding of the chemical component is insufficient, and we must test and evaluate the effect in vivo.

5. Conclusions

The philosophers of science have much more rigorous debates about the nature of supervenience, but Public Health assumes the existence of independent population-level (upstream) causes. The FDA has correctly set up a standard for evaluation based on public health, but reaching that standard may be more challenging than we realize [20]. Our methodologies for studying individual effects are much more developed with more advanced methods for identifying and addressing confounding. We need to address the challenge of improving our ability to study population effects, and the first step is the recognition and acceptance of real, independent effects on the population level.

Funding: This research received no external funding.

Data Availability Statement: No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Conflicts of Interest: The author declares no conflict of interest.

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