When are complex interventions ‘complex’? When are simple interventions ‘simple’?

Mark Petticrew

Professor, Social and Environmental Health Research Unit, London School of Hygiene and Tropical Medicine (LSHTM), UK

Correspondence: Mark Petticrew, Social and Environmental Health Research Department, Faculty of Public Health and Policy, LSHTM, London, UK, WC1E 7HT. e-mail: mark.petticrew@lshtm.ac.uk

The issue of complexity, and how one evaluates complex interventions, remains a key one for health service and public health researchers. The underlying question—what makes complex interventions complex?—is particularly interesting, and it has implications for the sorts of research we do. The MRC guidance provides one detailed answer: complexity resides (among other things) in the number of interacting components; the number and difficulty of behaviours required by those delivering or receiving the intervention; the number of groups or organizational levels targeted by the intervention; the number and variability of outcomes; and the degree of flexibility or tailoring of the intervention permitted. Other definitions also emphasize other aspects including the degree of flexibility and non-standardization which complex interventions are subject to; as explained by Hawe et al. (2004), complex interventions are non-standard, having different forms in different contexts, while still conforming to specific, theory driven processes.

There are many other definitions of complex interventions. These tend frequently to emphasize that they have multiple interacting components, and non-linear causal pathways. Complex interventions are often contrasted in the health literature with ‘simple’ interventions, in particular medical interventions, which are generally seen as having simple linear pathways linking the intervention and its outcome.

Such definitions are important because they have direct implications for the sorts of evaluative and other research we do to investigate complex interventions. If researchers see ‘simplicity’ in an intervention, then they may be more likely to argue that randomized controlled trials (as opposed to other sorts of research) are feasible and appropriate. However if they see complexity (non-linear pathways, multiple synergistic components, feedback loops and so on) as the key features, then by implication other types of research may be necessary for illuminating those complex processes.

Underlying most definitions is the assumption that ‘simplicity’ and ‘complexity’ are inherent characteristics of interventions. However, there is another possibility: that in fact there are no ‘simple’ or ‘complex’ interventions, and that simplicity and complexity are instead pragmatic perspectives adopted by researchers to help describe and understand the interventions in question. After all, on close examination most interventions, including medical interventions, reveal themselves to be complex. Despite this, it is often helpful to treat them as simple, in order to answer appropriately simple questions.

Many interventions may be equally open to either simple or complex forms of analysis. Take a complex public health intervention as an example—urban regeneration programmes. These programmes of structural and social change in urban environments have been often used to improve health and reduce health inequalities via the socio-economic determinants of health. They usually involve complex packages of ‘components’, such as employment, education, income, crime and housing interventions. Research conducted from a complex perspective might consider how and whether these components work individually and together. It might consider the synergies between them, phase changes and feedback loops, and the interactions between multiple health and non-health outcomes, as well as the process by which these components bring about change in communities.

However, such research could also adopt a simpler perspective, by ‘unpacking’ the complex intervention into its component parts, and examining the effects of single ‘components’, and perhaps focussing on questions of effectiveness alone. Complexity can therefore be simplified for the purpose of assessing outcomes. A simpler approach might involve simply asking whether investment in urban regeneration as a whole ‘package’ is associated with improved health, or not, and many evaluations have been done this way. While the intervention remains the same, the researcher’s perspective on the intervention is different, and the research methods that she/he will adopt will accordingly differ. Thus, simpler and more complex perspectives on the same question will yield different, and probably complementary, answers. A simpler perspective may focus on individual-level outcomes alone, whereas a more complex perspective may focus on outcomes at different levels—not just at the level of the individual, but also at the family, and community-level. These different analyses may be more or less useful to different types of user. Some users may want to know about outcomes; some are more interested in processes; many want information on both aspects. Some researchers and users of research require simpler answers, while some want more complex explanations.

The world therefore does not after all divide up easily into ‘simple’ or ‘complex’ interventions. Instead there are simple and complex explanations of those interventions. The answer to the question posed in the title of this editorial is therefore a pragmatic one: when are complex interventions complex? When it is helpful to see, and analyse them as such. The perspective that is adopted informs the type of analysis that is performed, and the explanation that is eventually produced is dictated by the original research question—whether it is about components, systems, processes, outcomes or about simpler pathways and outcomes.

In analysing complex interventions it may often therefore be helpful to start not with definitions of ‘simple’ and ‘complex’—as there are few (if any) truly ‘simple’ interventions—but with the research question. Not every complex intervention requires a complex analysis—unless the research question demands it.
Acknowledgement

This was originally presented at the 17th Cochrane Colloquium in Singapore, October 2009 http://www.cochrane.org/multimedia/multimedia-cochrane-colloquia-and-meetings/colloquium-singapore-2009.

References


doi:10.1093/eurpub/ckr084

Psychosocial environment and health

The assumption that there is a direct influence of the psychosocial environment on health is of increasing interest in partially explaining health inequalities. Recent works have shed light on mechanisms linking the psychosocial environment and biology. As an example, in 2010 a special volume of annals of the New York academy of sciences focussed on 'The biology of disadvantage: socioeconomic status and health'. This volume summarizes evidence from the literature regarding the strong link existing between environment, in the broadest sense of the term, and biology.

Biological mechanisms

Taking the example of cancer, several biological mechanisms are involved in cancer development and progression, which occurs over a chronic time period, and specific risk factors have been identified (i.e. alcohol, tobacco, chemicals agents). Causal relationships between environmental factors and biological mechanisms involved in cancer have been well demonstrated in animals. In human, evidence is more difficult to obtain, therefore available studies are sparse and mainly based on correlations. However, an increasing literature suggests links between psychosocial factors, like stress, depression or social isolation, and cancer progression through activation of neuro-hormonal systems and epigenetic mechanisms. Evidence of the role of psychosocial factors in cancer initiation is more equivocal, although recent articles linking environment, DNA hypomethylation and cancer development suggest that the environment may modify DNA and gene expression.

Biological mechanisms and the reduction of social inequalities in cancer

If the psychosocial environment can influence cancer progression, and even the onset of disease at a given level of exposure to a risk factor, this implies that people exposed to a stressful psychosocial environment are at a higher risk of developing cancer at an earlier age and more rapidly. Furthermore, if the way in which the environment is embodied is more biologically damaging for people with low socioeconomic status (SES), this might, in part, explain social inequalities observed in cancer incidence and mortality. In fact, it has been shown that measures of allostatic load, a measure combining biological parameters of multiple regulatory systems (cardiovascular, metabolic, inflammatory parameters), follow a SES gradient. This may have consequences for epidemiological research and public health policy aiming at reducing health inequalities in cancer.

In epidemiological research, the assumption that some individuals are biologically more at risk of developing cancer raises questions about the way in which risk factors are considered. Classically, in cancer their effect is related to dose and duration, like for alcohol or tobacco consumption. For a fixed dose and duration, the effect is assumed to be the same in each group of a given population. If we assume that people who live in a poor psychosocial environment are at risk of biological damage and therefore more susceptible to developing cancer, the risk associated with smoking could be higher for these individuals, as is the case for individuals with a lower SES.

At the individual level, the impact of risk factors should be considered according to the way in which the environment has been embodied by measuring for example key biological mediators contributing to the allostatic load and by analysing the interaction between risk factors and allostatic load. At the population level, for which data from biological samples are sparse, the effects of exposure to risk factors should be analysed by considering their interaction with social characteristics. Studies including clinical, biological, psychosocial data are thus urgently needed if we want to explore how the psychosocial environment impacts cancer risk and progression.

Regarding public health policy, assuming that the psychosocial environment can participate in producing social inequalities via biological mechanisms, modulating the effect of risk factors raises important questions:

In prevention, should interventions aiming to reduce the biological burden caused by environmental embodiment be