

# Can epidemics be noncommunicable?

Reflections on the spread of ‘noncommunicable’ diseases

Jens Seeberg and Lotte Meinert

## Abstract

This article argues that the concept of communicability that is central to the distinction between communicable diseases (CDs) and noncommunicable diseases (NCDs) is poorly conceptualized. The epidemic spread of NCDs such as diabetes, depression, and eating disorders demonstrates that they are communicable, even if they are not infectious. We need to more critically explore how they might be communicable in specific environments. All diseases with epidemic potential, we argue, should be assumed to be communicable in a broader sense, and that the underlying medical distinction between infectious and noninfectious diseases confuses our understanding of NCD epidemics when these categories are treated as synonymous with ‘communicable’ and ‘noncommunicable’ diseases, respectively. The dominant role accorded to the concept of ‘lifestyle’, with its focus on individual responsibility, is part of the problem, rather than the solution, and the labelling of some NCDs as ‘lifestyle diseases’ is misleading. Founded on a critical understanding of global health and globalized medicine, we propose to explore the dynamics of the phenomena of contamination and biosocial contagion in networks. An analytics of biosocial epidemics needs to be developed by a medical anthropology that is engaged in a critical dialogue with both medicine and biology.

## Keywords

noncommunicable diseases, biosocial epidemics, social networks, contamination, life conditions, lifestyle

## Introduction

By definition, a noncommunicable disease (NCD) is a disease that is not infectious or transmissible. In recent decades, the global balance of communicable versus noncommunicable diseases has tilted towards the latter; the World Health Organization (WHO) estimates that 60 percent of all deaths globally are attributable to NCDs (WHO 2009, 5), and that 80 percent of these occur in low- and middle-income countries (WHO 2011, vii). Accordingly, global policies are developed to address the epidemic spread of the most prevalent NCDs, listed as cardiovascular disease, cancer, diabetes, and chronic lung disease (WHO 2011, vii). The WHO suggests that a ‘major reduction in the burden of NCDs will come from population-wide interventions’, but acknowledges that these ‘are not implemented on a wide scale because of inadequate political commitment, insufficient engagement of nonhealth sectors, lack of resources, vested interests of critical constituencies, and limited engagement of key stakeholders’ (WHO 2011, vii). Despite these structural and larger political issues, the WHO maintains its focus on healthy lifestyle as the single most important preventive strategy. Key to this strategy is the reduction of:

the level of exposure of individuals and populations to the common modifiable risk factors for NCDs – namely, tobacco use, unhealthy diet and physical inactivity, and the harmful use of alcohol – and their determinants, while at the same time strengthening the capacity of individuals and populations to make healthier choices and follow lifestyle patterns that foster good health. (World Health Organization 2009, 10)

This article questions the understanding of communicability that forms the basis of global health strategies such as these. We argue that epidemic diseases must be understood as communicable, even though they may not be infectious in the biological sense; within this framework, a number of NCDs should be seen as communicable, and their noninfectious modes of transmission should be explored.

## Classifying CDs and NCDs

The CD-NCD distinction is intrinsically linked to different forms of biosociality and ideas about causation and lifestyle. Within the realm of communicable disease, chronic bacterial

and viral infections such as tuberculosis (TB) and human immunodeficiency virus (HIV) have formed the biological basis for global treatment regimes that temporarily or permanently reorganize the lives of potential and actual patients and their relatives. These regimes also introduce the concept of risk to individual lives and households, and they create new kinds of people (Hacking 1986), such as HIV risk groups (Fordham 2001) and TB treatment ‘defaulters’ (Seeberg 2014), as well as therapy management groups and other types of patient networks (Meinert 2013; Meinert, Mogensen, and Twebaze 2009; Nguyen 2010).

NCDs, for which, by definition, a single infectant cannot be identified, are understood as having a multifactorial cause that usually cannot be effectively addressed with a single intervention. The major form of biosociality linked to NCDs is the ‘healthy lifestyle’ regime, which is presented as a moral imperative, requiring personal efforts to help keep society’s health costs down. It is a combined expression of the individual and greater common good. Hence, control of NCDs is primarily linked to health campaigns that seek to regulate the major risk factors associated with cardiovascular disease, cancer, diabetes, and chronic lung disease. The focus on lifestyle in health promotion has played an important part in establishing forms of biosociality that emphasize individual choice, for example, through healthy living campaigns and patient schools (Grøn 2004; Lupton 1995).

Although lifestyle may be an unusual focus in the management of treatable CDs, control of the most common NCDs tends to have the individual and his or her lifestyle as a primary target. Inherently moral constructions of undesired lifestyles, and the benign rewards of intrinsically healthy lifestyles are widely circulated by public health campaigns (Kelly and Charlton 1995). Sometimes, lifestyle interventions are accompanied by other, more complex interventions that resemble vector control. For example, in the case of malaria, rather than targeting the disease itself, some interventions target the vector – mosquitoes that transmit the disease – through insect repellent or insecticide treated bed nets. Aspects of public health measures against tobacco-induced lung-disease may resemble vector control. Some interventions, such as smoking-cessation campaigns, follow the standard lifestyle approach, trying to enable individuals to stop habits and dependencies that are perceived as undesirable. At the same time, the creation of the concept of ‘passive smoking’ (Jackson 1994) transcends the focus on individual lifestyle, and establishes smoking as health hazard in contexts of work, public transportation, family life, and pregnancy. But another strand of public health control has attempted to limit the spread of tobacco use by regulating the tobacco market through advertising restrictions and taxation, as proposed in the 1985 WHO report discussed above. This is more similar to the classic CD approach, implicitly positioning tobacco as an inorganic ‘vector’ of lung cancer and cardiovascular disease and attacking the spread of this vector in a poorly regulated market.

The case of epidemic (and endemic) tobacco-induced lung disease directs us to more useful ways of understanding the spread of disease than the current distinction between NCD and CD seems to allow. This approach is linked to the distinction between macro- and micro-parasitism, proposed by Baer, Singer, and Susser (2003). Although micro-parasites such as bacteria define the class of CDs, macro-parasites such as harmful industries may significantly influence the spread of NCDs. Following this line of thought, the global market has become an effective channel for communicating a number of diseases that are classified as NCDs, such as certain forms of cancer, certain mental illnesses, diabetes, and chronic obstructive lung disease. Although we recognize that such diseases are clinically defined by their microbiological characteristics, we suggest that their epidemic potential – that is, their ability to spread epidemically in populations – may be better understood in terms of their biosocial dynamics, as these are embedded in political economy and human biologies.

## Contamination, configuration, predisposition

The elimination of the concept of ‘contagion’ from biomedical discourse may be seen as a result of the discovery of microbiological infectants, such as bacteria, which led to the creation of the category of ‘infectious diseases’ (Pernick 2002). This reclassification involved the reduction of a broad social and moral domain of contagion to one of biology. In the early twentieth century, this evolution of overarching medical concepts of noninfectious versus infectious diseases resulted in the authoritative distinction between noncommunicable and communicable disease, guided by the definition of communicable disease published in *Control of Communicable Diseases*, by the American Public Health Association in 1920 (Pernick 2002). As a category, NCD was an unsatisfactory definition from the outset; it was a residual category that was defined negatively, in terms of not being infectious. Could contagion have been kept as a meaningful category for understanding how some of these diseases may have epidemic potential?

Historically, contagion has been a problematic concept because of its heavy moral connotations, which have facilitated a process of ‘othering’ directed against those believed to be contagious. The concept often conflated the disease and the identity of the afflicted person, and served to justify stigmatization and social exclusion. In the absence of effective health care, such mechanisms may have protected some from disease. Yet, the distribution of both disease and health care is impacted by social forces, including deeply moralizing ones (Brandt and Rozin 1997). We suggest that contagion and the related notion of contamination may be used as analytical categories to capture these social dimensions of the spread of disease.

Rosenberg (1992, 295) sees ‘contagion’ as part of the more general concept of ‘contamination’, that is, ‘an event or agent that might subvert a health-maintaining configuration’. Thus, contamination subsumes the categories of contagion, infection, and pollution (for example, poisoning). He contrasts ‘contamination’ with the concept of ‘configuration’, which implies a focus on environment and social life. In Rosenberg’s view, as orientations that inform explanations of disease (and their epidemic occurrences), the former tends to be reductionist and monocausal, whereas the latter emphasizes system and interconnection – but they are not necessarily mutually exclusive.

Rosenberg (1992, 296) notes that a third term, ‘predisposition’, is used to explain why not all become ill when they are similarly exposed to contamination within a given configuration: ‘healers and laypeople have always needed to explain the immunity of some individuals from the epidemic “influence” surrounding them’. Where explanations of predisposition have historically been open to moral and religious interpretations, genetic explanations and the identification of biomarkers have more recently radically changed the understanding of predisposition.

The foregoing three concepts are, in Rosenberg’s historical account, types of explanations. However, they may also be used as analytical concepts. As such, their relevance has not decreased with the technological advances of medicine; an exploration of the relationships among contamination, predisposition, and configuration in epidemics of NCDs may be more useful, in terms of explanatory power, than is the idea of individualized and optional lifestyles.

## Lifestyle epidemics

The concept of lifestyle may be traced to Weber and others, who distinguished among *Lebensstil* (‘style of life’), *Lebensführung* (‘conduct’), and *Lebenschancen* (‘life circumstances’, ‘chances’). The last is primarily conceived in socioeconomic terms (Weber [1922] 1999, 709–10) and linked to associated concepts, such as ‘class’, while the first two concepts are linked to choice. As summarized eloquently by Cockerham, Rütten, and Abel (1997, 324), ‘*Lebensführung* refers to people’s choices in their selection of lifestyles, and *Lebenschancen* is the probability of realizing those choices’. Weber’s definitions of social groups were informed by his analysis of European industrial society, and he clearly acknowledged that not everybody had the same chances, since, as Cockerham et al. (1997, 325) put it, ‘chance is socially determined, and social structure is an arrangement of chances’. Yet, given the chances that one had, the realization of *Lebensstil* was understood as one of choice. Choice was seen as constrained, but not determined, by chance (or social structure), and lifestyle was defined as a social performance that distinguished social groups.

In the early 1970s, a link was established between cardiovascular disease and ‘mode of life’ (Stamler 1970), in which prevention was emphasized, but the concept of ‘lifestyle disease’ took another two decades to develop. In 1980, the WHO’s Regional Committee for Europe called for a new strategy for health for all, in which ‘healthy lifestyles’ played a key role; in 1985, the WHO/Europe office published its targets for this strategy (WHO 1985). This document associated lifestyle with a range of conditions, including accidents, poisoning, and other violent deaths; smoking-related cancers, certain respiratory diseases, ischemic heart disease, and cirrhosis of the liver (WHO 1985, 25); lifestyle-related cancers (32); undesired pregnancy/abortion (34); and dental caries and periodontal disease (35). With regard to cardiovascular disease, respiratory diseases, cancer, and accidents, lifestyle interventions were expected to be particularly important for increasing the life expectancy of men (WHO 1985, 39). The report goes on to define ‘lifestyle’ in ways not dissimilar to Weber’s three concepts:

A person’s particular way of life is shaped by patterns of interpersonal interaction and social learning that interrelate with and depend upon the social environment. Thus, lifestyles, shaped by experience and environmental factors, are not simply individual decisions to avoid or accept certain health risks. There are limits to the choices open to individuals – limits imposed by their physical, social and cultural environment and by their financial means. (WHO 1985, 53)

Interestingly, although defining lifestyles as individual choices, this document and its recommendations for interventions focused less on individuals than on government regulation of industries that produce unhealthy products (tobacco, certain foods, etc.). Harmful industries should not receive subsidies, and their products should be subject to high levels of taxation. Although this approach has not disappeared, the disaggregation of structural conditions into ‘risk factors’ has gradually come to define the understanding of the links between lifestyle and health. A healthy lifestyle has been equated with exercise, a low-fat diet, not smoking, and low alcohol consumption, tilting the balance in favour of a focus on individual choices, rather than on structural factors. It is interesting to note how, as an explanatory model for NCDs within the WHO’s strategic documents, the meaning of ‘lifestyle’ has changed over the course of a few decades. In the 1985 targets, lifestyle implied a broader configuration that included emphasis on the environment in general; twenty-five years later, WHO suggested a much more narrow configuration, which emphasized decontextualized factors that may be tacked onto individuals who are supposed to be in control of their micro-environments.

As a key concept of ‘new public health’, lifestyle has been politically attractive for a number of reasons. According to O’Brien (1995, 191), ‘the association between “health” and “lifestyle” is fundamentally a political achievement, supported by an institutionalised

consumerism, validated by a liberal political ideology and nurtured by a technocratic professionalism increasingly oriented towards problem solving approaches to health and social life'. This achievement has allowed for the development of health promotion in a wide range of forms, and it has provided clinicians with nonmedical tools, in the form of lifestyle counselling and referral to healthy living programmes.

From a medical/anthropological point of view, the idea of lifestyle disease could be particularly interesting, since the attention given to bodies and style is profoundly social (Glassner 1995). If an 'unhealthy' lifestyle is truly seen as a pathogen, then the understanding of epidemics of lifestyle diseases should take as their point of departure how lifestyles spread – as forms of representation, through consumption of lifestyle-defining products, and following lifestyle-defining practices – thereby positioning lifestyle diseases as highly communicable. Following this train of thought, a number of conditions resulting from the pursuit of a healthy lifestyle, such as exercise addiction (Berczik et al. 2012), should be classified as 'lifestyle diseases' in a much stronger sense: the promotion of healthy lifestyles – and their aggressive marketing by companies that sell goods that come to define such lifestyles – is potentially pathogenic.

In the commonly accepted configuration, where lifestyle diseases are defined as those that may be avoided by pursuing a 'healthy lifestyle', the construct of 'lifestyle' carries a moral load that resembles that of contagion. Whereas the concept of contagion has historically associated the contraction of infectious diseases such as syphilis, gonorrhoea, HIV, and plague with guilt, shame, stigma, and social exclusion, the concept of lifestyle has similar moral potential when it is used to assign individual responsibility to the development of disease. Although this sense of guilt may lead to disciplining the body in ways that some consider desirable, it is also a potential mental health pathogen. The active promotion on the Internet of eating disorders as a lifestyle choice could be viewed as an example of this, but research on this topic is inconclusive (Harper, Sperry, and Thompson 2008).

An analysis of lifestyle as a form of contagion has certain merits, but it also presents us with a number of unresolved issues. The first has to do with causality. As pointed out by Young (1995, 7), posttraumatic stress disorder (PTSD) is defined by its aetiology, and the same may be said of lifestyle diseases: they are – tautologically – diseases caused by unhealthy lifestyles. But PTSD is 'a disease of time', as it 'relives itself in the present, in the form of intrusive images and thoughts and in the patient's compulsion to replay old events' (Young 1995, 7). Lifestyle diseases have no clear temporal relationship, apart from the simultaneity that allows for statistical association, therefore, it may be difficult to establish whether a risk factor actually predates the disease. Furthermore, as NCDs in the lifestyle-disease cluster become increasingly common in the global South, including among populations that do not share

lifestyles that have been classified as unhealthy in the global North, the limitations of 'lifestyle' as an explanation of disease become even clearer.

## Epidemics of diagnoses and representations

Based on her long-term ethnographic research in Uganda, Susan Whyte (2012) indicates the need for studies of NCDs, as these interfere with and change people's lives and identities to the extent that new diagnostic techniques, counselling, and treatment options become available. She points out that "Non-communicable diseases" are communicable in a broader sense. *Awareness of them is contagious*' (Whyte 2012, 66, emphasis added). Clearly, such awareness is linked to the spread of diagnoses (not necessarily disease) that may at times be misinterpreted as a new epidemic, even if it is only the discovery of a pre-existing disease pattern. Hacking's (1992) work on the social dynamics of classification may help to understand important aspects of such epidemics of diagnosis. With his example of multiple personality disorder (MPD), Hacking shows how social categories and diagnoses come into being, historically. Institutions and therapies that define diagnoses – often simultaneously presenting solutions to an epidemic – also drive them. Even though some of the underlying practices perceived as causally linked to MPD, such as child abuse, may have been relatively stable historically, public interest in and awareness of the phenomenon may take on epidemic proportions, and lead us to think that child abuse is on the increase (Hacking 1992). When a bodily sign is given a name, and this is linked to certain moral characteristics of the person who exhibits that sign, a looping effect with epidemic potential may be created. Hacking's framework may be usefully applied to analysing and understanding the variation in frequency of diagnoses related to diseases such as ADHD, autism, PTSD, diabetes, and hypertension over time. However, this type of analysis does not address questions regarding the possible spread of an underlying disease, irrespective of its classification.

Focusing on representations rather than diseases, Sperber (1985, 3) points out that 'the human mind is susceptible to cultural representations, in the way the human organism is susceptible to diseases', and raises important questions regarding why some representations are more successful in a human population – more contagious, more 'catching' – than others. Representations may be transmitted slowly over generations, for example, through the continuous reinvention of traditions, and may be compared to endemic conditions. Other representations, such as fashions, spread rapidly through a population and have a short lifespan, resembling epidemic patterns (Sperber 1985).

Sperber (1985) points out that representations are transformed every time they are transmitted. Thus, the social process of transmission involves important micro-mutations, as



well as contingencies. In exploring how and why certain representations become epidemic, he notes that human beings remember a story much better than a text or a list of items: stories are ‘stickier’ than facts, because one thing leads to the other, and a process is started. Thus, representations that are part of a story may have greater epidemic potential than representations of abstracted facts. Another ‘stickiness factor’ (Gladwell 1996; Sperber 1985) in representations becoming epidemic is the evocation of emotions and the mobilization of a moral register. Stories, however, do not preclude statistics: numbers may be crucial in telling a story about an epidemic, even vested with moral importance, both at the macro-level, when an epidemic is constructed and addressed epidemiologically and politically, and at the micro-level, when individuals monitor their biomedical disease statistics, such as CD4 counts or blood sugar measurements (Meinert et al. 2009; Nielsen and Grøn forthcoming).

## Social networks

Social representations may move within, across, and outside social networks, and a number of researchers have attempted to study the prevalence of NCDs with social network mapping. For example, Christakis and Fowler (2007) have studied how various NCDs (including heart disorders and obesity) spread through social networks. Their research group found that contributing factors to NCDs, such as obesity (Christakis and Fowler 2007) and alcohol consumption (Rosenquist et al. 2010), and inhibiting factors, such as smoking cessation, tend to cluster in social networks. Their work shows that the types of relations within such networks are significant: friendship is a much more contagious relation than a family or neighbourhood relationship. In an online social network experiment, Centola (2010) demonstrated that simple and easy-to-do behaviours spread much faster in clustered networks (comparable to friendships) than in random networks. These approaches support our claim that phenomena hitherto assumed to not be contagious (such as obesity) may be understood as such.

Although Christakis and Fowler have generated substantial interest in this new ‘network medicine’, so far their contribution has been limited to demonstrating that contagion does occur through social networks, and that the type of network relation matters. It tells us less about why this happens; some of the effects may be due to confounding factors: strong social networks may share exposure to conditions or factors that could account for the health outcomes (Cohen-Cole and Fletcher 2008). Although Christakis and Fowler claim to control for them, we suggest that so-called confounding factors should be included in the analysis in a systematic way, based on qualitative research. We need to pursue what actually happens in these social networks: what is transmitted, and how? After all, obesity has to be linked to eating *something*, and the extent to which eating takes place as a social activity (within the family, at the workplace, or with close friends) may contribute to understanding

the patterning of obesity in social networks and among socially isolated individuals, as well. Also, this approach assumes the existence of a network, and cannot be used to understand what happens outside networks. As pointed out by Strathern (1996) in her critique of actor-network theory, social reality is as much created by cutting networks and connections as by linking them, but there is little evidence to suggest that social isolation protects one from being part of an NCD epidemic. Furthermore, it seems that the approach adopted in 'network medicine' provides interesting insights into the role of social networks as effective channels for biosocially communicable epidemics, but it does not promote a critical perspective regarding the societal dynamics that produce the 'pathogens' or 'vectors' that travel through such networks. Hence, despite the communicability inherent in the capacity of lifestyles to spread through social networks, the problem of causality, as discussed below, renders lifestyle a weak explanation for the epidemic occurrences of these so-called noncommunicable diseases.

## Life conditions

Clearly, an emphasis on Weberian *Lebenschancen* or 'life conditions' does not lend itself as readily to the health promotion ideology of 'new public health' as does the lifestyle concept, and the criticism that emphasis on the latter has been privileged to the near-exclusion of the former in health policies is not new (see, for example, Parish 1995). These criticisms have primarily focussed on prevention and treatment regimes that seek to discipline the self (see, for example, Petersen and Lupton 1996) and medicalize human life (see, for example, Rose 2007). Less critical attention has been given to the possible impact of lifestyle on the epidemic spread of those NCDs labelled as lifestyle diseases. If the causal link between lifestyle and disease is assumed a priori, this assumption will lead to a preference for epidemiological studies that look for associations between 'lifestyle factors' and 'lifestyle diseases', to the possible exclusion of other questions.

In a discussion on NCDs in Uganda, Whyte (2013) offers an ethnographically based analysis of the dynamic between life conditions and lifestyle (both as viewed by public health officials and as perceived – quite differently – by her interlocutors in rural and urban areas). For example, in terms of eating practices, she identifies cooking oil as a strong marker of social distinction, as access to frying food is considered a luxury, and hence a marker of an attractive lifestyle. However, the quality of oil varies from expensive (and 'safe') olive oil to cheap cottonseed and coconut oil that, according to Whyte's (2013, 198) interlocutors, 'goes in the veins and clots'. In rural areas cooking oil is 'ladled out of large containers into plastic sacks or bottles they have brought', sold in small amounts to people who 'may not know or care what kind of oil they have bought, as long as it is cheap' (Whyte 2013, 198). This example warns us that it is not necessarily the 'lifestyle' of cooking with oil that is unhealthy:

the life conditions under which this practice is enjoyed, combined with the hidden and unknown content of locally available products, may contribute to the spread of certain NCDs. It does not take much to identify similar scenarios worldwide. Importantly, here, all three elements at work (lifestyle, life conditions, and the market) may be seen as communicable: the 'lifestyle' valuation of cooking oil is spread through ads and other media that link it to smart and modern life in the city; life conditions may be shared across generations and neighbourhoods and may reproduce themselves through processes of structural violence; and potentially harmful molecules are introduced into the body through food items via the market.

This example directs us to a different classificatory logic than that of the CD-NCD dichotomy – one that distinguishes among infectious, genetic, and environmental disease – and generates other sets of questions. Research in environmental obesogens is an example of biomedical research that could complement ethnographic research, by qualifying the impact of various products, such as different types of cooking oil, on the human body. At a larger scale, there is increasing suspicion that the obesity pandemic is at least partially caused by synthetic, environmental contaminants, such as 'intentional food additives (e.g. artificial sweeteners and colours, emulsifiers) and unintentional compounds (e.g. bisphenol A, pesticides)', but more research is needed in this field (Simmons, Schlezinger and Corkey 2014, 273). Bisphenol A is a hormone-like compound that is widely used in food and drink containers and to line water pipes; its use in baby bottles has been banned in the European Union and Canada. It has been found to be positively associated with the development of obesity (through its disruption of the endocrine system), a number of cancers, and a range of other diseases. This may support the use of a metaphor of vector-borne disease as a way of understanding the global spread of obesity, as we suggested for tobacco-induced lung disease, above, even though the obesity epidemic is more complex. While political battles, such as the regulation of the tobacco market, or the one currently being fought between the government of France and the plastics lobby over the banning of Bisphenol A (Martin 2014), knowledge about the harmful effects of this compound – as well as the capacity to avoid them – is likely to follow the fault lines of privileged and underprivileged life conditions.

## Syndemics

The concept of syndemics developed by Singer and his colleagues (Singer 1996; Singer and Clair 2003; Singer 2009) is an important contribution to the critique of disease-specific approaches; they argue against the conceptualization of diseases as bounded entities, isolated from one another in individual bodies and populations, and distinct from the social conditions that create conducive environments for disease to develop and spread. Singer and

Clair (2003, 425) define syndemics at the population level as the co-occurrence of two or more epidemics that interact synergistically, and they maintain that, at the level of the body, the conditions that constitute a syndemic reinforce one another. Singer and Clair (2003, 425) offer the syndemic relationship of TB and HIV as an example of this: ‘coinfection with HIV and Mycobacterium tuberculosis (MTb) augments the immunopathology of HIV and accelerates the damaging progression of the disease’, while HIV also creates a biological environment that dramatically enhances the opportunity for TB (and other opportunistic infections) to develop and spread.

The concept of ‘co-infection’ is different from the concept of ‘syndemic’ in at least two ways: in syndemics, the epidemics involved need not be biologically infectious, and (related to this) the concept implies an analysis of the social conditions and political economy that strengthen the synergistic relationships between the conditions involved. As has been shown by medical anthropologists who have adopted a syndemic approach, this makes it possible to analyse nested epidemics, such as the substance abuse, violence, and AIDS (SAVA) syndemic (Singer 2006); food insecurity and AIDS in South Africa (Singer 2011); and the violence, immigration, depression, diabetes, and abuse (VIDDA) syndemic among Mexican immigrant women in Chicago (Mendenhall 2012). Reframing the understanding of the co-occurrence of epidemics in a given population in this way involves substantial methodological and analytical challenges, as it encourages the simultaneous exploration of infectious, genetic, and environmental disease. Hence, it also calls for medical anthropologists, and the multidisciplinary teams of which they may be part, to develop ways to link ethnographic data with epidemiological associations and biological constructs. The syndemic theory thus allows free movement across the CD-NCD divide, showing that this distinction is not necessary. More work is required, however, to understand the dynamics underlying the way in which NCDs become communicable and epidemic in the first place.

## Understanding the biosocial communicability of disease

Above, we suggested that it may be misleading to consider so-called lifestyle diseases as both noncommunicable and caused by lifestyle, and that doing so effectively marginalizes other important research questions. We believe there is a need to enhance our basic understanding and conceptualization of noncommunicable disease, and how it may spread and develop into epidemics. Although interdisciplinary collaboration is certainly required across traditional boundaries, anthropologists may also contribute within their disciplines. From the position of anthropology, we propose pursuing this agenda with ethnographic research that connects

different levels of analysis,<sup>1</sup> that is, seeking to understand the biosocial and structural dynamics of the large-scale, epidemic spread of NCDs between mutually constitutive local and global scales that articulate certain types of ‘friction’, as conceptualized by Tsing (2005). Attending to such levels and scales would focus our analysis on not only the intimate processes in living bodies, including cellular and molecular levels, but also on phenomenological and reflective aspects of contamination, susceptibility, immunity, and development of disease. These levels and scales would call our attention to social interactions, networks, relations and connections, and interactions with the physical world; the natural environment and material culture as assemblages of potentially contagious encounters; and, socioeconomic, systemic, and political factors, such as consumption, access to and promotion of diagnoses, treatment, control, and prevention of disease, as well as the statistical, discursive, and political making and framing of epidemics. Understanding the dynamics of contamination and predisposition, and epidemic potential, for specific NCDs would require the development of a new analytics that would be able to look at particular configurations across these scales. This analytics would qualitatively explore how contamination processes may mutate, sustain themselves, and disappear in cross-cutting connections, thereby redefining the concept of contagion from one that is focused on essence and substance to a broader notion that includes processes, relationships, and connections.

In anthropology, and more broadly in the social sciences, there is a long tradition of looking into processes of contagion and diffusion in social and cultural domains, and – partially linked to the expansion of the Internet – this research focus is currently taking on new forms, and inspiring new theoretical and analytical approaches. This fast-growing literature promises important insights into cultural processes of the nature that we have indicated here. To our knowledge, however, none of these contributions have suggested applying their insights to understanding the riddle at hand: how can noncommunicable diseases be epidemic? We suggest exploring the epidemic potential of NCDs: What constitutes this potential? What kinds of similarities, differences, and syndemic effects are to be found across different disease categories? And how will the answers to these questions help us redefine the concept of ‘noncommunicable disease’, and thereby develop better categories for disease classification?

1 At the research Centre for Cultural Epidemics at Aarhus University (EPICENTER), established in 2012, we are developing this approach. EPICENTER currently conducts research projects on a number of conditions, such as cancer, diabetes, obesity, PTSD, autism, suicide, and drug use. See <http://epicenter.au.dk/>

## About the authors

Jens Seeberg is Associate Professor and Head, Department of Anthropology, Aarhus University. He has directed a number of multisited interdisciplinary research projects in Asia and has published on health ethics, the role of the pharmaceutical industry, drug-resistant tuberculosis, and the aftermath of the 2006 Central Java earthquake. He is the director of a project on transnational migration in (and out of) Nepal; director of a project on strengthening research capacity in Nepal, Uganda, and Kenya; co-director of the Center for Research on Cultural Epidemics (EPICENTER); and coordinator of an interdisciplinary research network on drug resistance.

Lotte Meinert is Professor of Anthropology at Aarhus University, Denmark, and currently a visiting scholar at Johns Hopkins University. She has carried out fieldwork in Uganda for more than seven years on topics related to health, education, and security. Her monograph *Hopes in Friction: Schooling, Health and Everyday Life in Uganda* was published by IAP in 2009. She is co-editor of *Time Objectified: Ethnographies of Global Youth* (Temple University Press, 2014) and co-editor of *In the Event: Toward an Anthropology of Generic Moments* (Berghahn Books, 2015). She conducts research on postwar recovery in Uganda (<http://enrecagulu.net>) and land conflicts (<http://trustland.me>). She and Jens Seeberg coordinate Epicenter: Center for Cultural Epidemics (<http://epicenter.au.dk>).

## References

- Baer, Hans A., Merrill Singer, and Ida Susser. 2003. *Medical Anthropology and the World System*. Westport, CT: Praeger Publishers.
- Berczik, Krisztina, Attila Szabó, Mark D. Griffiths, Tamás Kurimay, Bernadette Kun, Róbert Urbán, and Zsolt Demetrovics. 2012. 'Exercise Addiction: Symptoms, Diagnosis, Epidemiology, and Etiology'. *Substance Use & Misuse* 47, no. 4: 403–17. <http://dx.doi.org/10.3109/10826084.2011.639120>.
- Brandt, Allan M., and Paul Rozin, eds. 1997. *Morality and Health*. New York: Routledge.
- Centola, D. 2010. 'The Spread of Behavior in an Online Social Network Experiment'. *Science* 329, no. 5996: 1194–97. <http://dx.doi.org/10.1126/science.1185231>.
- Christakis, Nicholas A., and James H. Fowler. 2007. 'The Spread of Obesity in a Large Social Network over 32 Years'. *New England Journal of Medicine* 357, no. 4: 370–79. <http://dx.doi.org/10.1056/NEJMsa066082>.

- Cockerham, William C., Alfred Rütten, and Thomas Abel. 1997. 'Conceptualizing Contemporary Health Lifestyles'. *Sociological Quarterly* 38, no. 2: 321–42. <http://dx.doi.org/10.1111/j.1533-8525.1997.tb00480.x>.
- Cohen-Cole, Ethan, and Jason M. Fletcher. 2008. 'Detecting Implausible Social Network Effects in Acne, Height, and Headaches: Longitudinal Analysis'. *British Medical Journal* 337: a2533. <http://dx.doi.org/10.1136/bmj.a2533>.
- Fordham, Graham. 2001. 'Moral Panic and the Construction of National Order: HIV/AIDS Risk Groups and Moral Boundaries in the Creation of Modern Thailand'. *Critique of Anthropology* 21, no. 3: 259–316. <http://dx.doi.org/10.1177/0308275X0102100301>.
- Gladwell, Malcolm. 1996. 'The Tipping Point'. *New Yorker*, 3 June 1996.
- Glassner, Barry. 1995. 'In the Name of Health'. In *The Sociology of Health Promotion: Critical Analyses of Consumption, Lifestyle and Risk*, edited by Robin Bunton, Sarah Nettleton, and Roger Burrows, 159–74. New York: Routledge.
- Grøn, Lone. 2004. 'Winds of Change, Bodies of Persistence: Health Promotion and Lifestyle Change in Institutional and Everyday Contexts'. PhD. diss., Aarhus University.
- Hacking, Ian. 1986. 'Making up People'. In *Reconstructing Individualism: Autonomy, Individuality, and the Self in Western Thought*, edited by Thomas C. Heller, Morton Sosna, and David E. Wellberry, 222–36. Stanford, CA: Stanford University Press.
- Hacking, Ian. 1992. 'World-Making by Kind-Making: Child Abuse for Example'. In *How Classification Works: Nelson Goodman among the Social Sciences*, edited by Mary Douglas and David Hull, 180–238. Edinburgh: Edinburgh University Press.
- Harper, Kelley, Steffanie Sperry, and J. Kevin Thompson. 2008. 'Viewership of Pro-Eating Disorder Websites: Association with Body Image and Eating Disturbances'. *International Journal of Eating Disorders* 41, no. 1: 92–95. <http://dx.doi.org/10.1002/eat.20408>.
- Jackson, Peter W. 1994. 'Passive Smoking and Ill- Health: Practice and Process in the Production of Medical Knowledge'. *Sociology of Health & Illness* 16, no. 4: 423–47. <http://dx.doi.org/10.1111/j.1467-9566.1994.tb00159.x>.
- Kelly, Michael P., and Bruce Charlton. 1995. 'The Modern and the Postmodern in Health Promotion'. In *The Sociology of Health Promotion: Critical Analyses of Consumption, Lifestyle and Risk*, edited by Robin Bunton, Sarah Nettleton, and Roger Burrows, 78–90. New York: Routledge.
- Lupton, Deborah. 1995. *The Imperative of Health: Public Health and the Regulated Body*. London: Sage Publications.

- Martin, Anne-Claude. 2014. 'French Government and Plastics Lobby Clash over Bisphenol A'. Brussels: Fondation EurActiv. <http://www.euractiv.com/sections/science-policy-making/french-government-and-plastics-lobby-clash-over-bisphenol-310509>.
- Meinert, Lotte. 2013. 'Antropologiske Perspektiver på Usikkerhed: Smitte som Analytik' [Anthropological Perspectives on Insecurity: Contagion as analytic]. *Tidsskriftet Antropologi* 68: 131–47. <http://tidsskrift.antropologi.ku.dk>.
- Meinert, Lotte, Hanne O. Mogensen, and Jenipher Twebaze. 2009. 'Tests for Life Chances: CD4 Miracles and Obstacles in Uganda'. *Anthropology and Medicine* 16, no. 2: 195–209. <http://dx.doi.org/10.1080/13648470902940697>.
- Mendenhall, Emily. 2012. *Syndemic Suffering: Social Distress, Depression, and Diabetes among Mexican Immigrant Women*. Advances in Critical Medical Anthropology 4. Walnut Creek, CA: Left Coast Press.
- Nguyen, Vinh-Kim. 2010. *The Republic of Therapy: Triage and Sovereignty in West Africa's Time of AIDS*. Durham, NC: Duke University Press.
- Nielsen, Mikka, and Lone Grøn. Forthcoming. 'Quantify Your Self! Numbers in Ambiguous Borderlands of Health'. *Tidsskrift for Forskning i Sygdom og Samfund*.
- O'Brien, Martin. 1995. 'Health and Lifestyle a Critical Mess? Notes on the Dedifferentiation of Health'. In *The Sociology of Health Promotion: Critical Analyses of Consumption, Lifestyle and Risk*, edited by Robin Bunton, Sarah Nettleton, and Roger Burrows, 189–202. New York: Routledge.
- Parish, Richard. 1995. 'Health Promotion Rhetoric and Reality'. In *The Sociology of Health Promotion: Critical Analyses of Consumption, Lifestyle and Risk*, edited by Robin Bunton, Sarah Nettleton, and Roger Burrows, 11–22. New York: Routledge.
- Pernick, Martin S. 2002. 'Contagion and Culture'. *American Literary History* 14, no. 4: 858–65. <http://www.jstor.org/stable/3568029>.
- Petersen, Alan, and Deborah Lupton. 1996. *The New Public Health: Health and Self in the Age of Risk*. London: Sage Publications.
- Rose, Nikolas. 2007. *The Politics of Life Itself: Biomedicine, Power, and Subjectivity in the Twenty-First Century*. Princeton, NJ: Princeton University Press.
- Rosenberg, Charles E. 1992. *Explaining Epidemics and Other Studies in the History of Medicine*. Cambridge: Cambridge University Press.



- Rosenquist, J. Niels, Joanne Murabito, James H. Fowler, and Nicholas A. Christakis. 2010. 'The Spread of Alcohol Consumption Behavior in a Large Social Network'. *Annals of Internal Medicine* 152, no. 7: 426–33. <http://dx.doi.org/10.7326/0003-4819-152-7-201004060-00007>.
- Seeberg, Jens. 2014. 'The Event of DOTS and the Transformation of the Tuberculosis Syndemic in India'. *Cambridge Anthropology* 32, no. 1: 95–113. <http://dx.doi.org/10.3167/ca.2014.320108>.
- Simmons, Amber L., Jennifer J. Schlezinger, and Barbara E. Corkey. 2014. 'What Are We Putting in Our Food That Is Making Us Fat? Food Additives, Contaminants, and Other Putative Contributors to Obesity'. *Current Obesity Reports* 3, no. 2: 273–85. <http://dx.doi.org/10.1007/s13679-014-0094-y>.
- Singer, Merrill. 1996. 'A Dose of Drugs, a Touch of Violence, a Case of AIDS: Conceptualizing the SAVA Syndemic'. *Free Inquiry in Creative Sociology* 24, no. 2: 99–110.
- Singer, Merrill. 2006. 'A Dose of Drugs, a Touch of Violence, a Case of AIDS, Part 2: Further Conceptualizing the SAVA Syndemic'. *Free Inquiry in Creative Sociology* 34, no. 1: 39–54.
- Singer, Merrill. 2009. *Introduction to Syndemics: A Critical Systems Approach to Public and Community Health*. Hoboken, NJ: Wiley and Sons.
- Singer, Merrill. 2011. 'Toward a Critical Biosocial Model of Ecohealth in Southern Africa: The HIV/AIDS and Nutrition Insecurity Syndemic'. *Annals of Anthropological Practice* 35, no. 1: 8–27. <http://dx.doi.org/10.1111/j.2153-9588.2011.01064.x>.
- Singer, Merrill, and Scott Clair. 2003. 'Syndemics and Public Health: Reconceptualizing Disease in Bio-Social Context'. *Medical Anthropology Quarterly* 17, no. 4: 423–41. <http://www.jstor.org/stable/3655345>.
- Sperber, Dan. 1985. 'Anthropology and Psychology: Towards an Epidemiology of Representations'. *New Series* 20, no. 1: 73–89. <http://www.jstor.org/stable/2802222>.
- Stamler, Jeremiah. 1970. 'Prevention of Atherosclerotic Coronary Heart Disease by Change of Diet and Mode of Life'. *Therapeutische Umschau* 27, no. 3: 133–44.
- Strathern, Marilyn. 1996. 'Cutting the Network'. *Royal Anthropological Institute* 2, no. 3: 527–35. <http://dx.doi.org/10.2307/3034901>.
- Tsing, Anna Lowenhaupt. 2005. *Friction: An Ethnography of Global Connection*. Princeton, NJ: Princeton University Press.

- Weber, Max. 1999 [1922]. 'Wirtschaft und Gesellschaft'. In *Max Weber im Kontext - Werke auf CD-ROM*. Berlin: Karsten Worm - Infosoftware.
- Whyte, Susan Reynolds. 2012. 'Chronicity and Control: Framing "Noncommunicable Diseases" in Africa'. *Anthropology & Medicine* 19, no. 1: 63–74.  
<http://dx.doi.org/10.1080/13648470.2012.660465>.
- Whyte, Susan Reynolds. 2013. 'The Publics of the New Public Health: Life Conditions and Lifestyle Diseases in Uganda'. In *Making and Unmaking Public Health in Africa: Ethnographic and Historical Perspectives*, edited by Ruth J. Prince and Rebecca Marsland, 187–207. Athens, OH: Ohio University Press.
- World Health Organization (WHO). 1985. 'Targets for Health for All: Targets in Support of the European Regional Strategy for Health for All'. Copenhagen: World Health Organization. [http://whqlibdoc.who.int/euro/hfas/EURO\\_HFAS\\_1.pdf](http://whqlibdoc.who.int/euro/hfas/EURO_HFAS_1.pdf).
- WHO. 2009. '2008–2013 Action Plan for the Global Strategy for the Prevention and Control of Noncommunicable Diseases'. Geneva: World Health Organization. <http://www.who.int/nmh/publications/9789241597418/en/>.
- WHO. 2011. 'Global Status Report on Noncommunicable Diseases 2010'. Geneva: World Health Organization. [http://www.who.int/nmh/publications/ncd\\_report2010/en/](http://www.who.int/nmh/publications/ncd_report2010/en/).
- Young, Allan. 1995. *The Harmony of Illusions: Inventing Post-Traumatic Stress Disorder*. Princeton, NJ: Princeton University Press.