



Introduction

# **Configuring Contagion in Biosocial Epidemics**

*Lotte Meinert and Jens Seeberg*

How do epidemics spread? The short, standard answer is through contagion. For most infectious disease epidemics, we are well aware that this happens through the transfer of microbes between people or between animals and humans. The COVID-19 pandemic has made this clear all around the globe, and many people have become increasingly aware of epidemic dynamics and concepts. Yet not all epidemics can be attributed to infection. A large part of the global burden of disease is due to so-called noncommunicable diseases.<sup>1</sup> How do these and other non-infectious conditions spread? Can they even be termed epidemic and contagious? This book offers ways to think about and expand our understanding of contagion beyond typical notions of infectious pandemics, beyond viral emergencies, to include the larger field of biosocial epidemics. Fundamentally we challenge the notion of noncommunicability (Seeberg and Meinert 2015), as it seems to render the epidemic spread of other forms of disease impossible. Some researchers have even proposed that we stop using the category of noncommunicable disease (Adjaye-Gbewonyo and Vaughan 2019; Blundell and Hine 2018) because many of these diseases are in fact communicable. In this book, we propose varied and detailed answers to questions about the epidemic and contagious potentials of specific infections and non-infectious conditions. We explore how inseparable social and biological processes configure co-existing influences which create epidemics, and we stress the role of social inequality in these processes. We use the term biosocial – in



one word and without a hyphen – to underline that these processes are indivisible. Epidemics do not spread evenly in populations and simply through coincidental biological contagion. They are socially structured and selective, and contagion happens under specific economic, political and environmental conditions in which various influences configure to make contagion possible. Even though this book is mainly – but not entirely – about so-called non-infectious diseases, we dwell for a moment on the corona pandemic because it underlines that something we usually perceive as entirely biological is in fact biosocial. Likewise, what we may assume to be fully social has inseparable biological sides.

The rapid escalation of the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), also known as new coronavirus, from its initial outbreak in Wuhan, China, into pandemic proportions in early 2020, is a good example of biosocial contagion. A virus does not constitute a form of life in itself but may be said to exist at the border of life. Coronavirus is an RNA virus and so depends on access to a living cell in which it can copy itself. It does not qualify as a species but may be considered as a quasi-species (Eigen, McCaskill and Schuster 1989) and, like influenza, it may jump between species and affect them in the process to become what Celia Low has termed a ‘species multiplier’ (Lowe 2010). In biosocial terms, it is a string of information whose existence depends on communication – a basic form of exchange that constitutes sociality – between human and/or non-human becomings (Ingold 2013). Indeed, the zoonotic origin of the human experience of hosting the virus is linked to the intensified social interaction between human and non-human species that were previously in less frequent contact in densely populated urban spaces, as exemplified by the original epicentre of the coronavirus epidemic, the now famous Huanan Seafood Wholesale Market in Wuhan, China. In other words, coronavirus cannot be understood as purely biological because it could not exist outside the realm of the social. Coronavirus is intrinsically biosocial. Whereas the theoretical implications of this fact have been somewhat overlooked during the pandemic, its ramifications for global control measures are obvious. All over the globe, barriers have been created to block the biosocial interaction between virus and humans through the political regulation of human social life by way of physical distancing, quarantine, improved hygiene and

the adoption of facial masks as well as through wider societal lockdowns of workplaces, sectors such as education, cities, regions and countries, disallowing most forms of physical movement and engagement. The combined impact of new coronavirus and strict control measures has reconfigured pre-existing vulnerabilities. In India, a strict societal lockdown with only four hours' warning sent millions of migrant workers on the move away from urban centres to rural places of origin, risking a rapid nationwide spread of the virus (Maji, Choudhari and Sushma 2020) and leaving desperate migrants without access to food or healthcare. People with chronic conditions such as diabetes in need of treatment experienced interrupted access to care due to either self-isolation or fear of COVID-19, or because healthcare services did not function as usual. Such dynamics had the potential to turn (the risk of) co-morbidities into new syndemics (see below), as coronavirus and its control came to interact with other epidemics along socioeconomic faultlines. We have not yet seen the full impact of the variations of configuring coronavirus-cum-control measures across and within countries, but it is likely that other public health concerns such as tuberculosis (TB) control have been severely affected, with a risk of widespread treatment interruption potentially leading to a surge in multidrug-resistant TB. Whereas the present volume does not address the coronavirus pandemic as such, since the book had been nearly completed prior to this event, we believe that it provides important contributions that may inspire future explorations of this pandemic and future ones.

## Epidemics

Our conception of epidemics is a specific one. There are at least two senses of the term epidemic: (1) The standard epidemiological definition of an epidemic as the rapid spread of infectious disease to an unusually large number of people in a given population within a short period of time (see, e.g., Center for Disease Control<sup>2</sup>); (2) The wider use of the term in public discourse as a metaphor for something – often alarming – that seems to have arisen quickly. The term 'epidemic' has been applied to the spread of noncommunicable diseases, such as cancer and obesity (Martin and Martin-Granel 2006: 979) in a metaphorical sense (Moffat 2010), thereby



assuming a distinction between ‘real’ epidemics involving the spread of infection, and *quasi* epidemics which are postmodern and socially constructed (Boero 2007; Grøn and Meinert 2017). Our use of the term epidemic refers to the spread of infectious and non-infectious diseases alike, and our use of the term is not simply metaphorical; we are interested in phenomena – of many different kinds – that actually spread in epidemic patterns, and in how this happens. In other words, we are interested in the factual and practical levels of how various communicable and non-communicable diseases spread epidemically. Rather than limiting the term epidemic to infectious diseases and using the term in a strictly metaphorical sense about other phenomena, we explore what we claim are actual epidemic patterns. With the concept of biosocial contagion, various vernacular terms for and understandings of spread and social influence, as well as theoretical conceptions such as imitation (Tarde 1903), affect (Stewart 2007) and resonance (Rosa 2019), we seek to widen the terminology employed to talk about the biosocial configuration of epidemics. This conception of epidemics enables us to explore how phenomena such as obesity, spirit possession, ADHD, suicide, trauma and some forms of cancer spread in certain populations at particular times, and to develop new insights into how these processes transpire.

Epidemics of various kinds always involve some kind of contagion, and are often configured by more than one kind of contagion and social influence that result in disease. For analytical purposes, we can distinguish roughly between biological kinds of contagion and social kinds of contagion. In reality, however, biological and social contagion are fundamentally inseparable. It is well known that infectious disease epidemics such as HIV and TB interweave biological factors such as viral, bacterial and fungal influences with factors such as human nutrition, gender relations, health system dynamics and phenomena such as love. Epidemics of non-infectious diseases like ADHD, autism and PTSD may not involve biological contagion as such, but they are biosocial phenomena that simultaneously involve forms of social and biological influence that in turn have biological and social consequences.

Diagnostic systems may in themselves contribute to the creation of what could be called ‘epidemics of attention’ regarding diseases such as ADHD or cancer. However, diagnostic attention

can seldom account for an entire epidemic. There are usually other dynamics at work as well. Some of the dynamics involved in epidemic phenomena such as suicide, self-harm, spirit possession, alcoholism and depression may involve social contagion processes such as affect, inspiration, aspiration and haunting (Folmann 2017; Nielsen 2017). Social, historical and political circumstances are always part of the configuration of a contagious landscape that influences members of social groups in various ways. An example of this is lung cancer, which is an epidemic in areas where the industrial production and promotion of tobacco is widespread, political regulation has been minimal, and smoking is regarded by particular population groups as socially attractive, and may later turn into addiction for some. Another example of how (the lack of) political regulation plays a significant role in the prevention or development of epidemics is the obesity epidemic and the way in which the industrial production and commercialization of food and beverages at particular places and points in history clearly influence the spread of obesity (see Grøn, this volume). Short-term as well as long-term political awareness and regulation or ignorance and *laissez-faire* policy clearly influence environmental factors, including those that lead to climate change – which affects health conditions as well. For some time now, we have been aware of how climate change influences the distribution of mosquitos and the risk of vector-borne diseases such as malaria, dengue fever and Zika virus (see Morgan, this volume). There is now also growing awareness of how climate change influences the spread of conditions such as asthma, anaemia, heart disease and depression (Coleman and Littlejohn 2019). The occurrence of (new) epidemics, including those that cross the problematic boundary between communicable epidemics and noncommunicable epidemics, calls into question what drives or slows down such epidemics and what configurations of contagion are at work in these processes.

Epidemics are the biosocial consequences of unique configurations of circumstances incorporating phenomena that are both biological and social. They are products of interconnections and interactions – they are epigenetic, intersubjective and contextual processes that happen over time. In this book we reflect on epidemic phenomena through the concepts of biosocial contagion and configuration to emphasize systemic relations between different



kinds of contagious and contaminating processes. The chapters in this book challenge and rethink the concepts of contagion and configuration in relation to specific diseases, conditions and social phenomena.

We build upon the medical historian Charles Rosenberg's distinction between configuration and contamination explanations (Rosenberg 1992). As a historian, Rosenberg described two kinds of explanatory models in time: (1) Configuration views that conceptualized epidemics as disturbing climatic and social balances which maintained health; and (2) contamination views that tended towards biological reductionism and placed emphasis on person-to-person contagion and micro-organisms. This notion of contagion was gradually eliminated from biomedical discourse 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). In this book, we work towards an anti-reductionistic return to the concept of contagion seen as a biosocial recovery of the idea of infection as embedded in its wider societal configuration. We propose analytical combinations of contagion and configuration theories, and we consider how local and regional epistemologies conceptualize the spread and causation of disease and epidemics. We apply Singer and Clair's (2003) important concept of syndemic, which indicates how various epidemics often play out together and are closely integrated – such as HIV, TB and certain forms of cancer, which are often defined as merely co-morbidity (Livingston 2020), or PTSD and depression, which often seem to accompany each other. This syndemic approach to health challenges conventional historical understandings of diseases as distinct entities in nature, separate from other diseases and independent of social contexts and environmental factors (Singer et al. 2017). The chapters in this book explore specific syndemic processes through a combination of experience-near approaches to the configuring of contagion in everyday lives, and analysis of how socioeconomic structures and social inequality shape syndemics. The chapters also explore how specific local epistemologies conceptualize and contribute to the configuring of such processes. We describe the two central concepts of contagion and configuration in some detail below, followed by a brief description of the chapters in the book.

## Contagion

The discovery of micro-organisms as pathogens meant that the term ‘contagious disease’ became too broad and imprecise, and medical science opted for the more specific term ‘infectious disease’. We suggest that we reconsider the term ‘contagious’ – not only when it is applied to disease, but also with regard to other phenomena (Fainzang 1996; Matthews 1968). We invoke and apply a broad and inclusive understanding of biosocial contagious processes because various epidemic phenomena and circumstances often happen in syndemic processes (Singer and Clair 2003) in which multiple epidemic phenomena, local epistemologies and socioeconomic, political and ecological circumstances work together.

*Social* contagion may describe various kinds of processes in which entities influence each other ‘with touch’ (from the Latin *con tangere*). These entities may be minds and mindful bodies (Scheper-Hughes and Lock 1987), body parts or larger entities of families, friends, communities or organizations. Gabriel Tarde’s early study of imitation from 1903 is notable in relation to social contagion because his most basic and radical idea was that sociality is imitation, and he describes some of these processes of social influence as contagion.<sup>3</sup> Tarde wrote about the triad of imitation (repetition), opposition (to imitation) and invention. He regarded imitation as the most common social form. He rejected ideas about oppositions such as subject-object and individual-society, and he regarded imitation as neither voluntary nor involuntary, but as a basic and universal dynamic of society. One of Tarde’s main contributions to our understanding of contagion is his point about how imitation happens between two or more units of analysis. Imitation is not necessarily intentional or directed, and the process is as much about the responding part (the imitator) as the imitated. Tarde’s interest in what might be termed the elementary structures of (social) processes is potentially useful for thinking about how contagion happens. Tarde pointed to the way in which currents of imitation happen over time – in history as well as in molecular processes of vibration. He saw all social resemblance as repetition, and wrote: ‘repetitions are also multiplications or self-spreading contagions. If a stone falls into the water, the first wave which it produces will repeat itself in circling out to the confines of its basin’



(Tarde 1903: 17). In his view, imitation is what drives diffusion and infusion, but it does so in diverse environments and is influenced by other waves. Waves affect each other and create interferences. We find Tarde's early ideas useful for thinking about how various forms of biosocial contagion and influence happen in practice and can be configured into biosocial epidemics. If we think about contagion as a form of communication, Gregory Bateson's famous insight that 'we cannot not communicate' becomes relevant. The idea that some diseases are 'noncommunicable' seems unlikely, because disease will always influence the system of which it is part (Bateson [1972] 2000). Bateson's systemic thinking in relation to selves, families, ecologies and minds and how they influence each other speaks directly to the perspective we cultivate in this book.

Social contagion processes often involve influence in the form of affect (Grøn 2017), responses to phenomena to which we cannot not respond (Waldenfels 2011), resonance (Rosa 2019), moods (Throop 2017), violence (Meinert and Whyte 2017), haunting (Good 2015; Meinert 2019), stigma (Lawlor and Solomon 2017; Mattingly 2017), and processes of imitation and diversification (Hollan 2017). The entities which influence each other may sometimes be hard to delineate: where does one self begin and another stop? Where is the limit between an individual and a group? Some forms of social contagion tend to run in the family through kinship connections (Meinert and Grøn 2019). Kinship connections highlight the fact that contagion is often ambivalent and tricky to deal with because it involves both positive and negative influence and nutritious and poisonous aspects (*ibid.*). Contagious kinship connections happen through various processes of belonging (Grøn 2019), child witnessing (Han and Brandel 2019), fear (Seeberg 2019; Stevenson 2019), love (Garcia 2014, 2019), and bitterness and affection (Oboke and Whyte 2019). Attempts to deal with contagious kinship connections may involve family therapy (Kuan 2019) or making cuts in physical bodies and family relations (Grøn 2019). Processes of social contagion may be unspecific and diffuse like the subconscious impact of subtle advertisements, yet when contagious processes happen there is often no doubt that they take place – we find ourselves buying products we have seen in advertisements, as do our friends – and we experience their effects, sometimes in the form of large-scale epidemics.

Whereas contagion often concerns processes involving human and non-human actors, sometimes connected in zoonotic epidemics, the concept of contamination involves a process (of a substance) that spreads in an environment, often with some kind of pollution being involved, at a pace which may be either rapid or diffuse and slow. Contaminating and contaminated areas may be large-scale, such as the environment, the market, the food industry or the pharmaceutical industry, but at the same time processes of contamination may be subtle, involving slow, poisonous contact, or marketing strategies directed towards permeable selves or bodies. Social contamination may involve processes of environmental influence, such as when the atmosphere in a room or the history in a landscape touches or affects us (Grøn and Meinert 2017). Processes of social contagion and contamination are contingent upon the perceived permeability of selves (Napier 2013; Taylor 1989; Throop 2017) and mindful bodies (Scheper-Hughes and Lock 1987); and upon the way in which social, historical and environmental influences saturate subjectivities and materialities. Actors – individuals, families, institutions, states – may take action to attempt to create protection against contamination, and may develop immunity and resistance to certain influences.

One common approach to the study of new epidemics has been inspired by Ian Hacking's theories of dynamic nominalism (Hacking 1986, 1992) to think about the spread of diagnoses, categories and representations. The spread of diagnoses has been pointed out in relation to various mental problems such as autism (e.g. Grinker 2009; Seeberg and Christensen 2017), ADHD (e.g. Rasmussen and Meinert 2019; Timmi and Taylor 2003), depression (Brinkmann 2010) and PTSD (e.g. Fassin and Rechtman 2009; Young 1996). In Uganda, Whyte has pointed to the related phenomenon of awareness epidemics in relation to certain noncommunicable diseases such as diabetes and high blood pressure (Whyte 2012). This social constructionist perspective is well researched and documents that diagnostic and categorizing processes are contagious and play an active part in the configuration of epidemics. However, in this book we regard social contagion as more than social constructions that create epidemics of awareness, definition and discovery. We are interested in (social) contagion as intersubjective processes of influence and affection that are intertwined into epidemic configurations.



## Configuring

We propose the analytical concept of configuring – employing the present progressive grammatical form to emphasize that a *process* is involved – in an attempt to form a framework for describing the figures and con-figures that create epidemic processes of biosocial contagion. As mentioned earlier, we build upon Rosenberg’s description of configuration theories. Rosenberg describes and contrasts configuration explanations as an empirical and historical phenomenon that was common before contamination explanations became dominant with ideas about infection and microbiological contagion. Configuration theories, writes Rosenberg, typically explained epidemic disease as something that was caused by a disturbance of the social order, with health described as a balanced and integrated relation between human and environment (Rosenberg 1992: 295). We have suggested the term configuring as an analytical concept to describe dynamics that collaborate when social contagion happens (Seeberg and Meinert 2015). Configuring can be regarded as a form of contextualization (Seeberg and Christensen 2017). But whereas a context analysis is potentially endless (Dilley 1999), configuration analysis is an attempt to foreground certain figures and relations and their influence.

An epidemic is an expression of a specific configuration of various forms of social contagion and contamination, as well as other biosocial and material processes that happen over time. Specific epidemic configurations often contain several diseases and assemblages of problems simultaneously, and are therefore syndemic (Singer and Clair 2003). Epidemic configuring is dynamic in that problems and diagnoses change according to which phenomena, actors, institutions, practices and material conditions are involved (Williams and Meinert 2017). Diagnostic categories and biological and social phenomena of very different kinds (e.g. PTSD, spirits, viruses, social media) can all play a part in specific configurations. But configuration analysis can also be applied to understand transformations of epidemics when an epidemic disease develops antimicrobial resistance (Seeberg 2020).

We use the concept of configuring in order to achieve a broader understanding of (social) contagion and epidemics that reaches beyond causal and essentialist explanations (Seeberg, Roepstorff

and Meinert 2020) as well as purely social constructionist models. Thinking in terms of configuration involves epistemological reconceptualization, with causal relations between various factors and units being supplemented by heuristic tools to explore a phenomenon. This perspective involves an epistemological shift from a correspondence theory about knowledge in which concepts work as mirrors of reality (Rorty 1980), to a pragmatic, testing and uncertain epistemology in which concepts are tried out as working hypotheses (Whyte 1997). In these contagious processes, local epistemologies, perceptions, assumptions, fears and apprehensions appear. Ideas about well-being that are historically grown, culturally specific and intersubjective in nature are all part of configuring processes.

In epidemiology, one effective way of thinking about processes influencing epidemics is in terms of disease agents, confounding factors and outcomes, and epidemiological analyses tend to single out specific agents and factors in lifestyles or life conditions as important for epidemic outcome. Policy approaches to the epidemics of non-infectious diseases have largely focused on lifestyle – understood as behaviour deemed risky in relation to specific conditions such as diabetes, lung cancer or obesity. Whereas these approaches focusing on lifestyle have no doubt been extremely important with a view to identifying specific dangerous practices, they have also been criticized for being behaviouristic, individualistic and mechanical (Coreil et al. 1985). Several Foucauldian analyses have pointed to the problematic promotion of prevention and treatment regimes, that seek to discipline the self (Petersen and Lupton 1996) and medicalize life itself (Rose 2007), whereas health issues often have to do with broader life conditions beyond individual lifestyle choices. Labelling certain illnesses as ‘lifestyle diseases’ can be thoroughly misleading (Seeberg and Meinert 2015). But in Weber’s original framework, lifestyle was intimately linked to life conditions, through the concepts of *Lebensstil* (style of life), *Lebensführung* (conduct of life) and *Lebenschancen* (life chances). Life chances were conceived in socioeconomic terms (Weber 1999 (1922): 709–10) and linked to concepts such as class, whereas the first two concepts were linked to choice (Seeberg and Meinert 2015: 58). In our conception of configuring contagion, we are inspired by Weber’s broad, sociological way of defining influence in our attempt to expand more narrow epidemiological definitions of agents, factors and outcomes.



We find the thinking of Singer and colleagues about syndemics useful for understanding how processes in which diseases or coinfections are intertwined reinforce each other at both biological and societal levels (Singer 2009; Singer and Clair 2003). Syndemic scholars have considered how infectious diseases create co-morbidity, and also how these and other diseases and conditions are intertwined with economic and social conditions as well as with ecological and environmental factors in what Singer defines as eco-syndemics (Singer 2012). Our thinking about configuring is in line with this school of thought, which reminds us that diseases are seldom separate units, but are interwoven phenomena in which life conditions and health problems are combined. This book's distinct contribution with the concept of configuring contagion highlights *the process of how* this interweaving and spreading happens in everyday lives, in networks of friends, family and social groups, under specific political and socioeconomic conditions, with an eye to how materialities and local epistemologies play into these processes.

## The Contributions

The chapters in this book are based on contemporary ethnographic studies from the global north and south that develop critical theoretical reflections about the dynamics of configuring contagion in global health. The chapters are based on empirical material from Denmark, India, Indonesia, Niger, Oceania, Uganda, the USA and Latin America. The chapters will appeal in particular to anthropologists and other scholars within the social sciences and humanities with an interest in epidemics, as well as psychologists, psychiatrists and global health specialists interested in cultural health studies. The insights developed in this volume cut across disciplines with the intention of exploring how specific contexts, cases and diseases dynamically co-configure contagion.

Each chapter focuses on the configuring of contagion in relation to a specific disease, condition or social phenomenon, including suicide, autism, ADHD, trauma, spirits, abortion, obesity, porn addiction, Zika virus and drug-resistant tuberculosis. The chapters develop a range of different theoretical approaches to understand the configuration of contagion, and they engage with local perceptions of, fears of and words for contagion or polluting and

contaminating substances, as well as local ideas about processes of configuring biological problems and social contexts. The chapters highlight the fact that contagion and configuration theories are not merely analyses that are externally imposed by the authors, but are shaped locally by historically specific and socially and ecologically attuned intersubjectivities. The chapters are developed from a selection of papers presented at a symposium on configuring contagion in biosocial epidemics organized by the Epicenter (the Centre for Cultural Epidemics) at Aarhus University in 2017.

The first two chapters of the book focus on suicide and its social configuration. Susan Reynolds Whyte and Henry Oboke write about gender discrepancies in completed suicides in northern Uganda. Based on archival material and contemporary data, they show that the gender discrepancy in this area has increased, with far more men than women committing suicide. The authors consider changing configurations of gender relations within families and between partners, and how masculinity has been challenged by war, encampment, disease, alcohol and poverty. Changes in gender configurations are related to broader patterns in political economy. Whyte and Oboke are not arguing for a single cause of the spread of suicide among men. Instead, they examine experience-near accounts in the context of these wider transformations in order to explore how contagious and contaminating affect relates to shifting configurations. Men talked about being bitter and angry because they felt they were not properly recognized and respected. The authors point out that configuring is also an endeavour undertaken by their interlocutors, who relate specific suicides to gender conflicts and failed filiation to fathers.

Edward Lowe's chapter draws on recent research on how the suicide problem is socially and experientially configured in the region of Oceania, particularly in Chuuk Lagoon. The chapter begins by presenting the way in which suicide cases are used in publicly mediated accounts as a metaphor for troubles associated with modernization and globalization, and the potential harm that these two processes cause in local indigenous populations. It then contrasts these generalizing epidemic readings of suicide to the way suicide is configured through lived experience as an endemic social practice in Chuuk and elsewhere in the region. One of the key points of focus in Lowe's ethnographic material is how suicide can grow out



of a larger set of practices that aim to restore relational harmony and balance in the aftermath of a sudden breakdown in key social relationships. Lowe contrasts this endemic reading of suicide in Oceania with the way suicide is understood in the specialist literature in the United States, where suicide is regarded as a way to end painful internal psychological suffering connected to disruptions in a person's autobiographic self-making. In the concluding section, Lowe discusses how suicide both as discursive metaphor and as a phenomenon in lived experience can be understood as varieties of what Heidegger called presences that become manifest out of the larger, dynamic lifeworld, as well as discussing the implications of this insight for the study of biosocial epidemics.

Cheryl Mattingly and Stephanie Keeney Parks take us into the world of autism and incarceration in the African American community. They take up the idea of syndemics by considering the configuration of intellectual difference (autism) and the rapid rise of incarceration in many minority communities within the United States. Mattingly and Keeney Parks consider autism as a mental health classification that has taken on epidemic proportions, as it intersects with the epidemic of mass imprisonment within the African American community. The authors explore how African Americans employ local epistemologies to conceptualize the interconnection between the two phenomena. More specifically, they explore how African American parents of boys at risk of an ASD diagnosis in particular *respond* to the threat of this syndemic and the inoculating strategies they employ as they try to protect their boys from the life-threatening spectre of a life of unemployment and potential incarceration. The parents feel haunted by their future prospects. In their chapter, Mattingly and Keeney Parks draw on hauntology perspectives to think not just historically, but in terms of the return of phantasmal forms from buried pasts.

Lone Grøn focuses on the configuration of biosocial affection in the obesity epidemic in Denmark by exploring relatedness maps drawn by Danish families in which obesity has run for generations. Building on earlier work (Grøn 2017), where she suggests 'affection' as a phenomenological theory of social contagion that is characterized by indeterminacy and intersubjectivity, Grøn adds two further features. Firstly, the family experiences of contagion highlight affection as mutuality, as love, as belonging which is,

however, both nourishing and poisonous, making contagion as affection especially haunting. Furthermore, the affective ties do not adhere to distinctions between what is biological and what is social, but emerge between material, biological, psychological, cultural, social and historical processes. Secondly, the configuration of diverse affections is specific and particular, which leads to different kinds of obesity, not only between families, but also within them. Obesity thus appears not as a monolithic category, but as obesities in the plural. Grøn argues that the notion of affection challenges prominent ideas about individual lifestyle and linear and demarcated causal pathways, which characterize contemporary debates and interventions as well as epidemiological approaches to obesity. The chapter presents instead an experience-near and anthropological approach that takes seriously the fact that academically demarcated territories between disciplines – and between individuals, others and the world – might not be helpful for understanding how obesity spreads.

While trauma may be seen to run in the family, like obesity, it can also affect much larger collectives in complex configurations. Jesse Hession Grayman, Mary-Jo DelVecchio Good and Byron Good explore changing social configurations of trauma in post-conflict, post-tsunami Aceh in Indonesia. This chapter interrogates the observation that a trauma triggered by conflict and/or a tsunami and experienced by the majority of Aceh's population reduced the stigma of mental illness, by reframing a 'fear of the other' (a contagion and confusion reaction to mental illness) as recognizable and deserving of compassion. High affect was associated with both the conflict and the tsunami, and the emotional intensity fed earnest approaches to reach out to treat those suffering from both disasters. This affective dimension is particularly powerful in energizing new ways of thinking about the mentally ill. Particularly notable is a professionalized willingness to confront the enormous challenges in caring for the seriously mentally ill, and to confront remainders of trauma embedded in historical events.

Lars Williams and Lotte Meinert discuss an extended case of trauma and spirit affliction after the war in northern Uganda to explore the configuration of affliction in a specific life, family and situation. They argue that being afflicted with trauma or spirits is a way of being 'touched' and contaminated by violence and death.



However, the experience is also configured as trauma and spirit affliction by psychotropic medication in the hospital, by local healers, and by prayer in church. In this way, contagious and contaminating elements from a legacy of violence con-figure – give figure to – afflictions of spirits and trauma. The authors describe how multiple treatment options for mental illness contribute to the configuration. When people combine different treatment options, the ways in which illness is expressed are transformed and configured through different paths of recovery. There is a market of healing with churches, NGOs, hospitals and traditional healers offering their services and tapping into each other as people travel through them in search of relief from their disorders.

Adeline Masquelier, Abouzeidi Maidouka Dillé and Ly Amadou H. Belko consider the relational dimensions of Nigerien girls' spirit possession as affliction through the analytical prism of contagion. By taking spirit possession and haunting to be a form of circulating affliction rather than individual resistance or somatization, or a way of dealing with conflict, Masquelier, Dillé and Belko consider schoolgirls' perceived susceptibility to others. They draw on the model of contagion elaborated by Gabriel Tarde to account for the rapid spread of ideas, fashions, technologies and crimes across a population. Through a focus on imitation – the ability to affect and be affected, consciously and unconsciously, by others – the authors highlight the extraordinary 'magnetic' power people can exert over each other in interactions. The authors argue that the mass possession of Nigerien schoolgirls indicates the way in which people incorporate others into themselves and are themselves embodied in others – a process that can be described as a contagion without contact.

Haunting may also be configured through internet-mediated fantasies and images, as shown in Doug Hollan's chapter on internet porn. He reflects on his meeting, as a therapist, with men who – although initially complaining about things such as depression, anxiety and various family and social problems – eventually shared their viewing of internet porn and the impact this had on their social, emotional and erotic lives. The chapter explores how contamination by sexual images and fantasies is configured and shaped by both social and personal factors. Hollan examines the role that culture, technology and institutions play in suppressing,

articulating, exploiting or denying human fantasy and imaginative thought, and in turn, how the protean and creative aspects of human fantasies feed into and transform the sexual images currently available on the internet. The contamination the men felt when encountering sexual images on the internet affected them in different and non-transparent ways, depending on the history and development of their sexual and erotic lives.

Social contagion may seep through international and government bodies in ways that worsen the health outcomes of an unfolding epidemic. Lynn Morgan has studied the management of the outbreak of Zika virus in Latin America that started in 2015. Health agencies responded with education and prevention campaigns focused mainly on mosquito control and travel advice. Conspicuously absent from US and international Zika control campaigns was any attention to the need for contraceptives or prenatal screening, and nor did campaigns inform women about pregnancy termination options even in places where this was legal. By amplifying only certain kinds of knowledge, the authorities were complicit in producing ignorance and extending the suffering. This chapter argues that the configurations of contagion surrounding the Zika epidemic in Latin America reveal a widespread pattern of reproductive governance that relies on discrimination, stigma, secrecy and political cowardice. Meanwhile, as of 9 February 2017, 2,700 children in the western hemisphere had been confirmed with a congenital syndrome associated with Zika virus.

The configuration of contagion of tuberculosis (TB) has established it as the 'quintessential social disease' (Cervantes 2016) due to its preference for the socially disadvantaged and the stigmatization it attracts. It is perhaps less obvious that its transition to increasingly drug-resistant forms itself constitutes a form of biosocial contagion. In their chapter, Jens Seeberg, Bijyalaxmi Rautaray and Shyama Mohapatra explore drug resistance as a product of particular patterns of interaction between microbes, medicines and humans. Their analysis focuses on frictions experienced in the lives of people infected with drug-resistant TB in India as seen through the figures of *Pharmacon*, *Regimen*, *Regula*, *Clinic* and *Oikonomia*. Seeing these figures as production sites of resistance adds a new angle to the discussion of biosocial relations of production (Palsson 2016) in the field of tuberculosis. While infection with DRTB places



the people affected in a situation of extreme vulnerability, individual life stories point to the mutuality of resistance and vulnerability. Actions of opposition, rejection and activism partially shape the configuration of the DRTB syndemic in its locality, pointing to resistance in vulnerability as much as vulnerability in resistance.

The volume concludes with an afterword by Byron Good reflecting on how the lens of configuring contagion may contribute to the understanding of very different kinds of biosocial epidemics and their mitigation.

### **Lotte Meinert**

Professor, Department of Anthropology  
Aarhus University, Denmark.

### **Jens Seeberg**

Professor, Department of Anthropology  
Aarhus University, Denmark.

## **Notes**

1. See <https://www.who.int/news-room/fact-sheets/detail/noncommunicable-diseases>. Accessed 1 June 2020.
2. <https://www.cdc.gov/csels/dsepd/ss1978/lesson1/section11.html>. Accessed 14 June 2021.
3. Gabriel Tarde's work is often opposed to Durkheim's owing to his idea that the whole is always less than the sum of its parts; but here we are particularly interested in his ideas about imitation as a basic form of sociality with a view to thinking about how we might conceive of contagion as social influence.

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