Community Dent Oral Epidemiol 2014; 42; 481–494

Community Dentistry and Oral Epidemiology

Unsolicited Narrative Review

Social oral epidemi(olog)^{2}y where next: one small step or one giant leap?

Sarah R. Baker and Barry G. Gibson

Unit of Dental Public Health, School of Clinical Dentistry, University of Sheffield, Sheffield, UK

Baker SR, Gibson BG. Social oral epidemi(olog)^{2}y where next: one small step or one giant leap?. Community Dent Oral Epidemiol 2014; 42: 481–494. © 2014 The Authors. Community Dentistry and Oral Epidemiology Published by John Wiley & Sons Ltd.

Abstract – Since the early 1990s, there has been heated debate critically reflecting on social epidemiology. Yet, very little of this debate has reached oral epidemiology. This is no more noticeable than in the field of oral health inequalities. One of the significant achievements of social oral epidemiology has been the persistent documentation of social patterning of oral disease. Nevertheless, where social oral epidemiology has fallen down is going beyond description to *explaining* these patterns. Thinking how and in what way things happen, not just in relation to oral health inequalities but also more broadly, requires a more creative approach which links to scholarship outside of dentistry, including the work from critical epidemiologists to that within the social sciences. The aim of this review study is to provide a critical commentary on key aspects of more general epidemiological debates in order to inform and develop social oral epidemiology theory and methodology. In the first section, '*Where are we now?*', six key debates are reflected upon: (i) analysis of variance versus analysis of causes, (ii) the fallacy of independent effects, (iii) black box thinking, (iv) theory and the understanding of mechanisms, (v) individualization of risk and (vi) the meaning of 'social'. In the second section, '*Where to next?*' we draw on a number of fundamental issues from within the social science literature in order to highlight possible channels of future inquiry. Our overriding goal throughout is to facilitate a critical engagement in order to improve understanding and generate knowledge in relation to population oral health.

Key words: causation; dental public health; health inequalities; lifecourse; oral epidemiology; risk factor; theory

Sarah R. Baker, Unit of Dental Public Health, School of Clinical Dentistry, University of Sheffield, Sheffield, UK
e-mail: s.r.baker@sheffield.ac.uk

Submitted 14 January 2014;
accepted 6 June 2014

Epidemi(olog)^{2}y or epidemiologogology is the study of epidemiology. Over the last two decades, many senior epidemiologists (1–12) have critically reflected on epidemiologogology. Surprisingly, very little of this debate has reached oral epidemiology. Indeed, whilst there have been some moves towards new approaches such as, multilevel modelling and a lifecourse perspective, epidemiological theory and method in dentistry still lags some way behind more general debates.

One field in which this is most noticeable is that of oral inequalities. One of the significant achievements of social oral epidemiology has been the persistent documentation of social patterning of oral disease. Indeed, there can no longer be any disagreement that social inequalities in oral disease are very large, very robust and global. Nevertheless, where social oral epidemiology has largely failed is going beyond description to *explaining* these patterns. To do this, we need to move beyond

doi: 10.1111/cdoe.12118

the almost exclusively methodological focus within some strands of social oral epidemiology. To move from studies which may be methodological rigorous but essentially pedestrian to more creative and bold social oral epidemiological research which attempts to *explain* and *understand*; to address the *why and how* questions and not always the *what?* Thinking how and in what way things happen, not just in relation to oral health inequalities but also more broadly, requires a more creative approach which links to scholarship outside of dentistry; including the work from critical epidemiologists noted above to that within the social sciences.

The aim of this paper is to provide a constructive but critical commentary on key aspects of these more general debates in order to inform and develop social oral epidemiology theory and methodology. In the first section, '*Where are we now?'* six key debates will be reflected upon; (i) analysis of variance versus analysis of causes, (ii) the fallacy of independent effects, (iii) black box thinking, (iv) theory and the understanding of mechanisms, (v) individualization of risk and (vi) the meaning of 'social'. In the second section, '*Where to next?'*, we will draw on a number of fundamental issues from within the vast social science literature in order to highlight possible channels of future inquiry.

Where are we now?

Much of social oral epidemiology today is underpinned by the risk factor approach. Within this approach, oral disease is viewed as resulting from multiple causes, determinants and risks involving a 'web' of interactions between individual and environment. In line with this, the traditional risk factor approach has been extended to incorporate not just clinical measures (e.g. DMFT) but also individual (e.g. smoking, self-esteem) and social factors (e.g. socioeconomic status) (see the 'eco-epidemiology approach', 11–12).

The goal of the risk factor approach is about quantification: identifying one or more risk factors and estimating their main effect whilst controlling for the effect of all other factors. To date, there have been some rigorous, high-quality studies with some important findings that have employed such techniques (13–18).

Nevertheless, despite these excellent examples and the importance of this approach, there are a number of problems in the field generally. Firstly, the risk factor approach has resulted in a plethora of cross-sectional analytic studies showing small associations between various risk factors and a few key outcomes (e.g. oral health quality of life, caries rates, periodontal disease). Secondly, oral epidemiology has primarily become a discipline of technique rather than substantive understanding. A consequence of this problem is that we have a pile of observations but with little understanding of how to 'join the dots' (i.e. the strands of the web). Yet, as Frost (1936), the first US Professor of epidemiology said 'Epidemiology is .... more than the total of its established factors. It includes their orderly arrangement into chains of inference' (19, p. 107).

Analysis of variance versus analysis of causes

This trend has mirrored that in epidemiology, more generally, wherein statistical prediction (the association between exposure/risk factor and disease) often passes for explanation (1, 2, 4, 11, 12). There is a crucial often overlooked difference, however, between explaining variance and explaining disease causation. As Ashley–Perry's Statistical Axiom Number five states 'the product of an arithmetical computation is the answer to an equation; it is *not* the solution to a problem' (Dickson, 1978 cited in 20). Analysis of variance should therefore be the first – but not the only – step in epidemiological analysis when testing for associations (and causation).

The discussion of causation in epidemiology is based on a number of criteria/models, for example, the nine criteria of causation (21), sufficient/component model (22) and the counterfactual model (23). Take as an example, the counterfactual model which suggests that the probability of disease in the exposed that would have occurred had they not been exposed. The goal of adjustment is to control for confounding. In this way, adjustment is equated with explanation. We would argue that this is the wrong definition of explanation. In addition, that when it comes to social risk factors, the counterfactual (i.e. the idealized unexposed) is problematic (24). Take as an example, the estimate of the effect of race on oral disease (i.e. the risk ratio of being black). Black people are the exposed group; so the right hand side of the equation is the probability of the outcome among black people that would have occurred if they had not been black. Leaving aside whether 'blackness' is the absence of 'whiteness', a black person who is not black cannot be considered the same person. They would

not have experienced the same environment (social, psychological) as someone who is white. So the question is, what finite set of covariates could logically make black and white people exchangeable? Should we adjust for neighbourhood SES or perceived stress or parental attitudes? All of which may shape 'black' identity (example adapted from 25).

As Kaufman and Cooper (25) state, there are two important points here: (i) these variables are not confounders but important in the complex context impinging on the outcome (e.g. oral disease) and (ii) it is not possible to control for everything to make two groups interchangeable with respect to all exposures, behaviours, etc. that bear on oral disease. In this way, there exists no logical counterfactual state (whites who are not exposed to whiteness) to support this model of disease causation.

Is there a similar problem when estimating potentially modifiable risks such as those associated with social inequalities? For example, what is the meaning of an independent effect of social class? In order to describe what proportion of the observed working–upper class difference in outcome is due to classism, this would imply a counterfactual equality between working class and upper class in the absence of all exposure differences; the only defined counterfactual is working class people exposed to some classism and working class people unexposed (25). This is also the case for behavioural risk factors, for example, a smoker, who does not smoke. If the counterfactuals held, would they otherwise be the same person? We would argue 'no' because other complex, interlinked determinants and concomitants of smoking are also going to change with the counterfactual (24). The widely held common risk factor approach would also suggest 'no' given evidence for the clustering of health-related behaviours (e.g. those who smoke are more likely to drink, to have a poor diet and so on) (26).

If one reads any of the vast literature on social inequalities in oral health, invariably the authors will state that to investigate, for example, the effect of income, they have adjusted for education, age, etc. so that 'important sources of confounding are controlled for'. But, in the real world, those other conditions are not and can never be held constant – they vary and interact in particular structured ways, that is, people do not arrive at an income through a randomization process – rather they arrive at an income through a dynamic life trajectory that is shaped by social relations and structures (25). Yet, the current approach within social oral epidemiology negates any such *social structure* that exists to condition the relation between variables.

In response to this, there have been since the early 1990s, a rise in multilevel modelling studies that have sought to bring social structure into oral epidemiological analysis (e.g. 15–18). However, the tendency in such studies has been to tack on a dummy variable representing a composite group-level ('neighbourhood') effect. This is little more than a residualizing effort to explain variance beyond that attributed to individual-level independent variables. Like all regression models, they still suffer from the fundamental problem of assessing relationships between 'independent' variables and 'outcomes'. Such models do not take into account dynamic and reciprocal relations (e.g. neighbourhoods influence individuals and not vice versa), discontinuous relations or changes over time (e.g. over the lifecourse). Furthermore, the residual 'contextual' variable may represent a wide array of potential mechanisms. Even if the approach results in significant effects, there are so many competing alternative explanations, and without any underpinning theory, it is not possible to interpret which might be correct (26). For example, is it the stress experience and the resulting coping behaviours, such as smoking or increased alcohol consumption, that increase the probability of disease outcome? If so, the stress experience is likely to be shaped by a person's social environment including their peer network, family or immediate neighbourhood. This creates multiple versions of the exposure; it is not possible to model or account for such dependencies in traditional epidemiological analysis based on the notion of independent effects (2, 4, 25).

The fallacy of independent effects

The multiple risk factor approach is dominated by the notion that the whole can be understood by breaking down into component parts, that is, the population into 'independent' individuals and the individual into 'independent' risk factors. What could be termed disease causation in chunks. Such reductionism is a process of simplification, which can and has been useful in pushing forward knowledge and understanding within social oral epidemiology. Nevertheless, as Levins (27) said 'the art of research is the sensitivity to decide when a useful and necessary simplification has become an obfuscating simplification' (p. 105). The risk factor approach *can* help towards our understanding

of the causes of oral disease, but the danger is in *only* doing such studies. This is because by dividing and isolating components (no matter how many you go on to add in at a later date), it will not be possible to understand a system in which there are a myriad of parts all of which are interrelated. Indeed, it could be argued that all that has happened is that the 'multicausal model' has been reduced to a collection of unicausal relations (2). The *relational* aspects of the system are largely ignored, and yet, there are massive multiplicity of connections between Xs and how Y is realized. Each connection is a *mechanism*.

The question for any particular oral health problem should not just be what diverse influencers there are for that outcome, but how they come together and interact (i.e. their mechanisms of action). Take as an example, the many influencers of a 'simple' problem such as tooth decay: (i) endogenous factors (enamel or dentin), (ii) individual factors (behaviour – dietary habits, sugar intake, going to the dentist, toothbrushing, beliefs about the importance of oral health, dental anxiety, income), (iii) neighbourhood factors (availability of grocery stores, advertising of certain foods, access to dental services, fluoridation policy), (iv) school/work factors (availability of sugary drinks, oral health education), (v) regional factors (regulatory marketing of high sugar foods, water fluoridation policy) and (vi) national factors (public policy on dental services, support for agricultural products) in addition to (vi) a lifecourse perspective (history of breastfeeding, maternal and paternal oral health and practices).

This example, adapted from the work of Galea et al. (28), highlights the difficulty for the current social oral epidemiological paradigm, that is, how to conceptualize and then analyse the contribution of all of these influencers when fixated on isolating independent causes? In order to move away from the linear causal thinking commonplace in, for example, upstream–downstream metaphors or proximal-distal factors, we need to begin to tackle inter-relationships. Identifying such inter-relationships will be necessary to understand the complexity of social systems in which individuals and populations live (i.e. the eco-epidemiological approach, 12). Causation will therefore not be the property of an agent (a factor), but one of complex systems in which the health process is embedded. This will require a paradigm shift away from the current static 'black box' thinking prevalent in social oral epidemiology.

Black box thinking

The black box paradigm (29), which originates from cybernetics, is used in diagrams as a quick way of alluding to some complex process: in its place a box is drawn with input and output arrows. Thus, the black box holds all of those mechanisms and pathways that tell us about how something works. As a tool, the black box is useful for simplifying complex processes to push forward knowledge. Indeed, our understanding in social oral epidemiology has increased with the use of multiple regression models which include a number of inputs and one output rather than the traditional simple one-in-one out.

Other recent advances in oral health research include structural equation modelling (SEM), an advanced statistical technique, which allows for the simultaneous testing of direct and indirect (mediated) relationships between many factors at the same time (e.g. DMFT, self-esteem, dental attendance, neighbourhood SES) (30). Compared with traditional regression models, the inclusion of biological, psychological and social factors and their inter-relationships allow for a better representation of the biopsychosocial model and may highlight potentially important pathways that could be investigated further. Yet, SEM is only as good as the *a priori* theory upon which it is based. Take for example, those SEM studies that have attempted to apply the Wilson and Cleary (31) model to further our understanding of the determinants of oral disease and oral health quality of life (e.g. 32–34). Even within these studies, individual processes are still housed in an 'individual' black box (sense of coherence, self-esteem etc.), and social processes are still housed in an 'environmental' black box (social capital, income, education etc.). Thus, theory-driven SEM still sidesteps the interior workings of the black box; it obscures *how* oral disease is produced and what exactly it is *about* income, self-esteem or sense of coherence that contributes to oral disease. So, despite the precision of its concepts, neatness of its prediction, strength of its method and significance of its statistical associations, the methodological 'toolbox' of social oral epidemiology remains black-boxed. We would argue that what is needed is a shift from thinking about 'variables' to an emphasis on 'mechanisms'.

Theory matters: Understanding mechanisms

The 'theory' underpinning much of social oral epidemiology to date is the web of causation, and the

multiple risk factor approaches the logical mathematical formulation of this 'theory'. First mentioned in the 1960s in the first US epidemiological textbook (35), the web of causation was never intended to be a theory. Theories attempt to explain why. Yet, the web tells us nothing about how the strands come together or influence one another; let alone in relation to public health, how to break selected strands of the web (4).

There is sparse talk or application of theory in social oral epidemiology. Of the 672 articles indexed by 'oral epidemiology' or 'dental public health' in web of science from 1900 to 2012, only three included the word 'theory'. Yet, theory is not just philosophical or part of academic debate. In relation to epidemiology, theory can help us explain causal connections, the who, where, when and how to intervene to improve the population's oral health. Take an example, there has now been a great deal of research describing oral inequalities in most parts of the world. Many studies tell us that poverty is linked to say higher caries rates; but what we do not have is a great deal of understanding of how the strands of the web actually 'work', that is, does poverty increase exposure to stress and adversity, or influence psychosocial resources such as sense of coherence, mastery, self-esteem or social support which may be mediators in the chain (36). Does it influence the development of unhealthy lifestyles (37) or do inequalities produce an unequal distribution of resources that allow people to avoid risks and adopt protective strategies (38).

By thinking in terms of black boxes, we are impeding our capacity for critically evaluating what works and what does not when it comes to reducing inequalities. As a consequence, we risk intervening in ways that widen rather than reducing inequalities and this means we could, in fact, be causing harm when we produce interventions to reduce inequalities (38). Thinking in terms of theory and concepts would be advantageous for many other reasons. At a basic level, it would avoid misspecification within our statistical models. If a factor is not in a causal chain, then we can control for it within a regression model but, and if it is in a causal chain (e.g. perceived stress links periodontal disease to quality of life), then it is statistically wrong to adjust or control for it. Far more, however, than the appropriateness of the analytic strategy, it is about improving *our ability to critically evaluate* what we are doing rather than research simply as a data collection exercise.

Ironically, the development of complex statistical techniques that allow for multivariate statistical tests without much knowledge of underlying mathematical principles has blunted our ability to draw on theory. By having the technique, and adding more and more into the model, the whole process of explanation is left implicit – the assumption being that the more we add into the model, the better or more comprehensive our understanding will become. This has meant that the causal model underpinning social oral epidemiological research and indeed the assumptions on which it hinges are very rarely discussed. At a more serious level, the types of questions being asked are being partly driven by the analytic methods available (rather than vice versa) and more recently being dictated by the data available (e.g. the exponential rise in secondary analysis of existing datasets). Rather than describing an observed difference, we need to move towards an explanation of the processes leading to the observed difference. Such explanation requires more complex models, which need to be underpinned by theory.

Why? Theory encourages us to think critically. We attempt to prove or disprove something rather than collect 'observations'. As Frost (1927) said many decades ago '. . . in collecting facts about the distribution of disease, the purpose and view is always to arrive at a better understanding of its nature, sources, means of spread and eventually its control. This implies that the facts must be related to each other in such an orderly way as to establish a theory or philosophy of the disease' (quoted in 39, p. 107). As Krieger and Zrierler (39) note, ideas for studies, formulation of hypotheses and emergence of knowledge begin with a theoretical framework. Epidemiological theory also determines what we know, what we consider knowable and what we ignore. Even within the most theorized field within social oral epidemiology, oral health inequalities, the discussion has not kept pace with key developments in wider epidemiology (see 40 for review of the nine theories of health inequalities). Engaging with these debates and viewing social oral epidemiology as a part of wider epidemiological endeavour would be greatly beneficial for our understanding. Take an example – the fundamental causes theory of health inequalities.

An example: Fundamental causation

In social epidemiological studies, socioeconomic status (SES) – typically conceptualized as income, education and/or occupation – is often used as a

proxy for measuring and then explaining social inequalities. Fundamental cause theory (FCT) would hypothesize that these are not 'true' causes of inequalities. 'True' causes are more distal in the causal chain; they involve access to flexible resources such as money, knowledge, power, prestige or social connectedness. The antecedent causes of these flexible resources are the social, economic and political structures of society evidenced by the sociological study of stratification (which indicates where fundamental causes come from in the first place). These, in turn, influence multiple risk factors and multiple disease outcomes (38, 41). Fundamental causes are therefore metamechanisms or 'factors that put people at risk of risks'.

One key premise of FCT is that the effect of these fundamental causes will not be removed by modifying the intervening variables in the pathway (38). This is because individuals with higher SES will always be able to utilize resources to mitigate ill health. Only when individuals cannot use resources will this inequality disappear, for example, a health condition where an individual cannot avoid risk or adopt protective strategies. One can see then how using a theory (FCT) helps derive the hypothesis (no inequality in nonpreventable disease), which in turn drives the analysis (preventable versus nonpreventable conditions). Interestingly, this preventable versus nonpreventable analysis of FCT has been supported in a number of areas (42, 43). Glied and Lleras-Muney (43) found, for example, that improvements in health technologies tend to increase disparities in health across educational groups because education enhances the ability to exploit technological advances, that is, the most educated can afford, make better use of and adopt new technologies first. From this, we can see that flexible resources are likely to be dynamic, that is, they may facilitate the creation of new mechanisms linking SES and health, for example, through new technologies.

What does the application of FCT suggest for future inequalities research particularly in relation to oral health? Over recent years, there have been changes in treatment strategies for edentulousness, most recently, with the rise in implantology. Is it possible that people with flexible resources could use these (money, knowledge, social connectedness) to gain access to such improved treatment strategies? If so, we could hypothesize that future years may see a differential change in the impact of edentulousness resulting from the development of this technology that, in turn, has the potential to lead to a social patterning in the experience of oral health in older age.

It is possible, however, that emerging technologies may not always be linked to increased inequalities. This will depend on the nature of the technological change and the extent of its diffusion and adoption (42). Some emerging technologies may act to contract rather than to expand social inequalities. This is because they act to minimize the beneficial effect of social resources such that being of high status confers little advantage. One possible example is fluoridation. Fundamental cause theory would hypothesize that the relationship between high status and disease (e.g. tooth decay) should be reduced when compared to the same geographical area before fluoridation and to other areas where the water supply is not fluoridated. The importance is to examine social inequality gradients over time and how these are changed when treatments or other health technologies emerge (e.g. 44, 45). The primary research question being – does the gradient change when an outcome or condition transitions from being (essentially) untreatable to treatable?

A related question of interest is whether there are social inequalities in the diffusion of new technologies. These may arise from costs, access to better-quality dental care systems or specialists, as well as due to beliefs about the benefits of the innovation (43). One example is the diffusion of fluoridated toothpaste. If, according to FCT, high status persons invest more effort, inequalities will decrease when advantages of this effort are reduced. With the introduction of fluoridated toothpaste, rather than changing one's eating and drinking habits or quitting smoking, people simply had to brush their teeth with this new 'technology'. Did the introduction of fluoridated toothpaste coincide with a contraction of inequalities in tooth decay by decreasing the value of dietary change more likely to be adopted by higher status people?

Where's the context? Individualization of risk

The individualism that is characteristic of the risk factor approach has been dominant within epidemiology since the 1950s, just as it has within wider public health and societal discourse. Consequently, rather paradoxically, social epidemiology, a population science, has been reduced to the study of individual risk or lifestyle factors (1, 46). Such research asks the question; what is it about individuals, which means they are more likely to 'get' disease? Much of the emphasis in social oral

epidemiology, to date, has been on behavioural risk factors: Do they smoke? Drink too much? Not brush their teeth frequently enough? Not visit the dentist for check-ups?

It could be argued that this focus has not arisen by chance but because those risk factors which are at the individual (rather than population) level are more controllable both by the individual themselves and by intervention strategies targeted at the individual (e.g. high-risk approaches). The difficulty is that such an individual-orientated risk factor approach leads to 'personal policy' changes (more toothbrushing, eating less, taking more exercise), which may have minimal impact on the health of the *population* (47).

This is not meant to imply that there is not excellent research in this area. Indeed, recent years have seen an increase in well-designed studies testing theory-driven tailored behavioural strategies aimed at individuals or 'high-risk' groups, with some notable positive results in improving oral health (48–50). Behaviour *is* an important determinant of oral health outcomes. Nevertheless, we would argue that the focus on behaviour and the individualization of risk have not been an overly successful approach *overall.* There are a number of reasons for this; firstly, whether documented changes are long lasting or translate into oral health improvements at a population level is far from certain. The theory of fundamental causation discussed above would caution that interventions targeted at individual behaviour might risk widening inequalities rather than reducing them. Secondly, most studies have been based on simplistic notions of behaviour that are treated as 'independent' effects devoid of any structural or social context (i.e. what places people 'at risk of risks'?).

Indeed, the study of the 'individual' within social oral epidemiology has been largely stripped of any context. The common risk factor approach identifies those things that a person does (their 'lifestyle') which mean they are more likely to get a range of diseases (26, 46). Their 'lifestyle' is almost exclusively focused only on behavioural aspects (smoking, eating sugar, oral health behaviours, not going for screening/check-ups). Lifestyles, however, are not just about behaviours and nor do they occur in a vacuum – they are not random or unrelated to structure – rather they are choices (not always conscious) influenced by life chances (51). By conceptualizing behaviours as simply activities under an individual's control inside a behavioural vacuum, it suggests that changing behaviour will come about through self-regulation whether that is cognitive (e.g. confidence in one's ability to act, strength of one's intention) or volitional/self-control. For example, the standard most influential health behaviour theories – health belief model, theory of planned behaviour, transtheoretical model and social cognitive theory – all use similar concepts that behaviour is rational and under individual control and that social context is exogenous to the individual rather than integral (see 52 for a review). Yet, the shortcoming of such theories has long been recognized within the psychology literature from where such models arise (53).

Furthermore, the focus on behaviour negates every other aspect of that person – their needs, motivations, emotions, thoughts, past experiences, desires, prejudices and expectations – as well as ignoring the structural (economic, political, familial) forces that shape that individual's exposure to risk. These forces cannot be reduced to a single entity that is, typically in social epidemiology, socioeconomic status. Socioeconomic status – material well-being, human capital and prestige – is a convenient summary term, but it should not be forgotten that it is just that (54). If we want to explain how the social becomes embodied, that is, how it 'gets into the mouth' to cause oral disease, we have to have a conceptual understanding of what is 'the social'.

The meaning of 'social'

SES categories, together with race, age and sex, are routinely collected in social oral epidemiological studies and always constructed as firmly fixed, 'natural' and *individualized attributes* (55). Typically, race, gender and even age are 'nuisance' variables to be controlled for statistically. Yet, as Krieger and Davey-Smith (56) succinctly state 'a person is not one day a woman, another day a Latina, another day heterosexual, another day a single mother etc' (p. 97). Race, gender, etc. are not objective variables, rather they are a set of relations and practices that inter-relate. These experiences cannot be neatly partitioned, nor are they confounders but are *in and of themselves of interest for our understanding*. 'Race', for example, could be a way to measure racism (9), physiological differences (57), possible cultural or behavioural patterns (58) or as a proxy of genetic differences (59) (example from 55). What then are the *causes* of *why* black men might have higher caries rates than white women?

Take another example – a 'simple' question – why do not kids in deprived areas go to the den-

tist? A traditional social oral epidemiological approach would be to include a measure of SES (one or more indices from income, education or occupation measured at an individual (maternal or paternal) level). More recent multilevel studies might add in a group-level effect typically, a measure of neighbourhood SES. In both approaches, there has been little attempt to conceptualize or consider what we mean by a 'deprived area' (54). Is it about the characteristics of individuals concentrated in particular places (a compositional resource-based explanation – families don't have the resources to take them) or the opportunity structures in local physical and social environments (a contextual resource-based explanation – too few dentists in the area and no good transport links to those further afield) or the sociocultural and historical features of communities, for example, shared norms, traditions and values [a collective explanation – within the local culture, oral health (and thus going to the dentist) is not seen as important] (example adapted from 60). There has been much work since the 1990s on such 'place-based' health research arising from geography and sociology on 'relational' views of context and space, that is, the interdependencies between people and places (60, 61). Much of this work may have direct relevance to oral inequalities research. We can already see, for example, that these three are not separate explanations; the collective is not separable from the contextual. In terms of translating this to research to understand the embodiment of such 'place' effects – how deprivation leads to higher caries rates – this is both methodologically and conceptually challenging as such effects will be cumulative and have a long time lag. What we need is a lifecourse perspective that resists transforming such 'layers of influence' questions methodologically into attributes of individuals at one point in their lives and thus loosing the properties (relatedness and connectedness) of the structure over time.

Where to next?

The previous section has highlighted some of the wider conceptual and methodological debates within epidemiology that might benefit social oral epidemiology. It has also introduced specific approaches that could be applied in the field such as, fundamental cause theory. In what follows, we consider further areas of interest that could add to and stimulate research in social oral epidemiology.

Complexity and dynamics: Lifecourse trajectories

Epidemiology is increasingly confronting the problems of complexity and dynamics. The lifecourse approach which centres on life trajectories as dynamic and shaped by many forces moves thinking from the two-dimensional static snapshot approach to one that considers an epidemiological triad – person, place and time. Such a perspective suggests that an individual's position is the end product of a life trajectory and that there are multiple risk exposures along the way (4, 8). Such a dynamic approach is the first step for social oral epidemiology in considering complex systems wherein disease or illness states arise from dynamic interaction within and between self-adjusting systems (psychological, emotional, cognitive, immune, nervous) not from a failure of specific components (62). Conventional analytic methods are unable to address situations where risk factors and resources are in flux and a state of interaction. The term dynamic complexity is used to describe such situations (63). Could dynamic complexity in social oral epidemiology be addressed by using systems modelling methodology in our future research programmes?

The central tenet of a systems approach is that complex behaviours of organic and social systems are the result of ongoing accumulations of people, material assets, biological or psychological states with feedback mechanisms (64). In systems in which different paths are dependent, actions at certain times called lever points or tipping points can have large effects on outcomes (65). As these authors outline, tipping points on the macro level are dramatic changes that arise quickly and usually unexpectedly (e.g. a slogan, political idea, a diet). A tipping point is a threshold effect (e.g. tooth whitening in particular subpopulations e.g. the media/advertising) at which individuals or groups adopt an idea or practice. At an individual level, these are likely to be influenced by social norms, whilst at a population level, by taxes or legislation (e.g. decrease in smoking following ban on smoking in public places in the UK). Is it possible by using such thinking to identify patterns both within individuals but also across individuals that predict the increasing likelihood of tipping into healthy behaviour lifestyles (e.g. going to the dentist, brushing twice a day)? This represents quite a

different approach – a move away from finding the 'magic-bullet' main effect and from a linear framework where unaccounted variance is relegated to 'error'. In complex systems, error is the thing of interest, as are the interactions. In complex systems, the interaction of factors may be analogous to higher-order interactions terms in regression models (5-, 10-, 15-way interactions), which cannot be examined traditionally because the research will be underpowered (most likely) as well as many of those interactions being nonlinear (65). As stated by Resnicow and Page, the blessing and curse of complexity is that it is conceptually and analytically complex! Complexity requires tolerance of heterogeneity, unpredictability and uncertainty; the opposite of the epidemiological paradigm and its drive for parsimony.

The conceptual basis of complex systems dynamic models has a long history, and these approaches are increasingly used in other disciplines (e.g. system biology, ecology, economics, organizational science, political science, 66). In epidemiology, there has been a growing call for such complex systems models (28, 67) although most have been limited to infectious disease with only a handful applied to noninfectious areas (e.g. smoking). Yet, they are the optimal analytic strategy for lifecourse perspectives in oral epidemiology where we are not concerned with disease as a static product at a given time (caries aged 10) but a result of circumstances *over* time.

Similarly, agent-based models, which are similar to testing 'what if' artificial counterfactual conditions, could be used in future social oral inequalities research to assess if certain public health interventions 'work', that is, whether such interventions influence patterning of say the use of dental services in a particular geographical area. In this way, they could be used to examine the distribution of resources relative to the distribution of inequalities. Using such analytic strategies in this way, we can examine a range of system effects an intervention or change in policy might be expected to have if implemented (68). The modelling of such dynamic processes related to place effects would therefore advance thinking that currently sees 'place' or the 'environment' as a static entity (see earlier discussion). One example of such an approach is a recent simulation study for designing effective interventions in early childhood caries (69). The authors used system dynamics to compare the relative effect and cost of six categories of early childhood caries intervention, applying fluorides, limiting cariogenic bacterial transmission from mothers to their children, using xylitol, clinical treatment, motivational interviewing and a combination of these. The resulting model predicted 10-year intervention costs and relative reductions in cavity prevalence with interventions targeting the youngest children having a greater benefit, those targeting high-risk children providing the greatest return on investment, and combined interventions showing the greatest cavity reduction.

By applying systems thinking to social oral epidemiology and then to interventions to improve population oral health, we would begin to target those causes that cannot be manipulated in a randomized-controlled trial. It is possible by using such methods, for example, to model those influencers on health outcomes (tooth decay) but also to evaluate public health policies (e.g. impact of investing in dental service provision on tooth decay under different assumptions about the importance of psychosocial orientations in influencing oral health) (example adapted from 28). Again, this approach is not a panacea but one tool that could help push social oral epidemiology forward both conceptually and methodologically.

Embodiment: How bodies register social experiences

In planning future research programmes, we need to move away from decontextualized and disembodied 'behaviours' and 'exposures' to understanding how the environment or 'society gets into the body' (70). This approach forms part of a long-standing tradition in sociology, exploring how physical bodies are shaped by the body social (71, 72). In this approach, bodies are seen as an important focus of social regulation. To this end, it has been argued that the principal mechanism of dentistry is disciplinary, that is, it is visited on the body in everyday life through the clinic in the form of the dental examination and through the home in the form of toothbrushing techniques (73, 74). In this research, the 'environment' is defined in terms of disciplinary knowledge and power and how this affects the body, which is in stark contrast to the SES variables commonly discussed as the 'environment' in oral epidemiology. In recent years, debates such as these have begun to enter epidemiology more generally. For example, recent work discusses embodiment as a multilevel phenomenon, and how processes become embodied

and generate population patterns of health and disease (7).

The problem of embodiment raises a central issue that might act as a catalyst for new collaborations between epidemiology and sociology. Through this theme, there are ways that both epidemiology and sociology can unpack 'how' the body and society are related and perhaps do so more carefully than has been the case to date. So, what then is the problem that underlies embodiment? The problem appears to be that if we start with the question of how society determines patterns of disease, we invariably end up confronting the difficulty of explaining how the acting feeling subject either resists or is less than determined by society. Another way of putting this is that describing how oral disease is socially patterned does not explain 'how' or 'why' it is patterned the way it is. Some room for the thinking acting subject is required. On the other hand if we start with the internal environment of the thinking acting subject, we end up struggling to explain social patterns of disease and illness (75).

There have been several attempts to overcome the dualism inherent in sociology's conception of society and the individual or the body. Writers such as Bourdieu (76) have proposed a way through the impasse with concepts such as 'habitus'. Habitus refers to the practices that we engage in that reflect regular problems encountered by people in their everyday lives. The practices reflect the regularity of everyday problems, but at the same time, they also reflect the structure of the environments we find ourselves in. So, for example, the practice of tooth brushing reflects the fact that we will all, more or less, experience tooth decay in the so called Western diet since the 'nutritional transition' (77, 78). The practice itself is a reflection of a generalized problem that our food environment presents to us, it is not, however, determined by that generalized problem. The concept of habitus seeks to enable us to reflect on the conditions of the environment *through the practices generated in reflection of how we have come to habitually deal with that environment*. There are as yet no serious explorations of the everyday habitus and how this relates to the mouth and oral health, and yet, the approach has received widespread attention in social science.

Other approaches may also prove promising. For example, Shilling (75) takes habitus as one of his points of departure into the pragmatic approach of Mead (79) in his attempt to unpick the relationship between society and the body. The pragmatism of Mead starts with neither the individual, nor the collective, but from the position that 'individuals are always already within a social and natural context, yet possessed of emergent capacities and needs that distinguished them from, and also enabled them to shape actively, their wider milieu' (75; p. 4). Our identities are shaped by ongoing interactions and transactions between the internal 'environment' of the embodied organism and the external social and physical environment. For Shilling (75), it is the ability of pragmatism to maintain a view of the internal and external environment that is distinctive. The suggestion then would be that the dental subject is therefore not determined by their social environment, as is often presented in the social determinants approach, rather they can, and often do, intervene creatively in the world to shape and change it.

We can draw on this approach in dental research. One starting point would be to adopt the 'transactional' approach of pragmatism between people in their environments. This might lead us to explore, utilizing in-depth qualitative methodologies, the different processes involved in different phases of interaction in oral health and society. For example, by looking at oral health-related habits, crisis and creativity that can combine at different times in the everyday lives of individuals (see 80 for an example). Such research involves being sensitive to the fact that there is more than one environment for social action and that we need to explore how the different environments constitute the different phases of action. The different environments are effectively the social and physical milieus of Mead (79).

The social milieu is constituted by people interacting on the basis of three things: their own desires and needs, what they think other people might think of them and from the standpoint of the group as a whole. This latter standpoint, termed the 'generalized other', places pressure on members of the group to conform to the standards of the group in terms of how they act. This approach filters the development of a bodily identity. Over time, we learn to evaluate ourselves according to the standards of the social group to which we belong. These organized set of attitudes to others is the way society influences its members. There are already existing data that can direct our attentions to such dimensions of oral health. Take the work of Sussex and colleagues (81) where it is clear that there was a generalized acceptance of edentulism

in New Zealand so that those with less than good teeth can live without stigma and can also support a symptomatic pattern of dental care. As a consequence, in the past, New Zealand society favoured extraction instead of restoration for dental disease.

The social milieu is vital, but it is not the whole picture that there is also a physical milieu that constrains and provides a restricted set of opportunities. For example, the key physical environmental influences on the adoption of these generalized attitudes in New Zealand appears to have been rural isolation (81). In this respect, the external environment has a social and physical dimension, and both of these dimensions are essential for understanding embodiment and corporeality in older New Zealanders in relation to their oral health.

We can also explore the internal environments of oral health and how this relates to emergent needs. For example, the approach of GH Mead and the pragmatists often begins with the internal environment of embodied action and then explores how our impulses are called out in particular ways by our environments. So, feeling low because of a glucose deficiency calls out a reaction to consume a sugary snack or drink. There is, in other words, a 'prereflective' tendency of our bodies towards survival, and we select stimuli that are basically relevant and depend on specific circumstances. Within this approach, the human body reaches out through its senses to manipulate the world around it in a multilayered way, and the senses become the embodied basis of our relationship to our environment (75). There is almost no detailed exploration of, for example, how sugar occupies a habitual space in the everyday environments of some populations and how its consumption might be related to the internal environment of the self. We are often called to reduce sugar consumption because of its associations with multiple forms of disease, for example, obesity and caries (78). Yet, this call often neglects or simplifies the complexities of the relationships between our internal and external environments and how these relate to embodiment.

For example, being asked to cut sugar consumption is similar to being asked to express self-control. Yet, recent research has shown that the exercise of self-control has a direct impact on our internal physical environment. In what is called the 'resource depletion' account, it is now being demonstrated that increasing self-control is significantly and specifically associated with the depletion of glucose as an energy resource (82, 83). The paradox, simply stated, is that self-control tasks, controlling ones diet, for example, can have direct impacts on blood glucose. Refraining from the consumption of certain foods can undermine ones will power to continue to control what one eats (82). This does not mean of course that eating sugary snacks is advisable; there will no doubt be better sources of glucose than sugar.

What this research shows is that the recommendation to cut sugar consumption within dentistry could significantly gain from being able to anticipate in more detail just what is being asked. Research in the social sciences can enable us to better appreciate the social and psychological dynamics behind resistance to such changes. So, for example, it is likely that there will be complex interactions between a person's everyday environments and self-control (82). As Gailliot and colleagues demonstrated, high degrees of self-control may be required in different occupations, and these in turn can have significant consequences for glucose depletion. Under such conditions, the sources of replacement glucose will no doubt have a significant impact on the health of the individual. Living under the conditions of an occupation where there are high demands in terms of self-control and limited choices in replenishing depleted glucose levels will have negative consequences for the things people can do to avoid the risks to their health and oral health. Reducing glucose intake under such conditions may well result in poorer performance in such roles, but also an added risk of significantly increased glucose consumption at another time.

Conclusion

There has been a chorus of dissenting voices rising in epidemiology outside of dentistry for some time. Within social oral epidemiology, the almost exclusive use of regression approaches constrains the questions asked, our hypotheses and the interpretations we develop. Our questions have the tendency to become narrower and narrower as we search for the 'truth'. Because of this, the field is becoming less relevant to understanding and intervening to improve the population's oral health. Understanding the links, the pathways and the processes by which factors such as inequalities influence oral health will require clear concepts, measures and methods. We need to stimulate

creative thinking about the processes involved and the questions being asked. This will require at the same time new tools, which can bring together traditional epidemiological methods and those from the social sciences. We are confined to work in the present and the past, but the changes happening with the discipline of social oral epidemiology will affect patterns of human oral health and disease in the future. To debate and engage with some of the conceptual and methodological issues raised here would be a small step for social oral epidemiology but potentially one giant leap for population oral health.

References

1. Diez-Roux AV. On genes, individuals, society, and epidemiology. Am J Epidemiol 1998;148:1027–32.
2. Diez-Roux AV. Bringing context back into epidemiology: variables and fallacies in multi-level analysis. Am J Public Health 1998;88:216–22.
3. Diez-Roux AV. Conceptual approaches to the study of health disparities. Ann Rev Public Health 2012;33:41–58.
4. Krieger N. Epidemiology and the web of causation: has anyone seen the spider? Soc Sci Med 1994;39:887–903.
5. Krieger N. Embodying inequality: a review of concepts, measures and methods for studying health consequences of discrimination. Int J Health Serv 1999;29:295–352.
6. Krieger N. Epidemiology and social sciences: towards a critical reengagement in the 21st century. Epidemiol Rev 2000;22:155–63.
7. Krieger N, editor. Embodying inequality: epidemiologic perspectives. Amityville, NY: Baywood Publishers; 2004.
8. Krieger N. Proximal, distal, and the politics of causation: what's level got to do with it? Am J Public Health 2008;98:221–30.
9. Krieger N. Epidemiology and the people's health: theory and context. Oxford: Oxford University Press; 2011.
10. Susser M. Does risk factor epidemiology put epidemiology at risk? Peering into the future. J Epidemiol Community Health 1998;52:608–11.
11. Susser M, Susser E. Choosing a future paradigm for epidemiology: I. Eras and paradigms. Am J Public Health 1996;86:668–73.
12. Susser M, Susser E. Choosing a future paradigm for epidemiology: II. From black boxes to chinese boxes and eco-epidemiology. Am J Public Health 1996;86:674–7.
13. Aida J, Ando Y, Oosaka M, Niimi K, Morita M. Contributions of social context to inequality in dental caries: a multilevel analysis of Japanese 3-year-old children. Community Dent Oral Epidemiol 2008;36:149–56.
14. Jamieson LM, Steffens M, Paradies YC. Associations between discrimination and dental visiting behaviours in an Aboriginal Australian birth cohort. Aust N Z J Public Health 2013;37:92–3.
15. Lamarca GA, Leal MC, Leao ATT, Sheiham A, Vettore MV. The different roles of neighbourhood and individual social capital on oral health–related quality of life during pregnancy and postpartum: a multilevel analysis. Community Dent Oral Epidemiol 2013;42:139–50.
16. Peres MA, Peres KG, Frias AC, Antunes JLF. Contextual and individual assessment of dental pain period prevalence in adolescents: a multilevel approach. BMC Oral Health 2010;10:20.
17. Sanders AE, Turrell G, Slade GD. Affluent neighborhoods reduce excess risk of tooth loss among the poor. J Dent Res 2008;87:969–73.
18. Turrell G, Sanders AE, Slade GD, Spencer AJ, Marcenes W. The independent contribution of neighborhood disadvantage and individual-level socioeconomic position to self-reported oral health: a multilevel analysis. Community Dent Oral Epidemiol 2007;35:195–206.
19. Cassel J. The contribution of the social environment to host resistance. Am J Epidemiol 1976;104:107–23.
20. Stallones RA. To advance epidemiology. Ann Rev Public Health 1980;1:69–82.
21. Hill AB. The environment and disease: association or causation? Proc R Soc Med 1965;58:295–300.
22. Rothman KJ, Greenland S. Causation and causal inference in epidemiology. Am J Public Health 2005;95:S144–50.
23. Holland PW. Confounding in epidemiologic studies. Biometrics 1989;45:1310–6.
24. McMichael AJ. Prisoners of the proximate: loosening the constraints on epidemiology in an age of change. Am J Epidemiol 1999;149:887–97.
25. Kaufman JS, Cooper RS. Seeking causal explanations in social epidemiology. Am J Epidemiol 1999;150:113–20.
26. Sheiham A, Watt R. The common risk factor approach – a rational basis for promoting oral health. Community Dent Oral Epidemiol 2000;28:399–406.
27. Levins R. Ten propositions on science and antiscience. Soc Text 1996;14:101–11.
28. Galea S, Riddle M, Kaplan GA. Causal thinking and complex systems approaches in epidemiology. Int J Epidemiol 2010;39:97–106.
29. Latour B. Science in action: how to follow scientists and engineers through society. Cambridge MA: Harvard University Press; 1987.
30. Kline RB. Principles and practice of structural equation modelling, 2nd edn. New York: The Guildford Press; 2005.
31. Wilson IB, Cleary PD. Linking clinical variables with health-related quality of life. JAMA 1995;273:59–65.
32. Baker SR. Testing a conceptual model of oral health: a structural equation modelling approach. J Dent Res 2007;86:708–12.
33. Baker SR, Mat A, Robinson PG. What psychosocial factors influence adolescents' oral health? J Dent Res 2010;89:1230–5.
34. Nammontri O, Robinson PG, Baker SR. Enhancing oral health via sense of coherence: a cluster-randomized trial. J Dent Res 2013;92:26–31.
35. MacMahon B, Pugh TF, Ipsen J. Epidemiologic methods. Boston, Mass: Little, Brown and Company; 1960.

36. Matthews KA, Gallo LC. Psychological perspectives on pathways linking socioeconomic status and physical health. Ann Rev Psychol 2011;62:501–30.
37. Cockerham WC. Health lifestyle theory and the convergence of agency and structure. J Health Soc Behav 2005;46:51–67.
38. Link BG, Phelan J. Social conditions as fundamental causes of disease. J Health Soc Behav 1995;35: 80–94.
39. Krieger N, Zierler S. What explains the public's health? A call for epidemiologic theory Epidemiology 1996;7:107–9.
40. Mackenbach JP. The persistence of health inequalities in modern welfare states: the explanation o41. f a paradox. Soc Sci Med 2012;75:761–9.
41. Link BG, Mary EN, Phelan JC, Ganz ML. Social epidemiology and the fundamental cause concept: on the structuring of effective cancer screens by socioeconomic status. Millbank Q 2008;76:375–402.
42. Chang VW, Lauderdale DS. Fundamental cause theory, technological innovation, and health disparities: the case of cholesterol in the era of statins. J Health Soc Behav 2009;50:245–60.
43. Glied S, Lleras-Muney A. Technological innovation and inequality in health. Demography 2008;45: 741–61.
44. McDonagh MS, Whiting PF, Wilson PM, Sutton AJ, Chestnutt I, Cooper J et al. Systematic review of water fluoridation. BMJ 2000;321:855–9.
45. Slade GD, Spencer AJ, Davies MJ, Stewart JF. Influence of exposure to fluoridated water on socioeconomic inequalities in children's caries experience. Community Dent Oral Epidemiol 1996;24:89–100.
46. Watt RG, Sheiham A. Integrating the common risk factor approach into a social determinants framework. Community Dent Oral Epidemiol 2012;40:289–96.
47. Rose G. Sick individuals and sick populations. Int J Epidemiol 1985;14:32–8.
48. Bonetti D, Young L, Black I, Cassie H, Ramsay CR, Clarkson JE. Can't do it, won't do it! Developing a theoretically framed intervention to encourage better decontamination practice in Scottish dental practices. Implement Sci 2009;4:31–5.
49. Jönsson B, Öhrn K, Oscarson N, Lindberg P. The effectiveness of an individually tailored oral health educational programme on oral hygiene behaviour in patients with periodontal disease: a blinded randomized-controlled clinical trial (one-year follow-up). J Clin Periodontol 2009;36:1025–34.
50. Halvari ALM, Halvari H, Bjornebekk G, Deci EL. Motivation for dental home care: testing a self-determination theory model. J Appl Soc Psychol 2012;42:1–39.
51. Cockerham WC, Rutten AT. Conceptualising contemporary health lifestyles: moving beyond weber. Sociol Q 1997;38:321–42.
52. Armitage CJ, Conner M. Social cognition models and health behaviour: a structured review. Psychol Health 2000;15:173–89.
53. Crossley ML. Rethinking health psychology. London: Open University Press; 2000.
54. Locker D. Deprivation and oral health: a review. Community Dent Oral Epidemiol 2000;28:161–9.
55. Shim JK. Understanding the routinised inclusion of race, socio-economic status and sex in epidemiology: the utility of concepts from technoscience studies. Sociol Health Illn 2002;24:129–50.
56. Krieger N, Davey Smith G. 'Bodies count', and body counts: social epidemiology and embodying inequality. Epidemiol Rev 2004;26:91–103.
57. Gump BB, Matthews KA, Raikkonen K. Modelling relationships among socioeconomic status, hostility, cardiovascular reactivity and left ventricular mass in African American and white children. Health Psychol 1999;18:140–50.
58. Crespo CJ, Smit E, Andersen RE, Carter-Pokras O, Ainsworth BE. Race/ethnicity, social class and their relation to physical inactivity during leisure time: results from the Third National Health and Nutrition Examination Survey, 1988–1994. Am J Prev Med 2000;18:46–53.
59. Williams RR, Rao DC, Ellison RC, Arnett DK, Heiss G, Oberman A et al. NHLBI family blood pressure program: methodology and recruitment in the HyperGEN network. Hypertension genertic epidemiology network. Ann Epidemiol 2000;10: 389–400.
60. Macintyre S, Ellaway A, Cummins S. Place effects on health: how can we conceptualise, operationalize and measure them? Soc Sci Med 2002;55:125–39.
61. Cummins S, Curtis S, Diez-Roux AV, Macintyre S. Understanding and representing 'place' in health research: a relational approach. Soc Sci Med 2007;65: 1825–38.
62. Lewontin R, Levins R. Let the numbers speak. Int J Health Serv 2000;30:873–7.
63. Sterman J. Business dynamics: systems thinking and modeling for a complex world. Boston: Irwin/ McGraw-Hill; 2000.
64. Homer JB, Hirsch GB. Systems dynamic modeling for public health: background and opportunities. Am J Public Health 2006;96:452–8.
65. Resnicow K, Page SE. Embracing chaos and complexity: a quantum change for public health. Am J Public Health 2008;98:1382–9.
66. Miller JH, Page SE. Complex adaptive systems: an introduction to computational models of social life. New York: Princeton University Press; 2007.
67. Koopman JS, Lynch JW. Individual causal models and population system models in epidemiology. Am J Public Health 1999;89:1170–4.
68. Auchincloss AH, Diez-Roux AV. A new tool for epidemiology: the usefulness of dynamic-agent models in understanding place effects on health. Am J Epidemiol 2008;168:1–8.
69. Hirsch GB, Edelstein BL, Frosh M, Anselmo T. A simulation model for designing effective interventions in early childhood caries. Prev Chronic Dis 2012;9:E66.
70. Connell R. Gender & power: society, the person and sexual politics. Oxford: Polity Press; 1987.
71. Featherstone M, Turner BS. Body & society: an introduction. Body Soc 1995;1:1–12.
72. Turner BS. The body and society. London: Basil Blackwell Publisher; 1984.
73. Lupton D. The imperative of health: public health and the regulated body. London: Sage; 1995.

74. Nettleton S. Power, pain and dentistry. Buckingham: Open University Press; 1992.
75. Shilling C. Changing bodies: habit, crisis and creativity. London: Sage; 2008.
76. Bourdieu P. Outline of a theory of practice. Cambridge: Cambridge University Press; 1977.
77. Watt RG. Strategies and approaches in oral disease prevention and health promotion. Bull World Health Org 2005;83:711–8.
78. Watt R, Rouxel P. Dental caries, sugars and food policy. Arch Dis Child 2012;97:769–72.
79. Mead GH. Mind, self and society. Chicago, Illinois: The University of Chicage Press; 1934.
80. Rousseau N, Steele J, May C, Exley C. 'Your whole life is lived through your teeth': biographical disruption and experiences of tooth loss and replacement. Sociol Health Illn 2013;36:462–76.
81. Sussex P, Thomson W, Fitzgerald R. Understanding the 'epidemic' of complete tooth loss among older New Zealanders. Gerodontology 2010;27:85–95.
82. Gailliot MT, Baumeister RF, DeWall CN, Maner JK, Plant EA, Tice DM et al. Self-control relies on glucose as a limited energy source: willpower is more than a metaphor. J Pers Soc Psychol 2007;92:325–36.
83. Vohs K, Baumeister R, Ciarocco N. Self-regulation and Self-presentation: regulatory resource depletion impairs impression management and effortful self-presentation depletes regulatory resources. J Pers Soc Psychol 2005;88:632–57.