



**Extended urbanisation and the spatialities of infectious disease: Demographic change, infrastructure and governance**

Journal:	<i>Urban Studies</i>
Manuscript ID	CUS-418-19-05.R2
Manuscript Type:	Debates Paper
<b>Discipline: Please select a keyword from the following list that best describes the discipline used in your paper.:	Geography
World Region: Please select the region(s) that best reflect the focus of your paper. Names of individual countries, cities & economic groupings should appear in the title where appropriate.:	Not Applicable
Major Topic: Please identify up to 5 topics that best identify the subject of your article.:	Agglomeration/Urbanisation, Demographics, Governance, Health, Infrastructure
You may add up to 2 further relevant keywords of your choosing below.:	infectious disease, landscape

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## **Extended Urbanisation and the Spatialities of Infectious Disease: Demographic Change, Infrastructure and Governance**

### **Abstract:**

This paper argues that contemporary processes of extended urbanisation, which include suburbanisation, post-suburbanisation and peri-urbanisation may result in increased vulnerability to infectious disease spread. Through a review of existing literature at the nexus of urbanisation and infectious disease, we consider how this (potential) increased vulnerability to infectious diseases in peri- or suburban areas is in fact dialectically related to socio-material transformations on the metropolitan edge. In particular, we highlight three key factors influencing the spread of infectious disease that have been identified in the literature: demographic change; infrastructure and governance. These have been chosen given both the prominence of these themes and their role in shaping the spread of disease on the urban edge. Further, we suggest how a landscape political ecology framework can be useful for examining the role of socio-ecological transformations in generating increased risk of infectious disease in peri- and suburban areas. To illustrate our arguments we will draw upon examples from various re-emerging infectious disease events and outbreaks around the world to reveal how extended urbanisation in the broadest sense has amplified the conditions necessary for the spread of infectious diseases. We thus call for future research on the spatialities of health and disease to pay attention to how variegated patterns of extended urbanisation may influence possible outbreaks, and the mechanisms through which such risks can be alleviated.

**Keywords:** Extended urbanization, infectious disease, governance, infrastructure, demographic change, urban political ecology

### **Introduction:**

To date, the literatures on urbanisation and globalisation have focused primarily on economic and demographic flows to, from and through cities and their regions (Brenner, 2014; Ren and Keil, 2017). More recently, there has been a growing academic and policy interest in connecting challenges of a majority urbanised world to questions of health and disease, including Ali and

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3 Keil's (2008) edited collection, *Networked Disease: emerging infections in the global city* as a  
4 groundbreaking publication (see also Elsey et al., 2019; Moore et al., 2003; Wu et al., 2017).  
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6 Meike Wolf's (2016) research on urban epidemiology also takes forward debate surrounding the  
7 role of changing geographies on urban health and disease. Furthermore, a recent article by Bollyky  
8 (2019) has more explicitly noted that the future of global health is urban health. Recognising these  
9 emerging conversations, we are specifically interested in this paper in new ways in which  
10 infectious disease is bound up with processes of *extended* urbanisation, paying particular attention  
11 to the socio-ecological flows and disruptions leading to increased incidence of infectious disease  
12 in peri- or sub-urban areas.  
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20 This paper, then, will focus on the impact more extensive forms of urbanisation worldwide have  
21 on increasing susceptibility to infectious disease, especially emerging infectious disease (EID), i.e.  
22 “an infectious disease whose incidence is increasing following its first introduction into a new host  
23 population” (Quammen, 2012: 43) and zoonosis which is “an animal infection transmissible to  
24 humans” (Quammen, 2012: 14). Notably, ecological pressures coupled with social and spatial  
25 change, have led to new forms of disease spread that are likewise contributed to the rise of EID  
26 epidemics. These include changes in water-borne EID spread, as was the case with *E. coli* 0157:H7  
27 (Ali, 2004); changes in food-borne EID transmission as brought on by changes in global  
28 consumption patterns (Hoffman, 2014); as well as changes in the distribution of vector-borne  
29 diseases such as malaria, wherein the distribution of mosquitos has been affected by global climate  
30 change (Epstein, 1998; Brisbois and Ali, 2010; Nading, 2014).  
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40 In examining these relationships between extended urbanisation and infectious disease, we suggest  
41 how a landscape political ecology (LPE) framework can be useful for integrating the key themes  
42 of spatiality, socio-natural metabolism and power relations central to undertaking such an analysis.  
43 As we discuss, LPE approaches are crucial to understanding the metabolic processes and socio-  
44 environmental implications bound up with extended forms of urbanisation. The concept of  
45 metabolism refers to the combination of social and natural processes to form socio-natural  
46 landscapes (Swyngedouw and Heynen, 2003). We posit that, while rapid and intensive forms of  
47 urbanisation (densification) are seen as enabling factors for the spread of infectious disease  
48 (Munster et al., 2018); it is important to study extended urbanisation because patterns of urban  
49 sprawl and expansion are more likely to lead to infectious disease outbreaks, as opposed to cities  
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3 which are generally assumed to reduce incidence of infectious disease for inhabitants (see Wood  
4 et al, 2017). This is, in part, because urban expansion might expose sub- and ex-urban areas to  
5 higher levels of biodiversity (and disease sources) than are found in central urban areas (Kaup,  
6 2018). Cities also have better health facilities and resources which can enable faster response times  
7 and enhance containment of disease outbreaks.  
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13 In other words, the new and evolving global peripheries have been particularly susceptible to  
14 diseases that jump the animal-to-human species boundary (zoonosis); have seen the introduction  
15 of new disease vectors; and dynamic changes to urban and spatial morphology and transformations  
16 over time (Brisbois and Ali, 2010). In demonstrating this argument, we highlight three key factors  
17 influencing the distribution of infectious disease burden that have been identified in the literature  
18 on this topic within various strands of urban studies. These interrelated dimensions are mobility  
19 and demographic change; infrastructure and governance. There are certainly more factors that  
20 could be identified (e.g. deforestation and climate change), but these are the three themes that have  
21 been most prominent in the literature on urbanisation and infectious disease, and also relate closely  
22 to processes of extended urbanisation.<sup>1</sup> We have kept these themes intentionally broad so as to  
23 capture as much of the disparate work that exists on this topic as possible. Moreover, we consider  
24 how these factors influence different phases of infectious disease management, from disease  
25 prevention, to mitigation and control of outbreaks, and possible responses.  
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36 In what follows, we will first establish the context for this paper, outlining why the relationship  
37 between extended urbanisation and infectious disease is important to study, and how this differs  
38 from existing work on the relationship between urbanisation and disease. Subsequently, we  
39 introduce the conceptual framework of landscape political ecology, indicating how this relates to  
40 (but differs from) urban political ecology, and how this is a useful lens through which to understand  
41 the emergence of infectious disease in peri-urban areas. We then survey the three themes of  
42 mobility and demographic change; infrastructure; and governance in turn, discussing their  
43 importance for addressing emerging urban challenges related to extended urbanisation. Finally,  
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53 <sup>1</sup> As one would expect, this is a highly interdisciplinary field, with contributions from social scientists, but  
54 also epidemiologists.  
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3 we reflect on how the expansion of the city can influence the spread of disease, and how this can  
4 be addressed in future research.  
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### 6 7 **Extended Urbanisation and Emerging Infectious Disease** 8

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10 Contemporary patterns of extended urbanisation fundamentally shift the vulnerability of cities to  
11 infectious diseases, in ways that differ from those that have historically been associated with  
12 urbanisation. Such processes of urban expansion are linked to the ubiquitous reordering of the  
13 global urban periphery through complex processes of displacement of central populations to the  
14 margins and the creation of new functional centralities (jobs, infrastructures, densities) away from  
15 the traditional core. As indicated, we use the term extended urbanisation as a summary concept for  
16 these developments. The processes captured under this term, originally informed by the urban  
17 theory of Henri Lefebvre (2003) predicts what he called “the complete urbanisation” of society.  
18 This phenomenon is partially caused by the rapid growth of the human population and the  
19 expanding geographical reach of capitalist accumulation over the past century, which has created  
20 an “urban revolution” and the creation of an “urban society” at the planetary scale (see Keil, 2018a).  
21 Relatedly, various scholars have argued that we are now witnessing a process of planetary  
22 urbanisation, which is premised upon expanding infrastructural networks and human settlements  
23 (Brenner, 2014).  
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36 In this broader context, we are specifically interested in what Lefebvre (2003) calls the spread of  
37 “the urban tissue” across the planet, which refers to the fluid relationships between urban and rural  
38 environments. Forms of extended urbanisation - such as suburbanisation - are an empirically  
39 recognisable process in this context. In many parts of the world, particularly in the global south,  
40 peri-urbanisation is the preferred term for extended urbanisation (de Vidovich, 2019). Some  
41 scholars have called the current phase of urban extension post-suburbanisation, which leads to an  
42 increasing complexity of structural form and daily life in the periphery of cities (Wu and Phelps,  
43 2011; Charmes and Keil, 2015). In this context, ‘peripheral’ can also refer to both the self-built  
44 structures and informal communities that characterise much of today’s urbanisation without being  
45 necessarily spatially on the margins (e.g. refugee settlements, mining camps, and indigenous  
46 reserves near urban centres) (Caldeira, 2017; Güney et al., 2019). Finally, extended urbanisation,  
47 refers to new and existing urbanisation and urban settlement in the periphery of cities *and* relations  
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3 that condition these spaces but also reach beyond them (e.g. mines, factories and infrastructures)  
4 (Keil, 2018a).  
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8 Such patterns of urbanisation - including connected processes of globalisation and  
9 neoliberalisation - can increase the qualitative conditions and the statistical odds that microbes are  
10 being spread, which has resulted in a tripling of the total number of disease outbreaks per decade  
11 has since the 1980s (Ali and Keil, 2007; Haggett, 1994). As Wald (2008: 14) has put it, cities have  
12 been known by public-health officials as “promiscuous” social spaces, with people “literally and  
13 figuratively bumping up against each other in smaller spaces and larger numbers than ever before”.  
14 Additionally, the most significant global disease outbreaks in recent years have originated in China  
15 and Africa, which are also amongst the most rapidly urbanising regions (Alirol et al., 2010). Both  
16 SARS and Ebola originated in peri-urban regions before traveling to and spreading between major  
17 cities like Hong Kong, Toronto or Kinshasa (Keil and Ali, 2007). As such, asking how and why  
18 the proliferation of suburban or peri-urban areas is conducive to disease spread is an important  
19 question to explore.  
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30 While other approaches situate communicable disease as a function of social interactions, we focus  
31 on changing spatial factors that drive changing patterns of disease. This can be cast as part of a  
32 general concern with the spread of risk as processes of peri-urbanisation and suburbanisation are  
33 arguably the defining forms in which global urban society is taking shape in the twenty-first  
34 century. In this context, Bloch, et al. (2013: 96) observed that “current urban growth patterns  
35 appear to have significantly amplified the exposure of urban populations to hazard risks, markedly  
36 but not exclusively those broadly characterised as the urban poor”. Therefore, identifying areas  
37 where the convergence of risk factors is occurring with greatest intensity, and at the largest scales,  
38 is a logical first step in the development of a mitigation strategy.  
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#### 46 *Chronic and (emerging) infectious disease*

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49 Before we continue, we need to acknowledge that much of the debate about urbanisation, or more  
50 precisely, extended urbanisation and health/disease is occupied by a burgeoning interest in chronic  
51 diseases associated with a lifestyle that is ascribed to suburbanisation, auto-mobility and related  
52 technologies. At the top of the list of these concerns are usually obesity (especially among young  
53 people), diabetes and heart disease (Hamblin, 2014). Importantly, attention has now also shifted  
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3 to mental health related to suburban life, for example in emerging work on people living with  
4 dementia in suburban environments (Biglieri, 2018). Much of the literature on chronic disease has  
5 as the tacit starting point the notion of “epidemiological transition”. That is, the argument that in  
6 Western, industrialised societies, more individuals are living to older ages, which consequently  
7 leads to increased incidence and prevalence of chronic diseases associated with (sedentary)  
8 “lifestyle” and “aging”, as opposed to infectious diseases. Bloch and co-authors (2013: 96) of  
9 course warn: ‘A closer examination of urban risk shows, in fact, that “sprawl” is not the problem,  
10 but rather the lack of adequate land use planning policies and infrastructure provision in rapidly  
11 growing and expanding settlements’.

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20 One of the key points of emphasis on approaches based on this premise is a focus on the physical  
21 environment found within such contexts. Much of the health research on the built environment  
22 reveals that the health of those in urban areas tends to be worse than those residing in less urbanised  
23 areas – a disparity referred to as the urban health penalty (Freudenberg et al., 2005). However,  
24 such findings are not wholly conclusive, as research has also pointed to certain features of urban  
25 life that benefit the health of urban-dwellers, including the availability of social support and better  
26 access to health and social services (Bollyky, 2019). Current health research does conclusively  
27 demonstrate, however, that one aspect of the built environment is detrimental to good health,  
28 namely, living in sprawling suburban neighbourhoods (Frumkin et al., 2004; Freudenberg et al.,  
29 2005). What has made this question more complicated is exactly the tendency, invoked in our  
30 usage of extended urbanisation and post-suburbanisation, of blurring the classical lines of  
31 distinction of city and suburb, town and country. It appears more important for the health of  
32 communities and individuals where in the world, and indeed where in the urban region they are  
33 located and how those particular areas are changing in relation to their natural and social  
34 environments (Wilson et al., 2008).

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47 As alluded to above, the tendency to date not to have focused concerted attention on infectious  
48 disease may have to do with the “epidemiological transition” model. According to this perspective,  
49 Western societies have undergone a health transition whereupon infectious diseases were no longer  
50 to be considered as major causes of mortality and morbidity (Omran, 1971). Thus, for example, it  
51 was in this light that in 1967, the U.S. Surgeon General publicly declared that it was ‘time to close  
52 the books on infectious diseases’ and to shift all national attention to chronic diseases such as  
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3 cardiovascular disease (Garrett, 1994: 33). Recent developments, however, appear to indicate that  
4 this may be premature as we now appear to face an onslaught of what are referred to as new and  
5 (re)emerging diseases (Morse, 1993; Haggett, 1994; Garrett, 1994; Mayer, 2000).  
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9 Emerging diseases are those which have become more prevalent during the last quarter century,  
10 while new diseases refer to not only newly-appearing ones, but also those that are spreading to  
11 new geographical areas (Mayer, 2000). Some examples of these include: yellow fever, the  
12 Marburg virus, Legionnaires' disease, the Ebola virus, Lyme disease, hepatitis C, HIV/AIDS,  
13 Hantavirus pulmonary syndrome, West Nile Virus and Severe Acute Respiratory Syndrome  
14 (Garrett, 1994; Heymann and Rodier, 1997; Drexler, 2002). On the other hand, re-emerging  
15 diseases, or those thought to have been eradicated due to aggressive antibiotic vaccination  
16 campaigns, have also begun to reappear with greater frequency in the population in recent years  
17 (for example, tuberculosis).  
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### 26 **Landscape political ecologies of health and disease**

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28 Spatialised political ecology of health approaches – paying specific heed to interactions between  
29 urban, suburban and rural landscapes - are important for their focus on the interaction between  
30 political interests, social institutions and the human-nonhuman environment, which can bring  
31 about a greater systemic understanding of health and disease (see King, 2010; Jackson and Neely,  
32 2015; Connolly et al., 2017). Given the interdisciplinary nature of health studies, political ecology  
33 is an ideal framework that allows for the use of mixed research methods and incorporates a range  
34 of conceptual approaches (Robbins, 2004; King, 2010). This is because of its deep concern for  
35 human/environment relations, and for its systematic study of the unequal distribution of socio-  
36 environmental harms and risks. More specifically, Connolly (2017) suggests that a landscape  
37 political ecology (LPE) perspective, can be a useful approach for examining the political ecologies  
38 of disease. This is because both political ecology and health geographies draw on ideas of place  
39 and landscape and utilise an understanding of place as a socially (re)constructed phenomenon (see  
40 King, 2010).  
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52 The concept of landscape is useful for studying processes of extended urbanisation, given the  
53 hybrid nature of the term, which allows for blurring distinctions between the urban and rural. This  
54 is one way in which sub/urban political ecologies have moved beyond critiques of 'methodological  
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3 cityism', by exploring socio-ecological processes on the urban periphery (c.f. Angelo and  
4 Wachsmuth, 2015; Connolly, 2019). The landscape lens is also important to understand how  
5 spatial factors and the physical ordering of the urban environment can directly influence the  
6 incidence of disease outbreaks, and possible responses to them (Lambin et al., 2010). Moreover,  
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8 Kearns and Moon (2002: 611) have argued that landscape serves as a metaphor for "the complex  
9 layerings of history, social structure and built environment that converge in particular places". This  
10 can be seen in the infrastructural (dis)connections; and changing nature-society interactions that  
11 are associated with urban expansion. As such, we posit that particular landscapes themselves can  
12 be structured in such a way that influence the likelihood of disease transmission.  
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20 Some scholars working on the political ecology of health and disease have used landscape as an  
21 analytical lens to consider how various health discourses can become materialised in particular  
22 places (Mulligan et al. 2012; Parizeau 2015). For example, Wald (2008: 2) has described how "the  
23 circulation of microbes materialises the transmission of ideas" regarding theories about how  
24 diseases spread and attitudes toward social change. In this way, disease is not only determined  
25 through biophysical factors, but also constructed out of a particular set of social and spatial  
26 relations which are mediated through the landscape. As we will discuss later in the paper, processes  
27 of extended urbanisation can increase risk to infectious diseases - which are themselves rapidly  
28 evolving - as the risks and mode of transmission are often neither well understood by science nor  
29 properly regulated by government, particularly in informal peri-urban settlements common in the  
30 developing world. Lambin et al. (2010) have examined specifically how landscape attributes and  
31 land use change can have a significant impact on re-emerging infectious diseases and/or zoonoses.  
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41 Others have used a landscape lens to examine the interconnections between social and  
42 environmental systems (Fairhead and Leach, 1996; Walker and Fortmann, 2003). In this regard,  
43 Wald (2008: 2) has observed how interactions between microbes, bodies and spaces have the  
44 tendency to blend together as they "animate the landscape and motivate the plot of the outbreak  
45 narrative". Such analyses draw upon a wide body of literature in science and technology studies  
46 (STS) and influenced by assemblage theory exploring the agency of non-humans in shaping urban  
47 environments and the regulation of public space (see Rose, 2007; Braun, 2008; Jackson and Neely,  
48 2015). Urban political ecology has also mobilised insights from STS to analyse the role of non-  
49 humans in shaping human health (see Jackson and Neely, 2015). This has been achieved through  
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3 the use of analytical and heuristic concepts such as Haraway's 'cyborgs' (Haraway, 1991) and  
4 Latour's 'quasi-objects' (Latour, 1993)—terms that are now commonplace in the literature on  
5 urban political ecology, and in the social sciences more broadly.  
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9 Braun (2008) for example, has argued that infectious diseases emerge from human-non-human  
10 relationships, circulation and exchange at a variety of scales from the molecular to global. This  
11 has caused various non-human animals including rodents to enter human settlements, which is  
12 partly to blame for the first Ebola case in Guinea. Processes of extended urbanisation have also  
13 facilitated the expansion of human settlements into former rainforest areas, exposing humans to  
14 new possible sources of disease (see Yong, 2018). Deforestation and human encroachment on  
15 wildlife habitats have increased interactions between wildlife, human beings, and livestock, thus  
16 heightening the potential for pathogens to cross the species barrier (Coker et al., 2011). As Yong  
17 (2018: np) has explained, such patterns have now become the general context for the spread of  
18 infectious disease due to zoonotic infection, noting that: "wherever people push into wildlife-rich  
19 habitats, the potential for such spillover is high" (Yong 2018). Such processes have been facilitated  
20 by the greater and more rapid movement of people, exposing human populations to a host of  
21 microbes, insects and other non-humans which were previously largely undisturbed by  
22 urbanisation.  
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34 Maria Kaika's (2005) city of flows analogy, born out of the urban political ecology (UPE) literature,  
35 is also a useful for conceptualising the relationship between extended forms of urbanisation and  
36 disease. UPE examines the multitude of socio-natural flows into and out of the city, often referred  
37 to as 'urban metabolism' including biophysical, technical, social and economic exchanges (Gandy  
38 2004; Loftus, 2006; Swyngedouw, 2006). Kaika and colleagues have also recently proposed a  
39 distinctive suburban political ecology lens which has considerable overlap with the perspective  
40 put forward here in our combined use of landscape and urban political ecologies (Tzaninis, et al.,  
41 forthcoming). As Coker et al. (2011: 599) have elaborated, cities are home to "dynamic systems  
42 in which biological, social, ecological and technological processes interconnect in ways that enable  
43 microbes to exploit new ecological niches". Moreover, "the particular sociopolitical contexts and  
44 spatial configuration of urban regions have strong implications for how these various non-human  
45 natures are urbanised" (Connolly, 2019). For these reasons, landscape political ecology thus  
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3 becomes an extremely useful tool for understanding the political, social, economic and cultural  
4 relationships between urban environments and public health.  
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### 8 **Extended urbanisation and infectious disease: Three dimensions**

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10 Meike Wolf (2016: 975) has proposed a number of “future challenges” of research into the “messy  
11 materialities” of (extended) urbanisation and (emerging) infectious disease research. In  
12 summarising her review of recent developments in the field, Wolf argues that “a reconsideration  
13 of analytical categories of space, time, climate or nature - which are of equal importance to both  
14 the social sciences and public health - goes hand in hand with accounts that ramify different sites  
15 and aim to capture new paths of connection and association” (Wolf 2016: 976). This can be more  
16 useful than making overarching processes such as globalisation or urbanisation synonymous with  
17 increases in mobility. With this in mind, we have isolated three dimensions of possible research  
18 on suburbanisation and infectious disease: dynamics of population change; infrastructure; and  
19 governance. As we will demonstrate, these concepts are well suited to a landscape political ecology  
20 approach, and are crucial for identifying spatial patterns and political-economic arrangements that  
21 influence the spread of infectious disease in the ongoing, and accelerating, process of extended  
22 urbanisation.  
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#### 36 A: Dynamics of Population Change:

37 “Dying alone in your hut isn’t an outbreak.”  
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41 (Khan and Patrick, 2016: 70)  
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44 This terse statement by former director of the US Centers for Disease Control’s Office of Public  
45 Health Preparedness and Response points the finger at an obvious truth: pandemic disease relies  
46 on population growth. Population growth in cities - driven primarily by rural-urban migration - is  
47 a major factor influencing the spread of disease (Coker et al., 2011). This is seen most clearly in  
48 rapidly urbanising regions such as Africa and Asia, which have experienced recent outbreaks of  
49 Ebola and SARS, respectively. Projections by urban scholars hold that sub-Saharan Africa’s  
50 urbanisation rates are higher than anywhere else in the world as the urban population in the region  
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3 “is expected to quadruple, from 295 million to 1.15 billion” (Angel et al., 2017: 169). Twelve  
4 million people now live in Kinshasa, capital of the DRC, which is three times the combined  
5 population of the cities affected by the 2014 outbreak in West Africa (Yong, 2018). Equally,  
6 regional towns in the DRC, where some of the recent Ebola cases have been recorded, have also  
7 been expanding, some under the influence of conflict and war. While the ecological consequences  
8 of this expansion are beginning to be better understood, we are only starting to shed light on the  
9 impact of dramatic and massive sub/urbanisation on health and disease.

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16 But, studies at the intersection of urbanisation and infectious disease have shown, it is not only  
17 population growth that leads to infectious disease spread, but also density. Jakarta, to take another  
18 example, is projected to become the largest city in the world in coming decades, with much of the  
19 population made up of rural-urban immigrants. Numerous researchers have thus noted that  
20 population density - which is highest in cities - strongly influences the likelihood of a disease  
21 outbreak (Ali and Keil, 2007; Alirol et al., 2010; Coker et al, 2011). For instance, Wilkinson and  
22 Leach (2014) have noted that the dense urban areas and slums in Monrovia and Freetown, Sierra  
23 Leone, have been prime sites where Ebola has thrived. While suburban areas are popularly  
24 understood as low density areas, such processes of extended urbanisation in developing regions  
25 often consists of densely populated ‘new towns’ of high-rise flats or peri-urban informal  
26 settlements with high densities (see Mabin et al., 2013). Such cases indicate the importance of a  
27 landscape political ecology lens in examining urbanisation and infectious disease, as it is often the  
28 lack of physical infrastructure, coupled with political-economic factors resulting in high density in  
29 such places that provide perfect scenarios for the spread of microbes.

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41 Research on urbanisation is also beginning to consider how mobility patterns between urban, peri-  
42 urban and rural areas influence infectious disease spread (see Herrick, 2014; Wolf, 2016). It should  
43 be noted that the first urban Ebola outbreaks happened in West Africa after almost four decades of  
44 rural outbreaks throughout the rest of Africa (WHO, 2015). Why, then was there a change from  
45 rural to urban outbreaks after this time and in this particular region? One factor is the high degree  
46 of population movement on the continent, which is seven times higher than anywhere else in the  
47 world (WHO, 2015). This migration is driven by a myriad of social and political economic factors  
48 that force people to travel daily in the search of food or work; extended families with relatives  
49 living in different countries; the traditional practices of returning to a native village to die and be  
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3 buried near ancestors; as well as travel to traditional healers who have the trust of community  
4 members (WHO, 2015). There are also the effects of civil war that have forced some family  
5 members to flee their home villages to other, usually more urban areas, for relocation and  
6 resettlement.  
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11 Disease transmission in large urban populations can also be affected by heterogeneity in health of  
12 urban dwellers, increased rates of contact, and mobility of people (Alirol et al., 2010). For instance,  
13 Alirol (et al., 2010) and Tong et al. (2015) have shown that rural to urban population movements  
14 can substantially increase risk of transmission amongst newcomers who may not have previous  
15 exposure (immunisation). It is also difficult to control migration between cities in many African  
16 countries, as Sierra Leone, Liberia and Guinea each have 5,000 border crossing points (Wilkinson  
17 and Leach, 2014). Thus, the monitoring of rural-urban and inter-urban migration will be crucial in  
18 order to stop the spread of disease in future outbreaks. Tong et al. (2015: 11029) further add that  
19 rural-urban migrants tend to be poorer and less educated than the permanent population in urban  
20 areas, live in lower quality housing with inadequate sanitation, have limited access to health  
21 services (see also Hynie, 2018). These migrants tend to settle in (often informal) places along the  
22 metropolitan edge. This can be problematic, as Wu et al. (2017: 21) have found that in many  
23 Chinese cities, public health management has not kept pace with demographic changes in rapidly  
24 urbanising areas.  
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36 As Wolf (2016: 965) has noted, infectious diseases are thus less of a ‘natural’ disaster, but emerge  
37 alongside social and spatial inequalities in housing, health education, or financial resources (see  
38 Kotsila, 2017). Such processes are particularly well suited to an urban political ecology framework  
39 which is not only useful for examining the ‘explosion’ of urban societies, but also the uneven and  
40 socially unjust power relations which amplify health inequalities in particular places and  
41 underlines the issue of governance that we will deal with later (see Houston and Ruming, 2014;  
42 Parizeau, 2015). Understanding the root causes of disease emergence in urban areas will thus be  
43 essential to preventing additional rural to urban spread and to containing outbreaks within urban  
44 centres (Fallah et al., 2018: 280; Richards et al., 2015).  
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### 53 B. Infrastructure:

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3 “Viruses have no locomotion yet many of them have traveled around the world.”  
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6 Stephen S. Morse in Quammen 2012: 24.  
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10 In many ways, the geographic spread, growing sophistication and colonising propensities of  
11 transportation networks are the hallmark of extended urbanisation in general (Keil 2018a).  
12 Specifically, peri-urban (transport) infrastructures are tremendously important for the functioning  
13 of the entire urban region (Filion and Pulver 2019). This is due to location of prime network spaces  
14 such as airports, recreational spaces, in addition to noxious or toxic industrial infrastructures  
15 including waste and water treatment facilities and incinerators, which are often in peri- or sub-  
16 urban areas (Keil, 2018b: 132). Diseases can spread rapidly between cities through infrastructures  
17 of globalisation such as global air travel networks. While this has been well documented before, it  
18 is relevant here because airports and other nodes of economic logistics and activity are often  
19 located in suburban municipalities, thus raising potentially complex governance and jurisdictional  
20 issues with regards to who has responsibility to control disease outbreaks in large urban regions  
21 (Ali and Keil; 2010; McNeill 2011; Addie 2014).  
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31 Ex-urban infrastructures have thus become the lynchpin of urban mobility and circulation and  
32 socio-natural metabolisms (see Monstadt, 2009; Filion and Pulver, 2019; Lin, 2019). As Lin (2019:  
33 76) writes, “infrastructures often figure as networked landscapes, constituting ‘spatial products’  
34 that script structural relations between places at the planetary scale”. For this reason, such  
35 infrastructures can facilitate the transmission of infectious diseases and make urban populations  
36 more vulnerable (Keil and Ali 2007: 848). Indeed, the spread of disease is enabled by the same  
37 infrastructures that carry people, resources and goods. For instance, Munster et al. (2018) have  
38 argued that road construction for logging, mining, and hydroelectric activities “continues to open  
39 access to remote locations”, making road development between major or minor urban centers a  
40 key factor in the spread of infectious disease.  
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50 Transportation infrastructure is thus a primary form of ex-urban infrastructure which can lead to  
51 the spread of disease, particularly in outbreak situations (Keil and Ali 2011: 131). During the recent  
52 outbreaks of Ebola in Central and Western Africa the increasing quality of transportation  
53 infrastructure, connecting African cities with each other and the world have been seen as a decisive  
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3 factor (McNeil, Jr., 2019). While Ebola is nothing new to the affected regions, previous cases have  
4 been contained by poor transport infrastructure, making travel between cities very challenging and  
5 time consuming. As Yong (2018) recently observed, the paving of the road between Kikwit to  
6 Kinshasa in the DRC decreased travel time from more than a week to just eight hours. Affected  
7 patients would thus leave Kikwit for Kinshasa seeking treatment, which could infect more people  
8 in Kinshasa. Such connections illustrate the central role of landscape in connecting infrastructures  
9 and local environments, and the ‘enviro-technical assemblages’ that can influence socio-ecological  
10 processes and the spread of infectious disease (see Ali and Keil, 2010; Keil and Young, 2009;  
11 Houston and Ruming, 2014).  
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20 We also need to take into account the *disconnections* that become apparent as rapid demographic  
21 and peri-urban growth is not accompanied by appropriate development of social and technical  
22 infrastructures. The rapid pace of urban expansion has meant that many emerging and existing ex-  
23 urban landscapes contain ‘infrastructure deserts’, especially in the global south, as infrastructure  
24 development has not been able to keep up with the spread of population (Keil, 2018b: 139). For  
25 example, Wilkinson and Leach (2014) have noted that the “precariously expanded urban areas” in  
26 West Africa have become populated by unemployed young people and lack basic municipal  
27 planning and services including access to fresh water or poor sanitation which would increase the  
28 potential threat of water-borne disease and low health indicators. Coker et al. (2011: 603) similarly  
29 found that population growth and urbanisation in Southeast Asia has meant the number of people  
30 using unimproved sanitation and drinking water systems in urban areas has risen by 20 million  
31 between 1990 and 2006. Finally, As Kotsila (2017: 99) found, there is a “considerable number” of  
32 people in Can Tho City who lack access to piped water, but are statistical minorities and as such  
33 do not receive as much attention as those in rural areas.<sup>2</sup>  
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45 Therefore, Filion and Keil (2017) have argued that suburbs in particular are important to study due  
46 to their rapid growth rate that is often coupled with an insufficient infrastructure development  
47 response. This echoes Mulvihill and Ali’s (2007: 356) observation underscoring the vulnerability  
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53 <sup>2</sup> The same is true for waste management in cities, as accumulated waste can be a breeding site for  
54 insect-borne vectors (D’Alisa, 2017).  
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3 of ex-urban places to an intensifying ‘urban shadow’ along the urban periphery. This is especially  
4 true in the global south, particularly in informal settlements whose needs are overlooked by  
5 governments, combined with lower income of residents. For example, Zhang et al. (2008) have  
6 highlighted the paucity of studies in developing countries which study the relationship between  
7 urbanisation and disease. However, ex-urban areas in developed regions are also rapidly growing,  
8 and can likewise be vulnerable if there is little knowledge about how to control a particular disease.  
9 This was evidenced in the case of an American healthcare worker who contracted Ebola in Dallas,  
10 Texas through treating an infected patient who had recently returned from West Africa (Courage,  
11 2014).

### 12 13 14 15 16 17 18 19 20 C. Governance:

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22 The crucial issues of governance and political-economic factors in relation to infectious disease  
23 have been a topic of scientific analysis since the middle of the nineteenth century, when Rudolf  
24 Virchow and John Snow demonstrated the connection between socio-economic context, natural  
25 resource management and outbreaks of various epidemics (Connolly et al., 2017: 3). Subsequently,  
26 health and medical geographers have examined the political economic factors shaping the spatial  
27 distribution of disease in order to achieve a more systemic understanding of health (see Haggett,  
28 1994; Kearns, 1993; Mayer, 1996). In particular, scholars such as Mayer (2000) and Ali (2004)  
29 have demonstrated how certain socio-political conditions associated with a physical setting can act  
30 as structural causes that play a central role in the number and intensity of disease outbreaks in a  
31 given area. In terms of extended urbanisation, this means that disease response mechanisms and  
32 other forms of governance may not be as well established in peri-urban areas, resulting in increased  
33 vulnerability to disease outbreaks.  
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44 One concept through which scholars have addressed the relationship between governance and  
45 disease is that of biopolitics, which refers to the ways in which health and disease has historically  
46 been closely associated with the modern (nation) state and its politics of governing (Braun, 2007;  
47 Rose, 2007; Collard, 2012). We cannot discuss this history in detail here. However, it has a bearing  
48 on our discussion directly through its link with settlement, urbanisation and density. In particular,  
49 it describes how the state controls populations for various purposes, including ostensibly for the  
50 purpose of disease management. Examples include public health, town planning and  
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3 administration, which sought to ‘improve’ the national population by eliminating risks to its future  
4 wellbeing (Braun, 2007). As Collard (2012) notes, biopolitical approaches examine how safe space  
5 is made, maintained and unmade, and how nonhumans (e.g. animals, bacteria, zoonoses) matter to  
6 the material and semiotic construction of ‘safety’ and space. As discussed above, urban political  
7 ecology approaches also discuss the ways in which governance decisions result in unequal and  
8 spatialised patterns of disease whereby particular spaces and population groups face a  
9 disproportionate burden of disease for various reasons (Rose, 2017; Sarasin, 2008).

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17 In this context, political ecology is a useful framework for considering issues of governance given  
18 that political economy and power is central to its analysis of the relationships between humans and  
19 their environment (Kaup, 2018; King, 2010; Turshen, 1984). For instance, King (2010: 42) has  
20 argued that political ecology of health frameworks can illustrate how key actors and institutions,  
21 and human-non-human relationships can influence the transmission of disease and ability of  
22 institutions to provide effective treatment. It can also help to understand how various power  
23 relationships and government policies at a variety of scales can reinforce social inequalities that  
24 influence vulnerability to disease. Kaup (2018), for instance, has drawn attention to  
25 neoliberalisation and privatisation or rolling back of government services as a factor influencing  
26 disease outbreaks, particularly in exurban areas. As he notes, this results in a decreased state ability  
27 to respond to outbreaks when they occur, and to create conditions in which outbreaks are less likely.  
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37 Future studies on extended urbanisation and infectious disease could therefore examine how  
38 government policies might seek to regulate patterns of sub- and ex-urbanisation in the interests of  
39 ‘healthy cities’. One area of focus here should be on the changing composition of ex-urban  
40 populations and communities includes the phenomenon of the “suburbanisation of poverty” which  
41 brings new health concerns to areas that had been traditionally been seen as privileged and well  
42 served by public health agencies and private providers of health care (Kneebone and Garr 2010).  
43 Highlighting spatial inequalities in healthcare provision and response in urban areas is a topic  
44 which is well suited to urban political ecology frameworks, given the field’s focus on  
45 environmental injustice.  
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3 Relatedly, the notion of “political pathology” has also been relevant in the governance of infectious  
4 disease. David Fidler (2004), in particular, has put forward the notion -- with respect to the SARS  
5 epidemic in 2003 -- that this “first severe infectious disease to emerge in the 21st century” was  
6 also the harbinger of a changing global landscape of health governance. Fidler argued that “SARS  
7 is the first post Westphalian pathogen because its nonrecognition of borders transpired in a public  
8 health governance environment radically different from what previous border hopping bugs  
9 encountered” (Fidler, 2004). Importantly, governance now had to recognise that the classical  
10 nation-state centric approach to global health had to adapt to changing realities in a world that  
11 became both more transnational and more localised. The debate on global health security has since  
12 been constantly in the foreground of governance on a rapidly changing planet especially after  
13 recent Ebola outbreaks in West Africa and the DRC (Halabi, et al., 2017).  
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23 As Priscilla Wald (2008: 17) has observed, drawing upon Rosen’s (2015 [1958]) earlier work on  
24 the history of public health in Europe, epidemics ‘dramatise’ the need for regulation with  
25 ‘terrifying urgency’. They further put in place the “administrative machinery for disease  
26 prevention” and protection of public health (Rosen, 2015: 47). As Keil and Ali (2011) found, it is  
27 typically conventional containment strategies, such as isolation and home quarantine, that proved  
28 most successful for controlling the spread of SARS in affected cities. As they note, this is based  
29 on the view of the bounded city with fixed, territorialised and restricted access, which contrasts  
30 with the unbounded and ‘topological’ character of contemporary urbanisation processes. The  
31 Wuhan Coronavirus (nCoV2019) epidemic that spread just as we were completing this paper  
32 sparked the “largest quarantine in human history” which resulted in the closure of the city’s airport,  
33 rail and bus stations to outgoing departures (Gollom, 2020). Wuhan’s urban periphery also became  
34 the setting for a ‘pop-up’ construction of a 1000 bed hospital facility to deal with affected patients.  
35 As Wuhan is locally known as the ‘thoroughfare of China’ (He, 2020), such spatial factors account  
36 for the need for a landscape political ecology approach that can interrogate the relationships  
37 between social actors across multiple spatial and temporal scales.  
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50 While the governance of disease control and prevention has often taken place at a municipal scale,  
51 the increasing porosity between urban, suburban and peri-urban places requires a new approach  
52 (see Houston and Ruming, 2014). Cities are thus reconceptualised “as unbounded and  
53 polyrhythmic spaces, no longer understood in terms of fixed locations in abstract space, but rather  
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3 in terms of a continuously shifting skein of networks, with their own spatiality and temporality”  
4 (Ali and Keil, 2007: 1217). Thus, the growth of megacities and mega-regions raises the critical  
5 question of who has the mandate to control outbreaks in peri-urban areas (see Keil and Ali, 2007).  
6 This issue of jurisdictional authority is particularly noteworthy in the context of public health and  
7 its connection to the unique type of governance relationships that may exist between urban and ex-  
8 urban centers. There is a need for future research in this area, to identify areas for improvement in  
9 urban health governance, which will assist in preventing future outbreaks.  
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## 16 17 **Conclusion**

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20 This paper has thus offered an initial attempt to theorise the relationship between processes of  
21 extended urbanisation and infectious disease, while also establishing the basis for a future research  
22 agenda in this area. The massive increase of the global urban population over the past few decades  
23 has been concentrated primarily in exurban areas, which has posed new challenges to the control  
24 of infectious disease. This includes processes such as population growth and movement between  
25 urban, ex-urban and rural areas, as well as infrastructure provision (e.g. water and sanitation), and  
26 land use change. As we have noted, these processes are especially pronounced in (but not limited  
27 to) developing regions, which have also been the source of recent major outbreaks such as Ebola  
28 and SARS. We have also noted how the governance of infectious disease is challenging, with  
29 overlapping institutional roles and responsibilities in urbanising regions, which poses questions as  
30 to who should do the work of managing (and preventing) potential outbreaks (see Coker et al.,  
31 2011). This is particularly problematic in developing regions, which are often faced with  
32 (inter)national political tensions and inequalities that can hinder effective control.  
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44 Given the scarcity of research on this topic, there remains a crucial need for both academic research  
45 and that which practically informs policy (Coker et al., 2011). In doing so, we have identified three  
46 key areas on which such research efforts should focus, namely: mobility and demographic change;  
47 infrastructure and governance. These have been identified based on existing research in these areas,  
48 at the intersection of urban studies and infectious disease. These three factors do not constitute an  
49 exhaustive list, however, as socio-environmental change - including deforestation and climate  
50 change - has been highlighted by authors as a key risk factor which could lead to the emergence  
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3 of new epidemics, and should form the basis of future research (see Brisbois and Ali, 2010; Tong  
4 et al., 2015).  
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8 We have also illustrated how a landscape political ecology framing which is more attentive to  
9 interactions along the urban periphery, can be useful for examining these topics along  
10 interdisciplinary lines given the holistic nature of the landscape concept, and the diverse  
11 methodological approaches comprising political ecology. The attention to socio-ecological  
12 metabolisms also allows for understanding how outbreaks of zoonoses and other emerging  
13 infectious diseases can be triggered by the expansion of urban settlements in previously forested  
14 or agricultural areas. For instance, the aforementioned outbreak of the new coronavirus first  
15 crossed the animal-human divide at a market in Wuhan, one of the largest Chinese cities with 11  
16 million people. Like in the SARS pandemic of 2003, the connectivities of accelerated urbanization,  
17 heightened mobilities and more extensive zoonotic risks became immediately apparent (Ali and  
18 Keil 2008).  
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28 Such transformations are producing new ecological niches for disease spread, meaning that  
29 exurban regions are likely to remain a hotspot for EIDs into the foreseeable future. This course of  
30 events, continuing as we complete this paper, urges upon urban researchers to seek new and better  
31 explanations for the relationships of extended urbanisation and the spatialities of infectious disease.  
32 This will require an interdisciplinary approach including geographers, health scientists,  
33 sociologists, while also developing possible solutions to prevent and mitigate future disease  
34 outbreaks.. As we have argued, landscape political ecology approaches can contribute to this goal  
35 by helping to identify the political-economic and bio-political factors influencing the spread of  
36 disease through a range of spatial scales in an age of extended urbanisation.  
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## 48 **Acknowledgements**

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51 The research for this paper was funded by a Canadian Social Sciences and Humanities Research  
52 Council (SSHRC) Major Collaborative Research Initiative on Global Suburbanisms: Governance,  
53 Land and Infrastructure. The authors thank participants in a workshop on 'Health and  
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3 Suburbanisms' who gave insightful feedback on an earlier draft of this paper presented at York  
4 University in Toronto in October, 2018.  
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