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The Urban Brain

> It is . . . time for an interdisciplinary neurourbanistic approach that connects public mental health to urban planning to create better environments that will improve the mental wellbeing of individuals and communities in cities, and strengthen the resilience of high-risk individuals and children.
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> —(ADLI, BERGER, ET AL., 2017: 184)

In 2017, a group of German psychiatrists, architects, and urban planners published a letter in *The Lancet* calling for the formation of a new discipline: 'neurourbanism' (Adli, Berger, et al., 2017).[1] Starting from the now familiar refrain about the proportion of the world's population living in cities and the links between urban living and mental health, they called for an interdisciplinary collaboration between the planning and health disciplines to develop tools that would meet the challenges posed by mental ill health in urban environments. Whether or not one needs a new discipline is not the important issue from our perspective; what is significant is the emergence of a new style of thought, in which 'the urban brain' functions as the meeting point between the citizen and the city. It is worth paying attention to the ways of seeing and thinking that have provoked this uneasy combination, as well as the new forms of intervention that are now being assembled within it.

In much contemporary research in neurobiology, the brain is taken out of its corporeal, interpersonal, social, cultural context and isolated in the laboratory, studied in model animals, and increasingly *in silica*—which is to

say, outside its location in time, space, and language (Rose and Abi-Rached, 2013; Mahfoud, McLean, et al., 2017).[2] But what is interesting about the emerging focus on 'the brain' in contemporary urban neuroscience is that brains are *not* imagined as isolated and enclosed organs. Rather, the complex of neural circuits that constitute the brain is understood to develop over time and in space, within an organism that is in receipt of continuous inputs from the environment, not just through the senses but through its interactions with its material environment and with its conspecifics, its encounters with other humans.[3] In this chapter we look in some depth at the contemporary scientific developments that are constituting this urban brain—new modes of visualization, new understandings of stress, recent work on the epigenetic modification of gene expression resulting from environmental exposures, findings on the social modulation of neuroplasticity, research on the role of the 'atmosphere' of the urban or the urban 'sensorium'; and evidence about the biosociality of exposures. We go into this in some detail to show how the urban brain has come to inhabit an explicitly *neurosocial*—even *neuroecosocial*—space of thought and intervention. While we will also point to the ways in which, all too often, the research on the urban brain falls back into various kinds of reductionist neurocentrism—nonetheless, taken together, and transformed through their relations with the social and human sciences, we argue that these new ways of thinking can enable us to re-cast the experience of living in cities as it becomes visible in the body and brain. This will then form the foundation for our final chapter, where we show how a livelier urban biopolitics, a new way of thinking about inhabiting urban space, might *also* be underwritten by this approach.

The Urbanicity Effect

In 1992, a new collaboration was established between researchers at the Institute of Psychiatry in London and the Karolinska Institute in Sweden. As is well known, many Nordic countries have kept meticulous church, census, and other records on their populations since the eighteenth century; this practice then morphed into the Central Population Registers in the 1960s, in which each resident was given a unique personal identification number, making it possible to link together a range of administrative information on each member of the population, including their health data. In their collaboration, the researchers used linked data drawn from the Swedish National Register of Psychiatric Care to challenge the "'geographical drift' hypothesis," that is to say, the argument discussed in a previous chapter that

the higher rates of psychosis in certain areas of the inner city arise because schizophrenics drift into these areas "because of their illness or its prodrome" (Lewis, David, et al., 1992: 137). Arguing that most psychiatrists had given too much weight to endogenous genetic factors in schizophrenia and neglected environmental factors, they found that "The incidence of schizophrenia was 1.65 times higher (95% confidence interval 1.19—2·28) among men brought up in cities than in those who had had a rural upbringing," and that this association persisted even when they adjusted for "factors associated with city life such as cannabis use, parental divorce, and family history of psychiatric disorder. This finding cannot be explained by the widely held notion that people with schizophrenia drift into cities at the beginning of their illness." Hence their unequivocal conclusion: "undetermined environmental factors found in cities increase the risk of schizophrenia" (ibid.). These factors, they went on, may have their effects through 'urban stress' along the lines of the 'stressful life events' previously identified in the work of George Brown and Tirril Harris (Brown and Harris, 1978).

By the turn of the century, this 'urban effect' on health had become widely known as *urbanicity*. In 2001, Carsten Bøcker Pedersen and Preben Bo Mortensen, drawing on data on 1.89 million people from the Danish Civil Registration System, classified everyone on a five-level scale of 'urbanization during upbringing' and linked this to the Danish Psychiatric Register—claiming to find what they termed a 'dose response' relation between urbanicity and schizophrenia. They concluded that "[c]ontinuous, or repeated exposures during upbringing that occur more frequently in urban areas may be responsible for the association between urbanization and schizophrenia risk" (Pedersen and Mortensen, 2001: 1039): their candidates for the unhealthy exposures of urbanicity include infections, diet, and exposure to pollution.

Four years later, Lydia Krabbendam and Jim van Os (Krabbendam and Van Os, 2005) agreed that research did not support the 'urban drift' hypothesis and suggested that a significant proportion of the incidence of schizophrenia may be related to as yet unknown environmental factors in urban environments interacting with genetic risk. While many studies offer clues about the key contenders for these environmental factors (drug use, size of social network, neuropsychological impairment, air pollution, childhood social position), Krabbendam and van Os argued that these tend to homogenize the urban environment and failed to recognize small neighborhood variations, the most important of which might be the level of 'social capital' in these small areas. Other groups of researchers developed this line of

argument, hypothesizing that high levels of social capital would be present when people find themselves among a community of others who came from similar backgrounds. Perhaps the migrants who suffered most from adversity were those who found themselves in a small minority in their new neighborhood, poorly scaffolded by previous generations of movers. These positive and negative influences are sometimes described as 'the ethnic density effect' (Schofield, Das-Munshi, et al., 2016: 3051). Peter Schofield and his colleagues, for example, once again made use of the unique identity number given to all Danish residents: they linked data from the Danish Civil Registration System, which includes demographic details and links to parents as well as continuous updates on place of residence and vital status, to data from the Danish Psychiatric Central Register, which covers all psychiatric in-patient admissions (Schofield, Thygesen, et al., 2017). And, indeed, they showed that living in an area where there are a large number of fellow migrants was associated with a lower rate of psychosis, while migrants who lived in areas with a low 'ethnic density' had higher rates. Despite the fact that the findings for psychosis did not hold for the so-called common mental disorders, they once more argued that the reasons that high ethnic density was protective against the urbanicity effect was because it was an indicator of 'social capital.'

But what is 'social capital'? The idea seems simple and plausible: if you live in an area where there are shared values, and where you trust your neighbors, that feels supportive, and perhaps it is a kind of 'resource' that you can draw on in times of stress. If you had such a resource, such 'capital,' and found yourself in a stressful situation, others would be around to offer comfort, a shoulder to lean on, giving you a sense that you are less alone with the trouble, providing moral, emotional, and even practical support and so forth. No wonder, then, that a common theme in research on mental health and urbanicity is that high levels of such 'social capital' protect against mental ill health, while low levels amplify it (Ehsan and De Silva, 2015).[4]

The term 'social capital' has a convoluted history, arising on the one hand from Pierre Bourdieu's attempts to suggest that the 'capital' that affected an individual's life chances was not merely economic, but also arose from networks of social relations of mutual recognition, group membership, and the symbolic accoutrements of being a valued member of society, etc. Here social capital "is the sum of the resources, actual or virtual, that accrue to an individual or a group by virtue of possessing a durable network of more or less institutionalized relationships of mutual acquaintance and recognition" (Bourdieu and Wacquant, 1992: 119). The American sociologist, James Coleman, had a similar idea: social capital, he argued, is a set of obligations and

reciprocities in networks of persons in social systems, arguing that individuals benefit in many ways from being part of networks of trust and reciprocity (Coleman, 1988). Arising from these twin sources, many others have sought to develop this idea that humans benefit from their embeddedness in groups, and that they suffer when there is an absence of such embeddedness. Soon the simple idea at the basis of this argument began to fragment, and researchers began to argue that there was not just one form of social capital, but a variety—bonding, bridging, linking, cognitive social capital. Unsurprisingly, these were then transmuted into a variety of instruments and scales claiming to measure them. In this process of 'operationalization,' social capital was transformed from a resource that inheres in social relations, culture, and material conditions, to a factor or variable that is 'possessed' by an individual (for helpful reviews, see Almedom, 2005; Moore and Kawachi, 2017).

But is it definitely the case that togetherness is beneficial? As Kawachi and Berkman (Kawachi and Berkman, 2001) point out, perhaps togetherness is important because of a kind of imitation effect, it may be beneficial when one is 'together' with others who espouse healthy or beneficial behavior—but of course, where others espouse unhealthy or dangerous behavior, togetherness might have just the opposite consequences. Extreme togetherness may help mitigate depression, as Brown and Harris found in the Outer Hebrides, but it also can lead to anxiety about whether one is able to conform to prevailing values (Brown and Harris, 1978). And what is this 'togetherness' anyhow? Is it the *reality* of communal support, or is it more a *feeling* of trust and reciprocity—that is to say a perception, a set of beliefs, or what some refer to as 'cognitive social capital'? If so, how is that to be understood—as a psychological condition or as a social relation that emerges from the rituals embedded in social encounters, as explored by Erving Goffman and Randall Collins (Goffman, 1967; Collins, 2004). And extreme 'togetherness' is often the flip side of extreme exclusion, marginalizing those who are not part of that tightly bonded group; so perhaps what is damaging to mental health is not the absence of social capital but social exclusion in and of itself (Morgan, Burns, et al., 2007; Wright and Stickley, 2013). All these complexities threaten to ruin a good hypothesis. But what becomes clear is that, for this literature, the mental health of those who migrate depends, not so much on the fact of migration itself, but on the conditions—physical, personal, social, economic, cultural—that people experience on their arrival, as they come to inhabit particular geographical and ecological places and spaces.

Understanding Urbanicity—To a New Style of Thought?

In the three decades since the publication of the *The Lancet* paper on "Schizophrenia and Urban Life" that we cited earlier (Lewis, David et al., 1962), thousands of articles have been published on the relationship between urbanicity and mental health or mental disorders. Almost all of the studies are epidemiological in character, using statistical correlations to evaluate associations between urban living, or one or another aspect of urban living, and particular psychiatric diagnoses. Almost all start by repeating the familiar theme that this issue is important because global urbanization trends mean that increasing proportions of the population live in cities. And almost all reviews of this body of research agree that it is difficult to draw firm conclusions because of the dearth of good evidence, the variations in measures and definitions between different studies, and the complexity of the associations that seem to be involved.

Let us take a few examples. In a recent special section of *Current Opinion in Psychiatry* on the theme of Urbanization and Mental Health (Szabo, 2019), a series of scholars reviewed the evidence on the relationships between urban living and psychotic disorders, mood disorders, substance misuse, eating disorders, and anxiety and stress-related disorders. Most of the studies reviewed point to the need for better data and the difficulties of undertaking comparisons between different pieces of research; thus, in the case of dementia—where rates seem lower in urban settings than in rural settings—there are pervasive problems of definitions of urban and rural, and definitions of the disorders in question vary greatly between studies (Robbins, Scott, et al., 2019). There are repeated references to the supposed benefits of 'green space'—in reviewing neighborhood-focused research on the consequences of urbanicity for anxiety and stress-related disorders, authors conclude that "physical (e.g. green space), social (e.g. social cohesion) and biological (e.g. stress response) factors—are directly linked to the presence and severity of anxiety disorders . . . architectural and space design elements . . . can either increase anxiety and lead to trauma triggers or relieve symptoms and reinforce safety" (Ventimiglia and Seedat, 2019: 248). The allure of the microbiome is powerful with "emerging evidence that being raised in urban environments with a wide range of microbial exposure dampens the immune response to psychosocial stressors" (ibid.).

As for mood disorders, it seems that only depression has been seriously explored; the reviewers find that "individuals residing in urban areas

experience increased risk of depression. Mechanistic pathways include increased exposure to noise, light and air pollution, poor quality housing, reduced diet quality, physical inactivity, economic strain and diminished social networks" (Hoare, Jacka, et al., 2019: 198). In a later review of the global literature on depression: Laura Sampson and her colleagues find "higher adjusted odds and/or severity of depression in urban areas compared with rural areas in the Netherlands, the United States, India, and Vietnam"; in China, depression was less common in urban than in rural areas, while studies in Ghana, South Africa, and the Netherlands showed no clear relationship (Sampson, Ettman, et al., 2020: 233). It is worth quoting their remarks on these complexities at some length:

> Given the many different aspects of urban living—some of which promote and some of which inhibit mental health—a focus on more manageable, modifiable factors may be fruitful, while simultaneously appreciating the full framework of multilevel influences on health. There are two major challenges to studying the relationship between urbanicity and depression. First, it is difficult to separate the effect of living in an urban area from the effects of higher income or other resources that are often necessary for someone to either migrate to or remain in an urban area. . . . Similarly, for those who migrate, it can be difficult to disentangle effects of urban living from effects of the process of migration itself or the underlying reasons for migration. . . . [The] ubiquity of urbanization presents a challenge for traditional epidemiologic study. Given the many possible mediators and moderators on the complex pathway from urbanization to depression (Galea and Vlahov, 2005) researchers should apply solid theoretical frameworks and causal thinking approaches . . . and move beyond single exposure–outcome association studies. At the same time, more granularity in defining and measuring specific exposures of interest may be warranted; simply comparing large urban areas to large rural areas may mix together many different exposures. (ibid.: 242)

A similar conclusion can be found in the review of urbanicity-psychosis associations in high-, middle-, and low-income countries by Anne-Kathrin Fett, Imke Lemmers-Jansen, and Lydia Krabbendam—showing "complex patterns of urbanicity–psychosis associations with considerable international variation within Europe and between low, middle and high-income countries worldwide" (Fett, Lemmers-Jansen, et al., 2019: 232). They suggest that social and economic stressors such as migration, ethnic density, and economic deprivation, exposure to nature, and access to resources could

only explain part of the urbanicity effects: "Urbanicity–psychosis associations are heterogeneous and driven by multiple risk and protective factors that seem to act differently in different ethnic groups and countries" (ibid.). The way forward, in their view, lies in interdisciplinary research "combining approaches, for example from experimental neuroscience and epidemiology . . . to unravel specific urban mechanisms that increase or decrease psychosis risk" (ibid.).

What might such an interdisciplinary approach look like? A later paper by Lydia Krabbendam and her collaborators sketches out one answer (Krabbendam, van Vugt, et al., 2021). The authors are moved by E. O. Wilson's 'biophilia' hypothesis that humans may not be evolutionarily equipped for urban living, given the short history of cities. Indeed, they suggest that humans have an innate love for nature that is disrupted by urban existence, and that urban environments tax our cognitive resources which are restored by contact with nature. One might wonder whether the words 'nature' and 'green'—while they may connote a certain bucolic vision of the forests and fields of northern Europe—adequately capture the evolutionary landscape within which the first hominids emerged. No doubt it is true that many people today find images or experiences of cultivated forests, parks, and gardens restful, yet it seems doubtful that they would feel the same if in an unmapped rain forest or on the featureless bush, however green they were. Indeed, we should hesitate before drawing universal conclusions about an innate love of green nature from research with modern urban humans, many of whom are themselves imbued with a historically and culturally shaped romance of the natural. Arguments about 'cognitive overload' in cities surely need a more subtle approach to the structured and systematic differences in inhabitation and the very different relations to urban space that they entail.

While in 2005, Krabbendam had speculated about the importance of genetic risk, by 2020 she and her colleagues doubted that genetics played a significant part in these urban-rural differences. On the contrary, they return to the theme that while city living may produce 'cognitive overload,' the natural environment has 'salutogenic effects.' These effects might include better immune function, lower blood pressure, and enhanced physical and social activity, as well as alterations in the volumes of grey and white matter in the brain; they might also affect the activity of the neural receptors responsible for social stress processing, and these might have effects beyond the individual exposed to their unborn offspring. Drawing on a number of neuroimaging studies, they suggest "stress sensitization through environmental stressors, neurotoxicity and neuro re- and degeneration as possible

neurobiological pathways that mediate urbanicity effects on cognitive functioning and mental health. Future studies need to systematically investigate multiple mechanisms that could underlie the urban effect on the brain (e.g. exposure to toxic or noise pollution, social stressors) . . . To improve our insight into which specific urban features are involved, experimental and experience-based studies that investigate immediate responses to specific physical and social characteristics of urban environments will be indispensable" (Krabbendam, van Vugt, et al., 2021: 1014).

Krabbendam and her colleagues thus recognize the importance of engaging with disciplines such as sociology, anthropology, urban planning, and geography; they conclude their review by suggesting that researchers need to start from the position that "the lived experience and sense-making of subjects are crucial for analysing the effects of urban or natural milieus on mental health." They thus suggest the use of ecological momentary assessments, which sample individual experiences of their urban environment in real time, and map these using GIS and other data, combined with the use of mobile EEG devices that can directly monitor the psychological effects of urban stress on the brain, and allow their correlations with psychological indices of stress—thus to unravel the "multiple interacting pathways and reciprocal relations of the urbanicity–mental health conundrum" (Krabbendam, van Vugt, et al., 2021: 1016). What would we find, then, if we really did venture beyond the correlational styles of thought in epidemiology and focused on what was really going on in individual urban brains in 'real time'—as their human bearers lived, moved, inhabited, experienced urban space?

Seeing the Urban Brain

Of course, there is a long history of brain visualization, of attempts to see human characteristics, and human pathologies in the cerebral tissues themselves. In the nineteenth and early twentieth centuries, the dead brains of criminals, the mad, and the brilliant were extracted, preserved, classified, mapped, and anatomized in attempts to correlate the intellect with the size or weight of the brain or the topography of the cortex (Hagner, 1997; Hagner, 2001; Hagner and Borck, 2001; Hecht, 2003). Those dead brains sadly failed to give up their secrets. Only in the twentieth century were technologies developed that could overcome the seemingly impenetrable barrier of the skull, to visualize the living brain in situ (Kevles, 1997). We moved rapidly from techniques that involved the painful injection of air or dyes into the vessels that entered the skull to technologies such as Positron

Emission Tomography (PET) and functional MRI (fMRI) that seem to show the activity of the living brain as it thinks, feels, desires, decides, and experiences the world around it (these are reviewed in Rose and Abi-Rached, 2013: ch. 2). Most of those technologies are 'heavy' and tied to the laboratory. PET scans require a person to be injected with radioactive tracers that bind to certain biological molecules, and then to lie in a large machine that takes images that show the take-up of the 'tagged' molecules in different areas of the body or—in our case—the brain (Dumit, 2003). fMRI requires the subject to remain motionless in a large and noisy scanner while a powerful magnet takes advantage of the fact that de-oxygenated hemoglobin is more magnetic than oxygenated hemoglobin, visualizing variations in oxygenation thus serves as a proxy to show increased brain activity in certain regions (Beaulieu, 2000). However, newer, 'lighter' technologies have been developed, such as mobile EEG devices that enable patterns of brain waves to be monitored and recorded during activity,[5] or Near Infrared Spectroscopy (NIRS), which can monitor and record levels of blood oxygenation in the layers of the cortex just beneath the skull, thus providing a proxy measure of brain activity, especially in the frontal cortex (Denault, Shaaban-Ali, et al., 2018). For researchers, despite the limited penetration of these devices into the depths of the brain, the advantage is that both increase the capacity for mobility, enabling the research subject to move around an environment (Bunce, Izzetoglu, et al., 2006).[6] It seems that, at last, one can see the urban brain in action.

In 2005, in Chiba prefecture in Japan, a team of researchers took seventeen female participants out for a walk.[7] First they went to a forested area. Then they walked around an urban station in Chiba City. At different points during the day, the researchers used NIRS to measure hemoglobin in cortical tissue in particular brain regions in real time—thus comparing the effects of walking in the two different areas, in terms of the participants' brain physiology (Tsunetsugu and Miyazaki, 2005). Much to the pleasure of the researchers—who were, not coincidentally, from Japan's Forestry and Forest Products Research Institute—significantly lower levels of oxygenated blood were detected for the forest as opposed to the city areas after walking—showing, the authors argued, "that in a forest environment, the activity in the prefrontal region was calmer than in a city environment" (ibid.: 469). Indeed, these researchers were not concerned with the neural effects of urban experience, but with that of the *forest*. This was just the start of a series of studies on 'the physiological effects of *Shinrin-yoku*'—that is to say, the effects of immersion in the forest atmosphere or forest bathing. Most

of these did not access the brain directly but used other physiological measures such as salivary cortisol, blood pressure, pulse rate, and heart rate variability (Park, Tsunetsugu, et al., 2009; Hansen, Jones, et al., 2017) to assert the importance of "increased awareness of the positive health-related effects (e.g., stress reduction and increased holistic well-being) associated with humans spending time in nature, viewing nature scenes via video, being exposed to foliage and flowers indoors and the development of urban green spaces in large metropolitan areas worldwide" (Hansen, Jones, et al., 2017: 895).[8]

Some continued to try compare the 'urban effect' and 'the rural effect' using brain imaging laboratory studies. Thus, a group of researchers at Chonnam University in Gwangju, South Korea, used fMRI to measure the brain activation of a series of participants, while they looked at images of variously rural ('forests, gardens, parks and hills') and urban ('high buildings, offices, electrical cables, garbage collections') scenes (Kim, Jeong, Baek, Kim, et al., 2010). In this case, while viewing rural scenery, the participants showed greater activity in areas of the basal ganglia, "important for positive emotions"; by contrast, when looking at urban scenes, participants showed activity in brain areas associated with aversive imagery and with evaluating cues that might predict danger (ibid.). Thus, the authors argued, participants showed "an inherent preference towards nature-friendly living" (ibid.: 2607). Their findings, they concluded in another paper published the same year,

> support the idea that the differential functional neuroanatomies for each scenic view are presumably related with subjects' emotional responses to the natural and urban environment, and thus the differential functional neuroanatomy can be utilized as a neural index for the evaluation of friendliness in ecological housing. (Kim, Jeong, Kim, Baek, et al., 2010: 507)

The belief that while the city jangles the nerves, swards of green have the opposite effect seems to have been the intuition behind an experiment conducted in Edinburgh a couple of years later, reported in an article entitled "The Urban Brain" (Aspinall, Mavros, et al., 2013). These researchers equipped their participants with a portable Emotiv EPOC™ 'wireless EEG' headset—which recorded electrical activity at fourteen different locations on the skull, using a proprietary algorithm that translated EEG data into four "emotional parameters," viz. frustration, engagement, excitement, and meditation. They then walked twelve people (individually) through three distinct areas of Edinburgh: an urban shopping street with light traffic, a

green space with lawns and trees, and a busy commercial district. Following the walk, the researchers correlated the output from the devices with the participants' presence in the different zones: how did different experiences of the environment correlate with the algorithms representing people's emotional states? The most significant finding showed a marked difference in activity as people moved from busy streets to quiet green areas: "the transition from Zone 1 to Zone 2 (urban shopping street to green space) . . . [shows] reductions in arousal, frustration and engagement (i.e. directed attention) and an increase in meditation" (ibid.: 5). The authors proposed that, in the future, studies like theirs might be "particularly beneficial in exploring the health improving potential of environments while people are on the move" (ibid.: 5).

Many similar studies followed, some seeking to directly measure urban effects in the brain, others using different physiological measures to support the view that, while urban living rattled the nerves, experiencing, seeing, walking in nature was good for the brain (Kondo, Jacoby, et al., 2018). There are enormous technical issues in these studies—in particular, issues around the sensitivity of mobile measures. Nevertheless they seek to go beyond a general belief that experiences of rural or urban environments must somehow be inscribed in the brain, to render such beliefs technical so that these inscriptions can be measured in real time by brain reading devices.[9] Urban experience has become neural.

These studies—and we have offered here only a small selection—provide a novel way of imagining and mapping the experience of cities. They encourage us to see those cities in terms of levels of stimulation, to map the spaces of the city in neural terms. We are urged to see the city *in the brain*, as urban citizens wind their way from place to place—here, noisily frustrated; there, calmly restored—often simplified into the differences between 'grey' and 'green' spaces. Stress is, once again, at the heart of these arguments. On the basis of their systematic review of studies of stress in relation to various outdoor environments, Michele Kondo and colleagues conclude that there is "convincing evidence that spending time in outdoor environments, particularly those with green space, may reduce the experience of stress, and ultimately improve health" (Kondo, Jacoby, et al., 2018: 136). They suggest that exposure to stress is "one of the ways that environmental and neighbourhood conditions 'get under the skin' and lead to poor health and associated health disparities . . . Conditions such as blight, segregation, poor social cohesion, and violence combine with personal experiences like job insecurity and discrimination to produce a range of persistent stressors"(ibid.: 148).

A novel field of intervention on urban mental health is thus opening up within the nexus of urban subjectivity, neural circuits, and urban design—and their associated regimes of governance. This argument was catapulted into popular debate by some research from Andreas Meyer-Lindenberg and his group based at the Central Institute for Mental Health in Mannheim, Germany. The study that they published in 2011 (which has since been cited more than 1,000 times) was reported not just in journals such as *Science* ('The Mental Hazards of City Living')[10] and *Nature* ('City Living Marks the Brain')[11] but also in *Wired* ('City Life Could Change Your Brain for the Worse)'[12] and many similar semi-popular outlets. The claim made by these researchers was that they had begun to identify the *mechanisms* that accounted for the fact that, while urban dwellers were, by and large, better off than their rural cousins, they experienced poorer mental health (Lederbogen, Kirsch, et al., 2011). And we will not be surprised to learn that these hypothesized mechanisms revolved around 'stress.'

Recruiting volunteers from cities, towns, and rural areas in Germany, the Mannheim group put their subjects into fMRI scanners, and set them a task of solving an arithmetical problem. They subjected them to various levels of 'stress' by putting increasing pressure on them to complete the task rapidly and to do well. As this pressure increased, so did heart rate, blood pressure, and cortisol levels—taken to be measures of stress. But also, as 'stress' increased, it seemed that different patterns of brain activation occurred depending on the extent and timing of exposure to an urban environment; in short, they processed stress differently:

> Our results identify distinct neural mechanisms for an established environmental risk factor, link the urban environment for the first time to social stress processing, suggest that brain regions differ in vulnerability to this risk factor across the lifespan, and indicate that experimental interrogation of epidemiological associations is a promising strategy in social neuroscience. (Lederbogen, Kirsch, et al., 2011: 498)

According to a report in *Nature* by Alison Abbott, one of their leading science journalists, Meyer-Lindenberg had first thought of the cerebral effects of city living while studying in New York, where he was "struck by the number of homeless mentally ill people on the streets," and began to wonder "if city living was somehow making the brain more susceptible to mental-health conditions" (Abbott, 2012: 164). Meyer-Lindenberg's surprise suggests that some neurobiologists are less than fully immersed in the heated debates on mental health policies and their consequences. Nonetheless, the research

itself painted a picture of mental life in the city that many commentators found compelling. Accompanying Abbott's report was a highly stylized and abstracted model of an 'urban habitat,' showing a small, triangular, green space, boxed in by grey buildings. A series of blacked-out human figures are dotted around, with captions describing their affective states: one, marked as 'Relaxed,' is depicted on a green square beside a tree; a huddle of figures in the middle distance are identified as 'Anxious,' while two more forlorn figures, far off to the right of the image, are marked as 'Isolated' and 'Lonely,' respectively. A caption above the last of these informs us that "feeling different to neighbours—owing to socioeconomic status or ethnicity—may be a factor. Immigrant populations have an increased risk of psychiatric disease" (ibid.). The urban, here, is not a space of cosmopolitan mingling, of civilized living, commercial vitality, cultural effervescence, and so on. On the contrary, reduced to a cartoon that bears no relation to the lived experience of any city dweller, it is represented as an array of potentially pathogenic neurobiological spaces.

If we return to the research itself, we can see that the way that stress was actually simulated in the laboratory experiments was as abstracted from lived experience as are these cartoons of urban life. The Meyer-Lindenberg group, like others, simulated stress in laboratory conditions because this was necessary if they were to visualize stress while their subjects were in fMRI scanners, which of course allow much more detailed mapping of brain activation than the EEG devices used in the naturalistic research described earlier in this chapter. The experiment used the Montreal Imaging Stress Task (MIST), "a social stress paradigm where participants solve arithmetic tasks under time pressure," giving them negative feedback on their performance by showing them a 'performance scale' and providing further negative feedback through headphones. This setup produced the fMRI images that were interpreted to show different patterns of brain activation for those who had been *brought up* in cities and those who *currently lived* in cities. They suggested that chronic activation of stress pathways at different stages in brain development had led to different ways of processing stress in the two groups. For those brought up in cities, they claimed to have found a "regionally specific effect on the pACC [the pregenual anterior cingulate cortex], a major part of the limbic stress regulation system that exhibits high neuronal glucocorticoid receptor expression, modulates hypothalamic–pituitary–adrenal axis activation during stress, and is implicated in processing chronic social stressors such as social defeat." On the other hand, the high levels of amygdala activity they found in those who currently lived in cities when

subjected to stress "signals negative affect and environmental threat" and might be related to "anxiety disorders, depression, and other behaviors that are increased in cities, such as violence" (Lederbogen, Kirsch, et al., 2011: 499).[13] This research thus seemed to show that there were indeed 'urban brains' that took different forms depending on the timing of exposure to urban stressors during one's life course.

We will, again, leave to one side the many technical objections one might make to this laboratory research and its extrapolation to everyday urban existence. What is more interesting is that, despite these issues, an array of psychiatrists, planners, architects, and urban policymakers began to argue—in scientific journals, the mass media, and an array of conferences and reports—that the research provided the basis for new practices for intervention and management. Launching a program by the International Council for Science in late 2014, for example, Anthony Capon explained to journalists how cities were associated with growing problems in noncommunicable disease and mental health: "The essence of this programme," Capon said, is about "scientists working with urban decision makers. It is about identifying problems together, and how we might better understand those problems and developing better ways of responding to rapid urban population growth" (quoted in Kinver, 2014). Richard Coyne, a professor of architectural computing at Edinburgh College of Art, and a co-author of the paper that tracked mobile EEG measures in Edinburgh, was clear about the implications of the kind of work that he and his colleagues were doing. 'Our study,' wrote Coyne on his blog in 2013:[14]

> has implications for promoting urban green space to enhance mood, important in encouraging people to walk more or engage in other forms of physical or reflective activity. More green plazas, parkland, trees, access to the countryside, and urban design and architecture that incorporates more of the atmosphere of outdoor open space are all good for our health and wellbeing.

This first wave of research, which compared neural responses to stress in those born in or living in cities, and which urged planners to provide access to green spaces to mitigate the stresses associated with noisy, crowded, and overstimulating urban environments, was hardly adequate to what we know of the multiplicities of urban habitats and the diversity of those who inhabit them. It did not give us much specificity about the kinds of urban experience that are hinted at in ideas like 'social exclusion,' 'social capital,' and 'social defeat' and their role in mental ill health. Nor did it help us understand who,

among those who inhabit cities, might fare best and who might fare worst, let alone why. The urban inhabitant was depicted as an interchangeable data point—as if the distribution of the environments of inhabitation across the contemporary city was not—and had not historically been—distributed unequally through hierarchies of wealth, race, and class.

Subsequent research has tried to offer something that we might consider more 'ecologically valid.' In a special issue of the journal *Current Opinion in Psychology* on socio-ecological psychology, Markus Reichert, writing with co-authors including Meyer-Lindenberg, argues for "ambulatory assessments," which use smartphone-based ecological momentary assessments, smartphone diaries, various monitors of physiological function such as accelerometers, ECG monitors, and fitness trackers combined with highly accurate GPS-based location tracking. Belatedly recognizing the limits of laboratory-based studies and retrospective reports of emotions, this group now argues for the ecological validity of real life measures that "capture how emotions, thoughts and behaviours fluctuate across time . . . modulated by biological, cognitive and contextual factors" (Reichert, et al., 2020: 159). But if the strength of the real-time ecological assessments is precisely that they map stress—and associate emotions and thoughts—in real time, this is also their limitation. 'Real time' is, of course, not a continuous flow of presence—humans live in the confluence of the currents of manifold pasts, ambiguous presents, and the shadow of multiple potential futures. For a sociologist or a philosopher, the presence of the past is a matter of memories, associations, sedimented meanings in streets and artefacts, stories, and myths. For neuroscientists, by contrast, the past is present because of its sedimented consequences in the human brain. And, for most, that sedimentation is a result of one major factor—stress.

The Biopolitics of Stress

If stress theories of mental disorder have achieved prominence once more, it is in part because of a hypothesis that has become widely accepted about the biological mechanisms by which a subjective perception of stressors can act on the brain. In the familiar 'fight or flight' response, something that is experienced by an individual as 'stressful' results in the increased production of cortisol, a glucocorticoid that is produced in the adrenal gland. Cortisol has a wide range of effects on the body, the most significant of which is the increased production of glucose, together with an inhibition of the production of insulin, thus preventing that glucose from being stored. This increase

in blood glucose prepares the organism for 'fight or flight' by providing the muscles with an easily available energy source and simultaneously narrowing the arteries, and generating increased production of epinephrine, which raises heart rate and blood flow. However, if high levels of glucocorticoids continue over time, this leads to the production of cytokines—small molecules that act as signals between cells and are especially important in immune and inflammatory responses. While the consequences are complex, continued production of cytokines upregulates the immune response, producing inflammation.[15] Sustained activation of these cytokines has a wider impact. In particular, it leads to changes in the size of the amygdala, the medial-frontal cortex, and other brain regions by modulating the HPA axis—the connections between the three adrenal glands of the hypothalamus, the pituitary gland, and the adrenal glands. Contemporary stress researchers argue that it is these effects on the brain that lead to changes in mood, emotion, and behavior, and that studies—once more mostly carried out in rodents—show that prolonged high levels of cytokine activation are associated with symptoms similar to those of certain psychiatric conditions (Dantzer, O'Connor, et al., 2008; Pariante and Lightman, 2008; van der Kooij, Fantin, et al., 2014; Sandi and Haller, 2015).

Bruce McEwen was one of the most tenacious researchers seeking to draw conclusions for physical and mental health from thinking about the neurobiological effects of stress. He argued that the evolved response to stress—'allostasis' or stability through change (Sterling and Eyer, 1988)—is protective; however, prolonged or repeated elevation of stress hormones has long-term consequences, increasing what he termed 'allostatic load,' that raises future risks of developing disease (McEwen, 1998; McEwen and Lasley, 2002; Lupien, McEwen, et al., 2009; McEwen, Bowles, et al., 2015). Further, seemingly appreciating the psychological and sociocultural evidence that what is crucial is not objective circumstances but the cultural shape of one's perception of a situation, McEwen insisted that "[s]tress is a state of the mind, involving both brain and body as well as their interactions," and it is the brain that is "the central organ of stress and adaptation" (McEwen, 2012: 17180). The social and physical environments get 'under the skin' through the neuroendocrine, autonomic, and immune systems, both in the course of early development and during later experience. Over time, stress has enduring effects through a hormonal cascade which acts on the structural plasticity of the hippocampus and other brain regions, notably the amygdala, the prefrontal cortex, and the nucleus acumbens. Its consequences for neuronal changes are not merely shown in animal studies but

are supported by neuroimaging studies in humans: both acute and chronic stress inhibit neurogenesis, and affect synaptic turnover, spine density, branching and length of neuronal axons (Davidson and McEwen, 2012). While a perception that one is in a dangerous, precarious, or threatening situation for oneself or those one cares about normally produces adaptive change, when such a perception is chronic, stress becomes 'toxic' and toxic stress has "implications for understanding health disparities and the impact of early life adversity and for intervention and prevention strategies" (McEwen, 2013: 673).

Thus, a mechanism by which a variety of subjective experiences, perceived as stressful as a result of individual socio-biographically and socio-culturally shaped meanings, can be translated into neurobiological configurations with deleterious consequences for brain and body. Of course, the argument that stress is a subjective response to external 'stressors' does not by any means imply that the distribution of such stress is independent of the actual distribution of potential stressors such as lack of money, insecurity of employment, bad living conditions, isolation, noise, pollutants, problems with traveling to work or walking to the shops, threats of violence, and so forth. While those external stressors inescapably increase the likelihood that individuals will perceive themselves as subject to stress, they do not inevitably determine those perceptions. On the one hand, as we saw in our discussion of the migrant experience in Shanghai, people may become inured to them or stoical in the face of them. On the other, experiences that many would find entirely acceptable, such as walking into a crowded space, taking public transport, or encountering strangers on the street, may appear exceptionally threatening to some.

This focus on 'toxic stress' and specifically 'toxic stress' in childhood, is shared by many who adopt a broadly 'neurodevelopmental' approach to mental disorders. Consider, for example, the way that the issue of stress, and of toxic stress, is framed by the World Health Organization Report on *The Social Determinants of Mental Health* (World Health Organization, 2014). This report first reviews the evidence that has unequivocally established that common mental disorders such as depression and anxiety "are distributed according to a gradient of economic disadvantage across society" (Campion, Bhugra, et al., 2013), and that there are consistently strong associations between poverty—in particular food insecurity, poor housing, financial stress, and similar indicators of a precarious and demanding form of life—and 'common mental disorders' (Lund, Breen, et al., 2010). Having painted social factors in such broad terms, and pondering which of these

broad demographic indicators might be most important, we will not be surprised to find that the authors of the WHO report settle for a common explanation for the effects of these different forms of adversity: stress.

Of course, stressful experiences do not always lead to mental disorders, and such disorders can occur in the absence of such experiences, so the report invokes the notion that social supports 'buffer' against stress: "the level, frequency and duration of stressful experiences and the extent to which they are buffered by social supports in the community" is what counts. And, in its view, those "lower on the social hierarchy" are more likely to be subject to such experiences and have access to fewer buffers and supports (World Health Organization, 2014: 17–18). Their choice of the term 'buffer'—rather than the psychological language of appraisal, adaptation, and coping—invokes familiar arguments about 'social capital,' now wrenched from its roots in the work of Bourdieu and Coleman in order to allude to the extent of strong social ties of reciprocity in particular communities. Indeed, echoing Richard Lazarus but without referring to his work, what turns out to be crucial is 'cognitive social capital'—the *belief* by individuals that such ties exist and that there are others to call on in times of difficulty.

The WHO report echoes the "ecobiodevelopmental" framework of Jack Shonkoff suggesting that "toxic stress" can occur "when a child experiences strong, frequent, and/or prolonged adversity—such as physical or emotional abuse, chronic neglect, caregiver substance abuse or mental illness, exposure to violence, and/or the accumulated burdens of family economic hardship—without adequate adult support" (Shonkoff, Garner, et al., 2012: e232).[16] The Report thus paints both 'the environment' and 'buffers' in terms of very broad factors, while recapitulating some rather old arguments about the importance of 'adequate' support in early life for future mental health.[17] However, we can see some of the problems of such an analysis in stress-based arguments that try to explain the fact that children from poor families tend to end up as poor adults, or, to put it more generally, why the social class of parents is predictive of the social class of their children. For example, Bruce McEwen,[18] in a paper co-written with his sociologist brother Craig McEwen (McEwen and McEwen, 2017), draws upon the work of a number of neuroscientists to argue that development of the prefrontal cortex in infants is "involved" in "observed SES disparities" (Hackman and Farah, 2009: 68).[19] They argue that toxic stress "alters its structure and functioning through the release of hormones and neurotransmitters by nerve cells" (McEwen and Morrison, 2013), and refer to the work of Shonkoff cited above in concluding that the consequences include compromised capacities

for behavioral and emotional self-regulation, including working memory and executive function.

The emphasis on the enduring neural consequences of events in the early years of a child's life are part of a wider style of thought that is having significant policy impact in a number of countries, including those in the Global South (see Pentecost, 2018). Shonkoff's own work (Shonkoff, Garner, et al., 2012) underpins the school of thought and intervention that focuses on Adverse Childhood Experiences, or ACEs.[20] A related school of thought, framed in terms of the Developmental Origins of Health and Disease (DOHaD), has its origins in David Barker's work on the correlations between birthweight and later ischemic heart disease (Barker, 2007), or 'the foetal origins of adult disease' (Cooper, 2013), proposes that exposures in utero or 'the first 1000 days' has a 'programming effect' on the brain that establishes the foundations of optimum health, growth, and neurodevelopment across the lifespan.[21] These movements seem full of virtuous intentions, for example wishing to draw attention to the need to remedy the social disadvantages experienced in early childhood that inhibit neural development. However, as Daniel Hoffman and his colleagues point out, "while a large number of studies provide evidence that supports the concept of DOHaD," most of these have focused on poor early nutrition, drawing on data from children exposed to famine or from longitudinal studies and arguing that these are 'risk factors' for later chronic diseases; researchers have had to extrapolate from animal studies to support their postulations of the potential epigenetic, metabolic, and endocrine mechanisms involved (Hoffman, Reynolds, et al., 2017). Whether or not extrapolation from laboratory research with rodents—mice, rats, and sometimes guinea pigs—is valid in the case of Type 2 diabetes, hypertension, or coronary heart disease, such extrapolations are certainly perilous in the case of mental disorder. The almost ubiquitous dependence on animal models for neurobiological conclusions is sometimes acknowledged in this literature (Shonkoff, Boyce, et al., 2009: 96), but the implications are almost never addressed.[22] For example, the widely cited 2013 paper by McEwen and Morrison, to which we have just referred, relies entirely on evidence from animal models for its claims about the vulnerability of the development of the prefrontal cortex to stress (McEwen and Morrison, 2013).[23,24]

More promisingly, perhaps, Susan Prescott and Alan Logan have called for attention to 'ecological justice' in what they term the 'dysbiosphere' (Prescott and Logan, 2016).[25] Their proposal takes a much broader view of the developmental origins of health and disease, addressing "the

forces—financial inequity, voids in public policy, marketing and otherwise—that interfere with the fundamental rights of children to thrive in a healthy urban ecosystem" while also focusing on the "grey spaces" of socioeconomic disadvantage that "insidiously reinforce unhealthy behaviour, compromise positive psychological outlook and, ultimately, trans-generational health" (ibid.: 1075). Not only do Prescott and Logan suggest that "greater focus on positive (beneficial) chemical and non-chemical, biotic and abiotic, family, neighbourhood and societal 'exposures' might uncover mechanisms of resiliency that go far beyond disease prevention and extend into health" (ibid.: 1076)—but also that we need to attend to how already disadvantaged populations bear a dual burden because they tend to inhabit urban "grey space", areas that "may include disproportionately higher industrial operations, commercial activity and major transportation routes, with resultant noise stress and excess light at night," as well as "residential proximity to higher levels of grey space and less equitable access to biodiversity," as well as large numbers of "bars, liquor stores, convenience stores, fast-food outlets, and tobacco vendors" coupled with "profit-driven marketing, billboards, sidewalk signage, in-store magnification of unhealthy products and targeted screen media delivery that make grey space an entirely different 'mental' environment" (Prescott and Logan, 2016).

The seemingly inescapable recurrence of the opposition of grey space to green space is at least given some substance here—grey space is not bad for one simply because it is not green, but because, because of the highly inequitable distribution of populations across urban space, the 'grey spaces' in which most disadvantaged people live are characterized by a high likelihood of toxic exposures. Here we can see how such an approach to what Prescott and Logan term "mental environments" might help us make our way through the pathways between specific modes of urban life and particular forms of physical, mental, and societal health. It is crucial not simply to pathologize neighborhoods because they do not conform to a certain bourgeois idea of good urban life. Yet we need to attend to the shaping and constraining of forms of life by discriminatory politics, policies, and planning over many generations; policies that have emplaced social exclusion and racism deep into the fabric of our cities, affecting not merely 'the first thousand days' but the whole course of the lives of those who have no option but to inhabit them. From this perspective, stress does not need to be an individualizing notion or one that roots all subsequent problems in early experiences of adversity. Precarity—in housing, employment, finance, and basic security—together with high levels of toxic ambient exposures,

is endemic in the lived experience of those most excluded, including many communities of migrants and refugees.

If we want to figure out the pathways for the embodiment of adversity, then we need to start from these ecosocial questions—rather than from the premise that the line of causation begins with toxic stress in inadequate families that damages the developing brains of individual children and dooms them to a suboptimal future in physical and mental health and intellectual development. An ecosocial account of the shaping of the urban brain would map out the shaping of stressors through collective experiences of poverty, exclusion, isolation, racism, violence, and environmental degradation. It would deepen such an understanding by exploring how these experiences get understood, and the ways that these are assigned meaning in specific groups and communities. It would seek to identify what there might be within urban life that mitigates against stress, whether that be the physical environment of individual apartments or houses, the layout of streets and the organization of public space, mundane experiences in cafes and shops and informal friendships, as well as more substantial forms of collective organization and mutual aid. And all of this leads us to a different question: what kind of biopolitics might such a neuroecosocial understanding of urban existence enable? Let us turn to some further research that can help us to think through these questions.

Epigenetics: Beyond the Genetic Program

As we know, there is a long history of the belief that madness—and all its synonyms from lunacy to mental disorder—is hereditary, or at least that individuals inherit a constitutional susceptibility to mental disorder that can be triggered by adverse circumstances of various sorts. From the inception of modern psychiatry in the mid-nineteenth century, most psychiatrists held to something like this view at least when it came to serious mental disorders. From the 1930s onward, there was much research that sought to demonstrate it—for example showing that if one of a pair of identical twins developed a mental disorder, the other was highly likely to develop a similar disorder, and that this concordance was much more likely than in the case of non-identical twins. Research tried to locate the gene or genes that were responsible, often announcing the discovery of 'the gene' for this or that condition, only to discover later that their findings were not replicated.[26] Many psychiatric geneticists, like other life scientists, thus moved away from simple genetic determinism to argue for the importance of gene-environment interaction,

in which individuals inherited, not a 'gene for' a condition, but a genetic sequence that increased the likelihood of developing such a condition where an individual was exposed to familial or environmental adversity at particular periods in their life course (Tabery and Griffiths, 2010). As sequencing of the human genome advanced, much effort was expended to try to identify the variations at the level of DNA sequences that were linked to susceptibility to mental disorders, again with little success.[27] While some thought that better results would be obtained with larger samples, better diagnoses, and more sophisticated algorithms, others sought to find different ways, for instance those based on large, long-term cohort studies that combined genetic data with information on key life events to disentangle themselves from simplistic genetic arguments about causation, some suggesting a reversal of terms, such that environmental factors like childhood maltreatment were construed as causes that led to mental disorders only in the presence of certain genetic variants (Caspi, McClay, et al., 2002; Caspi, Sugden, et al., 2003; Caspi and Moffitt, 2006).

But another way to think of the relations between genetics and environment has come to the fore in recent years, with significant implications for thinking about urbanicity and mental health. The basic argument is far from new: from the 1960s onward, evolutionary and developmental biologists were highly critical of the 'genetic program' approach which assumed that inherited DNA simply expressed itself in the process of development of body and brain (Jablonka and Lamb, 2014). They argued that it painted an entirely misleading picture of the ways in which genes and environment interacted with one another from the moment of conception onward—environment here embracing everything from the cellular to the social milieu. And, as we moved into what some have called 'the postgenomic era,' it turned out they had the evidence on their side. Today, even the most committed proponents of psychiatric genetics recognize that, with the exception of a few very rare conditions, there are no 'genes for' mental disorder, and at the very least many genetic variations of small effect are combined in the development of mental disorders, even for those diagnosed as psychoses, let alone for the more common forms of distress such as depression and anxiety (Plomin and McGuffin, 2003). In addition, it has become clear that what is crucial is not merely the inherited DNA itself—the genome—but the ways in which particular genetic sequences are expressed or suppressed over the course of development, and the ways that this process is shaped by the constant transactions between the developing organism and its environment. It is this that has come to be termed epigenetics.

As usual, epigenetics is a contested concept (Carey, 2012; Landecker and Panofsky, 2013; Lappé and Landecker, 2015; Lock, Burke, et al., 2015). For present purposes, we will take epigenetics to mean the processes by which gene sequences are activated or deactivated across the life course, largely through mechanisms of methylation and demethylation, in certain cells in particular regions of the body, under environmental influences.[28] Some of the most widely referenced epigenetic research relating to brain development came from a Canadian group led by Michael Meaney: their experiments with rodents showed that the earliest relation that a mother has to her pup can shape the expression of genes in its brain and therefore shape the way in which that pup's brain develops into adulthood; this shapes the way in which that pup treats its own pups, which in turn shapes the development of the next generation of pup's brains and so on and so on, down the generations (Meaney and Stewart, 1979; Meaney, Aitken, et al., 1985; Champagne, Chretien, et al., 2004; Pruessner, Champagne, et al., 2004; Weaver, Cervoni, et al., 2004; Cameron, Champagne, et al., 2005; Champagne and Meaney, 2006; Szyf, Weaver, et al., 2007; Szyf, McGowan, et al., 2008). Meaney and his colleagues were not slow in arguing that these findings could be extended to humans, for example arguing that specific epigenetic patterns could be found in suicide victims with a history of child abuse (McGowan, Sasaki, et al., 2009; Meaney and Ferguson-Smith, 2010). This research led to much publicity. An article in *Nature* playing on a reversal of the normal cliché of nature versus nurture was entitled 'in their nurture' (Buchen, 2010). Other research, also carried out in rodents, seemed to show that factors such as diet and exercise led to epigenetic changes that might increase brain health, for instance generating epigenetic changes that increase the production of a protein called BDNF (brain-derived neurotrophic factor) which enhances the growth of nerve cells (Sleiman, Henry, et al., 2016). These and many other similar experimental findings, usually on animals such as mice, rats, and guinea pigs, led to a widespread belief that genes—in the sense of the inherited sequences of DNA bases—are *not* determinant—that the way in which they play out in any cell of the body is also a matter of milieu.

For many social scientists, epigenetics signals a radical revolution in genetic thought (Lock, 2013; Meloni, 2014; Lock, Burke, et al., 2015; Niewöhner and Lock, 2018). However, a number of cautions are in order. First, we need to remind ourselves that, as developmental biologists have long known, the contributions of extra-corporeal environmentally triggered epigenetic changes over the life course, while undoubtedly significant in the development of pathologies, are dwarfed by the role of inherited gene

sequences, with largely standard and predictable patterns of gene activation and de-activation, thus shaping the basic features of an organism in regular and predictable ways (Jacob and Monod, 1961).[29] This regularly ends up in the emergence of a stable and fully formed organism, despite the fact that, for example, no two lungs are identical, even for monozygotic twins. This is also true for brain development: a large proportion of coding sequences in the human genome are expressed in the brain, and there is certainly much similarity between individuals in the overall architecture of brains, but no two brains are identical, either in general structure or in patterns of connectivity and neuronal circuitry, even for 'identical' twins (Van Essen, 1997; Devlin and Poldrack, 2007). Epigenetic processes are responsible as much for broad uniformity across individuals as for variations between them.[30]

Second, and perhaps more important for our question, we must point out once again that most of the research that underpins these arguments has been carried out in laboratory experiments on small animals, bred specially for research over dozens of generations, and kept from birth (and before) in laboratory cages (Rader, 1998; Rader, 2004). Extrapolation from the brains of such animals to 'wild type' animals, let alone to wild type human brains, needs to be undertaken with great caution, if at all. As we have already pointed out, there are huge differences between rodent and human brains, and these differences are not just in scale, but in the fundamental organization and capacities of 'the brain.'[31] Even if one was to stay with the etiolated conceptions of the social environment common to much psychiatric research, most would find it difficult to draw a parallel between the feelings that a guinea pig pup might have to its con-specifics and evidence that an individual's trust in the social support offered by others in their community might mitigate the effects of stressors on mental health. The slide from evidence about the effects of environmental adversity in rodents to those in humans is highly problematic, but nonetheless routine (to take just one of numerous examples, see Yam, Naninck, et al., 2015).

Some researchers avoided the many problems of such extrapolations by looking directly at epigenetic changes in adult humans who have experienced various forms of urban stress. Thus, Sandro Galea and his colleagues carried out a number of studies on urban mental disorder, which sought to examine such epigenetic changes in adult humans who have experienced urban stressors. Galea's group examined the epigenetic profiles of residents of a Detroit neighborhood and found distinctive gene methylation profiles in residents who had been assaulted: they suggested that "cumulative traumatic burden may leave a molecular footprint in those with [PTSD]" (Galea,

Uddin, et al., 2011: 402), and more generally that "different aspects of the urban environment are distinctly and variably linked to brain structure, function, and hence phenotype" (Goldmann, Aiello, et al., 2011: 859). Further research from Galea's group linked long-term epigenetic changes to risk and resilience in trauma (Sipahi, Aiello, et al., 2013), argued that childhood maltreatment was associated with epigenetic differences in hypothalamic-pituitary-adrenal (HPA) axis genes (Bustamante, Aiello, et al., 2013), suggested that epigenetics may explain the prevalence of respiratory symptoms in those responding to disasters such as that at the World Trade Center (Gonzalez, Guffanti, et al., 2014), and proposed that psychosocial stress accelerates immunological aging, perhaps through epigenetic mechanisms (Aiello, Dowd, et al., 2016). Indeed, a decade ago, on the basis of a review of studies of schizophrenia, major depressive disorder, posttraumatic stress disorder, anorexia nervosa, and substance dependence, this group argued that "dysfunction of epigenetic mechanisms offers a plausible mechanism by which an adverse social environment gets 'into the mind' and results in poor mental health" (Toyokawa, Uddin, et al., 2012: 67).

There are, therefore, plausible mechanisms linking environmental exposures to neurobiological changes that can be taken seriously by thoughtful social scientists. Yet not all agree that social scientists committed to countering the effects of social inequity on mental health should concern themselves with a search for pathways and mechanisms. Thus, for more than a quarter of a century, Bruce Link and Jo Phelan have argued that we should not spend our time and energy trying to identify specific risk factors or untangling the minutiae of causal pathways, but we should continually stress the macro-social character of social inequality, notably socioeconomic status. We should focus on 'the causes of causes,' they argue, and recognize that socioeconomic status is a 'fundamental social cause' of mental ill health (Link and Phelan, 1995; Phelan and Link, 2010; Phelan and Link, 2013). Dismissing the view that such measures are 'mere proxies' for the true causes in the causal chain leading to disease, they argue that it is crucial to attend to the processes that lead people to be exposed to these more proximate risk factors, that put those of lower SES "at risk of risk" (Link and Phelan, 1995: 81).

Few would disagree with the general argument that poverty and lack of material and social resources negatively influence health—indeed, this is the basic premise of arguments about the social determinants of health, and of mental health (Allen, Balfour, et al., 2014; World Health Organization, 2014; Marmot, Allen, et al., 2020). But the links with socioeconomic status

are actually an artefact of the ways—actually the variety of ways—in which epidemiologists have created and used versions of this measure as a variable in their correlational approach to mapping diseases across populations. It is true that this way of highlighting the impact of social conditions on health sounds compelling in contrast to the individualistic focus on health behavior that has become so common in advanced liberal societies such as the United States. But it has many weaknesses—not least failing to recognize that, as Jonathan Wolff and Avner de-Shalit put it, the many determinants of well-being are "not all reducible to a common currency" (Wolff and de-Shalit, 2013). Moreover, while Link and Phelan's position may well be radical in the United States, the social determinants of health, and of mental health, are well accepted by researchers and policymakers in most other regions of the world. There is no doubt that increasing social equity would have significant beneficial consequences on the incidence of ill health. But, given the likely persistence of a heterogeneous array of inequities in the distribution of resources, powers and capabilities for the foreseeable future, the question of pathways and mechanisms remains highly important, conceptually, politically, and for specific policies such as those involved in planning and managing urban spaces.

In 2013, Bruce Link collaborated with Sandro Galea, in an article setting out six paths for the future of social epidemiology (Galea and Link, 2013). The two authors agreed on the weakness of approaches that limited themselves to factors and correlations and on the need to move beyond this correlational style of thought. Four of their six potential paths focus on the need for more research on the social determinants of population health. However, two of these paths move further in the direction that interests us here: first, the need for the development of theory to open the 'black box' of correlational thinking, and second, a focus on 'mechanisms.' Galea and Link argue that the emphasis on macro-social factors has now been widely accepted, but it has led to the dominance of "social epidemiologic work that constrains itself outside the skin, with an interest in social processes without much concern with why these processes may matter . . . attending to mechanisms can help epidemiologists sort out which of their plausible social determinants are the most compelling ones" (Galea and Link, 2013: 846). As we have seen, Galea's work, with its focus on epigenetic changes consequent on trauma, had already made contributions in this respect. In a later paper, Galea and Katherine Keyes argued that what was needed was attention to what they termed "causal architecture" which enables us to move away from the epidemiological focus on risk factors. Rather than focusing

on associations between factors, "a causal architecture approach . . . would ask: What is the structure of causes that underlie disease? Do these causes work together or separately? And most importantly, which causes are the most prevalent in the population" (Keyes and Galea, 2017: 4).

There are many methodological and conceptual difficulties to overcome in thinking through how one might establish such a causal architecture in any particular case. Nonetheless, the work of Galea's group on the epigenetic pathways that embody—or 'embrain'—the impact of urban exposures to trauma provides a compelling example of how we might begin to understand some of the biological pathways and mechanisms entailed in the experience of adversity. This might enable us to work out ways in which the everyday experience of social adversity and social suffering that is so well described in ethnographic research can engage with the new 'social' styles of thought just beginning to take shape in the contemporary neurobiology of mental disorder, in which the human brain in particular is 'plastic'—modulated by its dynamic transactions with its milieu, at timescales ranging from the millisecond to the decade, from conception onward.

Neuroplasticity: The Modulated Brain

We have known for many years that while the human brain develops significantly in utero, it develops further, and rapidly, in the first two years of life, reaching around 80 percent of its adult weight by the age of two. While there is little change in the volume of the brain after five years of age, at the gross level of neuroanatomy there are changes in the relative proportion of grey matter (which reduces after 12 years) and white matter (which increases over development up to puberty and indeed well into adulthood). There are also changes in the size of various brain regions, for example in the hippocampus and the amygdala, which appear to be sex related and may be hormonally controlled (Casey, Giedd, et al., 2000). But crucially, synapse formation—that is the formation of the webs of connections between neurons—is central in neurodevelopment and occurs well before birth and throughout early life. There is an overproduction of synapses during this period, many of which are 'pruned' in the first years of life, presumably in response to experience and in the process of learning. To put it simply, synaptic connections that are regularly used will strengthen—as argued long ago by Donald Hebb (Hebb, 1949) and summarized in the well-known aphorism 'what fires together, wires together.' On the other hand, those connections that are not used will be pruned. There is extensive synaptic plasticity—that is to say, the formation

and pruning of synaptic connections—in the period leading up to puberty, linked to experience, habit formation, and learning, and, around the time of puberty, there is another intense phase of synaptic re-organization (Blakemore and Choudhury, 2006).

It is thus well recognized that the formation and stabilization of synapses is experience dependent, and that in this sense there is synaptic 'plasticity' throughout the life course. However, for a long time it was thought that *structural* plasticity—the development of new neurons and their integration into functional circuits—was limited to early childhood and a few 'critical periods' in development, notably in 'adolescence.'[32] Only relatively recently has it been accepted that there is considerable structural plasticity in the human brain through adulthood. This first became evident in neurological studies of patients who had suffered stroke or other forms of brain damage, which showed that, on occasion, patients could recover some of the functions that had been lost, presumably because other areas of the brain somehow acquired the ability to undertake them. Hence, researchers began to suggest that, in certain circumstances, perhaps with specialized training and exercise, the brain was capable of 'rewiring' itself.[33]

There is, however, a significant distinction between such 'rewiring' and the development and incorporation of new neurons, that is to say neurogenesis. Until the 1980s, most leading authorities argued that neurogenesis occurred rarely if ever in the adult mammalian brain, even though it had been found in the brains of birds and a few other species (for a good review, see Fuchs and Flügge, 2014). There were good reasons for believing this, because it seemed very likely that the generation of new neurons would disrupt existing neural circuits, for example those that had developed as a result of learning (Rakic, 1985). However, Elizabeth Gould and her group, overcoming much scientific skepticism and considerable technical challenges, demonstrated clearly that such neurogenesis did occur in adults, working initially with rodents (Gould, Tanapat, et al., 1999) and then with primates (Gould and McEwen, 1993; Cameron and Gould, 1994; Cameron, McEwen, et al., 1995; Gould, Reeves, et al., 1999). Rodent experiments also showed that neurogenesis was enhanced by training and exercise and adversely affected by early adversity and by adult social isolation and stress (Mirescu, Peters, et al., 2004; Stranahan, Khalil, et al., 2006; Opendak, Briones, et al., 2016).

Gould and her colleagues were initially cautious in any extrapolation to humans. However, some human studies, for example of examination of postmortem brains, did suggest that neurogenesis occurred in some regions

of the adult human brains, and some suggested that measures of stress, as shown by levels of the glucocorticoid cortisol in human blood and saliva, provided indirect evidence to support the extrapolation of the rodent research on environmental stimulation of neurogenesis to humans (for a good review, see Chen, Nakagawa, et al., 2017). Many, however, remained skeptical of such extrapolation, given the differences in brain size and complexity in rodents that we have already discussed. In 2018, a study by a large international group of researchers concluded that "recruitment of young neurons to the primate hippocampus decreases rapidly during the first years of life, and that neurogenesis in the dentate gyrus does not continue, or is extremely rare, in adult humans" (Sorrells, Paredes, et al., 2018: 377). This critical conclusion provoked Gould, together with a number of researchers, to review the field and argue that, to the contrary, "there is currently no reason to abandon the idea that adult-generated neurons make important functional contributions to neural plasticity and cognition across the human lifespan" (Kempermann, Gage, et al., 2018: 25).[34]

Despite these unresolved controversies, a new popular image of 'the plastic brain' is taking shape, with authors enthusiastically claiming scientific support for their claims that 'the brain' is open to molding by its milieu from conception up until late adulthood, and that we can influence this by our own actions (Doidge, 2007; Begley, 2009; Arden, 2010). The suggestion that aspects of 'lifestyle' might increase neurogenesis has been enthusiastically taken up in the fantasy world of health on the internet. However, we need to exercise extreme caution. Aside from the problems of extrapolation, there is a strong temptation for individualization when the researchers seek to draw social and policy implications from this research. As with the use of rodent evidence in arguments about the developmental origins of health and disease, some researchers have found it a short leap from ambiguous experimental evidence produced in laboratory studies of the effects of various environmental manipulations on rodents to speculation that poor parenthood might inhibit neurogenesis, and hence lead to lifelong disadvantage (Leuner, Glasper, et al., 2010) leading to interventions that focus on those parents—usually the mothers—themselves, and sometimes even on young women prior to conception (Sharp, Lawlor, et al., 2018; Pentecost and Meloni, 2020). Perhaps the most we can say at this stage is that, while it is clear that neural circuits in the human brain transform in timescales from milliseconds to decades across the life course, and are thoroughly enmeshed in a milieu that is not bounded by skull or skin, we need collaborative research to explore the mechanisms and consequences for humans in the real world

of their lived experience, whether in cities or elsewhere. And, of course, if plasticity does occur in particular situations and is inhibited in other situations, we still need to identify the forces that place people, and hold people, in those plasticity-inducing or -inhibiting situations in the first place.

The Exposome: An Urban Sensorium

We may now smile knowingly at the miasmatic theories of disease that were prevalent for so long, with their explanations of ill health in terms of the potent mixture of noxious odors from corrupting matter and the foul exhalations of one's fellow citizens (Nash, 2006), exacerbated by defective morals and lax habits in the perilous but seductive lanes and alleys of the 'cities of dreadful delight' (Walkowitz, 2013). But perhaps we should pause before consigning everything involved in those past beliefs to ignorance and prejudice. Districts really do have their own atmospheres after all: on the one hand, a mixture of affects and emotions, of feelings of calmness or excitement, of melancholy or joy, holiness or eroticism (Anderson, 2009).[35] We are becoming increasingly aware of another component of atmospheric exposures—of noxious pollutants in the air, the water, the ground, the buildings and more. For our current purposes, we can think of an 'atmosphere' as the sensory and affective, bodily, cerebral, largely non-conscious milieu that suffuses those who inhabit a particular spatial position or trajectory. To understand such urban atmospheres, we need something like an ecology of the senses.

We can start from the classic arguments of Jakob von Uexküll (von Uexküll, [1934] 2010) that require us to recognize that humans, like other organisms, inhabit and create a specific *Umwelt*, a 'biosemiotic' world made up of the aspects and attributes that are meaningful for us humans with our specific senses of touch, taste, smell, hearing, and vision, attuned by evolution, development, and our own cultures to those dimensions that are salient for our own existence (Kull, 1999; Kull, 2003). When we recognize the characteristics of the *Umwelt* within each human organism exists, the distinction between the neurobiological and the cultural ceases to make sense, for together these create our own perceptual and sensory universe. The human *Umwelt*, the ways we attend to and make meaning of that which threatens us and that which we desire, the perceptual cues that enable us to locate ourselves, our own bodies, and to navigate ourselves through our own milieu, is not that of a tick, a fly, a mouse, or a dog. What, then, of the *Umwelts* of the cities that we humans have created for ourselves? What role do the different human senses—sight, touch, smell, hearing—play in

our urban *Umwelt*, in the spatializations of life in cities (Urry, 2003)?[36] The senses that orient people in space and in time, flowing through the 'portals' to the body and mind—the eyes, the ears, the nose, the skin—bring present and past inextricably together. Each hour, in each room, house, street, office building, shop, or factory, is not only visually specific but carries sounds, smells, the touch of other bodies or their absence—individually or together, these can evoke a whole scenario made real though myth and memory.

Vision and visibility has long been considered the primary sense, and this has certainly been the case for those concerned with urban governance. City planners have often sought to govern by vision, opening up dangerous agglomerations of persons, habitats, and habits to the purifying and civilizing influences of visibility. City spaces also brand themselves by their visual appearance, from the neon lights of city centers, to the little theaters of shop windows, or the enticing lights of cafés and bars. And the visual negotiation of the city is not just a matter of managing one's way across the physical topography of roads, pavements, parks, subways, but of negotiating the gaze of others, creating intimacy or rejecting it by meeting or avoiding eye contact, intensifying encounters with challenging gazes or de-escalating potential conflicts with downcast eyes. Vision, as John Urry remarks in his provocative essay (Urry, 2003), also marks class, not just through the familiar framing of wealth, work, gender, and age by dress, but by the form of the gaze itself. The wealthy watch the heaving masses on the streets from their cars; the genteel enclose themselves in newspapers, books, and reading devices on buses and the metro, the tourists gawk from their coaches and buses. Visitors from afar and travelers from nearby no longer struggle with paper maps furling and unfurling in the breeze, but walk with their gazes fixed on their devices, obeying the instructions as to when to turn or cross a road, inhabiting a space simultaneously physical and virtual.

Moreover, as has been remarked by authors from the earliest times, cities are noisy—the clatter of hooves, the shrieks of street vendors, the whoosh of cars or buses, the blare of radios from cars or of piped music from stores—so much so that the absence of noise, the preternaturally dark and silent street, summons anxiety in the stranger, but familiarity in the local. And in the cities of today, one is never far from the sounds of diggers, pile drivers, road drills, and all the other noises associated with the incessant repair and upgrading of urban material infrastructures. No wonder, then, that so many create their own soundscape, as we have moved from the speaker systems of the 1980s, through the Sony Walkmen of the 1990s, to today's ubiquitous mobile devices. Sound can locate an individual in space or seem to render

them placeless, to transport them from their particular patch of asphalt into a despatialized world that they carry in their pockets.

Touch, and the avoidance of touch, also requires mastery of movement in the crowded city, on the pavements, in the shops, in the transport systems. We have some important contributions to the cultural history of touch (Classen, 2005; Classen, 2012) and some discussion of the management of touch in specific professions such as nursing and teaching, but there is little research on the *management* of touch in the city, and in modern life more generally. Such management requires more than a mastery of movement, it requires a reorganization of intimacy—how, for instance, to negotiate the presence of other bodies on public transport, not all of them innocent,[37] or crammed shoulder to shoulder in a small, enclosed elevator with a dozen strangers (Goffman 1963; Finnegan 2005).

And what of smell? Despite it being long considered as the least important sense for humans, the nose plays a very significant role in the differentiations of urban niches (Classen, Howes, et al., 1994; Kiechle, 2017; Barwich, 2020).[38] Smell can evoke places, from a city as a whole to the scents of particular individuals—as the 'hero' of Patrick Süskind's *Perfume*, who has memorized all the smells of Paris, knew well (Süskind, 1986). And there are the characteristic smells of shops and stores—from the Fierce tang emanating from the stores of Abercrombie & Fitch to the sugary aroma characteristic of Lush—now increasingly managed as 'scent marketing' in the service of consumption (Gulas and Bloch, 1995; Krishna, 2011; Spence, 2015).[39] The city has long been associated with inescapable encounters with the smells of others with whom one is almost inevitably forced into contact—an olfactory reality that has long been the friend of racists and xenophobes (Rhys-Taylor, 2013). If modernity is characterized by a war on smell, the smell of human waste confined to the water closet, the smell of perspiration effaced by baths, showers, soap and unguents, the smell of rotting rubbish transported outside the city to spaces of invisibility inhabited only by scavengers, this is a war that, for many, including dwellers in inadequate and overcrowded apartment blocks, let alone in polluted and insanitary slums and favelas, is far from won.

Such atmospheres of lived experience are central to our own neurosocial approach to the vital city, and to its potential transformation in the name of mental health. And the biotic and sensory exposures that characterize each locale are now being captured in the emerging interest in the microbiome and the exposome. As researchers on the human microbiome project remind us, "the microbes that live inside and on us (the microbiota) outnumber our somatic and germ cells by an estimated 10-fold. The collective genomes of

our microbial symbionts (the microbiome) provide us with traits we have not had to evolve on our own . . . understanding the range of human genetic and physiologic diversity means that we must characterize our microbiome and the factors that influence the distribution and evolution of our microbial partners" (Turnbaugh, Ley, et al., 2007: 804). Researchers have argued that activities of these microbes shape development, modulate the capacities of the organism, and affect both health and disease, not least through the gut-brain axis (Human Microbiome Project Consortium, 2012; Mayer, Tillisch, et al., 2015), and some have suggested that the "emerging links between our gut microbiome and the central nervous system (CNS) [could be] regarded as a paradigm shift in neuroscience with possible implications for not only understanding the pathophysiology of stress-related psychiatric disorders, but also their treatment" (Kelly, Kennedy, et al., 2015: n.p.)—arguing indeed that stress can affect the permeability of the intestinal gut barrier, permitting "a microbiota driven proinflammatory state with implications for the brain," in particular in relation to depression and other 'stress related psychiatric disorders' (ibid.). However, once more, caution is needed: there has been much premature speculation about the significance of the human microbiome in maintaining physical and mental health (Valencia, Richard, et al., 2017), and there is "a relatively limited understanding of the broader environmental factors, particularly social conditions, that shape variation in human microbial communities" (Herd, Palloni, et al., 2018: 808). Significant methodological shortcomings in this research will need to be overcome if it is to fulfill its promise to be "a new frontier in understanding the biology of human health" (Renson, Herd, et al., 2020: 63). As Herd and her colleagues remark, "fulfilling the promise of microbiome research—particularly the microbiome's potential for modification—will require collaboration between biologists and social and population scientists" (Herd, Palloni, et al., 2018: 808) and such collaborations are currently in their infancy.

Despite these challenges, researchers on the microbiome agree that it is acutely sensitive to changes in the internal and external milieu, in other words to the exposome—a concept formulated "to draw attention to the critical need for more complete environmental exposure assessment in epidemiological studies [and to provide] a comprehensive description of lifelong exposure history" (Wild, 2012: 24). The exposome is "composed of every exposure to which an individual is subjected from conception to death" (ibid.), and research suggests that immersion in an environment, suffused with chemicals, gases, pollutants, pharmaceuticals, radiation, and much more, shapes an organism in ways as fundamental as those attributed

to the genome. We thus need to consider the ways that humans' capacities, and health and disease, are shaped and reshaped not just by "processes internal to the body such as metabolism, endogenous circulating hormones, body morphology, physical activity, gut microflora, inflammation, lipid peroxidation, oxidative stress and ageing," and not only "the extensive range of specific external exposures which include radiation, infectious agents, chemical contaminants and environmental pollutants, diet, lifestyle factors (e.g. tobacco, alcohol), occupation and medical interventions," but also by "wider social, economic and psychological influences on the individual, for example: social capital, education, financial status, psychological and mental stress, urban–rural environment and climate" (ibid.). Indeed exposures such as tobacco or environmental pollutants "have specific 'omics' profiles," in other words, they show distinct patterns of transcriptomics, epigenomics, and metabolomics (Wild, Scalbert, et al., 2013: 480). It is exceptionally difficult to measure the exposome, despite the potential combinations of instruments such as sensors linked to geographic information systems (DeBord, Carreón, et al., 2016), and major challenges remain in identifying and distinguishing the multiple vectors involved (Guloksuz, Rutten, et al., 2018). Nonetheless, perhaps miasmatic theory should not be wholly banished to a misguided past: these conceptions of the microbiome and the exposome reframe miasma for the twenty-first century.

Toward a Conception of the Neurosocial City

Many have criticized George Engel's 'biopsychosocial model' (Engel, 1977) on the grounds that, while virtuous in its aims, it is vague, all-encompassing, and can cover a multitude of different and incompatible approaches—so much so that at one and the same time some can claim that both clinical and experimental medicine has long been biopsychosocial, while others can claim that this has never been the case. Borrell-Carrió, Suchman, and Epstein, writing twenty-five years after Engel's initial proposal, try to clarify what this approach entails: "Philosophically, it is a way of understanding how suffering, disease, and illness are affected by multiple levels of organization, from the societal to the molecular. At the practical level, it is a way of understanding the patient's subjective experience as an essential contributor to accurate diagnosis, health outcomes, and humane care" (Borrell-Carrió, Suchman, et al., 2004: 576). But it is hard to see what might be specific to what they term "biopsychosocially oriented clinical practice" except an attention to the patient and their history. From another direction, we

are fully in agreement with Nancy Krieger's arguments for an 'ecosocial approach' stressing that health and ill health arise out of the 'embodiment' of a fractal web of transactions between biological and socio-environmental vectors (Krieger, 1994; Krieger, 2001). It is indeed important to understand "how we literally biologically embody exposures arising from our societal and ecological context, thereby producing population rates and distributions of health . . . socially patterned exposure-induced pathogenic pathways, mediated by physiology, behavior, and gene expression, that affect the development, growth, regulation, and death of our body's biological systems, organs, and cells, culminating in disease, disability, and death" (Krieger quoted in Palm, Schmitz, et al., 2013). These are, as Krieger rightly points out, 'theoretical' issues (Krieger, 2014). Yet it would not be too harsh a judgment to suggest that the theories and concepts that are required remain embryonic, and that the studies carried out within this framework are, in the main, descriptive rather than analytical. Hence, as we have suggested, conceptualization of mechanisms and pathways is crucial if we are to identify tractable points within this 'web of causality' where effective transformations should be directed.

We have used the term 'urban *brain*' in this book to stress that our approach is neurosocial, not 'psychosocial.' This is not because we wish to reduce or ignore the role of 'mind' or mental events in these pathways, but because we want to suggest that many, perhaps most, of the processes that are at stake here, even though they are shaped by experience and in turn shape that experience, operate below the level of consciousness that is often implied in references to the mind. In this chapter, we have tried to specify some of the pathways now emerging within contemporary neuroscience: new modes of visualization, new understandings of stress, recent work on the epigenetic modification of gene expression resulting from environmental exposures, findings on the social modulation of neuroplasticity, the role of the 'atmosphere' of the urban or the urban 'sensorium,' and evidence about the biosociality of exposures. We have done so at some length as an attempt to unfold some new ways of thinking that might underpin a new 'neurosocial' conceptualization of the 'urban brain' and, indeed, underpin a new urban biopolitics that addresses the ways that these are lived together in the experience of a form of life actively lived across space and time. Can we give this argument an empirical specificity that would enable analysis and action? It is to this task that we now turn.