Abstract

**Background:** The environment is inextricably related to mental health. Recent research replicates findings of a significant, linear correlation between a childhood exposure to the urban environment and psychosis. Related studies also correlate the urban environment and aberrant brain morphologies. These findings challenge common beliefs that the mind and brain remain neutral in the face of worldly experience.

**Aim:** There is a signature within these neurological findings that suggests that specific features of design cause and trigger mental illness. The objective in this article is to work backward from the molecular dynamics to identify features of the designed environment that may either trigger mental illness or protect against it.

**Method:** This review analyzes the discrete functions putatively assigned to the affected brain areas and a neurotransmitter called dopamine, which is the primary target of most antipsychotic medications. The intention is to establish what the correlations mean in functional terms, and more specifically, how this relates to the phenomenology of urban experience. In doing so, environmental mental illness risk factors are identified.

**Conclusions:** Having established these relationships, the review makes practical recommendations for those in public health who wish to use the environment itself as a tool to improve the mental health of a community through design.
Keywords: psychosis, urban environment, dopamine, amygdala, caudal cingulate, neuroscience, risk factors, public health, behavioral health, psychiatric conditions, architecture, epidemiology, nature, health care

Key Concepts

The environment is a causal factor in mental illness, especially the designed environment, which is particularly rich with meaning and perceptual demands. Because the designed environment is ubiquitous in urban contexts, the impact of the urban milieu on an individual’s psyche is both pervasive and intense. This interactivity potentially explains the linear and significant correlation between childhood exposure to the urban environment and psychosis (Golembiewski, 2013a; Kelly et al., 2010; Lederbogen et al., 2011; Lederbogen, Haddad, & Meyer-Lindenberg, 2013; Peen, Schoevers, Beekman, & Dekker, 2010; van Os, Kenis, & Rutten, 2010; Vassos, Pedersen, Murray, Collier, & Lewis, 2012)—observations that have been identified repeatedly since “lunacy” was first mapped by Faris and Dunham (1939). Similar correlations have also been identified between urban upbringing and aberrant brain morphology (Haddad et al., 2015).

An analysis of this morphology against the putative neural functions of these areas gives a glimpse both into how design may become toxic and into which symptoms design decisions may potentially trigger. More importantly still, this analysis provides a schematic guideline for design praxis to help manage psychiatric illness on a noninvasive but population-wide level.

Organization

This article looks at current neuroscience as a way to understand the effects that the built environment may have on health—mental health and its symptoms especially. The article starts by discussing the origin of the considerable resistance this approach is likely to face, largely because it defies common practice and the canon of 18th and 20th century logic. The review then looks at the implications of recent discoveries in psychology and neuroscience and how these implicate the built environment—potentially as a causal factor in mental and somatic illness. The following section looks more deeply into the neuroscience to establish a cogent etiological mechanism, and the two sections beyond that identify and discuss how the environment can be used to improve health—mental health especially. The first of these in abstract and technical terms; the latter in the practical ways that may be useful for designers. The review finishes with a frank discussion of the limitations of current research and the reasons why an environmental approach should be adopted regardless.
A Background of Scientific Skepticism

In the early 17th century, Descartes proposed that “there’s nothing corporeal that belongs to our essence… Man is just the mind, and the body is no more than a vehicle for the mind” (Descartes, 1641[tr. 1986]). This concept was propelled by a booming interest in science and soon became doctrine. Accordingly, concepts that entertain a relationship between the physical world and mental health are still largely written off as spurious before they are ever properly investigated, except where evidence is taken to support truisms that hold to an even older but contradictory ancient doctrine, which somehow has always remained acceptable (that a healthy body is key to a sound mind); “mens sana in corpore sano” (Juvenal, ca. 200 AD).

Perhaps the greatest challenge to dualism was probably the famous (and possibly exaggerated) change in the personality of a construction worker following his remarkable recovery after a 900 mm long, 25 mm diameter, sharpened iron rod passed through his frontal cortex, effectively lobotomizing him (Damasio, 1994; Harlow, 1848). Since then, further developments in neurosurgery and psychopharmacology have brought further weight to the concept that mind may actually be a product of neural process (Andreasen, 1992; Grossberg, 2003; Hohwy & Frith, 2003; Taylor, 2007, etc.). And while scientists argue about the fundamentals, the unhinging of the central tenant of dualism means that mental processes need not be regarded as independent of the physical environment. Gibson (1979), for example, found that the “cognitive” process of observation is inextricable from the world which is observed and that visual perception and action are ecologically related. In other words, the choices we make, our behavior, and the things we notice—in loose terms “the mind” is a product of not only the brain but of the physical environment beyond the body also.

Gibson’s theory of ecological perception explains why the mind reacts to visceral stimuli: when it comes to experience and perception, the brain does not process information, but it engages in action. The difference is simple: “processing” models tend to conceive the brain as an impervious machine that impartially processes experience for the mind to consider. The ecological model acknowledges that experience is an action to which the mind reacts. At a neural level, this is palpably evident—neurons physically grow through 2 Health Environments Research & Design Journal the coincident activation of neurons, and the opposite—neurons atrophy through lack of perceptual engagement (Hebb, 1949).

Science thus has no problem with accepting that experience affects the brain—it is the kind of basic neuroscience that has been taught to undergrads for generations (Johnson Abercrombie, 1960) but acknowledging that these effects are continuous and occur outside the lab is apparently still a stretch. Dualist logic still has cache even today, because the implications of an ecological model are both contrary to the canon of cognitive science and because its implications are apparently impossibly enormous (Ullman, 1980).
All we experience affects the brain, and through the brain, the mind. Indeed, scientists have found that certain familiar stimuli will activate learned and instinctive processes prior to any cognitive process (Bargh & Dijksterhuis, 2001; Damen, van Baaren, & Dijksterhuis, 2014). If the details are set aside, the neurological impacts of perception cannot be denied; the early blind (Liu et al., 2007) and early deaf (Bavelier et al., 2000) have pronounced and distinctly different brain morphologies from the nondisabled.

**Perception, the Brain and the Designed Environment**

If the brain and mind are indeed permanently engaged in, and are being continually changed by external experience (at least whenever we are awake), then this puts designers collectively at the helm of a major force of neural functional dynamics. The fact that designers are generally unaware of the effects that their inventions have on the brain and therefore take no responsibility of this role is deeply worrying. The world of design is like a highway, where each and every driver is asleep at the wheel.

Fortunately, humans do not behave like frogs and insects, with little choice but to snap at bait dangled before them. Humans have developed neural mechanisms to prevent us from reacting uncontrollably to whatever experience is presented to them: these are the most distinctively humanoid parts of the brain—the great swelling of the frontal cerebral cortex. But when this area is damaged, people lose the ability to resist environmental cues. Among other behavioral changes after a lobotomy (See Damasio, 1994), the presence of a gun will trigger an irresistible desire to fire; food, an impulse to eat; a shop, an impulse to buy, and so on. These impulses are known as environmental-dependency syndrome and imitation and utilization behaviors (Lhermitte, 1983, 1986; Lhermitte, Pillon, & Serdaru, 1986).

These bizarre responses are reactions to the behavioral triggers that are purposefully designed. The human-made world is designed for human use and is replete with representations and meanings that are lost on any other species: symbols, words, icons, and implied uses and functions. These are easily identified by humans and are intrinsically connected to human psychic activity (Csikszentmihalyi & Halton, 1981).

Nowhere are the designed meanings, triggers, symbols, and so on more prevalent than in the urban environment. In the urban environment, just about everything is designed, manipulated by design, or caused by accidents and misalignments of design—even the weather is affected in local microclimates, by phenomena like heat islands and wind turbulence (Arnfield, 2003). In the city, even the detritus that flies around the streets is designed—bits of packaging, reading matter, food, cigarette butts, and the litter from landscape-designed street planting and gardens; nearly every crumb has had a human hand with some kind of design intention at some point and nearly every crumb is loaded with identifiable meaning.
Given this context, it should come as no surprise that people who grow up in the city have brain morphologies and reactivity patterns that are distinct from those who grow up in the countryside.

Specifically, there is a remarkably linear correlation between the “dose” of urban environment and stress-related activations of the amygdalae and the anterior cingulate cortex (Lederbogen et al., 2011), and there are also significant inverse correlations in the gray matter volumes of the right dorsolateral prefrontal cortex and anterior cingulate cortex—at least in males (Haddad et al., 2015). In itself, this is as predicted, and should not be a concern, except that the specific morphologies in question are closely linked to mental illness—depression, post-traumatic stress disorder (PTSD), and schizophrenia in particular (Boehringer et al., 2015). Furthermore, it appears that the same regions are those that are associated with surplus reactivity in highly symptomatic psychiatric conditions: the anterior cingulate cortex and the lateral prefrontal cortices (Golembiewski, 2012).

Unlike somatic conditions, mental illnesses including schizophrenia, bipolar disorder, PTSD, and many others are diagnosed exclusively by symptomatology. This means that at a very minimum the environment has a causal effect on psychiatric illness to the extent that the environment triggers symptoms. The degree to which this is so cannot be easily quantified because both the environment and the mind are extremely complex and heterogeneous systems, but even so we can predict a corresponding increase in psychiatric disorders in the urban environment. In fact, some prominent epidemiologists claim there is no more reliable or replicable epidemiological data—at least for schizophrenia (Kelly et al., 2010; van Os, Rutten, & Poulton, 2008) and PTSD (Liebschutz et al., 2007). The only data that have been presented as potentially more significant are easily confounded by the features and location of the built environment such as childhood exposure to sunlight and social dysfunction and may have thus been poorly identified (Golembiewski, 2013a).

**Actions and Reactions: The Brain and Exposure to Design**

The physical and designed milieu is almost certainly causally linked to a broad spectrum of psychiatric illnesses. So why don’t all urbanites suffer from mental disorders? The answer brings us back to the protective natural defenses that we have evolved. And again, the foremost of these is the inhibitory function of the prefrontal cortex.

Northoff et al. (2004) discovered that people with severe mental illness are highly reactive to the environment, even if they are arrested by a state of catatonia. Further analysis of Northoff et al.’s data by Golembiewski (2012) suggested that the symptoms of psychiatric illness appear to have caused a “spillover” of excess neural excitation in response to negative stimuli whenever the frontal inhibitory process is insufficient. Just like in physics, every neural action has an equal and opposite reaction. Whenever the inhibitory reaction of the prefrontal cortex is less than the event potential of the initial excitatory impulse, there is a surplus of neural excitation. This surplus excitatory potential is not lost. Like excess forces cause movement in physics, excess excitatory potential feeds into other parts of the
brain to emerge as uninhibited actions and thoughts: to the motor cortex to emerge as stereotypy and other automatic actions and also into the orbitofrontal cortex, where the surplus is experienced as invasive thoughts. The psychiatric cohorts that were measured in this study showed extremely high surplus reactivity to negative perceptions (a volume of 1,212–1,385 voxels of excited gray matter vs. 21 in healthy comparators; i.e., approximately 66 times higher).

Interestingly the nonpsychiatric controls were far more reactive to the positive emotional stimuli (the psychiatric cohorts demonstrated 470–598 vs. 881 surplus voxels for healthy comparators; i.e., nearly half). So when stimuli is positive, a surplus is healthy, and it is more likely to be laughter or other signs of a healthy disposition, the very same behaviors that are notably reduced in psychiatric conditions (American Psychiatric Association, 2000).

This brings us to the other discovery of Lederbogen et al. (2011) that stress-related activations of the amygdalae are stronger among urban dwellers. The function of the amygdalae is apparently to monitor personal involvement (ipseity as it is called) in any emerging narratives that can be “read” from the fabric of existence. The amygdala reacts according to how much an emerging story of ordinary events involves “me” (Le Hunte & Golembiewski, 2014). It appears that Lederbogen et al. have identified a neural signature of urban existence—that urbanicity is a concentration of meanings for humanity; a concentration of human design for human needs, in a way that the countryside and jungles just are not.

In the narratives surrounding urban existence, it is entirely expected that there will be a greater propensity to identify stories that work against us. It is much easier to speculate about malevolent intentions behind a closed-circuit television camera that is trained on the street outside your house 4 Health Environments Research & Design Journal than it is to identify malevolence in a spinney of trees. Of course, a rural or natural environment does not preclude the assertion of malevolent intentions, there can be real fears that the spinney might be the home of a witches coven or a werewolf.

The issue here is that urban amygdalae are likely to be more sensitive to the designed world, because design is deliberately intended to trigger human responses, whereas the reactions that people have toward nature may have evolved through the millennia but are not intentionally designed (at least by humans!). The demands required by the designed environment are diverse, complex, and very specific—a product on the counter of a store is to be bought, a seat is to sit, cash is to spend, moving cars are to be avoided, and new ones in a dealer’s lot are to be desired. The demands that the natural environment make on humans tend to relate to play, nurture, and discovery—a tree is to admire, feed us, or climb on, and a mountain is to explore (Golembiewski, 2013b).
Environmental Antipsychotics

The designed world is not only a set of perceptual demands waiting to trigger psychotic and other symptoms of mental illness. It can also protect the human psyche. The attention we provide to familiar, predictable, and nonaversive surroundings is very minimal, meaning that as long as we are in a place with such qualities, we have the opportunity to “let-go,” or as scientists say, attenuate to statistical regularities based on prior experience of the environment (Horga, Schatz, Abi-Dargham, & Peterson, 2014). This is a significant protective neurological and environmental interaction because the failure to attenuate to the environment— or worse, perceptual sensitization is widely thought to be causal in psychotic illness (Fletcher & Frith, 2009; Horga et al., 2014; Kapur, 2003).

There is a distinct environmental condition that is needed to enable the attenuation process, and this has a very particular chemistry. First and foremost, environments must be readable as nonaversive.

Theoretically, as long as environments present potential dangers or other negative features, we cannot attenuate because our “fight or flight” reflexes are attuned. This is undoubtedly due to a function of the limbic region: together the amygdalae and hippocampi recognize narratives and make a determination about potential threats. If dangers are present, instinctive and well-learned patterns of protective behavior take precedence. This enables latent attention and intelligence (learned reactions) and very fast actions, which provide a clear evolutionary advantage, potentially even before a genuine threat is even revealed (Le Hunte & Golembiewski, 2014). This automatic intelligence allows us to use the environment to our advantage: Objects can instantly become weapons, niches can become shelter, paths become escape routes, and a person will emit warning signals (screams) to others quite involuntarily; these actions all take place without the need for time-consuming cognitive processes (Ashby, Turner, & Horvitz, 2010).

As useful as it is, automatic reactivity also features in psychiatric symptoms as perseveration, spontaneous screaming, lashing out, and running (symptoms known as “disorganized behavior”). Automaticity is also possibly a feature of more mysterious symptoms like delusions and hallucinations. This is because automatic actions bypass a sense of self-agency and are rarely properly remembered—declarative attention being an inhibitory response to a percept, whereas latent, automatic actions are excitatory. Thus, too much attention will pause or arrest automatic actions whilst the percept is attended to. This enables memory, but disables action, which if sufficiently uninhibited, may even go by “unexperienced,” as episodic amnesia, or if over inhibited, will cause performance choking (Baumeister & Showers, 1986). Hypothetically, this amnesiac effect can explain hallucinations, because events seem to happen without self-agency; or as delusions if events are spuriously post-rationalized (Golembiewski, 2014). Examples of delusions that are triggered by an unawareness of one’s actions are Cotard delusions (where people become convinced that they are dead); or paranoid delusions when people think they are being controlled by others using remote electronic technology (Kean, 2009).
This switching between fully aware, deliberate behavior and reactive behavior is putatively a function of the dopamine neurons in the mesocortical pathway, a neural network associated with reward processing (Wise & Rompré, 1989) and a range of other attentional processes, the details of which depend on the various dopamine receptor types and their location (Bromberg-Martin, Matsumoto, & Hikosaka, 2010). A current hypothesis proposes that declarative attention (action associated with highly aware thought processes) is a product of occasional postsynaptic bursts (called phasis), and latent attention (cognitive processes that take place automatically and “unthinkingly,” such as typing or driving) is speculated to be the phenomenal product of more or less continuous, rhythmic dopaminergic tone, mostly in the nucleus accumbens and other mesolimbic areas (Golembiewski, 2013c; see Figure 1).

![Figure 1](image_url)

**Figure 1.** This is a schematic model of the various dopamine frequencies, which putatively moderate both latent and declarative attention: The continual tone moderates latent attention while phasis-spikes moderate declarative attention. Image © the Author, with permission.

Phasic actions (the ones that are thought to moderate “declarative” attention; Bromberg-Martin et al., 2010) consume about 10 times more dopamine than actions of the tonic “latent” condition (Seeman, 2008), meaning that declarative attention should keep functional dopamine supply relatively low. The way phasis reduces available dopamine is important because dopamine is the primary target of most antipsychotic agents (Ginovart & Kapur, 2010), and a normal supply is considered to be essential for mental health. For this reason, environmental conditions that enable declarative attention potentially exert a powerful antipsychotic effect. This effect is largely conditional on the affective response to circumstances— because the high-consumption mode will be inhibited (in most cases) by adverse circumstances, unless they are genuinely novel and unexpected (Bromberg-Martin et al., 2010).

In a demanding environment, such as an operating theater, a great deal of this declarative attention will be needed to process statistical irregularities. At home or another familiar place, whenever the environment is positive enough to really relax, the same attention can be deliberately focused elsewhere; on creative or thoughtful tasks, as a person desires. In either case, the receptors can become engaged in the high-consumption “declarative” mode. In these conditions, general dopamine supply is predicted to be consumed— an absolutely safe antipsychotic effect.
If this “downtime” is spent (as it often is) engaged in surprising narratives and other aesthetic engagements through writing, art practice, music, theater, and film, there potentially may be an additional benefit—an opportunity to refresh the foci of the amygdalae and hippocampi. This is predicted to have a wide range of additional benefits, including improving the plasticity of the regions that process narratives—it potentially may even “flush out” the monothematic narratives that partly define psychotic delusional thinking (Gallagher, 2007). Although this area of environmental psychology is novel and needs a lot more exploration, there is already considerable evidence that the effect is as predicted: narrative-enriched downtime considerably improves both mental and somatic health outcomes (Pennebaker, 2000).

Positive experience may also directly addresses the negative signs of psychosis such as emotional flatness (American Psychiatric Association, 2013), even if the results are only marginal, they are still predicted to be worthwhile (remember that psychiatric patients are about half as reactive to positive experience but are reactive nonetheless; Golembiewski, 2012). This particular set of symptoms is otherwise notoriously difficult to target using traditional antipsychotic medication (Buckley & Stahl, 2007).

The perceptual opportunities offered by unstimulating environments and routines aren’t likely to be problematic in themselves, provided that they are sufficiently interspersed with others that will trigger declarative thought— perhaps this quality is the “delight” (venustatis) that was famously prescribed for architecture by Vitruvius (ca. 15 BC: Bk. 1, Ch. 3, St. 2) at a time when concepts about a healthy body and healthy mind were still fresh (Juvenal, ca. 200 AD) and hadn’t yet been reduced to a platitude.

If there are insufficient phasic opportunities to consume surplus dopamine, it may be consumed by increasing the number of automatic, practiced tasks and thought activities. This will be particularly true if natural metabolizing agents have been inhibited by environmental agents like cigarette smoke (Fowler et al., 1998) or particular genetic factors like the methionine allele of the COMT gene (Bilder, Volavka, Lachman, & Grace, 2004). Whatever the cause, a surplus is predicted to increase mental strain and troublesome symptoms associated with automaticity whenever there is a risk of mental illness.

**Conclusions and Limitations**

This article draws together theory and empirical data, much of which was generated in labs under microscopes or simply through literature review. The product is a review of an immensely complex area of knowledge, but one with enormous scope.

Currently mental illness represents a major financial and social burden the world over, and it is very difficult to treat and there are no established nonsocial interventions to curb the epidemic. While this
theory will prove difficult and costly to verify emphatically, the benefits of investing in design-based interventions in high-risk environments such as health facilities, community housing, and the prison system can only be of benefit to all stakeholders involved.

If this theory were to be more prescriptive, if there were a risk of side effects or were it to recommend a reversal of humanist approaches to design, then rigorous empirical testing would be critical before these principles were employed; but when the risks are low and the potential benefits are high, sometimes critical medical interventions must employ the theory now and test it later, just as many countries have done with sudden infant death syndrome (Mitchell, Hutchison, & Stewart, 2007). This is also such a case where there is no risk in embracing the recommendations and findings contained within this review. The review supports the principles of current best practice and encourages designers to extend these practices to make wholesome and high-quality environments, so the risk of implementing the concepts herein are low.

**Implications for Practice (Designing Protective Factors)**

We must design for genuine respite if we are to achieve an environmental antipsychotic effect through design. We also have to acknowledge that the subjects of the design are going to be supersensitive to negative environmental features (indeed, this is a feature of paranoia—perhaps the most typical symptom of psychosis and also very common in mood disorders; Freeman & Freeman, 2008). Designers are well practiced at creating “nice” and “homely” environments, but in this case, because patients suffer from supersensitivity to the environment, designers should make extra allowances for this. There is no limit for the creative possibilities this theory presents, but designers can consider:

- Taking steps to remove environmental features that might trigger someone with paranoia.

- Taking steps to make places better than just nice or homely; designers might consider much higher quality finishes, views, furniture, and materials than they otherwise would in a domestic milieu.

- Designers and their clients should also discuss how the environment can protect vulnerable people from undesirable environmental effects including overexposure to other people, pollution, and cigarette smoke.

- A range of modalities. Think about visual impressions, a soundscape, the olfactory sense, about textures and comfort. Good wayfinding, access, and egress. People with paranoia will not want to feel trapped.

- Because an important behavioral gateway appears to be perceptions about whether a place is safe or potentially dangerous, based on how the amygdalae/hippocampi deal with prevalent narrative information, these too can be designed: design a compelling and positive story. The intention should be
to use the language of design to suggest that the subject is safe and comfortable—not just on this room, but outside and elsewhere also. Narratives can be designed using thematic expressions, much like a stage set.

Surplus subcortical dopamine does not have to be consumed on unwanted automatic tasks. Positive automatic reactions also consume dopamine. Positive and rewarding activity is therefore also a useful tool—wholesome things to do and play with. But remember that gaming, TV, and other mindless tasks, even if notionally positive, may not be sufficiently engaging and may leave surplus presynaptic dopamine to be consumed in symptomatic ways.

It is not sufficient for the environment to just offer respite, because the predicted full antipsychotic effect may only be achieved with conscious engagement. So design in a choice of things to think about: learning, writing, making art and music, gardening, cooking, animal husbandry, performance, religion, hobbies, reading, and strategic sports.

A further benefit of designing opportunities for engagement in wholesome ideas and activities is that such interests are infectious (so there is a greater social benefit) and they are ongoing—vulnerable people can take these ideas and interests with them wherever they go, and these skills will prove protective against unwanted automaticity, paranoia, and other symptoms. This is the true meaning of recovery-centric design.

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Note

1. Voxels are a volumetric measure of activated neurons.
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