The four dimensions: a model for the social aetiology of psychosis

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Summary
Recently, there has been increasing focus on prevention of mental illness, early intervention and the promotion of mental health. The social determinants of health and public health approaches are considered key. Early intervention has focused on psychotic disorders but prevention has not. This may in part reflect the fact that public health planners do not have a clear model for how social determinants influence the risk of developing a psychotic illness. Drawing on biological, genetic and epidemiologic evidence regarding the relationship between social risk factors and psychosis, this paper outlines a conceptual framework for understanding how individual and ecological factors contribute and interact to modulate the risk of developing psychotic illness. The framework asserts that there are four dimensions: individual factors; ecological factors; the interaction between individual and ecological factors; and time. It may help those considering interventions to understand the multilevel and multifactorial effects of social factors on the aetiology of psychotic illness, to develop targeted strategies for the prevention of psychotic illness and serve as a template for the assessment of initiatives.

Declaration of interest
None.

Social factors and psychotic illness

There are a variety of explanatory models for the impact of social factors on psychotic illness. Social factors may change the exposure to or action of biological risk factors or they may have a direct toxic effect on individuals and their families. Genetic and epigenetic processes interacting with social factors at the individual, group and societal levels over a life course produce a complex and interconnected web of causation.

Understanding how different social factors change the risk of psychosis has been problematic, in part because of the numerous possible mechanisms and multiple levels through which this could occur. For instance, there is a reported association between prenatal malnutrition and an increased risk of schizophrenia in adolescent life. This could reflect the impact of nutritional stress on the hypothalamic–pituitary–adrenal (HPA) axis or direct effects of the low levels of nutrients on deoxyribonucleic acid stability or expression (to name just two theories). However, at a different level, urban or rural residence during famine may change the risk of exposure by altering access to food to supplement the diet.

The complex nature of this enquiry is demonstrated here by consideration of one of the explanatory models for the impact of social factors on psychotic illness: the ‘vulnerability–stress model’. This model posits that genetic or developmental vulnerabilities interact with social adversity to culminate in a common pathway leading to stress.

Genetic, biochemical and neurological evidence supports the link between stress and psychosis. The physiological effects of stress are demonstrated by findings of activation of the HPA axis to produce excess cortisol and adrenocorticotrophic hormone. In addition to genetic factors, acquired vulnerabilities such as prenatal exposures, perinatal complications, drug misuse and childhood abuse or neglect may regulate the response to stress. Links between the physiology of stress and that of psychosis have been reported. The hippocampus, known to play a key role in dampening HPA activity, is reduced in volume in psychotic disorders. Stress also leads to mesolimbic release...
of, dysregulated mesocortical response to and overall sensitisation
to dopamine, a neurotransmitter that may mediate psychotic
symptoms.18–20

Experimental support for this is found, for example, in studies
in which psychotic symptoms are associated with increased
dopamine release after amphetamine challenge.21 Over time, in
individuals who are at risk, exposure to moderate levels of stress
can lead to an excessive and persistent dopamine response.
Building on this theory, those with higher rates of life stressors
may be at particular risk of developing a psychotic illness.

Epidemiological studies support this at multiple levels. Cumulative
exposure to traumatic life events, or the number of
life events experienced, is associated with an increased risk for
psychosis22–25 and the British National Psychiatric Morbidity
Survey has reported that adverse life events are associated with
subsequent psychotic experiences in the general population.26,27
In addition to traumatic major life events, the accumulation of
minor events or ‘daily hassles’ have also been linked to psychotic
illness.28

However, it is not just the accumulation of life events that is
important. As stated previously, a life event may trigger a train
of other events. For instance, loss of a parent as a child (which
has been associated with increased risk of later psychosis) could
increase the chance of living in poverty and thus exposure to other
environmental risk factors associated with psychosis.

At the level of gene–environment interaction, minor life events
and daily hassles interact with polymorphisms known to be
involved in dopamine neurotransmission to cause differential
stress reactivity and psychotic experiences.29 Of note, the same
polymorphism has been shown to correlate with differential risk
for psychosis in individuals who use cannabis,30 further
highlighting the complex interplay between multiple biological
and environmental risk factors. And genetic variants and
environmental exposures can interact in ways that are protective
and not just harmful. Instead of focusing on ‘vulnerability genes’
that confer increased risk in the presence of certain environments,
Belsky et al call for an appreciation of ‘plasticity genes’ that confer
a nuanced differential susceptibility: increased risk in some
environments, and decreased risk in others.31

Physiologically and cognitively, exposure to early developmental
stressors (such as childhood trauma) may act by sensitising people
to later adverse events, major or minor. They may increase the
likelihood of adverse events for instance by shaping the capacity
of individuals to form relationships.7,32 Alternately, they may
interface with a person’s attributional style – potentially making
them more prone to psychotic thinking.33

Moving from the individual to the ecological, a major branch
of investigation into the social aetiology of psychotic illness began
in 1939 with Faris & Dunham’s work on the urban environment.34
Since then, research has focused on better defining its role as a risk
factor for psychosis. Studies have demonstrated that the higher
risk of psychotic illness for those living in cities is not the result
of social drift but is associated with being born and growing up
in an urban environment in childhood. Moreover, the bigger the
city the bigger the risk.35–38

Living in an urban environment must, however, be a marker
for a number of exposures. Allardyce et al found that after
adjusting for social fragmentation and deprivation, there was only
a non-significant trend towards an association between urbanicity
and schizophrenia.36

Other factors may be important for the association between
place and psychosis. For instance, Silver et al documented that
the proportion of people moving in and out of an area was
associated with higher rates of schizophrenia.39 Another pilot
study has reported that increased social cohesion and social
efficacy in areas in London, UK were associated with a reduced
incidence of psychosis,40 and after controlling for individual
deprivation, Boydell et al have shown an increased rate of
schizophrenia with increasing neighbourhood inequality, but only
in more deprived areas.41

Kirkbride et al recently used multilevel Poisson regression
simultaneously to model individual- and neighbourhood-level
factors.42 They reported that 23% of the incidence of
schizophrenia could be attributed to neighbourhood-level social
risk factors including socioeconomic deprivation, voter turnout
(proxy for social capital), ethnic fragmentation (proxy for
segregation) and ethnic density (95% CI 9.9–42.2). A 1% increase
in voter turnout (incidence rate ratio (IRR) = 0.95, 95% CI 0.92–
0.99) and ethnic segregation (IRR = 0.95, 95% CI 0.92–0.99) were
both independently associated with a reduced incidence of 5%
indepedent of age, gender, ethnicity, deprivation and population
density.42

However, there are a number of different mechanisms through
which ecological factors could have their impact on psychosis.
Stress may be just one of a number of mechanisms.5

Taken as a whole, the literature demonstrates that social
factors are associated with psychosis risk at an individual and
an ecological level. There is evidence of an increased risk with
greater exposure to some individual risk factors and with a greater
number of risk factors. There are multiple mechanisms and an
interplay between genetic, epigenetic and environmental factors.

Conceptual framework

A framework for categorising or organising different associations
between psychotic illnesses and risk factors may be helpful to
those who are considering developing prevention/promotion
initiatives. It may also further research in the field. Although we
have highlighted the stress hypothesis, this umbrella conceptual
framework we present would allow the inclusion of a variety of
perspectives on causation and different mechanisms. A simplified
conceptual model would follow what we know from the literature
and posit that there are four dimensions of interest:

(1) exposure to individual-level social factors linked to psychosis;
(2) exposure to ecological-level social factors linked to psychosis;
(3) the interaction between individual and ecological social
factors; and
(4) time.

Exposure to individual-level social factors linked
to psychosis

At the individual level, the model would be similar to heart
disease: there is an inherited risk, but whether one develops a heart
attack or not depends on other risk, protective and health
promotion factors that one encounters. Accordingly, the risk of
developing a psychosis for any individual would depend in part
on inherited vulnerabilities, but also would rest on the balance
between exposure to factors that either increase or decrease risk
or enhance mental health. In addition to genetic risk and
biological risk factors, a list of social risk factors could include
the use of certain drugs (especially cannabis), racial
discrimination and factors in development such as bullying and
psychological trauma in childhood, separation from parents and
other childhood adversity.31 The amount of exposure or number
of factors to which a person is exposed would need to be sufficient
for a psychotic illness to develop. Previous work has shown that
the greater the number of risk factors the higher the risk of psychosis.32-37

**Exposure to ecological-level social factors linked to psychosis**

At the ecological level, social factors change the risk on a population basis or change environmental risk. The model would be similar to that of diabetes, where a change in the availability and calorific content of certain types of food and cultural changes in exercise have led to increased rates of the illness. Groups with similar individual-level risk factors may have different rates of illness dependent on the environment. Environmental risk factors have been less well studied but they would include city birth and city living, famine, social cohesion, social fragmentation, minority groups living in areas with low-population densities of their group, and migrants from countries that are predominantly Black living in countries that are predominantly White.32-41

**Interaction between individual and ecological social factors**

However, in the real world, individual and ecological risks may interact. For instance, some ecological factors could decrease the rates of illness, either by decreasing the impact of individual risk factors or by reducing the possibility of exposure to individual social risks. The social safety net, for instance, may mitigate the impact of life events. Conversely, some ecological factors could interact with other ecological or individual factors to increase risk. Individual and ecological factors may not simply be independent variables acting in synergy, but may amplify each other’s effects thus further increasing risk. For example, at an individual level use of cannabis may increase a person’s risk of developing a psychotic illness. But at an ecological level, the availability of cannabis in the community could increase the risk of that individual using cannabis in the first place. The social context is also important in other ways. Regular use of cannabis in some jurisdictions may offer access to a subculture or environment which changes the risk of exposure to other social factors associated with psychosis. The environment of some regular drug users is characterised by more daily hassles and life events than that of the rest of the public. Exposure to more life events and daily hassles may lead to higher levels of perceived stress and thus further increasing the risk of psychosis. In this way, environment and individual-level risk factors may amplify each other.

**Time**

The fourth dimension is time and this can be considered in a number of different ways. First, sufficient exposure to an individual or ecological risk factor may only occur over time. Second, time may be needed for the interaction between individual-ecological factors to amplify. Third, there are sensitive periods in brain development at which time exposure to certain risk factors may be more important. For instance, being born and brought up in a city is aetiologically more significant in schizophrenia than living in a city per se and other risk factors such as separation from parents may be more important in childhood than adult life. Fourth, there may be a delay in time between the exposure to a risk factor and the development of a psychosis. For instance, the impact of maternal malnutrition on psychosis risk may only be evident when offspring reach early adult life.3

**Developing interventions**

This simplified model may be useful in considering possible prevention and promotion initiatives, since each dimension can be linked to types of strategies. Prevention at an individual level will aim to decrease the amount of exposure or limit the number of different social factors to which a person is exposed. Promotion efforts would enhance behaviours and expose individuals to social factors known to strengthen mental health. At an ecological level, the aim would be to develop healthy environments and limit exposure to psychologically noxious social environments. Where interactions between individual-ecological factors lead to an amplification or spiralling of risk, targeting such interactions may be important. If neither individual risk factors nor environmental risk factors can be reduced, then remedial strategies aimed at uncoupling the links between factors could be effective. And, of course, time is all-important. Understanding the stages of development at which particular risk factors act is important for targeting prevention strategies for maximal effect. Understanding the longer-term impacts of particular risk factors may give an indication of how long preventive strategies should last. Finally, understanding the timescales over which social factors may have their effects helps evaluators to pick the right time frame for assessment of the effectiveness of interventions.

**Conclusion**

We have outlined here a framework for considering how a range of social factors, acting over time, could individually or together make a contribution to the development of psychotic illness. The importance of such a model for conceptualising the social aetiology of psychosis lies in demonstrating the complex and multifaceted interplay between individual and ecological dimensions, their interactions, and time, while taking into account gaps in knowledge or the current evidence base. We hope that this model will help in research; in the design and planning of interventions aimed at particular risk and protective factors; in the training of health professionals; and in the development of health and social systems that more effectively support populations at risk of developing psychosis.

**References**

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