

# Identity in relation to the “excess of psychosis” phenomenon within the ethnic minority population

Nicole Casali, Scuola Galileiana di Studi Superiori di Padova, Classe di Scienze Sociali

## Abstract

The aim of this paper is to provide a picture of the main psychological and anthropological features related to the increased prevalence of psychosis in immigrant ethnic groups, in the attempt to combine scientific research with theoretical thought. We will argue an integrated and culture wise approach is needed when interpreting scientific evidence, as well as ethnopsychiatry and community psychology principles should guide research and clinical practice. In particular, the paper will focus on trauma, dissociation and nostalgia as part of the subjective migration experience. Second generation immigrants will be more closely considered, given their further proneness to psychosis (a risk 4-5 times higher than natives, compared to a 3 times increased risk in first-generation; McGrath et al. 2004, Malzeberg 1995). The multifaceted construct of identity may prove a valuable means for this survey, having been dealt from multiple perspectives and because of its association with psychosis, historically as well as statistically. For instance, research suggests both an increased (Reininghaus et al. 2010) and a weak (Veling et al. 2010) ethnic group identity worsen the negative effect of ethnicity on psychosis. Therefore, these findings will be discussed taking into account concepts such as urbanicity, gene-environment interaction and social support, in order to uncover possible mechanisms at work along the pathway from identity crisis to psychosis onset and chronicization.

## 1. Excess of psychosis: description and epidemiology

Research into psychosis has been increasingly focused on the role environmental issues may have on the pathogenesis and maintenance of symptoms. For instance, a growing body of evidence points out the condition of immigrant is a risk factor for developing severe mental health problems, a phenomenon known as “excess of psychosis”.

Ødegaard was the first in the 1930s to observe the increased prevalence among Norwegian immigrants in the United States compared with indigenous Norwegian population. His explanation referred to genetic liability –those who were prone to develop schizophrenia were more likely to emigrate. Several studies disconfirmed the “selective migration” hypothesis and underlined the role played by environmental factors in order to explain second generation further increased risk and the similarities observed in the epidemiology between homeland and immigration countries. Furthermore, as reported by Sharpley (2001) in her review, the incidence of schizophrenia among African-Caribbeans living in England is raised not only relative to the host English population but also to their population of origin in the Caribbean.

More intriguingly, this increased risk cannot be explained away as just the artefact of culturally insensitive diagnostic processes, as originally demonstrated by a study held at the Institute of Psychiatry in London, involving a Jamaican psychiatrist, Fred Hickling (Hickling et al. 1999).

At a psychological level, possible explanations involve racial tension, exposure to overt discrimination and institutional racism, together with experiences of alienation and isolation, possibly affecting attributions and self-attributions, psychological processes that appear to influence paranoia and mania (Bentall, 2003).

### 1.1. Second generation further risk: possible explanations

These issues become even more apparent when dealing with second generation. According to Richard Bentall, one possibility is that practical and economic problems associated with migration constitute a hinder for families to provide their offspring with ideal child-rearing experiences that would act as protection factors from these influences. For instance, early separation (before 16 years-old) from parents often observed in immigrant experience has been identified by AESOP study as a risk factor, as well as younger age at migration and duration of migration (reported in Tarricone et al. 2015).

Another possibility is that questions of culture identity are especially taxing for second-generation immigrants. According to the Canadian psychologist John Berry (1990), the so-called ethnocultural identity conflict (Baumeister, Shapiro, and Tice, 1985) can be solved through four possible outcomes: integration (the individual identifies with and exhibits some features of both cultures), assimilation (the host culture is embraced and the culture of origin is disowned), separation (the individual retains their culture and rejects the host culture) and marginalization (the individual feels uncommitted to either culture). Integration is associated with the most adaptive outcomes, including psychological wellbeing (Berry, Kim, Minde, & Mok, 1987; Dona` & Berry, 1994; Sam & Berry, 1995; Ward & Kennedy, 1994; Ward & Rana-Deuba, 1999). In a study by Veling et al. (2010), psychotic subjects “*significantly more often had an assimilated or a marginalized identity and less often had a separated identity*”.

More recently, Colleen Ward has questioned this approach to acculturation as too reductive and has proposed a new construct, motivation for ethno-cultural continuity, to emphasize the agency of migrating individuals in the long-term acculturation of their group, including commitment to cultural persistence over time. Not only does the group exert influence on acculturating individuals but also individuals’ attitudes and behaviours have consequences for the group, both in the present and the future (Gezentsvey, Ward, Liu, 2013).

## 2. Ethnopsychiatry and community psychology principles

The point made by Ward is methodologically interesting, both because it shows the need to progress in acculturation research and because it aims at considering both the individual and the area/group levels. A couple more premises, drawn from two different disciplines, ethnopsychiatry and community psychology, shall be made to methodologically frame the discussion.

Ethnopsychiatry is grounded in Georges Devereux’s *Etnopsychanalyse complémentariste* (1972). Devereux brought inspiration from the notion of complementarity by Niels Bohr, which is in turn a generalization of Heisenberg’s uncertainty principle. Following this hint from physics, the French anthropologist maintains a double discourse has to be developed, a psychological and an ethnological one, in order to study a phenomenon not simultaneously, but complementarily, therefore respecting each discipline’s borders.

According to Nathan, one of Devereux’s main disciples, ethnopsychiatry derives its clinical utility from this concept of double and that of frontier: the researcher has to have the mastery over two different scientific discourses and the frontier becomes an object of study, thus epistemologically founding this discipline. Another principle we could assume from ethnopsychiatry is the similarity that can be drawn between the psychotic and the stranger: both “force us to accept and appreciate the different ways of how we understand reality, the relationships between people, and to re-think our interpretative and therapeutic models” (Mandaglio & Maierà, 2014).

Community psychology approach is fundamental in order to interpret immigrant psychosis. It focuses on the individual-in-the-context, meaning the person can never be tackled separately from the environment and the historical experience in which they have been placed.

That separation would produce a capital misinterpretation of our phenomenon: by considering genetic liability alone, it would be difficult to explain why there is an increase in psychosis in first generation immigrants, given the prevalence in origin countries is similar to the one of immigration destinations.

Conversely, genetics plays an important role in the further increase observed in second generation population, and biology is the key to describe possible pathways connecting environmental changes, psychological transformations and psychosis onset and maintenance.

### **3. Nostalgia, dissociation, trauma: psychological mechanisms implicated in immigrant psychosis**

Nostalgia or home-sickness as a specific form of “immigrant psychosis” (Frost, 1938) can be interpreted thanks to ambivalence (Coppo, 2003). On the one side, there can be a feeling of criticism towards the new context that is expressed through resistance at integrating. On the other side, there is the drive for migration, e.g. intolerability for the previous condition, but the homeland is now idealised and becomes an object of desire and longing.

This dynamics affects identity, namely the identity of the immigrant is hanging in the balance between two opposite tendencies, that of closure, resulting in a radicalization of one’s ethnic sense of belonging, and that of deculturation, the loss of cultural landmarks and the fall into aspecificity.

This possibility is particularly dangerous in terms of identity stability. According to Beneduce (1999), deculturation occurs when hybridisation is forced, violent or too rapid, and consists in the loss of the vital connections acting as protection mechanisms and means of reproduction of personal identity and one’s sense of belonging. The crucial point is that those vital connections (e.g. cultural and ethnic references) are lost without being replaced by alternative devices, thus leaving the individual devoid and lost. In Freudian lexicon, we could refer to this dynamics as a complicated grief, in which the object of love is lost and cannot be replaced.

In other words, we could also look at the phenomenon as an immigrant “double loneliness”, because not only they are orphans of their culture (as Tahar Ben Jelloun puts it), but also they may be incapable to develop strong social and affective relationships. Moreover, home-sickness can be more precisely addressed as “territorial anxiety” a term coined by De Martino to indicate the removal of indigenous reference points.

Following De Martino conceptualization of mental illness, *“the madman is detached from the present, precisely because he cannot fully “be-there” (esserci) in the present, being still anchored or polarized in an undecided critical moment of his own personal history, where the chance of any overcoming is reduced. Thus the person stands non-dialectically in presence; no longer as an instance of conscious awareness, or active memory, but as symptom”* (translation from 1956 article, 2012).

He coined the expression “crisis of presence” to indicate the condition of an individual being devoid of the vital possibility to make decisions, *“losing the ability of being the meaning and norm of this process [the historical one]”*, hence social withdrawal and incompatibility with any form of cultural life.

Trauma and dissociation can be discussed together since exposure to trauma is thought to exacerbate and precipitate dissociative experiences, such as altered memory, perception and identity (Seligman & Kirmayer, 2008) and dissociation predicts psychotic symptoms, such as hallucinations, in patients with a history of trauma (Kilcommons & Morrison, 2005). This relation has to do with migration experience, since it represents a critical event of life that can often be traumatic, with refugees reporting PTSD (Post Traumatic Stress Disorder) and dissociation symptoms. Dissociation is an adaptive response to threat, as long as it is flexible; it becomes object of clinical attention when it stands as the only and primary method of coping with situations that are experienced as traumatic.

### **4. Biological pathways involved**

From a neurobiological point of view, the underlying explanation refers to the effect negative adverse social experiences could have on the hypothalamic-pituitary axis, whose dysregulation has been implicated in psychosis onset. Facing severe or chronic social stress, such as isolation, low socio-

economic status or perceived discrimination may result in long-term alterations of the biological stress-response system. Wickham et al. (2014), by using IMD (Index of Multiple Deprivation), found it significantly predicted psychosis, specifically paranoia, but not hallucinations or hypomania. In *The Spirit level* (2008), Wilkinson and Pickett gathered together an impressive array of evidence showing that social inequality (not wealth) is related to poor mental health, possibly due to effect of negative social comparison on the HPA axis.

Hyperactivity of the HPA axis includes low cortisol, increased glucocorticoid sensitivity and functional desensitisation of the glucocorticoid receptor (whose activation would inhibit inflammation) and has been linked to emotional trauma leading to immunosuppression (reported in Rhen & Chidlowski, 2005). Individuals confronting long-term social adversities have repeatedly exhibited increased expression of pro-inflammatory immune response genes and decreased expression of genes involved in inflammatory and antiviral responses (reported in Tarricone et al. 2015). Interestingly, a recent meta-analysis by Miller et al. (2013) has suggested the presence of an inflammatory syndrome in schizophrenia.

Another line of evidence is that social isolation can lead to dopamine dysfunctions, thus increasing the risk of developing psychosis, specifically positive symptoms, e.g. delusions and hallucinations, as in Kapur (2003) model of schizophrenia, in which dopamine dysfunction is connected to aberrant salience assigned to experiences and thus delusion formation as an attempt to make sense of them.

None of the common levels of inquiry in schizophrenia revealed useful: most neurodevelopmental factors (such as obstetric complications, vitamin D deficiency or neurological markers) have proved unlinked to excess of psychosis, as well as substance use (primarily cannabis) hypothesis (Morgan et al., 2008).

## 5. Environmental issues

Among environmental factors associated with schizophrenia, urbanicity, gene-environment interactions and social support are ones more closely related to our phenomenon. As

for urbanicity (e.g. negative impact of urban environment over health), Vassos et al. (2012) conducted a meta-analysis and concluded that the risk of schizophrenia at the most urban environment was 2.37 times in the most rural environment. Kirkbride et al. (2012) used environmental data for 427 FEP subjects to estimate the incidence of disorders across 56 neighbourhoods in East London area: incidence of nonaffective psychosis was independently associated with increased deprivation, income inequality and population density. Schofield et al. (2011) found a negative association between ethnic density and psychosis incidence. In those neighborhoods where black people were less well represented, their relative risk increased nearly threefold (odds ratio OR=2.88).

Another critical factor emerged in a study carried out in different neighbourhoods in London, where it was found that non-white people living in white neighbourhoods were more likely to become psychotic than those living in non-white areas (Boydell et al. 2001). The relative risk (RR) of psychosis for minorities who live in areas with low ethnic density (less than 22%) was about twice (RR 4.4) the risk of those living in areas of high density (RR 2.4). Interestingly, lower rates are observed in more cohesive and less fragmented neighborhoods, not the poorest but rather the most disorganized. This means that social capital could mediate the effect of ethnicity and density in increasing the risk of developing schizophrenia, buffering negative aspects of migration experience, such as perceived discrimination or unsuccessful acculturation.

Gene-environment interactions are to be considered here in connection with epigenetics (Tahira & Agius, 2012): epimutations and epigenetic polymorphisms (e.g. reversible and heritable changes in DNA methylation and histone deacetylation) would be induced by psychosocial stress related to migration, and it would be possible to reverse negative effects by intervening on the environmental

context. Social support could have a normalizing effect (Veling et al. 2008) and act as well as a network in which immigrants can be embedded to make their experience less traumatic and find areas of interplay between original and hosting culture (Coppo, 2003).

### **Conclusions. Ethnic identity: which role?**

Research suggests both an increased (Reininghaus et al. 2010) and a weak (Veling et al. 2010) ethnic group identity worsen the negative effect of ethnicity on psychosis. In the former study a comparison was made between Black and minority ethnic (BME) and White British population. The association between strong ethnic identity and psychosis in BME individuals was attenuated and nonsignificant when controlled for perceived disadvantage. This means perceived disadvantage may explain the effects of strong ethnic identity as a contributor factor of increased psychosis.

The two results are not in contradiction: on the one hand, strong ethnic identification may be associated with increased isolation and social distance from other members of the society, for instance the White majority group (Boydell et al. 2001). On the other hand, according to Veling et al., positive identification with one's own ethnic group is not intrinsically protective, but rather its value depends upon the social context. As previously mentioned, we should consider the conflict between assimilation and identity radicalization.

To conclude with, we could highlight excess of psychosis is a complex phenomenon, which can be dealt with from several perspectives, each explaining one or a subset of its features. Ethnic identity may be central in explaining the onset of psychosis, but for even opposite reasons, as we have seen. A study by Bhugra et al. (2010) found BME cases reported weaker ethnic identification in some life domains than BME controls, but stronger ethnic identification in others, underlining the importance of closely study how the same phenomenon (in this case, excess of psychosis) is declined in different ways due to different historical and cultural contexts.

### **Bibliography**

-Books

Beneduce, R. (2007). *Etnopsichiatria: sofferenza mentale e alterità fra storia, dominio e cultura*. Carocci

Bentall, R. P. (2003). *Madness Explained: Psychosis and Human Nature*. Penguin UK

Coppo, P. (2003). *Tra psiche e culture. Elementi di etnopsichiatria*. Torino, Bollati Boringhieri

-Journal articles

- Boydell, J., Van Os, J., McKenzie, K., Allardyce, J., Goel, R., McCreadie, R. G., & Murray, R. M. (2001). Incidence of schizophrenia in ethnic minorities in London: ecological study into interactions with environment. *Bmj*, *323*(7325), 1336.
- De Martino, E. (2012). Crisis of presence and religious reintegration. Prefaced and Translated by Tobia Farnetti and Charles Stewart. *HLAU: Journal of Ethnographic Theory*, *2*(2), 431-450.
- Hickling, F. W., McKenzie, K., Mullen, R., & Murray, R. (1999). A Jamaican psychiatrist evaluates diagnoses at a London psychiatric hospital. *The British Journal of Psychiatry*, *175*(3), 283-285.
- Kilcommons, A. M., & Morrison, A. P. (2005). Relationships between trauma and psychosis: an exploration of cognitive and dissociative factors. *Acta Psychiatrica Scandinavica*, *112*(5), 351-359.
- Kirkbride, J. B., Morgan, C., Fearon, P., Dazzan, P., Murray, R. M., & Jones, P. B. (2007). Neighbourhood-level effects on psychoses: re-examining the role of context. *Psychological medicine*, *37*(10), 1413-1425.
- Mandaglio, C., & Maierà, E. (2014). Psychosis and migration: does a correlation exist?. *Psichiatria Danubina*, *26*(1), 119-122.
- Morgan, C., Charalambides, M., Hutchinson, G., & Murray, R. M. (2010). Migration, ethnicity, and psychosis: toward a sociodevelopmental model. *Schizophrenia bulletin*, sbq051.
- Ødegaard, Ø. (1932). Immigration and Insanity: A Study of Mental Disease Among the Norwegian born Population of Minnesota. *Acta psychiatrica Scandinavica, Suppl*, *4*.
- Reininghaus, U., Craig, T. K., Fisher, H. L., Hutchinson, G., Fearon, P., Morgan, K., ... & Morgan, C. (2010). Ethnic identity, perceptions of disadvantage, and psychosis: findings from the AESOP study. *Schizophrenia research*, *124*(1), 43-48.
- Rhen, T., & Cidowski, J. A. (2005). Antiinflammatory action of glucocorticoids—new mechanisms for old drugs. *New England Journal of Medicine*, *353*(16), 1711-1723.
- Schofield, P., Ashworth, M., & Jones, R. (2011). Ethnic isolation and psychosis: re-examining the ethnic density effect. *Psychological medicine*, *41*(06), 1263-1269.
- Seligman, R., & Kirmayer, L. J. (2008). Dissociative experience and cultural neuroscience: Narrative, metaphor and mechanism. *Culture, medicine and psychiatry*, *32*(1), 31-64.
- Sharpley, M. S., Hutchinson, G., Murray, R. M., & McKenzie, K. (2001). Understanding the excess of psychosis among the African—Caribbean population in England. *The British Journal of Psychiatry*, *178*(40), s60-s68.
- Tahira, A., & Agius, M. (2012). Epigenetics and migration—considerations based on the incidence of psychosis in South Asians in Luton, England. *Psichiatria Danubina*, *24*, S194-196.
- Tarricone, I.; Tosato, S.; Cianconi, P.; Braca, M.; Fiorillo, A.; Valmaggia, L.; Morgan, C., *Migration history, minorities status and risk of psychosis: An epidemiological explanation and a psychopathological insight*, «JOURNAL OF PSYCHOPATHOLOGY», 2015, 21, pp. 424 – 430
- Veling, W., Hoek, H. W., Wiersma, D., & Mackenbach, J. P. (2009). Ethnic identity and the risk of schizophrenia in ethnic minorities: a case-control study. *Schizophrenia bulletin*, sbp032.

Veling, W., Susser, E., Van Os, J., Mackenbach, J. P., Selten, J. P., & Hoek, H. W. (2008). Ethnic density of neighborhoods and incidence of psychotic disorders among immigrants. *American Journal of Psychiatry*, 165(1), 66-73.

Ward, C. (2008). Thinking outside the Berry boxes: New perspectives on identity, acculturation and intercultural relations. *International Journal of Intercultural Relations*, 32(2), 105-114.

Wickham, S., Taylor, P., Shevlin, M., & Bentall, R. P. (2014). The impact of social deprivation on paranoia, hallucinations, mania and depression: the role of discrimination social support, stress and trust. *PloS one*, 9(8), e105140.