

ENVIRONMENTAL TRIGGERS OF PSYCHOPATOLOGY: GENDER & DEPRESSION AS A CASE STUDY

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Abstract. In this paper I set out to explore a subset of environmental factors that have been found to be significantly correlated with the development of psychiatric symptoms. More specifically, I argue that situations of *disadvantage* such as the exposure to repeated discrimination or persistent social isolation may act as triggers of mental disorders, thereby causing or exacerbating psychiatric symptoms. The paper is divided into three sections: in §1. I briefly discuss the important role played by environmental factors as triggers of psychopathology; in §2. I take the high incidence of depression among women as a case study to illustrate the points outlined in §1. In §3. I introduce the general notions of *at-risk population*, *vulnerability* and *triggers* and I suggest that they can be helpful in explaining how a pathological constellation comes about.

1 INTRODUCTION

Exploring the role played by *environmental factors* in the onset of psychopathology is crucial for a view of psychiatry that goes beyond the neurobiological level and takes personal as well as social phenomena into account (see Murphy 2006). In this paper I set out to explore a subset of environmental factors that have been found to be significantly correlated with the development of psychiatric symptoms. More specifically, I argue that situations of *disadvantage* such as the exposure to repeated discrimination or persistent social isolation may act as triggers of mental disorders, thereby causing or exacerbating psychiatric symptoms. My main goal is to defend two claims:

- a) Some etiological factors that are crucial for the onset of psychopathology cannot be reduced to traits or habits *internal* to the patient. Indeed, external conditions such as social pressures or discrimination may also play an important role.
- b) Specific forms of disadvantage can be mapped onto specific disturbances: in other words, it is possible to connect the emergence of some symptoms with significant life events or conditions (see Bentall et al. 2014 for a similar point). Notably, this allows us to go beyond the general formulations offered in the literature – e.g. “social disadvantage” – to uncover more specific mechanisms through which environmental factors affect psychological well-being.

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b). In §3. I introduce the general notions of at-risk population, vulnerability and triggers and I suggest that they can be helpful in explaining how a pathological constellation comes about.

2 BACKGROUND: ENVIRONMENT AND PSYCHOPATHOLOGY

Within the history of Western psychiatry, Freud has probably been the strongest defender of the key role played by environmental factors in the onset of mental disorders. Indeed, psychoanalysis offers a multi-factorial account of how mental disorders arise and distinguishes itself from competing views of mental illness as “organic inferiority” (Adler 1907) or “degeneracy” (Janet 1894). Yet, Freud’s skepticism against purely organic accounts should not be interpreted as a wholesale rejection of the role played by biological factors. Indeed, on his view two different kinds of pathogenic determinants need to be present: *dispositions* (or constitutional factors) on the one hand, and *experiences* (or accidental factors) on the other. The former are described as those elements that “a person brings along with him into his life”, whereas the latter are the ones that “life brings to him” (Freud 1913, p. 2623). The very idea that accidental factors would play an important role in the development of psychopathology has been introduced by Freud & Breuer in their *Preliminary Communication*: “[Our results] are valuable theoretically because they have taught us that external events determine the pathology of hysteria to an extent far greater than is known and recognized” (1893, pp. 3-4). A few years later, Freud insists that mental disorders should not be treated on a par with cases of “mental degeneracy” but that they can be seen as motivated responses to traumatic life events. Thus, the main novelty of such an account consists in the idea that mental disorders seldom have a uniquely identifiable cause and should rather be seen as constellations of pathogenic elements.

In the past few decades, several researchers in psychiatry and clinical psychology have focused on the impact of environmental factors and explored the correlation between situations of social disadvantage and the onset of psychopathology. A pioneering work in this sense has been conducted by the Dutch psychiatrist Jim van Os (1998), whose research on the eco-genetics of schizophrenia has uncovered a complex system of interactions between genetic and environmental factors. On one hand, there is interplay between genetic elements – e.g. a family member affected by the disorder – and environmental “risk-increasing” elements, such as stressful life experiences. On the other, some interactions that are significant for psychopathology do not depend strictly on genetic factors: van Os dubs them

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“environment-environment interactions” (van Os 2003, pp. 291-292). For example, migrant populations have been found at increased risk of developing schizophrenia but *only* under specific conditions, such as being part of a visible minority (Ottesen et al. 2014) or reporting high levels of discrimination (Janssen et al. 2003). This distinction between gene-environment and environment-environment interactions is crucial because it allows us to see that the environment can act upon the individual at multiple levels. For example, it may work in combination with genetic predispositions and liabilities (e.g. familiarity plus traumatic event) or interact with other environmental elements thereby giving rise to pathological constellations (e.g. minority status and discrimination).

In what follows, I discuss the case study of gender and depression as an example of environment-environment interaction. First, I briefly discuss some recent empirical evidence in order to isolate some candidate factors that may be – at least partially – responsible for the high incidence of depression among women. Then I show that specific disturbances or symptoms can be reliably connected with specific forms of disadvantage. This takes us a step further with respect to the observation that some populations exhibit a high incidence of mental disorders. Indeed, it allows us to sketch an environmental-level explanation of such an incidence: for example, the repeated exposure to discrimination – and at times violence – experienced by *women* in most societies may trigger feelings of hopelessness and frustration that are characteristic of depression (see Ussher 2010).

3 GENDER AND DEPRESSION

The high incidence of depressive disorders among *women* has been reported by several clinical and epidemiological studies throughout the last few decades (see Bebbington 1996; Jenkins 1987). Most of these studies stress the fact that women experience depressive symptoms at higher rates than men – the numbers agree on a 2:1 ratio – but also that they are more likely to seek medical attention and thus more likely to receive pharmacological treatment (see LaFrance 2007 for a review). Generally speaking, some of the researchers appeal to biological or constitutional differences to explain the gender gap whereas others see it as the direct result of a social construction. Among the biological and constitutional factors we find hormone levels (see Seaman 1997) as well as the tendency towards rumination and a low sense of mastery (see Nolen-Hoeksema, Larson & Grayson 1999). Among the constructivist accounts, most regard depression as the mere product of discursive trends that shape conceptions or expectations on gender and thus pathologize women’s emotions and experiences. For example, Emmons (2008) argues that the pervasive characterization of women as “the emotional sex” lends itself to the idea that depression would be an intrinsically feminine disorder. As she puts it: “While depression in men tends to be presented as a stark departure from ‘normal’ feelings or emotions, depression in women is more likely to be understood as an *outgrowth* of women’s complex emotional lives” (p. 112. *Italics mine*).

Drawing on Freud’s idea of mental disorders as constellations arising from a variety of factors, I suggest a more complex etiological picture to explain the higher incidence of depression among women. Indeed, focusing on environmental factors does not necessarily entail embracing a social constructivist account. In particular, one can maintain that the disadvantaged condition of

women in most societies makes them more liable to developing depressive symptoms without thereby denying the reality of the disorder. Similarly, one could acknowledge the great impact of socio-economic factors on the higher incidence of somatic ailments in certain populations – e.g. diabetes in African Americans – without regarding these diseases as mere social constructions. Such an approach would allow us to recognize that environmental factors play a key role in psychopathology while acknowledging that they crucially interact with personal-level elements (e.g. emotional reactions) and neurobiological processes (e.g. amygdala regulation). Thus, in order to see the relationship between gender and depression as an example of environment-environment interaction, it is not sufficient to isolate one sub-group of individuals because of their heightened vulnerability towards a specific mental disorder. It is also crucial to understand how the onset of psychopathology connects with a host of factors *within* the relevant sub-group. Indeed, nuanced distinctions can be made between different risk-factors affecting women’s well being and cross-cultural studies can be especially informative in this sense.

Recently, some interesting studies have been exploring the reasons behind the gender gap in depression by investigating more specific environmental factors such as relationship status, employment and social support. For example, in an Austrian study that takes into account gender, relationship and employment status, *marriage* has been found to act as a protective factor against depression for men but not for women. Indeed, among married subjects women exhibited an incidence rate that was more than twice as high with respect to men – i.e. 72 versus 31 patients out of 100.000 (Gutierrez-Lobos et al. 2000, p. 204). The same study also reports that *employment* status significantly affects depression rates – both for men and women – with higher incidence among the unemployed (p. 206). However, when marital and employment status are considered together the gender gap comes to the surface again, with unemployed married women showing higher rates of depression than their male counterpart – i.e. 88 versus 41 out of 100.000 (p. 204). The situation appears reversed among the unmarried, with unemployed men displaying higher rates of depression – i.e. 140 versus 128 (p. 204). This study is particularly interesting for our purposes because it goes beyond the “gross oversimplification” of women being more liable to depression, and explores more specific environmental factors that could be responsible for the gap (p. 207). These results also point towards more profound reasons that may explain women’s heightened vulnerability to depression. Indeed, the fact that married *and* unemployed women form the sub-group with highest depression rates is consistent with other studies that stress the unequal workload distribution within the household and childcare. For example, in a research measuring the emotional states and degree of happiness experienced by a group of North-American mothers and fathers, Larson, Richards and Perry-Jenkins (1994) found that “women who are full-time in the traditional homemaker role” report more negative emotional states and lower degrees of happiness across the board (p. 1044). Again, more recent studies on post-partum depression indicate that new mothers lacking adequate social support are more likely to develop depressive as well as psychotic symptoms (see Fairbrother & Woody 2008).

At the same time, the data concerning gender and depression also vary *cross-culturally*, with studies reporting higher rates among employed women in India (Das, Das & Das 2012) and in

rural European areas (Vazquez-Barquero *et al.* 1992). These results are – again – interesting because they suggest that cultural background and social pressures may play a significant role in explaining the high incidence of female depression. Indeed, it is plausible to assume that unemployment would affect women more negatively in societies where they are expected to contribute to the workforce and in which gender roles are somewhat fluid (e.g. Austria). The opposite may be the case in societies where women are expected to adhere to strictly defined gender roles that often comprise housework and childcare (e.g. India). In such contexts, working women may be more exposed to discrimination and stereotyping because their status could be seen as “threatening the authority of men” (Das, Das & Das 2012, p. 1661).

4 AT-RISK POPULATION: VULNERABILITY, TRIGGERS, & PATHOLOGICAL CONSTELLATIONS

In the discussion above I have stressed the importance of different kinds of factors that often interact with one another and facilitate the development of psychopathology. In particular, I have briefly explored the case of gender and depression to show that specific forms of disadvantage can be mapped onto specific disturbances. Here I introduce the notions of *at-risk population*, *vulnerability* and *triggers*, suggesting that they could be applied more generally to illustrate the influence of different factors on psychopathology. Roughly speaking, *at-risk populations* can be characterized as groups of individuals under the influence of environmental factors that are highly correlated with psychopathology. Notably, this notion goes hand in hand with the one of *at-risk individuals*: these can be characterized as people exhibiting personality traits, metacognitive habits and life experiences that make them particularly liable to the development of pathological combinations of symptoms.²

Three important notions need to be explored further in order to garner a better understanding of the interplay between various risk factors:

- 1) *Vulnerability*. This is a state or condition that constitutes a “weak spot” either in an individual’s biological makeup (e.g. having a biomarker connected with a specific disorder) or in her socio-cultural environment (e.g. being a member of a disadvantaged group).
- 2) *Triggers*. These are events or processes that work as causal influences operating on existing vulnerabilities. Just like vulnerabilities, triggers can be found at different levels: sub-personal (e.g. experiencing a neurological trauma), personal (e.g. experiencing negative emotional reactions) or environmental (e.g. being discriminated for belonging to a certain group).
- 3) *Pathological constellation*. This usually emerges from some combination of 1) & 2) and can be characterized as the sum of multiple vulnerabilities or triggers experienced by an individual. On the one hand, every constellation would be highly individualized and deeply connected with the

patient’s life history. On the other, it should be possible to draw significant parallels among different cases due to common neurobiological and environmental features (e.g. same biomarker; same culture or community).

Applying this framework to the case study discussed above, one could see “being a woman” as a *de facto* vulnerability for depression. Generally speaking, this means that some individuals or groups can be regarded as more vulnerable simply due to socio-cultural pressures and constraints. This point ties then into a more general social critique: if some environmental factors play a key role in the onset of psychopathology, therapeutic approaches that are solely person-centered may fail to address the issue at the appropriate level. Yet, vulnerability *per se* is clearly not sufficient for the development of pathological symptoms: after all, many women are not affected by depression despite belonging to a vulnerable group. Some events may thus act as triggers – e.g. “being discriminated *qua* woman” – and can be characterized as processes extending over time and exercising a causal influence upon the existing vulnerability. Triggers can then be seen as a set of actions, perceptions and experiences: e.g. being denied a raise, experiencing sexual harassment, being subject to social pressures concerning traditional gender roles, etc...In our case study, a pathological constellation would then appear as the outcome of a certain combination of vulnerability and triggers. For example, a woman could start developing paranoid or obsessive thoughts and exhibiting certain behaviors (e.g. avoiding social contact) or affective states (e.g. fear or anxiety) as the result of repeated experiences of discrimination and social pressures.

5 CONCLUSION

To sum up: this discussion shows that the interplay between environmental factors and psychopathology is a complex and nuanced one. First – in a Freudian spirit – I have proposed to regard mental disorders as constellations of pathogenic elements where different kinds of factors are operating at the same time (i.e. dispositions and experiences). Second, I have argued that it is not sufficient to isolate some sub-groups of individuals because of their heightened vulnerability towards specific mental disorders (e.g. women and depression). It is also crucial to understand how the onset of psychopathology connects with some relevant factors *within* the sub-group. While some of these factors may turn out to be constitutional, some of them can be characterized as environment-environment interactions (see van Os 2003). Moreover, special attention should be paid to cross-cultural results: indeed – in the case study discussed above – a similar disorder may appear in two different societies as the result of opposing pressures (i.e. women are expected to work/not to work). Finally, I have introduced the general notion of at-risk population and I have suggested that it may help to capture the various environmental influences at a finer-grained level of analysis. For instance, being able to distinguish between structural vulnerabilities and local triggers may allow clinicians to tailor therapeutic interventions to their patients and adequately assess the impact of different risk factors in specific situations.

belonging to at-risk populations would be particularly prone to developing clinically relevant manifestations.

² I cannot discuss the notion of at-risk individual in detail here, but I want to stress the fact that these two notions should not be seen as mutually exclusive. In other words, it makes sense to think that at-risk individuals

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