INTRODUCTION

• Genetic and environmental factors both contribute to individual differences in schizophrenia.

• The evidence linking environmental risks to psychotic syndrome (group of symptoms), and to what degree such associations may be valid and indicative of causal influence is examined; which aspects of the environment actually explain, or mediate (as factor between the environmental exposure and the outcome that actually occasions the effect).

• Things like the variation in places or environment; and which biological and cognitive mechanisms may be the source of such effects are being examined. This explains how the gene-environment goes hand in hand.

• Efforts have been made to improve upon data collection methods in order to systematically identify and test for a number of risk factors during childhood and adolescence in relation to risk for schizophrenia.

• Many studies of schizophrenia have begun to incorporate measures of the environment into animal models.

A neural diathesis stress model of schizophrenia concludes with the importance of the genetic component. The environment can contribute to the expression of genetic predispositions. It is possible to have high levels of genetic risk and never develop schizophrenia, but for those who do develop it, the environment plays a crucial role.

ABSTRACT

This literature review examines the biological and environmental risk factors in the etiology of schizophrenia. A well-researched theoretical framework behind the etiology of schizophrenia is the dopamine hypothesis which states the elevated levels of the brain chemical dopamine can contribute to the development of schizophrenia. The diathesis-stress model describes the heightened cortisol releasing the potential to intensify schizophrenia symptoms while amplifying dopamine activity in the environment. More recent findings show that genetics influences act in cohesion with environmental factors. Based upon the findings of this literature review; genetics, prenatal and postnatal factors, brain abnormalities, and environmental factors all have an impact on the etiology of schizophrenia. Further research should focus on how biological and environmental factors impact the etiology of schizophrenia to aid in better understanding of the illness’s development.

RISK FACTORS

Psychological

• Maternal depression (mental illness) and stress are huge risk factors. (Alan S. Brown 2011)

• Developmental trauma can be a risk factor of schizophrenia. (Jim van Os, Bart P. F. Rutten 2010)

• Prenatal nutrition in the etiology of schizophrenia has a number of known neuropsychiatric disorders related to nutritional deprivation. (Alan S. Brown 2011)

Biological

• Having a parent(s) with schizophrenia increases the risk of psychosis. Having a family history is the strongest relative risk. (Alan S. Brown 2011)

• Adolescent cannabis exposure with the genetic variant in the COMT gene synergistically increased their risk of psychosis. (Jim van Os, Bart P. F. Rutten 2010)

• Dopamine changes in the brain has shown an increased risk for schizophrenia. (Olivier D Howes, Robin M Murray 2014)

Environmental

• Growing up in an urban environment have high rates of schizophrenia. (Jim van Os, Bart P. F. Rutten 2010)

• Postnatal and prenatal exposure to infections have been implicated in the pathogenesis of schizophrenia. (Alan S. Brown 2011)

• Migration and ethnic minority status may be other risk factors. (Jim van Os, Bart P. F. Rutten 2010)

• Childhood trauma in detrimental environments is a risk factor of schizophrenia. (Alan S. Brown 2011)

FINDINGS

• Early neglect in detrimental environmental situations can stop stress signaling pathways that lead to impaired cognitive responsiveness. Drastic prefrontal cortical decline can occur due to the environment and the cognitive impairments shown in psychotic symptoms.

• These findings may have specific risk factors that suggest that urban environmental regions sometimes with minorities can link to schizophrenia.

• A diathesis-stress model of schizophrenia states that the illness develops due to stress exposure. It acts on premeditated weaknesses which are genetic factors and early environmental situations.

• Findings in my research showed increased striatal (part of the brain) dopamine activity in the case of the dopamine occurring within the prefrontal terminals and release of dopamine in the brain as the major place of dopamine impairing in schizophrenia.

• Brain abnormalities are definitely shown in schizophrenia in these areas of my research: reduced grey areas, matter volume in various brain areas as well as increased ventricular volume (cavities filled with cerebrospinal fluid).

• Findings in my research have shown that exposures to maternal infections can lead to schizophrenia.

• The animal models show that prenatal vitamin D deficiency is linked to patients with schizophrenia. Iron depletion during pregnancy can also impair dopamine.

EVIDENCE-BASED INTERVENTIONS

• Many studies are focusing on genes in the development of the brain. Environmental risk factors are the best chance of prevention, but genetic factors do not have a high chance if prevention (Oliver D. Howes, Robert McCutcheon, Michael J. Owen, and Robin M. Murray 2016)

• Examining the environment requires updated technology to point the way to possible interventions. (Jim van Os, Bart P. F. Rutten 2010)

• An animal development model in schizophrenia shows that certain treatments can reduce stress and prevent dopamine to dysregulate. These interventions have to be treated early in schizophrenia and before dopamine starts to dysregulate. (Olivier D. Howes, Robin M Murray 2014)

• The prevention of schizophrenia can be solved by interventions and treatments that are already out. Many of these interventions and treatments are inexpensive and cover large populations. (Alan S. Brown 2011)

• Antipsychotics have shown tremendous amounts of treatments towards patients. Most of the help with dopamine regulations. Also, the antipsychotic medicines can reduce cortisol in treating patients. (Olivier D. Howes, Robert McCutcheon, Michael J. Owen, and Robin M. Murray 2016)

• While antipsychotic treatments may reduce increased dopamine activity, other psychotic symptoms like inflammation, cognitive decline, and abnormalities may become worse over time. (Olivier D. Howes, Robert McCutcheon, Michael J. Owen, and Robin M. Murray 2016)

• For example, Mirtazapine is used as a treatment in any disorders relating to stress and it is possibly a successful result for treatment with negative symptoms in schizophrenia. (Olivier D. Howes, Robert McCutcheon, Michael J. Owen, and Robin M. Murray 2016)

• In one research it states the updated model that reviews all aspects of vulnerability, psychosis progression, and neurobiological processes. Also, in this research article, future research will be discovered to help any undeveloped treatment in schizophrenia. The future research will implicate findings for treatment options. (Olivier D. Howes, Robert McCutcheon, Michael J. Owen, and Robin M. Murray 2016)

• A neural diathesis stress model of schizophrenia concludes with future research and treatment strategies. (Olivier D. Howes, Robert McCutcheon, Michael J. Owen, and Robin M. Murray 2016)

• One articles states the various treatment strategies including antipsychotics medication with serotonin re-uptake inhibitors (SSRIs), stress reduction and behavioral interventions corticotropic (CHC), and environmental exercises. (Marita Proussnera, Alexia E. Cullen, Monica Aas, Elaine F. Walker 2017)

IMPLICATIONS

• Assessing the animal and human imaging have shown gene expression, molecular and receptor changes in relation to environmental and genetic experiences.

• Implicating the avenue of future research suggests that risk factors like family history, post natal and prenatal infections and deficiencies, maternal stress, dopamine changes, and cannabis all have a negative impact on mental health pertaining to schizophrenia.

• Examining the many complexities in the assessments for environment/gene impacting schizophrenia has shown that it can be developed for different individuals.

REFERENCES

• References are available upon request.