Case Study

Name

Institution
1. The Possible Biological Causes of Mental Illness

A. Mental illness may be caused by abnormalities in the structure and function of neural circuits.

1) Reduction in the capacity to maintain activated neural circuits that would result in a positive emotion (Heller, Johnstone, Shackman, Light, Peterson, Kolden, Kalin, and Davidson, 2009)

2) Significant decrease in the number of neurons that expressed receptors for the proper glutamergic neurotransmission, resulting in the development of schizophrenia (Bitanihirwe, Lim, Kelly, Kaneko, and Woo, 2009).

3) Electroencephalography showed that the auditory response of the auditory cortex is reduced among patients positively diagnosed with schizophrenia, resulting in the occurrence of hallucinations (Spencer, Niznikiewicz, Shenton, and McCarley, 2009).

4) Neural circuitry in the frontostriatal region of the brain is impaired among patients with bulimia nervosa (Marsh, Steinglass, Graziano, O’Leary, Wang, Murphy, Walsh, and Peterson, 2009).

B. Mental Illness may be Caused by Defective Brain Development

1) Certain regions of the brain, such as the lower occipital complex, fail to be activated in patients positively diagnosed with a mental illness (Green, Lee, Cohen, Engel, Korb, Nuechterlein, and Glahn, 2009).

2) Lesions in the prefrontal-subcortical regions of the brain result in the
development of obsessive-compulsive disorder (Salinas, Dávila, Berthier, Green, and Lara, 2009).

3) Larger cerebellar volumes were observed among teenage marijuana users (Medina, Nagel, and Tapert, 2010).

4) Larger amygdala volumes were observed among children who suffered from poor emotional regulation (Tottenham, Hare, Quinn, McCary, Nurse, Gilhooly, Millner, Galvan, Davidson, Eigsti, Thomas, Freed, Booma, Gunnar, Altemus, Aronson, and Casey, 2010).

C. Genetic Mutations may Result in the Development of Mental Illness

1) Mutations in the dopamine D2 receptor gene are associated with alcohol dependence (Prasad, Ambekar, Vaswani, 2010).

2) The presence of a third sex chromosome of either X or Y is considered as a common feature among Klinefelter children who are neurocognitively impaired (Ross, Zeger, Kushner, Zinn, Roeltgen, 2009).

3) Mutations in the TRAPPC9 gene are related to the development of intellectual disability (Mochida, Mahajnah, Hill, Basel-Vanagaite, Gleason, Hill, Bodell, Crosier, Straussberg, and Walsh, 2009).

4) Mutations in the 7q36.1 chromosomal region are associated with schizophrenia (Atalar, Acuner, Cine, Oncu, Yesilbursa, Ozbek, and Turkcan, 2010).

D. Abnormal Levels of Specific Proteins may Cause Mental Illness
1) The excessive deposition of amyloid beta proteins in the brain cause Alzheimer’s disease (Murphy and LeVine, 2010).

2) Low levels of the selenoprotein P results in the development of Alzheimer’s disease (Takemoto, Berry, and Bellinger, 2010).


4) Reduction in the production of the protein glutamic acid decarboxylase-67 kDa is associated with the development of depression (Karolewicz, Maciag, O'Dwyer, Stockmeier, Feyissa, and Rajkowska, 2010).

2. The Function of the Neurotransmitters in Depression

A. Neurotransmitters relay messages that are generated from the brain of an individual.

B. Neurotransmitters serve as chemical impulses that transmit messages between one neuron to another.

C. Neurotransmitters serve as the connecting matter between two neurons that are in synapse.

D. Three primary neurotransmitters are associated with depression, namely serotonin, norepinephrine and dopamine.

E. The neurotransmitter serotonin regulates the emotions of an individual.

F. The neurotransmitter norepinephrine is responsible for the reaction of an individual to stressful situations.

G. The neurotransmitter dopamine influences the sleep and the appetite of an individual.
3.1 The Cause of Margaret’s Mania

A. Margaret’s mania is mainly due to the excessive amounts of fluoxetine hydrochloride in her body, due to her self-medication of doubling the dosage of the drug.

B. It is highly likely that Margaret is also suffering from bipolar disorder, also known as manic-depressive syndrome, which has been undiagnosed by her attending physician.

C. Margaret’s mania may also be due to the imbalance in the levels of dopamine in her body, resulting in her lack of sleep and her hyperactivity. The excessive amount of dopamine may overstimulate the dopamine receptors of the brain, resulting in a disconnection in the neural circuitry of the prefrontal cortex of the brain (Hairns and Arnsten, 2008).

3.2 Other possible causes of mania

A. Mania may also be due to abnormal brain development, resulting in this particular behavior when triggered by stress or medication. Abnormal brain development may result in an impairment of cognitive performance, as well as the functional recovery from a mental illness (Gogtay and Thompson, 2010).

B. Mania may also be possibly due to mutations in certain genes that are associated with the specific mental illness. For example, the lipid transporter gene ABCA13 has been implicated as a susceptibility factor to bipolar disorder because variations in this gene have been found to be associated with clinical cases of bipolar disorder (Knight, Pickard, Maclean, Mallo, Soares, McRae, Condie, White, Hawkins, McGhee, van Beck, MacIntyre, Starr, Deary, Visscher, Porteous, Cannon, St Clair, Muir, and Blackwood,
C. Mania may also be triggered by exposure to environmental stimuli that would result in the differential expression of genes that are associated in the etiology of this mental illness. It has been reported that nicotine from tobacco smoke, as well as drugs, may significantly increase the expression of SLCA3 and SLCA4 genes, generating the manic phenotype (McEachin, Saccone, Saccone, Kleyman-Smith, Kar, Kare, Ade, Sartor, Cavalcoli, and McInnis, 2010).

D. Mania could be caused by the dysregulation of neural circuitry, such as that of the paralimbic network, which control the prefrontal and, parahippocampal and amygdala, resulting excessive neural activity. The continuous excitation of the corticolimbic network would then result in the symptoms of mania (Brooks, Hoblyn, Woodard, Rosen, and Ketter, 2009).

4.1 The DSM IV Diagnosis of Bipolar Affective Disorder is as follows: (DSM-IV, 1994, p. 350)

A. Bipolar affective disorder is diagnosed when an individual has experienced at least one manic or mixed episode.

B. Bipolar affective disorder is diagnosed when an individual has experienced at least one occurrence of depression.

C. Bipolar affective disorder is diagnosed when an individual presents as least one episode of a mood disorder that was triggered by a substance such as a medication, illegal drug or toxin.

4.2 Margaret meets the criteria for DSM IV diagnosis for bipolar affective disorder due to the
following observations:

A. Margaret is already showing symptoms of mania, including poor sleep and excessive activity.

B. Margaret also presents with rapid pressured speech and racing thoughts, symptoms of the manic condition.

C. Margaret is also reports to be recently highly active in sexual activities, which is another corollary of the manic condition.

D. The case of Margaret is associated with a long history of depression, thus indicating that she may be diagnosed with bipolar affective disorder.

5. i) Three different medications which may be used to treat Margaret as described in the case study:

A. Lithium decreases the occurrence of mood swings in a patient who is positively diagnosed with bipolar affective disorder.

B. Topiramate is a sulfamate-substituted sugar moiety that could serve as a psychotropic drug for patients with bipolar affective disorder.

C. Carbamazepine is an anticonvulsant that lessens the occurrence of manic episodes and tranquilizes a patient with bipolar affective disorder.

ii) Side effects of the medications

A. Lithium

   1. May cause drowsiness and general weakness as it controls the hyperactivity of the neural circuits of the patient (Bolamperti, Mula, Varrasi, Tarletti, Cavanna, Monaco, and Cantello, 2009)
2. May affect the cardiovascular system by causing depolarization of the cardiac muscles, resulting in variations in the electrocardiogram (ECG) readings (Pacítl Slavicek, Dohnalova, Kitzlerova, and Pisvejcová, 2003)

B. Topiramate

1. May result in cognitive impairment, especially when administered for a long duration of time (Park and Kwon, 2008)

2. May induce aggression and violence in the patient due to the modulation of gamma amino butyric acid receptors of the central nervous system (Lane, Gowin, Green, Steinberg, Moeller, Cherek, 2009)

C. Carbamazepine

1. May result of weight gain due to general fatigue that sets in from the administration of carbamazepine (Gaspari and Guerreiro, 2010)

2. May cause urinary retention due to the anticholinergic properties of the carbamazepine (Hmouda, Ben Salem, Grira, Slim, and Bouraoui, 2007)

6. Current research relating to the biological considerations associated with suicide and affective disorders.

A. Brain imaging facilitates in the identification of specific regions of the brain that may be hyperactive (Jollant, Lawrence, Giampietro, Brammer, Fullana, Drapier, Courtet, and Phillips, 2008).

B. Studies on the electrodermal and active potential parameters of the brain and its circuitry may assist in the establishment of connections between high excitability and suicidal attempts (Jandl, Steyer, and Kaschka, 2010).
C. Immunohistochemical studies of tissue sections of brain autopsies help in determining the levels of brain-specific proteins that regulate brain cognition and function (Gabbay, Klein, Guttmann, Babb, Alonso, Nishawala, Katz, Gaite, and Gonzalez, 2009; Savitz et al., 2009).

D. Immunoautoradiography allows the examination of specific brain regions of autopsied individuals who committed suicide for any significant increases in the expression of specific enzymes such as tryptophan hydroxylase (Boldrini et al., 2005).

E. Respiratory conditions have been examined in terms of its influence on the risk of committing suicide (Giltay, Zitman, Menotti, Nissinen, Jacobs, Adachi, Kafatos, Kromhout, 2010).

F. Genetic studies attempt to identify specific genes that may have been mutated and thus result in the abnormal behavior of a patient (Brent, Melhem, Ferrell, Emslie, Wagner, Ryan, Vitiello, Birmaher, Mayes, Zelazny, Onorato, Devlin, Clarke, DeBar, and Keller, 2010; Grohman, Hammer, Walther, Paulmann, Büttner, Eisenmenger, Baghai, Schüle, Rupprecht, Bader, Bondy, Zill, Priller, and Walther, 2010).

G. Genetic association studies may assist in the establishment of risk factors among individuals showing signs of mental illness (De Luca, Hlousek, Likhodi, Van Tol, Kennedy, and Wong, 2006).

References

Atalar, F., Acuner, T.T., Cine, N., Oncu, F., Yesilbursa, D., Ozbek, U & Turkcan, S. (2010). Two four-marker haplotypes on 7q36.1 region indicate that the potassium channel gene HERG1(KCNH2, Kv11.1) is related to schizophrenia: A case control study. *Behavioral and Brain Functions, 6*, 27-37.


suicide victims with major psychoses. *Genes, Brain and Behavior, 5*(1),107–110.


Heller, A.S., Johnstone, T., Shackman, A.J., Light, S.N., Peterson, M.J., Kolden, G.G., Kalin,


Biochemical Behavior, 92(2), 357-362.


