THE EPIGENETIC OF THE PANIC DISORDER

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Abstract

From this review, I discussed the epigenetics of panic disorder. Epigenetic is the changes in the heritable phenotype without any change from the DNA sequence. From one study to the next, there has been disagreement over the functions of many epigenetic processes, including DNA methylation and chromatin remodeling. The focus of the study was on how potential genes might contribute to the emergence and progression of panic disorder. Numerous candidate genes on various chromosomes, including 1q, 2p, 2q, 3, 7, 9, 11, 12q13, 12q23, and 15, may be utilized as markers in the future to diagnose panic disorder in children, are present. The fundamental value of therapies including exposure, cognitive therapy, relaxation training, and breathing retraining has not yet been determined, and recent research has not shown gene therapy's significance in treating panic disorder. Affected protection signal processing and anterior cingulate cortex-amygdala coupling can be used to distinguish between these patients to determine the effectiveness of exposure-based cognitive-behavioral therapy and associated neuroplastic alterations.

Keywords: Panic disorder, Epigenetics, candidate genes, hypermethylation, Hypomethylation, chromatin structure, and chromosomal regions

Introduction

Panic disorder is an anxiety disorder characterized by extreme, unpredictable attacks of severe fear and anticipatory anxiety, frequently comorbid with agoraphobia, with a prevalence of 1-3 percent in life (Ziegler et al. 2016). Patients provide a clinical model of stress with panic disorder. They illustrate persistent stress-related changes in sympathetic nerve biology on a good day free from a panic attack (Esler et al. 2008). Panic disorder etiopathogenesis remains largely unknown, but the risk is attributed to both genetic and environmental factors (Iurato et al. 2017). Although no clear etiology has been recognized, several factors such as genetic, environmental, neurobiological, and psychopathological variables have been proposed, like other psychiatric disorders (Kim 2018).

Epigenetics represents a change in the heritable phenotypic expression of genetic records except for changes in DNA sequence (Smail 2016, Smail 2019, Smail et al. 2022).In anxiety, affective and stress-related disorders, epigenetic mechanisms, For example, DNA methylation, have been suggested to play a vital role in the intersection of genetic and environmental factors in the pathogenesis of diseases and mediation of treatment response (Ziegler et al. 2019). Epigenetics is known to play an essential role in the etiology of complex characteristics and diseases, and one of the main types of epigenetic modifications is DNA methylation (Shimada et al. 2017, Smail et al. 2022).

For anxiety-based phenotypes, e.g., the NPSR1 gene, which has been primarily associated with panic disorder, genes related to peptide and hormone signalling have been suggested (Gottschalk et al. 2016). The Krüppel-like factor 11 (KLF11; alias TGFB-Inducible Early

Growth Response Protein 2 [TIEG2]) has been highlighted by recent studies as a novel transcriptional MAOA gene expression activator (Kollert et al. 2020). IL-4 gene methylation levels were positively linked to panic and anxiety severity, and hypermethylation of the genes CSF2, CXCL8, and IL-4 significantly correlated with higher childhood panic disorder (Zou et al. 2020).

Panic disorder symptoms

Generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), and panic disorder (P.D.) often co-occur (Chantarujikapong et al. 2001). Intolerance to uncertainty (I.U.) was inferred as required in group samples for catastrophic interpretations. The current research explored the associations in a clinical sample between symptoms of I.U., AS, and panic disorder. Participants had a major panic disorder diagnosis, with or without agoraphobia (Carleton et al. 2014). The subsequent development of signs of fear, panic attacks, panic disorder, and anxiety characteristics (STAI-T). The ASI was the best predictor of panic symptom growth and panic attacks. The ASI was not predictive of the development of panic disorder after managing trait anxiety (Plehn et al. 2002). Popular characteristics of panic attacks include respiratory symptoms, including shortness of breath, coughing sensations, and lightheadedness. Patients with respiratory conditions, such as asthma and chronic obstructive pulmonary disease (COPD), show an elevated risk of developing the panic disorder (Mills and Searight 2020). During therapy and six months of follow-up, primary panic disorder symptoms and secondary depressive symptoms improved dramatically. The latent growth curve simulation of the parallel mechanism showed that the trajectory of depressive symptoms and the trajectory of panic disorder symptoms are significantly linked (Walderhaug et al. 2019).

No meta-analysis explicitly assesses the efficacy of Internet-based and mobile-based (IMI) treatments in adults with diagnosed panic disorder and agoraphobia (PD/A) to date (Domhardt et al. 2020). A disproportionate number of participants met the requirements for PTSD, P.D., and AUD. With 25% showing symptoms of all three conditions, co-occurrence was prevalent among detainees booked for violent offences. P.D. has emerged as the most potent single violence-related disorder, whereas the combination of PTSD, P.D., and AUD has dramatically increased the risk of violent offences (Barrett et al. 2020). In Latino populations, or comorbid asthma and panic disorder with treatment response, heart rate variability (HRV) and final tidal CO_2 (ETCO2) were not studied (P.D.). The current study investigated psychophysiological variables as potential mediators of treatment response, an extension of previously published studies (Nelson et al. 2020). Results indicate that patients with a short disease span may experience substantial relief in the severity of panic symptoms during the postpartum period (Aydogan et al. 2020).

Genes related to the panic disorder

A role for miRNAs in psychiatric disorders is indicated by the involvement of microRNAs (miRNAs) in neuronal differentiation and synaptic plasticity; interaction studies and functional approaches were used to assess the effect of miRNAs on panic disorder susceptibility (Muiños et al. 2011). In people of European origin, the genetic basis of anxiety disorders overlaps with that of other psychological disorders and their intermediate phenotypes (Ohi et al. 2020). Seven SNPs that were located in or adjacent to genes, including PKP1, PLEKHG1, TMEM16B, CALCOCO1, SDK2, and CLU (or APO-J), were significantly associated with P.D. (Otowa et al. 2009).

Linkage studies have so far indicated that the transmission of panic disorder phenotypes is associated with chromosomal regions 13q, 14q, 22q, 4q31-q34, and possibly 9q31. In association studies of Panic disorder, more than 350 candidate genes have been tested, but most of these findings remain contradictory, negative, or not reproduced (Olsson et al. 2004). The

variant of SLC6A4 rs140701 polymorphism may be correlated with P.D. susceptibility, and in P.D. care, 5-HTTLPR polymorphism may be a predictor of sertraline response (Zou et al. 2020). A risk factor for Panic disorder contributing to sex-specific dysfunction in women could be polymorphisms in the CNR1 gene (Peiró et al. 2020). The TERT gene can play an essential role in the pathogenesis of accelerated ageing-related Panic disorder functional disability. These results could potentially enable us to establish new ways of predicting the outcome of panic disorder and enrich the field of telomere genetic imaging research in Panic disorder (Ding et al. 2020). A risk factor for panic disorders and phobic anxiety disorders, the CRH gene affects inhibited disposition. A significant technique for understanding the genetic basis of anxiety disorders is genetic studies of anxiety-related temperaments (Smoller et al. 2005).

Higher scores on dysthymic, cyclothymic, irritable, and nervous temperaments (Schiele et al. 2020). were correlated with less active 5-HTTLPR/rs25531 S/LG alleles. Findings help classify anxiety symptom severity biomarkers to examine startle-related genetic variants (Tomasi et al. 2020). The gene CAMKMT is located in the shared panic disorder region 2p21 (Hettema et al. 2020).

Neuropeptide S (NPS) and its cognate receptor (NPSR) molecular genetic research in humans to mediate anxiety-related actions and anxiety disorders (Domschke et al. 2011). The risk of panic disorder was increased by a variant within the respective ADORA2A gene (rs5751876) (Hohoff et al. 2010). Genetic variants of several neurotransmitter system candidate genes, each with a minor individual influence, can contribute to panic disorder susceptibility (Maron et al. 2005).

"Imaging genetics" is a groundbreaking modern research approach. Imaging genetic studies are designed to assess the effect on the cerebral function of genetic variants (polymorphisms) in regions important for P.D. In addition to supporting the importance of serotonergic and noradrenergic transmission in the etiology of P.D., recent imaging genetic studies have also shown the significance of neuropeptide S receptor, CRH receptor, channel 2 (ACCN2) gene transmembrane protein (TMEM123D), and amiloride-sensitive cation (Sobanski and Wanger 2017). As evidence from numerous studies and experimental methods points to the presumptive importance of TMEM132D in phenotypes of anxiety, some research made the decision to check whether the first association's findings might be confirmed in distinct P.D. cohorts. The top related TMEM132D SNPs from Erhardt et al. were genotyped in five additional P.D. samples obtained from the Panic Disorder International Consortium (Erhardt et al. 2012). Both a strong association between LSAMP SNPs and MDD and a possible association between LSAMP SNPs and P.D. This is the first evidence of the LSAMP gene's possible contribution to human mood and anxiety disorders (Koido et al. 2012). The ultimate goal of such investigations is to outline disease pathways and transfer this knowledge to the bedside. Such an individualized, genetically-informed approach does not only involve experiments in pharmacogenomics (for example, utilizing MAO inhibitors primarily in long allele carriers) but may also adapt the psychotherapeutic approach to the patient (Reif et al. 2014). After adaptation for age and sex, the results persisted, and there was no proof that the correlation was due to population stratification. The promoter region of the gene provided no evidence of association, 5-HTTLPR, irrespective of whether evaluated as a triallelic or biallelic locus nor did any of the other four candidate genes tested (Strug et al. 2010).

DNA methylation in the panic disorder

DNA methylation patterns have been examined for a role in anxiety disorder pathogenesis, and the role of DNA methylation-DNA methyltransferases (DNMTs) enzymes has yet to be investigated (Berking et al. 2020). Twenty-four studies documenting the relationship between antidepressants and eight genes (BDNF, MAOA, SLC6A2, SLC6A4, HTR1A, HTR1B, IL6, IL11) and whole-genome methylation resulted in a systematic literature review. The predictive

of the antidepressant response was methylation of certain sites within BDNF, SLC6A4, HTR1A, HTR1B, IL11, and the entire genome (Webb et al. 2020). Methylation of SLC6A4 may therefore constitute a potential early biomarker that predicts biologically mediated clinical changes specifically caused by the exposure (Schiele et al. 2020).

SKA2 genetic and epigenetic variation may affect the chance of developing panic disorder and the severity of its symptoms (Lisoway et al. 2020). The most promising genes for diagnosing P.D. may be COMT and SLC6A4 (Tretiakov et al. 2020). Researchers are investigating the DNA methylation epigenetic process as a result of the identification of the potential biological response mechanisms. Early research in this field has demonstrated that changes in DNA methylation may underlie the responsiveness to psychiatric therapy (Roberts et al. 2019). Because of their pivotal role in the metabolism of monoamines and as pharmacological targets of potent antidepressant drugs such as tranylcypromine, phenelzine, or moclobemide, monoamine oxidases A and B (MAOA/MAOB) are prime candidates for research into the role of DNA methylation in mental disorders (Ziegler et al. 2018). In epigenetic studies using locus-specific assays, several candidate genes (e.g., BDNF; FKBP5; SLC6A4; AVP; NR3C1; CRH; COMT; MAOA; OXTR, and APOE) for psychiatric disorders have been cited (Bortoluzzi et al. 2018)

Panic disorder patients and FOXP3 hypermethylation may potentially reflect impaired thymus and immunosuppressive Treg function in female patients with panic disorder, which may partially account for the known increased morbidity and mortality of anxiety disorders, such as cancer and cardiovascular disorders (Prelog et al. 2016). DNA hypermethylation of the NET gene promoter region. The clinical concordance is perhaps underlying and epigenetic 'comorbidity' (Esler et al. 2006). Examine the broad epigenome differences in peripheral blood in patients with P.D. Interestingly, our findings point to possible changes in panic disorder in sex-specific and functional methylation (Iurato et al. 2017). HECA hypermethylation for women with panic disorder and ASB1 hypermethylation associated with symptoms of a generalized anxiety disorder (Mufford et al. 2020). Hypermethylation of the GAD1 gene encoding the glutamic acid decarboxylase1 enzyme, the enzyme that catalyzes glutamic acid decarboxylation into GABA, has been identified. DNA hypermethylation correlates with gene expression inhibition (Peedicayil 2020). Compared with AN-Rem and NED participants, hypermethylation of a number of C.G. sites was seen in AN-Active participants (Thaler et al. 2020).

Epigenetic markers such as monoamine oxidase A (MAOA) gene DNA methylation have previously been shown to be altered in disorders linked to anxiety and stress (Schiele et al. 2020). Analysis suggests that in Caucasian patients with panic disorder, DNA hypomethylation of the 5-HTT transcriptional control region, possibly through increased serotonin transporter expression and consecutively reduced serotonin availability, could impair antidepressant treatment response (Domschke et al. 2014). Data indicate a potentially compensatory function of Hypomethylation of the GAD1 gene in panic disorder that may mediate the impact of adverse life events and rely on genetic variation (Domschke et al. 2013). The study suggests that epigenetic alterations have a potentially female-specific function, i.e., Hypomethylation of the MAO-A gene in interaction with environmental factors in panic disorder pathogenesis (Domschke et al. 2012).

Recent advances now enable the complex path of P.A.s outside the laboratory to be better tested in patients' natural environment. This will provide new insights into the fundamental processes and the effects of environmental factors that can alter gene regulation by altering the methylation of DNA (Leibold and Schruers 2018). The current pilot data do not indicate that MAO-A DNA methylation has a substantial effect on the response to antidepressant therapy. However, the current trend towards CpG-specific MAO-A gene hypomethylation, likely through increased gene expression and consecutively decreased availability of serotonin and/or

norepinephrine, may be worth following up in larger pharmacogenetic studies to potentially drive impaired antidepressant treatment response in female patients (Domschke et al. 2015). Age of DNA methylation is associated more strongly with chronological age and core psychosocial, behavioral, and health factors than relative telomere length (RTL) or copy number of mitochondrial DNA. For psychosocial and neurobehavioral factors, signals were observed for associations with epigenetic aging (Vyas et al. 2019). To further examine the predictive diagnostic value of DNA methylation reliably, longitudinal studies in animal models and patients with depression are therefore required (Chen et al. 2017). The development of genome-wide techniques capable of differentiating 5-methylcytosine (5mC) from 5hmC has shown that increasing behaviors correlate with independent disturbances of 5mC and 5hmC levels, further emphasizing the special significance of each of these brain changes (Rustad et al. 2019). Human literature is in its infancy, but it indicates some epigenetic relations with behaviors and disorders of anxiety. In particular, the effects of monoaminergic systems are seen in conjunction with findings from studies into etiology and treatment. There is also evidence that epigenetic variations may be transmitted to impact subsequent generations (Nieto et al. 2016).

Chromatin modification or chromatin remodeling in the panic disorder

At the molecular level, epigenetic mechanisms control developmental processes. Recent clinical and pre-clinical evidence obtained by ourselves and others indicates that epigenetic variations are correlated with several psychiatric disorders in different regions of the brain, including those that are stress-related (Dudley et al. 2011). Chromatin structure alteration (histone protein and DNA complexes) by acetylation (typically promoting gene expression) are epigenetic changes that are thought to affect behavioral phenotypes (Akiyoshi 2012). Transcription control closely involves the contribution of processes modifying chromatin, such as histone modification and chromatin remodeling based on ATP, but their role in pathological panic disorder is not established (Wille et al. 2016).

It has recently been found that p11 is associated with traumatic stress and depression, and the expression of the p11 gene is regulated by glucocorticoids. Two glucocorticoid response elements (GREs) in the p11 promoter region interact with the ligand-activated glucocorticoid receptor (G.R.) to up-regulate the p11 gene (Zhang et al. 2011). BAHD1 is a heterochromatinization factor that has recently been identified as a multiprotein complex component associated with HDAC1/2 histone deacetylase. BAHD1's physiological and pathophysiological functions are not well described (Pourpre et al. 2020). Histone modifications have a role in the progression of panic disorder (Wang et al. 2020). CpGenome DNA modification, the ABI Prism 7700 Sequence Detection System, and chromatin immunoprecipitation (El-Sayed et al. 2012). In high-trait anxiety mice, data show fresh evidence for localized differences in certain ATP-dependent chromatin remodeling components that may potentially result in aberrant transcriptional programs that appear as pathological anxiety (Singewald 2011). In response to stressful experiences and environmental factors, histone modification and microRNA expression could improve panic disorder (Saavedra et al. 2016).

Chromosomes of panic disorder

Some genes affect the susceptibility to pleiotropic syndrome that include panic disorder, bladder disorders, extreme headaches, mitral valve prolapse, and thyroid conditions on chromosome 13q, and likely on chromosome 22 as well (Hamilton et al. 2003). Panic disorder association studies have shown regions of interest in chromosomes 1q, 2p, 2q, 3, 7, 9, 11, 12q13, 12q23, and 15 (Logue et al. 2012). Proof that chromosome 19p13.2 contain candidate genes that contribute to the risk of panic disorder growth. Besides, the effect on other mental disorders

of the associated genes can suggest shared genetic vulnerability between mental disorders (Gregersen et al. 2016).

Amiloride-sensitive cation channel 1 (ACCN1) is located as a possible candidate gene for panic disorder on chromosome 17q11.2-q12 (Domschke et al. 2011). While several chromosomal regions, including 1q, 2q, 4q, 7p, 9q, 12q, 13q, 15q, and 22q, have been involved in linkage studies of panic disorder, they have not yet been able to establish a major gene responsible for panic disorder (Jacob et al. 2010). Several chromosome regions, including 1q, 2q, 7p, 9q, 12q, 13q, 15q, and 22q, have been involved in PD linkage studies. Candidate genes, including HTR 1A, 2A, CCK, ADORA2A, MAOA, and COMT, have been investigated in association studies (Otowa et al. 2010).

Molecular diagnosis of panic disorder

GABRB3 is associated with autism, schizophrenia, panic disorder, Asperger's syndrome, and epilepsy (OMIM 137192). CHRFAM7A (OMIM 609756) and CHRNA7 (OMIM: 118511) are linked to schizophrenia, bipolar illness, attention deficit hyperactivity disorder, Alzheimer's disease, autism, epilepsy, and learning difficulties (Chen et al. 2020). The A1 and A2a adenosine receptor (A.R.) genes differ, which influences genetic predisposition to panic disorder (Deckert et al. 1998). The polymerase chain reaction method with the study of restriction fragment-length polymorphisms (PCR-RFLP) (Inada et al. 2003). has identified HTR1A, HTR2A, and HTR2C polymorphisms. Normal diagnostic models have been used to observe linkage signals (genome-wide significance) on chromosomes 10q25, 10p12, 16q24, 16p13, and 16p12 (Cheng et al. 2006).

Treatments for panic disorder

While a great deal of research has been carried out on the effectiveness of psychiatric treatment for panic disorder with or without agoraphobia, the fundamental contribution of interventions such as exposure, cognitive therapy, relaxation training, and breathing retraining has not yet been identified (Sánchez et al. 2010). When the therapist directs the patient through the exposure activities, patients with a high behavioral propensity for active and passive avoidance react better to exposure therapy (Hamm et al. 2016). For this debilitating condition in adolescents, cognitive-behavioral therapy for panic disorder in adolescence is a feasible and potentially successful intervention (Pincus et al. 2010). Differences between these patients that assess the efficacy of exposure-based cognitive-behavioral therapy and related neuroplastic changes can be demonstrated by altered protection signal processing and anterior cingulate cortex-amygdala coupling (Lueken et al. 2013). Growing evidence indicates that cAMP response element-binding protein (CREB) and brain-derived neurotrophic factor (BDNF) are correlated with alternations under escitalopram care for patients with Panic Disorder (Yang et al. 2020). The therapeutic response for panic disorder in clinical settings is significantly less effective than it is in our imaginations. Increased data suggests that panic disorder has a chronic or remitting-relapsing clinical history (Chen and Tsai 2016).

Future of panic disorder

In the current systematic review and meta-analysis, the usefulness and efficacy of cognitive-behavioral therapy (CBT) administered via the internet on the severity of panic disorder and agoraphobia symptoms were reviewed. 27 studies have been discovered (Stech et al. 2020). The pilot study suggests that attentional bias could be investigated in future research using attention bias adjustment (ABM). A larger sample will allow the mechanisms by which ABM operates to be further studied, along with possible moderating factors and the use of psychophysiological measurements in panic disorder (Baker et al. 2020). In the COVID-19 setting, many respondents reported panic and generalized anxiety. The results indicate the need

for increased panic and generalized anxiety screening by longitudinal evaluations. During this stage (and possibly later stages) of the COVID-19 pandemic, evidence-based intervention programs and supporting resources to overcome panic and generalized anxiety seem important to Bangladeshi individuals (Islam et al. 2020). Many facts that otherwise remain isolated elements without a systematic context can be explained in perspective, i.e., the association with agoraphobia, the initiation of P.D. during puberty and young adult life, the need to be accompanied, the association with air hunger and other respiratory abnormalities, the efficacy of antidepressants and the absence of Hypothalamic-Pituitary-Adrenal (HPA) activation (HPA)(Francesetti et al. 2020). Limited focus has been given to Relapse prevention in P.D. There's a lack of recent development and definitive signs. It could be productive to re-think pharmacological research in P.D. It can help to classify accurate predictive models by collecting a wide variety of clinical and individual features/biomarkers in large-scale multicenter, long-term naturalistic studies and applying for recent technological advances (e.g., electronic medical records/'big data' platforms, wearable devices, and machine learning techniques) (Caldirola et al., 2020).

Biomarkers of panic disorder

A biomarker is characterized as an indication of normal biological processes, abnormal processes, or biological responses to a therapeutic intervention that is objectively assessed and evaluated (Cosci and Mansueto 2019).

To test the serotonergic dysfunction theory, researchers have used peripheral serotonergic indicators (such as serum serotonin levels and platelet indices). Among the platelet indices, the mean platelet volume (MPV) and platelet count have received the most research, whereas platelet distribution width (PDW) and plateletcrit have received less attention. Despite the substantial research on MPV in P.D. patients, the results are contradictory and show that MPV is a subpar diagnostic indicator. MPV is a poor biochemical marker, according to Almis et alstudy, .'s which also included a small number of individuals with generalized anxiety disorder. This finding suggests the necessity to look into other diagnostic markers (Leibold et al. 2020). Despite the fact that a lot has generally been accomplished, no perfect/specific psychiatric disease marker has yet been fixed, and medical experts concur that the search for such a marker would rank among the most difficult challenges ever encountered by researchers. The most significant factors that decrease the usefulness of potential markers include (a) the fact that some markers' levels can be affected by psychiatric disorders as well as psychiatric conditions, environmental and lifestyle factors, including stress, diet, activity level, and the use of psychoactive drugs (such as alcohol), as well as comorbidities or medications (including psychotropic disorders), and (b) incorrect or incomparable study methods (technical or material-peripheral/centra) (Waszkiewicz 2020). The results of the study suggest that the proper tapetum may act as a potential neurological marker of early sexual trauma in P.D. patients, which could make them more personally vulnerable and result in worse treatment outcomes following pharmacotherapy (Kim et al. 2020). Numerous psychiatric, psychological, physiological, sociodemographic, and lifestyle variables are associated with the progression of illness. For instance, worse outcomes at 1-year, 2-year, 6-year, and 12-year follow-ups are associated with higher baseline intensity of anxiety symptoms, the presence of somatic or psychiatric comorbidity, and higher levels of disability (Bokma et al. 2020). While the specific pathomechanism of changes in the regenerative system in mental illness is not well known, our findings suggest a disruption in regenerative processes in B.D. Patients. Stem cell activity in bipolar disorder (B.D.) varies from anxiety disorders. It also varies according to the treatment of lithium salts and other medical products. This topic needs research on peripheral blood stem cells in other mental disorders, especially depressive disorders, and also on B.D. subjects during various stages of this disorder. In the search for biomarkers helpful in the differential diagnosis

phase of mental illness, this field seems to be full of hope (Reginia et al. 2020). Results showed that people with pathological anxiety display cognitive bias in the accumulation of data, which may explain why patients with anxiety overestimate risk in their everyday lives. This explanation illustrates the value of perceptual bias measures, such as improving the clarity of desirable probabilities of outcomes (Kim et al. 2020).

In patients with MDD, greater pre-treatment levels of leptin were linked to a better response to treatment for panic symptoms, whereas higher pre-treatment levels of IL-6 were linked to a worse response to treatment for panic symptoms in patients with P.D. Even in cases where the same symptoms are seen in distinct illnesses, unique predictive biomarkers must be found, according to a variety of peripheral predictive biomarkers reported in MDD and P.D. (Kim et al. 2019). The search for biomarkers to aid in the diagnosis, prognosis, and prediction of response to therapy of psychiatric diseases is a top priority of twenty-first-century medicine. The existing method of diagnosing mental health diseases, which relies on symptom explanations rather than causal biological evidence, contributes to the current lack of biomarkers in widespread use. Along with the immense advancements in genomic, epidemiological, and neuroscience studies, new ways of conceptualizing mental health conditions educate the brain pathways and neural processes that underpin behavioral frameworks that cut through existing diagnostic structures (Pratt and Hall 2018).

Conclusions

The cause of the panic disorder is still unclear, but it may be caused by genetic, environmental, neurological, and psychopathological factors. Numerous investigations have shown that several potential genes exist for panic disorder. Epigenetic processes such as DNA methylation and chromatin remodelling play a part in how panic disorder develops. Early panic disorders can be recognized using a variety of markers on various chromosomes.

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