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Review Article

Neuropsychiatric Approaches for Human Suicide

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Abstract

Some widely accepted medical knowledge suggests that suicide events or episodes are associated with a series of neuropsychiatric changes (a pattern of pathology origin, progress and therapeutic responses). We speculate herein that neuropsychiatric interference may turnover suicide rates and mortality. To find some useful evidence, cutting off unnecessary suicide-induced genes or molecules linking with disease pathogenesis will reduce suicide-induced mortality. This article discusses a framework of neuropsychiatric changes and interferences in patients.

Introduction

Early hypothesis

A great number of risk factors may trigger human suicide ideation and episode [1-3]. After a length period of psychiatric onset and pathophysiology turmoil, "patients" may seek suicide action to end their painful emotion and depressive feeling. Through this lengthy duration of psychiatric abnormality, the functional change of "patient brain" may transform unto insane condition that drive then ending life unconsciously [4-7]. This medical hypothesis can answer why human suicide emerge after a sequential and cascade of pathological pathways and networks [3].

Clinical evidence

Medical evidence suggests that suicide events or episodes are associated with a wide variety of risk factors. It is proposed that mental condition diagnosis and prevention may more or less overturn outcome and condition of suicide-induced attempts and mortality [8-10]. In order to find useful measures for curbing suicide-induced mortalities, mental-related genes or molecules linking with disease pathogenesis and therapeutic responses should be identified first. Only by this pathway, suicide prediction and prevention can be possible.

Neuropsychiatric Field

Neuropsychiatric behaviors

It is easy to note that outside force may lead to human suicide. But, we disparately need to know why someone chose suicide while most others stay calm. This personal variation may be identified by clinical clue and association of interpersonal variation of biochemistry and neuropsychiatry. Whether therapeutic responses can be promoted by pharmaceutical and neuropsychiatric approaches [7-11]? Neuropsychiatric approaches may provide biomedical insights between human suicide risks, mental disorder co-morbidity and therapeutic selection (schizophrenia and mood disorders in particular).

Knowledge emerges

Symptom similarity and morphological overlapping between suicide ideation and mental disorders—identical signs of depressive or manic, brain images, biochemical parameters and instrumental data should be carefully investigated for knowledge progress. As a result, several dimensions of "mental diseases" have been estimated for possibility of suicide risks and mortality by scientists, clinicians and psychiatrists.

Until now, scientists do not well confirm for suicide origins (framework of psychiatric syndrome and neuropathy in image data and biochemical assays) by existing techniques. Human suicide events and mental illness dimensions are comparable for biochemical parameters and environmental variables. Different evaluative systems should be given for high-quality suicide diagnostics and therapeutic selection (Table 1). Biomedical diagnostic paradigms and novel ideas should be promoted in the future.

Table 1: Diagnostic translation from symptoms to biochemical parameters.

Psychiatric Symptoms	Syndromes	Biochemical Parameters
Low mood	Somatic complains	Genomic changes
Diurnal mood variation	Psychiatric abnormal	Genotypic abnormal
Insomnia and early awaking	Social problems	Phenotypic difference
Lack of interests & energy	Genetic alteration	Brain image
Poor concentration	Epigenetic abnormal	Neural circuitry
Weight loss	RNA change	Cerebral density

Human suicides were previously managed by chemical drugs, mainly antidepressants and other anti-psychiatric agents. However, these drug therapies like a double-edged sword that has both strengths and weaknesses [11-19]. Only part of depressive patients is effective with chemical drugs. Some of depressive patients even commit suicide. To many depressive persons, current antidepressants and drug selective systems are useless [11]. As a result, pharmaceutical companies begin to seek new types of antidepressants. In order to achieve higher therapeutic responses, therapeutic mechanisms and new medication should be explored.

From psychiatric symptoms to biological parameters

Before high-quality therapeutic drug development, biomedical diagnostic paradigms should be pursuit for benefiting therapeutic responses and selection. Previously, suicide/mental illness diagnostics came from patient's psychiatric symptom and behaviors [6,7]. These psychiatric symptoms can only be diagnosed by well-trained psychiatrists. These symptoms (depression and mania) have been classified into different disease dimensions. Mental health diagnosis and confirmation is based on patient's symptom checklist. Transition of symptom diagnosis into biomedical parameter prediction is highly needed [3,4].

Formally, worldwide diagnostic guidelines have been established and widely applied. Detail diagnostic information can be found in Diagnostic and Statistical Manual of Mental Disorder—from DSM-I to DSM-V of mental problems and Hamilton Depression Rating Scale (HAM-D) of suicide risks. From DSM-1 to DSM-5, it contains more diagnostic symptoms and biochemical parameters. Accordingly, these two diagnostic systems shared same clinical symptoms and molecular translation (Table 1).

To find and identify interaction and association between psychiatric symptoms, biological parameters (genetic or molecular variation), synaptic activity (hormone or transmitter levels) and neuropathy (structure and function) play key roles of neurobiology in patients at suicide risks and normal people in Table 1. By doing this etiologic/pathologic evaluation, clinical evidence can be gradually transformed into well-established therapeutic guidelines in brain structural and functional output and connection.

Etiopathologic Origin

Psychiatric characters for mental disorders and suicide

Genetic-induced pathogenesis for suicide behaviors might come from genomic transition by consistent outside attack and forces. From psychiatric scope, the depressive or mania symptoms are reviewed from negative symptoms, parameters and domains, such as hopeless feeling, self-deny and mania. Since pathogenesis evidence has not been well confirmed in suicide risk persons, genetic elements and variations (inheritable, mutation, relocation, copy number and epigenetic) may be characterized from features of suicide risks, events or drug toxicity. Human neuropathy evidence may profoundly impact for human suicide study [8-10].

Neuropathy evidences

To evaluate human suicide in neuropathy (nature, pathways and network), new neurobiology knowledge (structure, function and chemistry) for suicide should be accumulated [18-20]. The mystery and complex processes of neuropathology have been evidenced in brain image and molecular analysis (genetics, biomarkers and cellular) in the past reports [21-28]. Many patterns of neural structural and functional elements have been investigated. This neuropathy characterized as brain lesion, density, structure and image may be as informative as psychiatric symptom analysis embarked 100 years ago.

Techniques for modern diagnosis

Knowledge breakthrough of suicide prediction and therapy is supported by different technology. Like many other biomedical disciplines, hospital equipment and technical supports play key roles for suicide prediction and therapeutic decision-making. Previously, an association between the severity of drug side-effects (suicidal incidence in juvenile) and drug responses (patients' depressive symptom alleviations) [11-18] had drawn unprecedented attention and technology progresses for repeated suicide episodes, self-harm or self-injures. Comparison and pharmacological study of similarity and diversity of drug toxicity, metabolism and responses should be

strengthened—including useful patents, diagnostic paradigms and modern techniques (Table 2). By upholding these efforts, advanced suicide predictive and treatment systems from patients' symptoms (depressive, cognitive and behavior) into biochemical parameters (blood, saliva fluids, urea and image) might be applicable in general hospitals.

Table 2: Genetic knowledge for psychiatric diseases.

Techniques	Discovery and Outcomes
GWAS	**PROVED** Schizophrenia Bipolar Autism spectrum disorders (ASD) **UNDER-INVESTIGATION** Major depressive disorder (MDD); Attention deficit hyperactivity disorder (ADHD); Obsessive-compulsive disorder (OCD); Post-traumatic stress disorder (PTSD); Tourette disorder
CNV	Schizophrenia Intellectual disability; Autism spectrum disorders (ASD) Specific language impairment Reduced cognitive ability
Whole-exome sequencing	Autism spectrum disorders (ASD) Cognitive function
SNP	**CO-MORBIDITY** MDD-bipolar disorders MDD-Schizophrenia MDD-ASD-ADHD

Mental disease treatment

Generally speaking, it is widely known that a treatment of any mental illness is not easy. In many clinical occasions, mental disorders are complex and mystery in disease onset and progress. They are difficult to be quickly diagnosed and high-quality symptom alleviation by drug management. Regarding a possible molecular-based diagnosis of depression or suicide entering into the hospitals, many environmental factors could be ruled out in therapeutic decision-making. Cerebral morphological change [21-26] and therapeutic decision-making by pharmacogenomics (PG) has been increasingly noticed [14,15]. To stratify pathology and clinical data regardless of environmental forces and variables, more clinical biomedical diagnosis should be statistically analyzed and applicable in general hospitals.

Genome-wide association study (GWAS)

Understanding the suicide-associated relationship at chemical, genetic, molecular, neural, environmental and therapeutic levels is the top priority in biomedical science [17]. Moreover, wide-range genomic information can be calculated from data of GWAS (genome wide association study) and other modern techniques (omics and metabolomics) [27,28]. Genomic information between normal and genetic vulnerable humans has to be scaled up globally in the future [8-10]. But less than half of these alleles are statistically significance by past large-scale genetic exploration. Governmental policy should be adjusted for supporting more genetic allele identification and statistics in patients with high suicide risks. GWAS has provided translational strategies for clinical data collection and disease hypothesis. Only human genetic data of neural dysfunction and family diseases in larger sample sizes (>100,000 human subjects) can obtain new psychiatric knowledge and diagnostic paradigms.

Neurosciences

Brain anatomy and circuitry

Different psychiatric symptoms, synaptic connectivity, neural circuitry and brain function continue to decipher; different cerebral locations, entities and domains are vulnerable to dysfunctional of mental cognitive, emotion and behaviors; Regional lesion or

destruct of brain circuitry may lead to abnormal mental and psychiatric symptoms, disease manifestations and human suicide.

Dopamine associated areas: ventral striatum (VS), amygdale, orbitofrontal cortex (OfC) and anterior cingulated (ACC)

Serotonin associated areas: amygdala, lateral OfC, insula and hypothalamus; [29] Cognitive disability, emotional processing problems and harmful behavior are either separated or overlapped. Further neurobiology exploration may clarify their relation, interaction, separation and different mechanisms step by step.

Brain biology

Brain genotypic and phenotypic alteration may associate with the occurrence of different dimensions of mental disorders [30-32]. This hypothesis was supported by clinical evidence of different psychiatric symptoms (mood disorders; opposite extremes of emotional symptoms and biomedical profiles—anxiety and depression), schizophrenia (high-level of dopamine) and neurodegenerative disorder (low level of dopamine; cognitive impair, such as Alzheimer and Parkinson's diseases). This brain biology evidence is very useful for patient's diagnosis and treatment.

Regional images (location or density in cerebral cortical and sub-cortical areas) have been associated with different mental illnesses respectively in the clinic. Correspondingly, these kinds of cerebral information can be used for varying molecular diagnosis in the future. Different modern techniques, instruments and tools are growing popular in both experimental and clinical study. Fronto-cinguls-striatal network is most interesting for revealing the underlying mechanisms of suicidal, social processing capability and other neurobiological behaviors in the past reports. More evidence should be accumulated and identified in the future.

Modern diagnosis

Since human suicide/mental illness are widely known as brain diseases, it suggests that brain image changes of both cerebral volume and regional lesion (prefrontal or cingulated cortex and so on) are useful ways for promoting imaging techniques, such as position emission tomography (PET), single-photon emission computed tomography (SPECT), electroencephalogram or functional magnetic resonance imaging (fMRI) [21-26]. The changes in cortical and sub-cortical areas have been especially relevant for suicide diagnosis in the future.

Divergent techniques of neural imaging are taken together to identify cerebrally morphological changes and guide therapeutic targets and selections consequently. To overcome false-positive or negative diagnostic data, software or psychiatric knowledge breakthrough is still an important pathway for high-quality diagnosis in the clinic.

Neural transmitters

Among present knowledge about signal pathway modulators (activators and inhibitors) in neurobiology, such as neural transmitter, its receptors, signal cascade processes, breakup-enzymes and signal transduction are especially important [29]. These fields of neurotransmitter knowledge are therapeutic paradigms and more straightforward than those of psychoanalysis for patients.

Dopamine network: dopamine network is associated with human reward machinery (cognitive); Decreasing of dopamine levels may lead to neurodegenerative diseases and increasing dopamine production may lead to schizophrenia symptoms, pathological gambling and hypersexual activity [29].

Serotonin network: serotonin network and pathway is associated with emotional activity and symptoms (anxiety—aversion); some emotional changes may greatly correlate with human suicide and mortality [29].

Normal human brains are soft and low density from outside observations. However, some aberrant macro-materials, such as Tau and β-amyloid are commonly observed in patients with neural-degenerating diseases (Alzheimer and Parkinson's diseases). In these neurodegenerative diseases, molecular dysfunctions really matter for disease progress and straightforward from brain image and circuitry observation.

Discussion

Psychiatry and suicide

The earliest reports for human suicide did not use the term of "suicide"'. It commonly describes clinically symptoms—melancholy (current language as depression) or other clinical term "mania". It is not until 17-th century (AC 1642) that word "suicide" was formally named [2].

Two extremes of psychiatric symptoms (depression and mania) are deeply rooted in suicide ideation and behavior. It needs advanced knowledge to categorization and integration of suicide, psychiatry and neurobiology in deeper insights.

Knowledge novelty

The relationship between chemical, genetic, molecular, cellular, environmental, social factors and therapeutic responses should be clarified in the future. The knowledge gaps between suicide and psychiatry should be filled soon. We look forward to a great integration and impacts under the same roof (kill several birds by one stone). Presently, many unresolved questions of why no unequivocally answers can be revealed between normal and suicide-risk humans. Environmental forces or genetic variation can be integrated into modern diagnostic parameters and therapeutic novelty for suicide prediction and prevention. A whole-some comparison between suicide and mental problems by modern technology will be highlighted. Some unexpected scientific discovery and technical advances may bring us into new horizons. As a result of all, these neuropsychiatric efforts cannot be in vain for clinical trials.

Future Direction

Major breakthroughs

1. Suicide risk prediction and diagnosis should be more straightforward (molecular based)

2. Neuropsychiatric knowledge about signal pathways, such as neural transmitter receptors, signal cascade and neurotransmitter activators/inhibitors should be especially emphasized because some well-established paradigms are based on that.

3. Mathematical, algorithmic, computational network and artificial intelligence for suicide should be emphasized (equation, modality and large databases of genetic, bioinformatics or brain image data available)

4. New animal models, state-of-the-art techniques and avant-garde lab instruments, such as gene knockout, optogenetics, genomic editing or genetic engineering mice should be used in drug developments, licensing and clinical utilities

5. Seeking genetic or bioinformatics variations from genomic data beyond protein-encoding sequencing regions (many repeat DNA in human genomes) [33]

Genetics-environmental interplay

How to accumulate advanced knowledge about suicide etiologic and pathologic needs a sequence of multi-disciplinary study of neuropsychiatry. Modern diagnosis should be based a sequence from genetic to bioinformatics to visual data or vice versa. Presently, we do not know which pathway is quicker and more effective. Yet, further medical exploration on suicide neurobiology should be great fruitful [34-38].

Conclusion

In summary, the clinical suicidal behaviors and events should not be confined in parameters of suicide episode and behaviors. Chemical structures of drugs, genetic databases in patients and environmental variables should be overall carried out. More cutting-edge technology, high-quality statistical analysis and artificial intelligence for larger number of patients should be integrated by different systems of modern diagnostics.

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Conflict of Interest

Author declare no conflict of interest.

References

1. Kapur N, Gask L (2009) Introduction to suicide and self-harm. Psychiatry 8(7): 233-236.
2. Shandilya S (2018) Suicide and suicide prevention: a historical review. The Research Journal of Social Science 9 (12): 35-40.
3. Lu DY (2017) Suicide Risks and Treatments, New Ideas and Future Perspectives. In: Ed Da-Yong Lu Nova Science Publishers New York, USA.
4. Lu DY, Wu HY, Cao S, Che JY (2020) Historical analysis of suicide. J Translational Genetics & Genomics 4: 33.
5. Marneros A (2009) Mood disorders: epidemiology and natural history. Psychiatry 5(4): 119-122.
6. McAllister-Williams R, Ferrier IN (2009) Pharmacological management of bipolar affective disorder. Psychiatry 5(6): 195-198.
7. McAllister-Williams R, Ferrier IN (2009) Pharmacological management of unipolar affective disorder. Psychiatry 5(6) 189-194.
8. Malhotra D, Sebat J (2012) CNVs: harbingers of a rare variant revolution in psychiatric genetics. Cell 148(6): 1223-1241.
9. Krystal JH, State MW (2014) Psychiatric disorders: diagnosis to therapy. Cell 157(1): 201-214.
10. Lu DY, Che JY, Wu HY, Lu TR, Putta S (2020) Suicide risks and prevention, neuropathogenic study. Psychiatry 4(1): 124.
11. Marshall M (2020) Roots of mental illness. Nature 581: 19-21.
12. Brent D, Melhem N, Turecki G (2010) Pharmacogenomics of suicidal events. Pharmacogenomics 11(6): 793-807.
13. Lu DY, Lu TR, Zhu PP (2012) How can we pinpoint genetic involvement in antidepressant-induced suicide? Adv Pharmacoepidemiol Drug Safety 1(1): e101.
14. Lu DY, Lu TR, Zhu PP (2012) Genetics in neural toxicities of drugs. Cent Nerv Syst Agent Med Chem 12(4): 250-253.
15. Lu DY, Lu TR, Zhu PP (2013) Pharmacogenetics in neural toxicities of drugs. Pharmacogenomics 14(10): 1129-1131.
16. Lu DY, Lu TR, Che JY, Zhu PP (2014) Genetics and bioinformatics studies of antidepressant drug therapeutic efficacies and toxicities, a current overview. Recent Pat CNS Drug Discov 9(3): 193-199.
17. Lu DY, Lu TR, Zhu PP (2010) Undesired neural side-effects of a drug, a chemical and genetic interrelated problem. Cent Nerv Syst Agents Med Chem 10 (2): 108-112.
18. Zhao Y, Xiong N, Liu Y, Zhou YH, Li NM, et al. (2013) Human dopamine transporter gene: differential regulation of 18-kb haplotypes. Pharmacogenomics 14(12): 1481-1494.
19. Youngtrom IA, Strowbridge BW (2012) Visual landmarks facilitate rodent, spatial navigation in virtual reality environment. Learn Mem 19(3): 84-90.
20. Read J, Runciman O, Dillon J (2016) In search of an evidence-based role for psychiatry. Future Sci OA 2(1): FS0101.
21. Frangou S (2009) Brain structural changes in mood disorders. Psychiatry 8(3): 105-106.
22. Roiser JP, Rubinsztein JS, Sahakian BJ (2009) Neuropsychology of affective disorders. Frangou S. Brain structural changes in mood disorders. Psychiatry 8(3): 91-96.
23. Desmyter S, Bijttelier S, Heeringen KV (2013) The role of neuroimaging in our understanding of the suicidal brain. CNS & Neurol Disord Drug Targets 12(7): 921-929.
24. Frangou S (2008) Functional neuroimaging in mood disorder. Psychiatry 8(3): 102-104.
25. Jiang JJ, Yan ZZ, Sheng C, Wang M, Guan QL, et al. (2019) A novel detection tool for mild cognitive impairment patients based on eye movement and electroencephalogram. J Alzheimers Dis 72(2): 389-399.
26. Miller G (2016) NEUROSCIENCE. Brain scans are prone to false positives, study says. Science 353(6296): 208-209.
27. Menke A, Samann P, Kloiber S, Czamara D, Lucae S, et al. (2012) Polymorphisms within the metabotropic glutamate receptor 1 gene are associated with depression phenotypes. Psychneuroendocrinology 37(4): 565-575.
28. Menke A, Domschke K, Czamara D, Klengel T, Hennings J, et al. (2012) Genome-wide association study of antidepressant treatment-emergent suicidal ideation. Neuropsychopharmacology 37(3): 797-807.
29. Ramsay TZ, Skov M (2010) How genes make up your mind: Individual biological differences and value-based decisions. J Economic Psychology 31(5): 818-831.

30. Lu DY, Zhu PP, Wu HY, Yarla NS, Zhu H, et al. (2016) Human suicide study, is there an associations between suicide and mental illness? Metabolomics 6(3): 186.

31. Lu DY, Zhu PP, Wu HY, Yarla NS, Xu B, et al. (2018) Human suicide risk and treatment study. Cent Nerv Syst Agents Med Chem. 18 (3): 206-212.

32. Na EJ, Lee H, Myung W, Fava M, Mischowlon D, et al. (2019) Risks of completed suicide of community individuals with ICD-10 disorders across age groups: A nationwide population-based nested case-control study in South Korea. Psychiatry Investig 16(4): 314-324.

33. Pennisi E (2010) Shining a light on the genome's 'dark matter'. Science 330(6011): 1614.

34. Freedman DH (2019) Hunting for new drugs with AI. Nature 576(7787): S49-S53.

35. Lu DY, Shen Y, Cao S (2020) High quality treatments for human suicidal events and mortality. Advanced Techniques in Biology & Medicine 8(2): 269.

36. Vanneste S, Ridder DD (2021) Chronic pain as a brain imbalance between pain input and pain suppression. Brain Communication.

37. Mann JJ, Michel CA, Auerbach RP (2020) Improving suicide prevention through evidence-based strategies: A systematic review. Am J Psychiatry 178(7): 611-624.

38. Orsolini L, Latini R, Pompili M, Serafini G, Volpe U, et al. (2020) Understanding the complex of suicide in depression: from research to clinics. Psychiatry Investig 17(3): 207-221.