We recently woke up to the death of Robin Williams, the famous actor of “the hope in hopelessness”. He was 63. He died of suicide. The energetic, idealistic teacher of the “dead poets society” who reminded us to “seize the day” was gone. Evidently, he could not see the way out just like his talented student in the movie did not. Why? The biter smile he carried on his face in almost every movie that I remember makes me wonder if it was nothing but the reflection of his own fragile spirit.

Robin Williams has long suspected to be suffering from either depression or bipolar disorder. Apparently he was experiencing a depressive episode which led him to commit suicide (1). Williams had an addiction to cocaine and alcohol during late 1970s and early 1980s. He was successful in his battle against cocaine but it had not been the case with his alcohol problem. After being sober for a while, he restarted drinking in 2003 and checked himself in to a substance-abuse rehabilitation center in 2006 with the declaration that he was an alcoholic (2). He was readmitted to another rehabilitation center for alcohol addiction two months before his death. Williams was diagnosed with early stage Parkinson’s disease a year ago but was not yet ready to share it publicly. His publicist comments that he was severely depressed prior to his death (3). This was not the first time that Williams thought about suicide. During an interview about his struggle with addiction in 2006 he talked about the similar impulsive quality of committing suicide and drinking: “you are standing at a precipice and you look down, there’s a voice and it’s a little quiet voice that goes, ’Jump’, ‘The same voice that goes, ’Just one’. And the idea of ’just one’ for someone who has no tolerance for it, that’s not the possibility’. More recently, Williams was so overwhelmed with fear and anxiety that he sought solace in alcohol (4).

The list of facts about Williams’ death constitutes the table of well known facts associated with suicidal behavior. Being severely depressed with co-morbid alcohol misuse sets the stage for exploring the relationship between suicide, mood disorders and impulsivity from a bio-psycho-social perspective. The mythical association between creativity and mood disorders as well as impulsive/addictive behavior deserves additional attention.

A more recent study from the same group with the inclusion of higher number of patients (n=1,172,763) revealed no connection with professions requiring creativity and major psychiatric disorders except for bipolar disorder (11).

Suicide: The Shark Of Psychiatry

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Structural and Functional Alterations in the Suicidal Brain

Magnetic Resonance Imaging studies reveal a high rate of deep and periventricular white matter hyperintensities and grey matter hyperintensities in the frontal, temporal and/or parietal lobes of individuals with a history of suicide attempt. Decreased volumes in the frontal and temporal lobe and reduced grey matter volume in the frontal lobe are among other findings from structural imaging studies on suicide. The grey matter hyperintensities are thought to be representing disruptive communication in critical neuroanatomic pathways, leading to a higher risk of suicide attempts. In support of this hypothesis is the enlarged amygdala volumes and smaller right and left orbitofrontal cortex grey matter volumes in suicide attempters. Such anatomical changes may be associated with impaired decision making and predisposition to impulsive behavior and suicide. Reduced prefrontal perfusion and metabolism in the brains of individuals with past suicide attempt may indicate a prefrontal dysfunctioning which is related to reduction in attention and executive functions as well as problem solving abilities and a higher potential to respond to negative emotions in these individuals. Such reduced frontal perfusion may also lead to absence of prospective anticipation to problems, resulting in hopelessness. Together with a dysfunction in the orbitofrontal cortex which leads to response inhibition deficit and impulsivity, such emotional and executive dysregulation may cause further risk for suicidal behavior (17).

Neurotransmitter Systems and Suicidal Behavior

Neurochemical properties of suicidal behavior involve mainly the serotonergic system. Decreased CSF 5-HIAA levels and increased platelet 5HT2A receptors may be considered as one of the important trait-related markers in suicide (7). Some neuroanatomical studies have suggested a link between serotonin receptor up- or down-regulation in depression and suicide (15). Reduced raphe nuclei echogenicity in patients with suicidal ideation, a negative correlation between impulsivity and 5-HT2A binding, reduced 5-HT2A binding in the frontal cortex in patients with a recent suicide attempt, are signs of the serotonergic deficiency in suicidal behavior (17). Noradrenergic system seems to be less affected in suicidal behavior. Despite equivocal data, there seems to be an increase in α2-adrenergic receptors supporting the monoaminergic reduction theory of depression and suicide. Additionally, significant reduction in dopamine transport coupled with an increase in D2/D3 receptors in the amygdala of patients with major depression supports the idea that regional changes in dopaminergic transmission may be involved in mood disorders and in suicide. Given the important role of Glutamate in the neuronal death and the non-specificity of any alteration in the GABA/Glutamate activity in various psychiatric disorders, further investigation is needed to explore subtle differences in the glutamate/ GABA system in suicide, beyond analysis of up/down-regulation. Up to date data on the involvement of cholinergic system is scarce and the opioid system has not been fully addressed in the brain of suicide (15).

Cell Signaling

Intra and extra-cellular signalling elements function in modulation of the cascade which regulates generation of nuclear gene products. Among those intracellular signalling components, PKC activity and related mRNA expression levels were found to be decreased in the prefrontal cortex and hippocampus of teenage suicide victims compared to that of healthy controls. A decrease in cAMP binding and PKA activity was also observed in both adult and teenage suicide victims (7). Brain derived neurotrophic factor (BDNF) and fibroblast growth factor (FGF), the two components of the extracellular signalling system, or their respective receptors have been shown to be involved in suicide or major depression (15). Beyond Neurochemistry and Cell Signalling

Further investigations on pathophysiology of suicide show evidence for a strong association between HPA axis dysfunction and suicide. DST non-suppression was found to be significantly related to committing and completing suicide compared to DST suppression. Some direct and indirect evidence suggests a relationship between suicide and immune dysregulation (7).

Final Statements

Suicidal behavior is a complex trait with an underlying and distinct genetic liability and related neurobiological and psycho-social components. Conceptualization of suicide within the “poetic romanticism” of the triangle of impulsivity, creativity and mood disorders is an underestimation of a preventable behavior. Coupled with its deadly consequences, and strong association with major psychiatric disorders, mainly mood disorders, suicidal behavior requires special attention in terms of research, detection, prevention and treatment of the underlying causes from a bio-psycho-social perspective.

References

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