Suicidal Behavior And Physiological Changes

Suicidal behavior is one of the most serious and life threatening symptoms of major depressive disorder. It is estimated that, worldwide, approximately 800,000 people die due to suicide every year (WHO, 2020). In the case of Ms. Wilkins, her suicidal behavior was specific to suicidal ideation and planning, including the thought process that if she were dead, everything would be better and she would no longer suffer.

Serotonin is a neurotransmitter frequently associated with suicidal behavior. In a study conducted by Mann (2003), it was concluded that there were abnormalities in the serotonin system within the prefrontal cortex region of patients who died by suicide. In this population, fewer presynaptic serotonin transporter sites were found in the prefrontal cortex, occipital cortex, hypothalamus and the brainstem. Within the prefrontal cortex, abnormalities were found specifically within the ventromedial prefrontal cortex, a part of the prefrontal cortex implicated in behavioral and cognitive inhibition, processing risk and fear, regulating the amygdala, inhibiting emotional responses and processing decision making and self-control. This definition clarifies how an abnormality in this region may lead an individual to suicidal behavior, where there will typically be disinhibition and a greater chance of acting on suicidal or aggressive feelings, as a consequence of lower serotonergic input (Mann, 2003).

An increased activity within the hypothalamic-pituitary-adrenal (HPA) axis has also been associated with suicidal behavior. The HPA axis stimulates the production of cortisol and is closely involved with our stress responses. Studies correlating the HPA axis and suicidal behavior have found either a hyperactivity or an abnormality in this stress response system, indicating that patients who present with suicidal behavior might have a biological stress response that reflects risk for suicide, including increased adrenal glands and less prefrontal cortical binding. Patients diagnosed with major depressive disorder have shown hypercortisolaemia (increased cortisol levels) and impaired suppression of cortisol secretion after administration of dexamethasone, a drug that suppresses HPA activity. Incorporating stress relief or reduction of stress effects as psychotherapy goals, may lead to increased efficacy of therapeutic interventions (Mann, 2003; Heeringen, 2003).

Interestingly, Mann (2003) explores the correlation between genes, environment and stress response, indicating that there appears to be a significant interaction between genes, one’s environment and the function of both the serotonergic and noradrenergic systems. A study comparing peer-reared monkeys to maternally raised monkeys found a lower serotonergic activity in the first group. This lowered serotonergic activity remained into adulthood and resulted in greater demonstrations of aggression and impulsivity. Consequently, it could be hypothesized that an adverse upbringing or environment may cause serotonergic function to occur at a lower level, an effect that could continue into adulthood and contribute to a higher risk for suicidal behavior.

Studies have also explored microglia density in the dorsolateral prefrontal cortex, anterior cingulate cortex, mediodorsal thalamus and hippocampus of depressed patients. These studies suggest that significant microgliosis, or the increased number of microglia, in patients presenting with depression and who committed suicide, when compared to healthy controls, could be

regarded as a marker of suicidal behavior. The substances released from the microglia may have altered serotonin receptors, therefore contributing to suicidality (Maletic & Raison, 2009).

Finally, degenerative alteration in the brain's white matter, or the nerve tissue that contains mostly myelinated fibers, has been reported by multiple studies to be associated with suicidal behavior (Gianluca Serafini, et al., 2012).