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BIOLOGICAL LANDMARKS IN UNDERSTANDING SUICIDAL BEHAVIOUR

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Abstract:

Background: Suicidal behaviour has had, over time, different explanatory meanings. In ancient and feudal times, it took philosophical and religious meanings. Upon entry into modern times appeared explanatory sociological, medical and psychological meanings. Among the attempts to explain suicidal behaviour, especially in the last half century, there are also the biological landmarks. **Objective:** Synthesizing the biological research results in a small explanatory set. **Material and methods:** Data is collected from literature. **Results and conclusions:** On the basis of suicidal behaviour, there are nonspecific genetic vulnerabilities, found also in specific mental illnesses that favour, at a different level, the developing of nonspecific neurotransmitter vulnerabilities which, in turn, favour not the suicidal decision, but the progress of this behaviour.

Key-words: suicidal behaviour, biological landmarks, explanatory models

Rezumat:

Premise: De-a lungul timpului, comportamentul suicidar a avut diverse semnificații explicative. În vremuri antice și feudale, a avut explicații filosofice și religioase. La intrarea în modernitate, au apărut semnificațiile explicative sociologice, medicale și psihologice. Între încercările de explicare a comportamentului suicidar, mai ales din ultima jumătate de secol, intră și reperele biologice. **Obiectiv:** Sintetizarea, într-un mic corp explicativ, a rezultatelor cercetărilor biologice. **Material și metodă:** Datele sunt culese din literatura de specialitate. **Rezultate și concluzii:** În bazele comportamentului suicidar, se găsesc vulnerabilități genetice nespecifice, găsite și în boli psihice specifice, care favorizează, la alt nivel, apariția unor vulnerabilități de neurotransmisie, la fel, nespecifice, dar care, la rândul lor, nu favorizează decizia suicidară, ci desfășurarea comportamentului de tip suicidar.

Cuvinte-cheie: comportament suicidar, repere biologice, modele explicative

INTRODUCTION

Over time, depending of culture and the dominant paradigm of the moment, suicidal behaviour meaning varied between extremes.

Thereby, for Socrates, a suicidal himself, this behaviour represented „rational control of death”, demonstrating it in 39 B.C. The Stoics gave a similar meaning to suicide. For Judas the Iscariot (33 A.C.) suicide meant self-punishment. Christianity defined it as the supreme sin. So, in ancient times and in the European Middle Ages, suicide had a philosophical and religious meaning. In 1644, Donne and then Huwe (1783) finally proved that suicide is a disease and not a sin.

But only in 1790, Moore, saying that there is “madness in every suicide”, opens the medical view of suicidal behaviour (1). In the same illuminist era, an assertion of the social significance of suicide comes out, Peuchet (1838) refers to suicide as “a deficient organization of our society”. Both paradigms: medical and social will develop laying the foundations of modern suicidology. In 1821, Esquirol and in 1828, Burrow state the relationship between suicide and mental illness, in 1879 Morselli proves the “relation of madness with suicide” which clearly states initially the paradigm and then the medical dimension of the suicidal behaviour. The social paradigm will finally impose Durkheim (2) after the foundations were expanded by Sundt, Lisle, Boismont and others (1).

Philosophers continue to approach suicide from their point of view starting with Kierkegaard, then Nietzsche, continuing with Heidegger and up to this day (Cioran) they state that “suicide is the expression of freedom and responsibility”. Then comes Freud who, by his immense prestige, focusing on guilt, humiliation, loss, revenge, depression..., at the beginning of the twentieth century lays the foundation for the psychological current (paradigm). So, throughout time, the dominant paradigm was either religious, philosophical, medical, social, a combination of the forementioned, or psychological. In fact, human behaviour has all these dimensions. The difficulty is to articulate the synthetically.

Today (3) suicide is not regarded as an illness but as a behavioural act, having in its deterministic roots cognitive, affective and communicational functions that are either normal or altered by disease. In addition, it is accepted that suicide is a deliberate act, but suicidal behaviour finalized in a suicidal act is based on psychological unconscious mechanisms as well as inherited/earned vulnerabilities or pending pathological processes. The progression to suicide can be acute, chronic or latent (*ibidem*).

OBJECTIVE

Summarized synthesis of the most experimentally based biological landmarks in a small explanatory set of the suicidal behaviour model.

MATERIAL AND METHODS

Data is collected from the suicide literature in its diversity focusing on the most notorious names in the field.

Types of explanatory models

Only until 1980, there have been over 31,000 papers on suicide published (4). Of these only about a third (9,951 papers) were published in psychiatric journals. The remaining number of papers were distributed to some 16 fields of research. It is impossible for someone to consult them all, therefore, in addition to up to date papers we resort to the most notorious and the newest monographies that summarize knowledge acquired in a particular area of interest.

One knows today that between risk factors (individual, domestic, social) and explanatory models there is a difference of species. Risk factors are just the “fuel for the decision making engine”, and the explanatory models try to explain the vulnerabilities that are part of the foundation of mechanisms and the mechanisms that lead to the decision of the behavioural action.

As an expression of the human being, researching this behaviour targets all four levels of a human being: the biological, the onto-psycho-genetic, the socio-cultural as well as the spiritual level (5). At the biological level we have researches and genetic and neurotransmitter explanatory models.

THE GENETIC EXPLANATORY MODEL

Genetic studies refer to molecular genetic studies, family studies and studies on twins, comparing suicidal concordance in monozygotic and dizygotic twins (6). Although studies are not always comparable because of different methodologies and difficulties in creating large batches, Kalman (7) reports that one, possibly three out of 18 pairs of monozygotic suicidal twins are concordant in opposition to none out of the 21 pairs of dizygotic twins. In terms of suicidal behaviour, Kringler (8) found that dizygotic twins of the same sex have a higher concordance in comparison to dizygotic twins of opposite sexes. The most notorious is the study of Danish authors Juel-Nielsen and Videsech (9). By comparing all twins born over a period of 50 years (1870 – 1920) in Denmark (19,484) the authors found 19 pairs of monozygotic suicidal twins of which 4 (3B and 1F) were concordant, and 58 pairs of dizygotic twins out of which no pair was concordant. Statham (10) states that monozygotic twins have the same social character more frequently than dizygotic twins and so it is marked as a behavioural pattern more evenly. He also states that the concordance of monozygotic suicidal twins is supported by mental illness diatheses, so we are talking about non-specific vulnerabilities. Wasserman (3) still states that genetic inheritance is important, but does not mean predestination. In some, but not all, not even in most cases an innate vulnerability is present which, however, is nonspecific, only favouring the developing of thinking, and finally suicidal decision mechanisms. Without the contribution of the other levels of a human being, the genetic level does not mean but a vulnerability with a probability to manifest itself depending on other factors.

THE NEURO-PSYCHO-BIOLOGICAL MODEL

Classical psychiatry observations (11) have shown that any brain injury: traumatic, infectious, vascular... determines from minor to major behavioural changes, sometimes leading to unrecognizable cases. These records imposed the idea that not only structural inframicroscopical but also functional neurosomatic changes could cause behavioural changes, including the suicidal type. Starting from the idea that suicide is also a frequent complication of mental illness, neurotransmitters (12, 13) that had become the foundation of biological models to explain mental illness were taken in turn. Consequently, a significant relationship between suicide in major depression and decreased serotonin function in various regions of the brain (12, 13) was found. Also a decrease in serotonin transporters in platelets, a decreased level of 5-Hydroxyindoleacetic acid (5-HIAA) in CSF, a weak prolactin response to oral fenfluramine or L-tryptophan intravenous were found. This low level is also found in balanced phases between episodes in depression. The effectiveness of antidepressants on suicidal thoughts is an additional argument in favour of the involvement of the serotonergic system in suicidal behaviour. Serotonin function is under genetic control (12). The fact that the decrease in serotonin function is in relation to suicide in several other mental illnesses in the genesis of which serotonin does not play a major role is again an argument (14). Most studies show that the most affected cortical regions from this point of view are those of the prefrontal, ventral and orbital cortex. These are areas involved in behavioural inhibition. Injury to this area leads to behavioural disinhibition, aggressive behaviour and self harm. It was also noted that noradrenergic overactivity predisposes to oversized behavioural reactions to stress. This system is also under genetic control (15). The two systems are in an inhibition/disinhibition report regarding behaviour but in a non-specific way. They favour disruptive, violent, uncontrolled behaviour, narrowing of consciousness. They do not contribute to the decision making process, but are just biological (technical) grounds. The role of Tyrosine hydroxylase (TH), an important factor in norepinephrine and dopamine synthesis has also been studied. Other researchers involved the opioid system and more specifically opioid receptors in suicidal behaviour starting from the brain analysis of 14 suicidals (16). A high density of receptors was found in the frontal cortex (16, 17). But all these neurotransmitter factors

are involved in the etiology (also nonspecific) of mental illnesses and much more nonspecific in favouring suicide (10).

CONCLUSIONS

Suicidal behaviour, or at least suicide accompanying mental illness, has, in its foundations, genetic vulnerabilities that encourage the development of neurobiological neurotransmitter vulnerabilities. These are also nonspecific, but they favour the explosive, disproportionately reactive, uncontrolled behaviour with narrowing of consciousness. The presence of this vulnerability will allow the contribution of a human being on other levels, from genetic to spiritual, to generating suicidal behaviour. Biological landmarks don't play a role in generating the decision of a suicidal behaviour but favour the progress of this behaviour.

Acknowledgments and disclosure

The author declares that she has no potential conflicts of interest to disclose.

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