Evolutionary Psychiatry Special Interest Group (EPSIG)

Newsletter February 2022

- Editor: Dr Annie Swanepoel (Consultant Child & Adol. Psychiatrist) annie.panzer@gmail.com
- EPSIG Chair: Dr Paul St John-Smith (Consultant Psychiatrist) paulstjohnsmith@hotmail.com
- Finance Officer: Dr Riadh Abed (Consultant Psychiatrist) abedrt@btinternet.com

- EPSIG YouTube Channel: https://www.youtube.com/channel/UCHyW9iyWB4jcQTequ-Nrq5w
- EPSIG web Pages: www.epsig.org

1. Notes from the Editor

In our last newsletter, we published the core trainee winner and runner-up. In this newsletter, I am delighted to include the winning and runner-up essays for our Charles Darwin essay competition in the specialty trainee category. Many congratulations to Dr Matt Butler and Dr Anna Eaton! Their respective essays on resignation syndrome and suicide are excellent and worth reading, as I am sure you will agree.

2. WPA, Section of Evolutionary Psychiatry Free Webinar Program.

The next free WPA evolutionary psychiatry webinar will be on 31st March 2022 at 4pm where Prof Henry O'Connell will be speaking about the evolutionary foundations for psychotherapy. Please register below.

https://www.wpanet.org/evolutionary-psychiatry

3. SPECIALTY TRAINEE WINNER: Dr Matt Butler

Resignation Syndrome: An Evolutionary Perspective

Submission for the Charles Darwin RCPsych Evolutionary Psychiatry SIG prize 2021

Dr Matt Butler ST4 in psychiatry, South London and Maudsley NHS Foundation Trust

Summary

Resignation syndrome is a recently described neuropsychiatric disorder which predominantly affects children who seek refuge in Sweden. Prominent features of resignation syndrome include a period of withdrawal followed by an unresponsive coma-like state which may require nasogastric feeding and full-time carer support. Resignation syndrome has been suggested to represent a form of functional coma, although the condition also shares similarities with other neuropsychiatric disorders such as depression, seasonal affective disorder, and catatonia. Several evolutionary mechanisms which once provided adaptive advantage have been implicated in the genesis or maintenance of these three disorders. These mechanisms include learned helplessness, hibernation, and tonic immobility. Due to overlapping clinical features and risk factors, similar evolutionary mechanisms may be implicated in resignation syndrome. A learned helplessness mechanistic model implicates previous trauma in the genesis of resignation syndrome and lends itself to identification of those at high-risk of the disorder, as well as using positive clinical signs to 'demonstrate an escape' to affected patients. A hibernation model suggests avenues for mechanistic research, including exploration of vagal tone and endocrine modulation. Finally, conceptualising resignation syndrome as a form of tonic immobility implicates naloxone as a potential treatment, particularly given the putative role of the endorphin system in the maintenance of tonic immobility in animals. This essay explores an evolutionary perspective on resignation syndrome and suggests potential hypothesis for future research into the condition and its treatment.

Introduction

Resignation syndrome is a neuropsychiatric disorder characterised by an initial depressive withdrawal phase followed by a coma-like state, in which affected children are almost entirely unresponsive (1). The illness falls outside of established psychiatric classification (e.g., the Diagnostic and Statistical Manual of Mental Disorders), although it was recognised as a distinct diagnosis by the Swedish National Board of Health and Welfare in 2014 (2). The disorder, encountered by Swedish researchers and doctors from the 1990s onwards, affects solely children, and is typically seen in those who have a history of significant traumatic experience(s) before moving to Sweden as asylum seekers (2).

Clinical features

Resignation syndrome is a debilitating disorder in which children and adolescents slowly become stuporous following a depressive period. The prodromal phase features social withdrawal, sleep disturbances and dysphoria. This progresses, via mutism and stupor, to complete unresponsiveness with double incontinence. In the latter stages, affected children are unresponsive even to painful stimuli (1). There are many clinical similarities between resignation syndrome and functional coma (3). Neurological examinations in affected patients are generally normal, however, patients may

have hypotonicity/flaccid paralysis with weak reflexes. Other neurological signs point towards a functional aetiology, such as closed eyes resistant to opening, and pupils which divert from the examiner, both of which are reasonably specific positive signs for functional coma (4). Further clinical signs may include tachycardia and elevated temperature, (1) although affected children often have heart rate and blood pressure within the normal range, and haematological markers are normal or non-specific. EEG findings are normal and show patterns of a sleep-wake cycle (5). In part due to a poor understanding of the disorder, treatment options are limited. During the illness, management is almost entirely supportive, with many children requiring nasogastric feeding and complete support for all basic activities of daily living. This support is often undertaken by the families of patients (2). The condition may last for months or years at a time before resolution, which may be partial or complete and may be precipitated by a change in living circumstances (1).

Conceptual challenges

Resignation syndrome affects children who almost invariably are caught in the midst of a familial application for asylum, and the disorder often arises during periods of uncertainty regarding residential status (2). Cases described in Sweden almost invariably affected children who arrived from particular geographical regions, and the vast majority had witnessed significant trauma pre-morbidly (2). Although resignation syndrome is unlikely to be an entirely novel syndrome (cases with similar clinical presentations have been described in the literature (1,6)), its specific demographic, political, and geographic distribution certainly point towards a culture-bound element to the disorder (1). A number of explanatory and precipitating aetiological theories have been advanced to conceptualise the disorder, including medical, family dynamic, psychological, political, cultural, and intentional models (7). The interplay of these remains a controversial issue, particularly with the political backdrop which surrounds the disorder; some have suggested that the disorder is pervasive refusal syndrome, malingering or even Munchausen's by proxy (7). There are, however, no firm data to support these assertions, and a discussion of the diagnostic and political controversies surrounding the disorder is beyond the scope of this essay. A model of symptom genesis which considers the multiplicity of sociocultural, biomedical, and psychological factors is likely to be the most robust means of understanding the disorder.

Evolutionary perspectives on resignation syndrome

Despite the similarities between resignation syndrome and other psychiatric disorders with proposed evolutionary perspectives, such as depression(8), seasonal affective disorder (SAD) (9) and catatonia (10) there have not yet, to the best of the author's knowledge, been any evolutionary perspectives on resignation syndrome in the English-language literature. Nevertheless, it is possible that resignation syndrome, although inextricably culture-bound, can be conceptualised in part as arising from situationally inappropriate use of evolutionarily preserved mechanisms of response to stress and fearful situations. The aims of this essay are thus as follows:

a) To explore the similarities and differences of resignation syndrome with the evolutionary models of related disorders including depression, SAD, and catatonia.

b) To evaluate potential mechanisms behind resignation syndrome from an evolutionary perspective. These mechanisms include tonic immobility, learned helplessness, and hibernation.

c) To propose testable hypotheses based on these features.

Depression: learned helplessness

Depression is one of the most common psychiatric disorders, affecting millions worldwide. Although the phenotype of depression varies between individuals and cultures (11), symptoms often include withdrawal from social interaction, changes to eating and sleeping habits, loss of interest and pleasure in activities and psychomotor retardation. Depression, in its severest form, can lead to a stuporous state in which the individual does not eat or drink and shows limited interaction or responsiveness to the environment. These severe cases of depression may require nasogastric feeding and interventions such as electroconvulsive therapy (12). Many of these symptoms of depression are shared with resignation syndrome. Depression is one of the psychiatric conditions with the most well-characterised evolutionary perspectives (8). These perspectives describe how depressive symptoms, such as withdrawal, may have been adaptive in certain situations in pre-modern humans, for example in the face of uncontrollable and hostile environments (13). It follows that withdrawal from situations, with a consequent reduction energy expenditure, may be an adaptive mechanism when humans are faced with repetitive stress or frustration. In humans, this type of behaviour may prevent excessive pursuit of an unachievable goal (8). An example of a specific proposed evolutionary mechanism by which depression arises is that of learned helplessness, a phenomenon which was first noted in animal models (14). When faced with repetitive and enduring stressors which are beyond their control, some mammals will enter a withdrawn and anhedonic state which has similarities with a human depressive syndrome. One experimental paradigm utilises electrical shocks, and when animals who have 'learnt' that they cannot escape the shock are placed in a new environment where a shock is present but escape is possible, many will exhibit passive attitudes and make no attempt, at least initially, to avoid the shocks (15). In some experiments, intervention by researchers to show that escape is possible does eventually lead to the animal escaping. Learned helplessness can be used as a putative model to explain some of the symptoms of resignation syndrome, such as withdrawal and apathy, and emphasises the importance of a history of previous traumatic experiences in the subsequent development of these symptoms. This is particularly pertinent, given that the large majority of children affected by resignation syndrome have a history of some form of life-threating trauma prior to their arrival in Sweden (2). It is possible, therefore, that using the framework of learned helplessness, that asylum-seeking children could be assessed for the risk of prospectively developing resignation syndrome based on their trauma history, particularly in traumatic events which have been perceived by the children as being uncontrollable and inescapable. This paradigm also, importantly, reiterates the importance of clinicians 'demonstrating an escape' to the affected children by compassionately eliciting positive functional signs (discussed later), which indicates to patients and their carers that the possibility for the resumption of normal function remains, even in the most severe cases. This form of therapeutic technique already has some evidence as a means of treatment in functional coma (4).

Seasonal affective disorder: attenuated hibernation

Seasonal affective disorder (SAD) is characterised by lethargy, hypersomnia, social withdrawal, and low mood. The disorder is thought to occur in increasing frequency at higher latitudes, although other influences such as climate and sociocultural factors may also predispose to or protect from the development of the disorder (16). Resignation syndrome, at least in the early stages, shares some clinical aspects with SAD. SAD has been postulated to arise from the evolutionary advantage gained

from lower fertility rates in winter, during which time it is much harder to provide for neonates in areas where food is scarce; the disorder in this sense has been compared to a form of attenuated winter hibernation (9). It is suggested that episodes of torpor, or at least relative inactivity, during periods in which energy conservation may be necessary (i.e., when food is scarce) has adaptive advantages, particularly 'in situations in which action would be futile or dangerous.' (8) Previous authors have shown evidence of increased vagal tone, measured by frequencies episodes of sinus arrhythmia, in subsyndromal SAD patients in comparison to controls (17). This heart rate alteration is shared by some animals entering hibernation, implicating the vagal nerve in commencing physiological cascades which cause the animals to enter the hibernation state (18). Hibernation is practised by numerous animals and occurs mostly in winter. Hibernation takes numerous forms, from extreme drops in core body temperatures sustained via a drastic reduction in metabolic rate, to lighter winter 'torpor' undertaken by, for example, brown bears (19). The animal with the closest phylogenetic related to humans which hibernates is the dwarf lemur, a primate which populates Madagascar and hibernates for around seven months a year, despite the high temperatures (20). This suggests that the genomic correlates of hibernation could be preserved in humans, a hypothesis which has been suggested by both animal and medical researchers (21). Indeed, archaeological evidence has suggested periods of hibernation in an extinct hominin species around 500,000 years ago. Researchers analysing the fossilised remains of a group of these hominins concluded that regular structural bone abnormalities in the skeletons were indicative of osteodystrophy characterised by severe hyperparathyroidism, which they suggest would have arisen during months spent in low-light environments (i.e., caves) during the intolerable winter season (22). They suggest that this provides indirect evidence of hibernation in pre Homo sapiens human species. Despite this, evidence of modern human hibernation is sparse. Periods of winter hibernation – known as lotska – may have been undertaken by Russian peasants in 18th Century Pskov area of Russia (a similar latitude to Stockholm), as described in the British Medical Journal around a century years ago, although this anthropological narrative may be apocryphal (23,24). Hibernation involves a reduction in metabolic rate, and a consequent lowering of body temperature, as well as additional physiological changes such as bradycardia (as well as sinus arrhythmia, as discussed above). The physiological correlates of hibernation are incompletely understood (25), however it is likely that a complex interplay of endocrine alterations are responsible for its induction and maintenance (26). Hypercortisolaemia has been associated with hibernation in brown bears (19), and alterations in other hormones such as leptin, ghrelin, insulin, and parathyroid hormone have been postulated to ensure homeostasis in other animals during hibernation (27). In the media, resignation syndrome has been described as akin to hibernation (28). Although evidence so far from resignation syndrome is limited, calorimetry measurements in some patients has demonstrated metabolic rates below that of expected basal rates, suggesting a state akin to hibernation (2). Despite this, there are some data from a small case series which diverge from animal studies; for example, there are suggestion that the duration of illness in resignation syndrome is negatively associated with markers of endogenous steroids (including cortisol) (29). Previous authors have drawn evolutionary comparison between hibernation and anorexia nervosa, and have suggested that markers of hibernation, for example heart rate variability, could be used to unmask at-risk patients. These physiological parameter alterations may represent an avenue for the mechanistic exploration of resignation syndrome. Research could help to unpick whether patients who are at-risk of the disorder manifest physical changes such as increase in heart rate variability. The putative endocrine alterations in hibernation,

particularly modulation of cortisol, leptin, ghrelin, insulin, and parathyroid hormone, also offer an opportunity to explore the neuroendocrine correlates of resignation syndrome (30).

Catatonia: tonic immobility

Catatonia is an acute neuropsychiatric disorder which arises in the context of medical or psychiatric illness. Catatonia may, in some cases, be described as functional or psychogenic, and there have been suggestions that resignation syndrome represents a form of functional catatonia (1). As well as a vast array of causes, catatonia has a heterogenous presentation; typically, however, patients enter a period, sometimes long-lasting, of immobility and relative unawareness of the outside environment. This is often accompanied by clinical signs such as catalepsy, rigidity, stereotypy, and refusal to engage or eat (31). In previous evolutionary perspectives, it has been suggested that catatonia represents an exaggerated fear response, with features similar to the animal defence of tonic immobility (10). Colloquially, this is referred to as being 'scared stiff'. This is supported by phenomenological accounts of catatonia: when able to remember the experience, patients often recount periods of intense fear which accompanies the physical symptoms (32). Evolutionary perspectives have suggested that catatonia may represent a form of tonic immobility, a physical defence mechanism in some animals characterised by paralysis and anaesthesia (10). Many animals, when faced with stressful or fearful situations, particularly if there is a threat to life (such as facing a predator) will suddenly freeze (10). Features of this behaviour - tonic immobility - include cessation of movement, initial hypertonia followed by hypotonia, suppression of vocalisation, and lack of response to the environment (including painful stimuli), as well as heart rate and body temperature decreases (33). It is suggested that tonic immobility evolved as many predators rely on movement perception when hunting, and thus a prolonged period of immobility would eventually lead to the predator ceasing to hunt the frightened animal. Tonic immobility should, however, be distinguished from 'attentive freezing', a more transient animal response which reduces the chances of detection by predator and shifts attention diversion in the prey animal to potential dangers in the surrounding environment (10,33). Features suggesting tonic immobility have been described in humans in response to extraordinarily stressful situations, such as sexual assault, (34,35) and dissociation is seen as a possible mechanistic correlate of this response (36). Research, primarily in animal models, has noted the analgesia which accompanies traumatic events, and suggests that this may be mediated by the endogenous opioid system. Indeed, in experimental models, tonic immobility (particularly the analgesic aspect) can be shortened via the administration of the opiate antagonist naloxone (37,38). If extrapolated, this data suggest that dissociative analgesia may be reversed by naloxone in humans. Indeed, there is evidence, albeit limited at present, that some symptoms of dissociation, particularly in the context of posttraumatic symptoms, may respond to naloxone (39). Patients with resignation syndrome have been noted, despite signs of lingering awareness and volition, to be able to tolerate significant noxious stimuli without response or arousal (1), a feature which is shared by patients with functional coma resulting from other causes (4). The shared features of resignation syndrome with tonic immobility lends the disorder to a novel treatment hypothesis: reduction of the level of dissociative anaesthesia via the administration of naloxone may allow for clinically-appropriate stimuli -such as Harvey's sign, the application of high-frequency vibrating tuning fork to the nasal mucosa - to improve arousal (4). This is particularly important, as we have seen, as demonstration of normal physiological functioning is one of the mainstays of the treatment of functional disorders, including functional coma (4,40).

Conclusions

Resignation syndrome is a culture bound illness which, in the opinion of this author, is best described as a functional coma with additional depressive and catatonia-like features. The interplay of sociocultural factors in its genesis is indisputable, however proximate mechanisms may involve evolutionarily conserved mechanisms, such as learned helplessness, hibernation, and tonic immobility. Whatever the cause of resignation syndrome, evolutionary perspectives on resignation syndrome lend themselves to testable hypotheses in conceptualising and treating the disorder. The learned helplessness model offers a chance to stratify at-risk patients, and is a hypothesis supported by the improvement of patients' conditions when asylum status is granted or when separated from the home and family environment (1,41). It is suggested that clinicians consider appropriate trauma histories in young refugees who arrive to countries where resignation syndrome has been described. This is particularly the case in patients who may have experienced repeated severe trauma which has been perceived as uncontrollable and inescapable. This model also reiterates the necessity for clinicians to offer an escape from the condition via demonstration of the capacity for normal physiological functioning. Although speculative, the overlap in clinical aspects of hibernation and resignation syndrome suggests potential avenues for mechanistic exploration. The influence of vagal tone could be monitored in patients who are identified as potentially at-risk of developing resignation syndrome. Furthermore, exploration of serum endocrine changes in patients with resignation syndrome may help to develop a deeper physiological understanding of a condition which is currently poorly understood. Finally, larger-scale studies into the metabolic rate of patients with resignation syndrome may offer insights into the physiological manifestations of the disorder and improve understanding of the similarities and differences between resignation syndrome and the hibernation state. Finally, the possible influence of tonic immobility, particularly the anaesthetic aspects (as in some cases of traumatic dissociation and functional coma), offer a potential treatment hypothesis. It may be prudent to trial the administration of naloxone, a safe and well-tolerated medication, in patients with resignation syndrome, followed by the use of clinically appropriate and brief painful stimuli, which could help to induce arousal. This induction of arousal may be a pathway for patients to begin their recovery from an extraordinarily debilitating condition.

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4. SPECIALTY TRAINEE RUNNER UP: Dr Anna Eaton

"Natural selection will never produce in a being anything injurious to itself,"

- Darwin '*On the Origin of Species*'

The suicide rate for females under 25 years in the UK is currently the highest on record[i]*, whilst the Millennium Cohort Study discovered that 7% of children had attempted suicide by 17 years of age with 24% having self-harmed within the year*[ii]*. Globally almost 800,000 people end their lives by suicide each year*[iii]*. For each adult worldwide that successfully takes their own life, approximately 20 have made an attempt and 200 have thought about it*[iii]*. Moreover,* the heritability of completed suicide is estimated at 43%[iv]. *How then, can we possibly begin to conceptualise this phenomenon in the context of adaptation to survive?*

As Darwin considers human and animals on a natural continuum[v]*, perhaps the first consideration is the manifestation of self-injurious behaviour across the animal kingdom. Traditionally*

conceptualising animal behaviour as deliberately harmful or suicidal has been dismissed as metaphorical or anthropomorphic[vi,vii]. But is it our human experience of the sequelae, socio-cultural and religious overlays that colour our understanding and interpretation of suicide as uniquely and tragically human – a by-product of a sophisticated brain[viii]? Particularly when these factors, in combination with stigma and legality chiefly shape broad societal 'anti-suicide defences'[ix].

"There is no fundamental difference between man and animals in their ability to feel pleasure and pain, happiness, and misery."

Animals experience many of the emotional states associated with suicide[x] and depression; helplessness[xi], lethargy[xii], anhedonia, chronic anxiety and sleep disturbance[xiii]. In fact, research has identified pathological behaviour akin to human psychiatric disorders including PTSD, eating disorders, antisocial and borderline personality disorders, diagnosed using human psychiatric models and treated in the same way[xiv,xv]– with positive results[xvi].

There is significant evidence for a large spectrum of self-injurious behaviour in animals, particularly so in captive or isolated animals[xvii]. These are exhibited with associated neurological, neurochemical and endocrine abnormalities, decision-making deficits, genetic, epigenetic interactions and interestingly exposure to early life stress and adversity[xviii]. These findings are in parallel human to 'stress-diathesis' models[1] of suicide[xix],[xx] and neurobiological changes identified post-mortem such as low levels of cerebrospinal fluid 5-HIAA, disturbances in the serotonergic system and hypothalamic-pituitary adrenal axis dysfunction[xviii, xxi]. Many animals have been documented to starve themselves voluntarily if captured, self-neglect or exhibit self-aggression, self-biting and self-mutilation in suboptimal living conditions 'perceived as uncontrollable'[xiii]. Self-destructive behaviours have been demonstrable in artificial laboratory based psychological and biomedical research but also within ethological observation of altruistic suicide, stress related self-harm and dispersal behaviours[xvi].

Durkheim posited the term 'altruistic suicide', motivated by self-sacrifice for the 'common good'. This seems to be present in the natural world and enacted in insects (hymenoptera), seen also in herding behaviour in cattle and fish, 'kamikaze'-like defensive behaviour in some bird species[2] and is also seen in other eusocial[3] animals. This is adaptive if providing survival benefit to one's kin or social group and may explain other human social behaviours such as self-sacrifice in the context of war or natural disasters and other dangers[xxii,vii].

It is therefore suggested that negative cognitive bias, typical of depression, may result in overvaluation of one's perceived burdensomeness and, therefore, increase the risk of erroneous altruistic suicidal behaviour by 'self-removal' or self-neglect[xxiii, xxiv]. These cognitive biases may be more prevalent in the depressed elderly and those suffering with chronic health problems[xxv]. This hypothesis led to the interpersonal theory of suicide; coexistence of high levels of perceived burdensomeness and low levels of belongingness with hopelessness regarding change[xxvi,xxvii].

There is some overlap with these ideas and the aforementioned dispersal behaviours - expulsion of an individual from a group due to population density pressures or resource availability – seen in small mammals such as rats, mice, lemmings and arctic ground squirrels [xvi,xxviii]. There may also be

[1] Proposes pre-existing vulnerability factors activated by stressors
[2] Blackbirds, warblers, jays, robins and hummingbirds
[3] Rely on multigenerational and cooperative care of young, utilize division of labour for successful survival

parallels between this and suicidality linked with social exclusion or isolation, marginalisation, poverty, bullying and harassment[xxix,xxx]. Or put another way, risk taking or suicidal behaviour may manifest as a result of lack of physiological, safety and belonging needs being met in the environment incuding those of love intimate and sexual relationships[4].

An alternative, and more commonly understood explanation is the antithesis of altruistic suicide – egoistic suicide – motivated by personal escape from intolerable circumstance[vii]. And here belies the problem. These acts of deliberate self harm, enacted by distressed individuals, as a communication of that distress or even as self-euthanasia[vii] cannot be understood as adaptive.

Many believe that animals lack the cognitive capacity requisite for suicidal acts. Specifically, reflexive subjectivity is felt to be a requirement that is lacking. However, there is not a standard which can be applied for determination across different species[xxxi] and empirical research suggests self-cognizance is a matter of 'degree rather than kind'[xxviii]. Some animals may even perform better than children[xxxii] in demonstrating reflexive self-conscious subjectivity, for example on Gallup's mirror test[xxxiii] (although a crude measure[xxxiv]). Moreover, how could we then account for suicidal behaviour in children or those with transient or permanent cognitive deficits or developmental impairments? Likewise in order to intentionally bring about one's own death, it is argued that one must be able to conceptualise death or one's own mortality. But again, there are humans that engage in self-injurious behaviour who may also lack this understanding. There is also no evidence that animals necessarily lack this concept. Burial rituals as well as bereavement reactions may suggest otherwise following the loss of companions and relatives[xxviii]. Other land and aquatic mammals have specific post-mortem rituals which hint towards an understanding of death further than the experience of grief, for example, Bonobos will provide end of life care to others with whom they share a bond[xxxv] and will risk harm to themselves whilst preventing interference with bodies of loved ones[xviii]. Whilst it is accepted that animals instinctively respond to fear and through learning from experience about danger, seek to avoid it, it is difficult to both prove and disprove concept of mortality.

Suicide must be a choice of 'free will'. But it has also been put forward that as free will is a metaphysical or abstract notion, the assumption is that if a self-induced death when applied to animals can be explained 'naturally' by physical, chemical, biological or psychological processes then it is not a 'true suicide'. But where does this leave us in classifying self-inflicted death that is explained in this way?[xviii] Or even those arising from indoctrination, so-called suicide martyrdom[5]? Roughly half of completed suicides are linked with alcohol or drug abuse and dependency and up to 10% with organic disorders and schizophrenia[xxxvi], impairing executive functioning and directly contradicting the above.

We then must consider intent. How can we prove that any animal intends to end its life or that the behaviour is goal directed further than death being the final outcome? There are obviously some advantages in drawing conclusions about humans that choose to end their life regarding reasoning and motivation, but still proving true 'intent' is nuanced. So much so that it is felt there is frequent mis-coding or attribution of causation or mortality worldwide leading to underreporting of completed suicide[xxxvii]. Even considering the above concepts of altruistic and egoistic suicide, in real terms there is little way of reliably separating one's motivation, which are likely to be mixed. It is also

[4] As per Maslow's hierarchy of needs.
[5] Which could be understood as an altruistic suicide

difficult to establish the intent of passive suicidal behaviour or self-neglect described above in animals and seen in severe depression, psychotic illnesses, substance addiction amongst others clinically. The motivating state does not typically map over to active suicidal behaviour should the means become available[xxxiv]. Clinically there is much difficulty in differentiating parasuicidal behaviour as a 'cry for help', risk of death by misadventure and serious intent, due to the similarity in clinical profiles and stated intent[xxxviii] whilst simultaneously appreciating the increased risk of completed suicide following a previous attempt[xxxix]. Similarly, despite the availability of risk assessment tools based on known risk factors there is limited evidence of any predictive value[xl].

Although controversial and heavily disputed, the research into animal self-harm behaviours seem to draw two broad conclusions: the first that there exists some form of continuity between self-destructive behaviour in non-human animals and homo sapiens[vi] and secondly that stress can change the behaviour of an animal in a way that can threaten its life.

"It is not the strongest of the species that survives, nor the most intelligent that survives. It is the one that is most adaptable to change."

The ramifications of 'stress' on behaviour should not be a surprise. 78% of suicides occur in low to middle income countries[xli], with the rates of these rising[6] over the past 50 years whilst western societies saw a simultaneous decrease[xxxvi]. Socioeconomic factors strongly influence suicide and are co-variables, for example; low income, income equality, unemployment, low educational level, urbanization and poverty[xxxvi]. Where individuals have not met the criteria for a mental illness, precipitating factors to crises have been identified such as; acute financial difficulty, loss or divorce from a significant other[7], discrimination, terror, violence and war[xxxvi], medically unexplained pain[8,xlii], disability[xliii] and chronic and/or lethal physical health problems such as HIV, cancer, MS, SLE, epilepsy amongst others.

Groups at higher risk of suicide are those working in the emergency services or first responder roles[xliv], in the military[xlv], imprisoned or incarcerated[xlvi], homeless[xlvii], refugees and asylum seekers[xlviii ,xlix], minority groups[l] and the LGTBQ community[li] - all groups exposed to higher-than-average levels of psychosocial stress, including personal endangerment.

Overall men are almost twice as likely to complete suicide[lii] and the rate of male to female suicide is only reversed in China and Bangladesh[9]. We can hypothesise that local factors and cultural gender roles and expectations may contribute to the impact and management of stressors[liii] including availability of broader support systems and avenues of coping. Although there is dispute as to the origin, traditional gender roles broadly place males as 'providers and protectors'[liv] which may add to the psychological burden of challenges or adversity that threaten fulfilment of this role[lv]. Sociocultural ideals and expectations of masculinity may lead men to be less likely to seek help[lv] and have a tendency toward externalising behaviour[lvi], impulsivity, aggression and lethal methods[lvii,lviii].

But the brain is evolved to protect the self from harm - to survive and reproduce. Navigating stress, challenges, threats, predictable and unpredictable danger are universal experiences for all living

[6] By 60%
[7] To a lesser degree, a lack of romantic relationship
[8] Although this is beginning to be understood as a somatoform illness
[9] M:F ratios of 0.8%

beings. Aptitude to problem-solving and adaptation through adversity has cultivated and shaped our modern environment[lix, lx]. It must then follow that the manifestation of suicide occurs generally and across species as an *absence* of adaptation in the context of overwhelming or cumulative stress or threat to the self or progeny.

Although suicide risk factors and warning signs have been identified these are imprecise and not sufficiently predictive. *Approximately 25% of those who have completed suicide are known to mental health services in the year prior to death*[lxi]. Between 60-98% of suicides are associated with mental illness on 'psychological post-mortem' studies although some caution needs to be taken with interpretation of these and the findings are contradictory with broad results[lxii],[xxxvi]. Most commonly identified was depression (particularly psychotic depression in the elderly), alcohol or drug abuse as aforementioned, and personality disorder. Organic disorders and Schizophrenia make up less than 10% of these[xxxvi].

Symptom-based diagnoses using ICD and DSM can be limited due to the lack of causal explanation for the presenting pattern of symptomology, which is often individually variable despite implication of an underlying, universal mechanism. There is high comorbidity between disorders[lxiii] *as well as poor diagnostic concordance between professionals and lack of specific treatment implications*[lxiv]*. We also know that the vast majority people with psychiatric disorders do not become suicidal, despite the presence and accumulation of these being an apparent risk factor, for example, less than 5% hospitalised with mood disorders go on to end their lives by suicide*[lxv]*. Mental illness is not a solely biological phenomenon, thought to originate from a complex interplay of biological, psychological and social precipitants, and is not predictive in its relationship to suicidal behaviour, but is often identified as a linear causative factor. Moreover,* psychobiological abnormalities are associated with vulnerability to suicidal behavior, independent of co-occurring psychiatric disorders[lxvi,lxvii]. Focusing on psychiatric diagnoses does not offer us any causal explanation as to suicidal behaviors despite mental health services and legal frameworks being structured around prevention of harm to self.

It has been suggested that symptoms of mental illness itself may be protective against suicide – for example anergia in depressed mood or that addiction, for example, may act as means of dampening down emotional pain and arousal and therefore be protective[lxviii]*. Certainly, poor mental health and wellbeing appears to be an indicator to self and others of psychological suffering or 'dis-ease' across the animal world.*

The disease-based model disregards the significance of personal individual experiences or exposure to threats and self-protective responses and insinuates an external locus of 'illness' or dysfunction which does not hold true for many non-organic disorders.

Successful adaptation to the environment, or resilience, involves learning from experience by changing behaviour, changing neural responses, changing immune responses and so forth. Each exposure to threat offers novel information with which to refine one's responses, by using the past to predict the future[lxix]*.*

This is a process which starts even prior to birth by intrauterine programming – shaping the foetal autonomic nervous system and HPA axis for the postnatal environment it is likely to encounter[lxx]*. Epigenetics allow for the transmission of transgenerational trauma*[lxxi] *whilst early life stress and adverse early experiences influence neuroplasticity*[lxxii]*. Unsurprisingly, adverse childhood experiences*

or ACEs have repeatedly been shown to be directly linked to suicidal behaviour in adulthood[lxxiii,lxxiv]*. Likewise, trauma is associated with suicidality- interpersonal trauma most strongly, followed by childhood trauma and sexual trauma, more pronounced in men. Men were also found to be more at risk of suicide related to non-interpersonal trauma*[lxxv]*.*

Vulnerable and entirely dependent on others, new-borns must adapt to their environment and interpersonally caregivers to maximise availability such that needs are met. Attachment theory proposes an innate drive to organise behaviour self protectively[10][lxxvi] *shaped by interactive effects of genes with maturational processes and individual-specific experiences of threat and dyadic relationships with attachment figures. The outcome are strategies, born from neurological transformation of sensory stimuli into relation of self to context, and application of these dispositional representations to motivate behaviour. These may be be normative or non-normative but always adapted to individual context - at risk of giving rise to later psychopathological behaviour*[11]*,*[lxxvii] *as a developmental process*[lxxviii]*.*

Broadly speaking the maturing brain processes two avenues of incoming information with relation to possible threat; temporal order of events (apparent causal sequencing) and affective or somatic intensity in relation to the context. Conditions of a generally safe environment, or atleast adequate protection from danger, comfort and understanding of dangerous events supported by attachment figures allow for, more or less, a normative balance of cognitive and affective information processing allowing flexibility around selected behavioural responses when faced with threat – a 'B' or balanced strategy. This internal working model gives accurate associations of past events to accurate predictions and representations of the self in environmental context to the future in motivating behaviour. These becoming increasingly sophisticated and complex through development to adulthood as the cortex matures and at puberty integrate to sexuality and intimacy into pre-existing strategic functioning.

Without such optimal conditions or interruptions to this process[12] *a bias to either cognitive (temporal) or affective processing motivates behaviour. Both over-estimate danger and dispose the individual to act in self-protective ways - which although originally may have been safety-promoting, are likely maladaptive in the context of relative safety. However, the function of the behaviour is to reduce danger as (mistakenly) represented by the individual in the face of percieved threat. The first, an array of 'Type A' strategies*[13] *prioritise cognitive information, temporal order and causality at the expense of omitting negative feelings. These manifest as a result of predictable rejection of attachment behaviours by child or predictable danger. The opposite 'Type C' strategies*[14] *organise entirely around feeling with little attention paid to causality or consequences, in other words, promote indiscriminately responding and attending to changes in autonomic arousal regardless of context. Development and experience allow for opportunities for 'reorganisation' or correction, however in the case of pervasive predictable, unpredictable or deceptive danger a risk of reinforcing the strategy (at the expense of flexibility) or increasing the distortion of information. The transition to*

[10] And sexually, post puberty.
[11] Related to psychiatric diagnoses such as personality disorder, anxiety etc
[12] By abandonment, rejection, seperation from or loss of attachment figures, or alternatively experiences of active or passive harm by attachment figures
[13] Overlapping with 'Avoidant Attachment' in ABCD Attachment model terms
[14] Overlaps with 'Anxious Ambivalent' attachment in ABCD Attachment model terms

adulthood is a particular opportunity for strategic reorganisation. Confounding these strategies for an individual using any strategy, is an experience of trauma (resulting from danger beyond the individual's zone of proximal development, from which they were not protected nor comforted). These use pre-cortical neural processing as a 'psychological shortcut' to a fixed, rapid behavioural response[lxix].

At face value, there are more obvious and immediate risks associated with using a 'Type C' strategy. Such behaviour is usually organised around unpredictable care or danger. For example, commonly presenting to CAMHS, these children may escalate their affect, using actions to communicate by harming themselves in order to elicit a response from parents to regulate their arousal, despite the nature of percieved threat often being ambiguous[lxxix]*. These individuals rely on others to modulate their affect. By adolesence and early adulthood the more extreme 'obsessive' C strategies are engaging in a relational care-seeking struggle that risks the self and obscures meaning, often seen in personality and eating disorders*[lxxix]*. However, these individuals who act in ways that are at risk the themselves (or others) often do self-present in some form to services. Despite this, the struggle endures with increasingly risky presentations and services duty bound to reliably intervene although with no answer to an irresolvable problem. Although the intention is not necessarily to harm the self but to draw others in, increasing risk is often necessary to keep the needs of the individuals met (or professional services engaged). In this we see an iatrogenic worsening of risky behaviour, often seen on admission to inpatient wards competitively or at the point of discharge.*

Type A inhibitory strategies conversely are likely to go unnoticed, even praised by pleasing others through inhibition of true negative affect. In adulthood, these individuals who idealise and exonerate others and blame themselves for negative outcomes, are more at risk of satisfying others at the expense of personal feelings of emptiness or failure. They are more at risk of depression and psychosomatic distress[lxxx] which are significantly less likely to present to services or meet the attention of a system that requires demonstrative risk before a defined intervention is given. But it is the compulsive A strategies who may find themselves quietly impulsively drawn to sudden serious suicidal acts, particularly in an intrusion of negative affect – a rapid rise in autonomic arousal (which is usually kept low through inhibitory processes) when extreme danger is imminent and escape required[lxxx]. This uncontrolled outburst of emotional behaviour may alert others to the individuals need for protection which may be obscured by compulsive behaviour (caregiving, performance, compliance) and pseudo-intimacy and allow for intervention – thereby remaining adaptive[lxxx].

Perhaps even riskier in both patterns is a state whereby the individual may become actively aware of the failure of the strategy as self-protective. But in the absence of an alternative or the reflective capacity to reorganise towards balance, despite being aware that the behaviour is no longer functional, these individuals use the pattern regardless, trapped in a position of non-agency regarding safety and comfort[lxxxi]*. Both strategies, although initially adaptive and self-protective, involve risk.*

For the majority, in the absence of reorganisation, psychopathological behaviour or enacted danger to the self or others, these non-normative strategies simply continue to be, for the most part, adaptive to the individual's circumstances, and thus the next generation are born. In the role of attachment figure, like all parents they construct a world for their children based on their own

experiences of safety and danger from which their children must construct self-relevant meaning through their own experiences of this environment[lxxvi].

These strategies fit neatly with previous hypothesis about two groups of suicidal individuals – those who may threaten, self-harm and make suicidal gestures but remain at risk of (perhaps unintended) death to others with objectively a higher risk of suicide completion[lxxxii], and is concordant with what presents clinically as well as accepted 'at risk' psychiatric disorders[15] whilst explaining why individuals may engage in this behaviour in the absence of known mental illness.

Understanding suicidal behaviour through a developmental lens allows us to conceptualise the function and emergence of deliberately self-injurious behaviour and the relationship between stressors with heritability, physiological and genetic factors of suicidal behaviour and psychopathology more generally.

However, if we are to consider and understand such behaviour from an evolutionary and adaptive perspective this does draw questions about how we are to best address it. Coordinating effective response would require restructuring of services into early intervention and prevention, investment in robust social services and early years support and more easily accessible systemic and psychotherapy provision toward customised, family-based treatments. This would be a marked shift away from the current model of care focusing on the treatment of adults, primarily through pharmaceutical means. Such work would require not only significant financial and political backing and infrastructure, for which there is already little appetite, but also a level of individual and societal introspection, engagement and accountability which feels aspirational but unfathomable in the current climate of health and social care in the UK.

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Many thanks for reading the Newsletter and please don't hesitate to get in touch with any contributions or suggestions.