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Research Article

Does Everyone Have ADHD? the Evidence for Attention-Deficit/Hyperactivity Disorder as an Evolutionarily Adaptive Behaviour Originating in the Middle-Upper Paleolithic Period

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Abstract

Attention-Deficit/Hyperactivity Disorder (ADHD) is a highly heritable condition with a prevalence of about 5% in children, and about half that figure in adults. With these epidemiological numbers in mind, how does ADHD evolutionarily persist despite it being a disadvantageous medical problem? This paradox requires explanation, and any solution must be consistent with evolutionary theory. This paper reviews the essential features of ADHD, and examines whether the condition should be better characterized as a disease or phenotype. Prior hypotheses concerning the possible evolutionary origins of ADHD are briefly reviewed. This paper proposes that ADHD is more likely to be a behavioural phenotype as opposed to a disease; and its presence in DSM-V as a disorder is mostly due to certain behavioural disadvantages that stand out in highly technological societies (i.e. Mismatch theory). It is also theorized that ADHD evolved primarily to enhance warring capabilities in Homo sapiens around the Middle-Upper Paleolithic period. The most penetrating ADHD behaviours appear localized in a small subset of the population; however, it is not yet known whether lesser amounts of ADHD permeate the rest of humanity in a Gaussian distribution.

Keywords: ADHD; evolution; anthropology; cross-cultural; diagnosis

Introduction

Over the last few decades, greater numbers of individuals have been diagnosed with Attention-Deficit/Hyperactivity Disorder (ADHD). The essential features of ADHD are inattention, impulsivity and hyperactivity - behavioural tendencies that are typically judged to be less desirable in the modern world. Some accounts suggest up to 10% of children could have ADHD (Cabral et al. 2020) [1]. Moreover, ADHD is a highly heritable condition (Franke et al. 2012) [2]. Simply put, how does a genetic-based condition persist at such a high prevalence rate if it is so disadvantageous? From an evolutionary perspective, ADHD requires greater examination.

When zoologists look at animal behaviour, they often try to understand whether it is adaptive or not, with the lion's share of behaviours eventually understood as being advantageous. So many perplexing behaviours are eventually understood in the context of evolution – for example, the waggle dance of honeybees or dolphin surface leaping (von Frisch 1953, Norris et al. 1994) [3,4]. Non-adaptive behaviours generally fall into one of two categories: 1) evolutionary trade-offs or 2) disease and/or injury. Genetic-based diseases rarely exceed 1% prevalence in fecund populations (with rare exceptions such as balanced polymorphisms seen in sickle cell anemia). As for evolutionary trade-offs, no adaptation

is perfect, but for a trait to persist, there must be in toto more advantages than disadvantages. For example, anger can sometimes be disadvantageous. Still, the sum total expressions of anger are likely to be advantageous in most individuals – otherwise, the trait would not have persisted. For heritable behaviours in hominids, the behaviour may have previously been advantageous in traditional societies but no longer. Evolutionary theorists refer to this as Mismatch theory (Lloyd et al. 2011) [5], and it seems to apply to certain psychiatric conditions like PTSD (Cantor 2009) [6]. Instilling semi-permanent fear in a hominid could be enormously useful to heighten vigilance around predators at specific locations like nearby watering holes; however, such an anxiety-inducing system may not serve much purpose for some modern threats.

So, how should we frame ADHD? Here, mainstream psychiatry has historically been negligent in differentiating behaviour from disease (or delving into the question of whether Mismatch theory applies to various psychiatric conditions). There are several reasons for this glaring blind spot among the mainstream psychiatric establishment. Part of the problem is that at its inception in the 19th century, psychiatry only dealt with extremely deviant behaviours; namely, psychosis, severe depression and severe hysterical reactions (Shorter 1997) [7]. However, as societies grew wealthier through the 20th century, psychiatry expanded and began to treat less severe conditions (e.g. marital therapy, substance use disorders, ADHD) – psychological ailments that perhaps were blending into normality.

The pursuit of a deeper understanding of psychiatric ailments was also perhaps derailed by the success of DSM-III in 1980 (APA, 1980) [8]. This respected manual focused on enhancing the reliability of diagnoses; however, applying scientific validity was almost abandoned (Greenberg 2014) [9]. The main architect of DSM-III, Robert Spitzer, realized there would be no consensus among psychiatrists around the possible origins of psychiatric conditions, especially between the Neo-Freudian and biological psychiatrists, who were almost speaking two completely different languages. It was also perhaps appropriate to side-step questions around scientific validity since, in the late 1970s, the study of neuropsychiatric pathology was still in its infancy. However, the continued atheoretical stance of DSM-III is arguably eroding psychiatry's reputation as a scientific discipline. By 2012, a landmark paper authored by leading evolutionary psychiatrists highlighted the problem (Brune et al. 2012) [10]. In their aptly entitled paper, The Crisis of Psychiatry – Insights and Prospects from Evolutionary Theory, this group of evolutionary psychiatrists suggested that psychiatry do what ethologists do when studying animal behaviour, and use evolutionary principles as their theoretical foundation.

Disease versus Phenotype

When it comes to nature's organisms, every discernible phenotype is either adaptive or it's not. In general, if a phenotype was once adaptive, it will completely suffuse the species. So, barring rare mutations, everyone has a nose, two eyes and a mouth – since having these features has historically been adaptive. Now, many phenotypes are expressed through polygenic effects (i.e. several genes), such as height, natural strength or dexterity. Such complex traits generally center around a Gaussian mean, which is usually the ideal form (or at least represents the ideal adaptation of the recent past). For example, larger individuals may have been especially vulnerable to starvation during periods of food paucity due to greater caloric requirements, but smaller individuals may have historically been vulnerable to being physically dominated. At such extremes of the bell-shaped curve, the phenotype can be highly disadvantageous or even be confused with outright disease (for example, being statistically very short versus dwarfism due to genuine hormonal dysfunction).

There are also many situations in nature where two or more phenotypes are simultaneously present, but one has not supplanted the other - like eye colour in humans. This problem has been intensely studied by evolutionary theorists (Hunt and Jaeggi 2022) [11], and a number of genetic/evolutionary mechanisms have been proposed to explain the presence of more than one phenotype in nature (Keller and Miller 2006) [12]. The evolutionary mechanisms - most relevant to psychiatric conditions – that possess the theoretical capacity to maintain polymorphisms in a species are 1) genetic drift, 2) negative frequency-dependent selection, 3) antagonistic pleiotropy and 4) heterozygote advantage (Polimeni 2012) [13]. Therefore, if ADHD is only present in a subset of individuals within our species, it ought to be maintained by one of these genetic/evolutionary mechanisms.

Randolph Nesse is one of first physicians to take evolutionary principles into account in the practice of medicine. In his seminal book (co-written with the evolutionary theorist G. C. Williams), Why We Get Sick (1994), Nesse [14] proposed that all medical ailments can be explained by one of six evolutionary mechanisms. This is a powerful categorical system because it has been around for several decades and can accommodate every medical complaint found in a doctor's office. It is not a purely algorithmic system (since some medical ailments can occupy more than one category), but it is certainly helpful in deepening our understanding of medical ailments.

The six general evolutionary causes of medical problems are 1) novel environments (for example, the modern diet causes a disproportionate amount of diabetes), 2) competition between organisms (for example, bacterial infections that parasitically use other organisms for their survival), 3) bodily defences mistaken as disease (for example, coughing, which is technically not a disease but a physiological mechanism to protect organisms from infiltrators), 4) evolutionary constraints or imperfections in design (for example, the eye's blind spot, or the unnecessarily circuitous path of the laryngeal nerve), 5) primacy of survival (for example, pulmonary emboli are partially due to our hair-trigger coagulation system designed to protect organisms from easily bleeding to death when lacerated), 6) trade-offs or design compromises (for

example, knee problems in hominids, evolutionarily tolerated to accommodate the needs of upright locomotion).

Psychiatric conditions can also be pigeonholed into Nesse's scheme. For example, generalized anxiety can be framed as a bodily defence to warn organisms of potential threats. PTSD, an extreme anxiety reaction, may be placed in the category of primacy of survival - since avoiding getting killed at all costs may be job-1 for any organism. At first glance, it seems that ADHD could be a result of novel environments and/or trade-offs. Before examining the possible evolutionary reasons for ADHD's existence, let's review the basic characteristics of ADHD.

Summary of ADHD: Essential Features, History, Epidemiology, Comorbid Conditions, Genetics, Neurobiology and Animal Models

Essential Features

The essential features of ADHD are hyperactivity, inattention and impulsivity. Most individuals with ADHD will demonstrate all three features, but some may only have one or two of these cornerstone symptoms. Individuals with ADHD tend to be impatient – having difficulties waiting in line or frequently interrupting others during conversations. They often throw caution into the wind, and make decisions with less thought (which could be advantageous during emergency or time-pressured situations). They are often distractible - both by external stimuli and internal thoughts. This sometimes causes them to forget a task at hand completely. Thus, they often forget their keys or miss appointments. It should be noted that some individuals overcompensate for their forgetful tendencies and become unfailingly organized to avoid making mistakes of omission (e.g. faithfully using alarms as reminders).

Many individuals with ADHD can become easily bored and require constant perceptual stimulation, whether it be playing video games or teasing others. Reading is often too unstimulating, and perhaps related, some have varying degrees of dyslexia. Hyperactivity is common, although less so in adults. Some fidget a lot, but not all. Severe cases are noticeably distractible, even in the physician's office. Individuals with ADHD often start projects but fail to finish them due to boredom or disorganization. They can sometimes be irritable and moody. Interestingly, in my office practice, I have noticed that many patients with ADHD have difficulty with the serial sevens subtraction test. Furthermore, how they make mistakes is telling – patients with neurocognitive disorders or depression will usually pause and be reluctant to provide a wrong answer, while those with ADHD tend to quickly recite mistaken numbers with no sense of reflection. On a positive ledger, individuals with ADHD may be more creative and outgoing, or excel in certain types of athleticism (chaotic sports like football or rugby).

History:

Perhaps the first clear description of ADHD symptoms in the medical historical record can be found in a medical book by Scottish physician Alexander Chrichton in 1798 [15]. In 1932, the concept of "Hyperkinetic Disease of Infancy" was introduced, and its symptoms resembled the modern formulation of ADHD (Lange et al. 2010) [16]. By the 1950s, the concept of "minimal brain damage" became commonly used to describe aberrant behaviours in children with neurological disorders such as epilepsy, postencephalitic syndromes and head injuries (with severe cases of ADD being clustered in this group). By the 1960s, this term was softened to "minimal brain dysfunction" - as it became evident that not all children with behavioural problems had evidence of brain pathology. In 1980, the DSM-III manual proposed a list of criteria to diagnose ADD (divided into those with or without hyperactivity). Then, in 1987, the modern formulation of ADHD was established in the revised DSM-III, with the hyperactive and non-hyperactive groups being combined, partly to increase the inter-rater reliability of the diagnosis.

Epidemiology and Comorbid Conditions:

In high-income Western nations, the most recent figure for the prevalence of ADHD in children is about 4-6%, with a male-to-female ratio of about 2:1 (Fayyad et al. 2017) [17]. It seems that about 65% of these cases persist into adulthood (about a 2.5% prevalence of ADHD in adulthood) (Cortese and Coghill 2018) [18]. The increase in prevalence rates over the last several decades is most likely due to greater awareness by both patients and clinicians. It should be noted that greater awareness among patients will allow a better description of symptoms, which can increase prevalence rates even if clinical rating scales have been consistent through the years. In general, ADHD prevalence correlates positively with the wealth of a nation (Fayyad et al. 2017) [17]. ADHD has been associated with an increased risk of divorce, less full-time work, and an increased risk of unintentional injuries (independent of substance abuse) (Katzman et al. 2017; Brunkhorst-Kanaan et al. 2021) [19,20].

There are many psychiatric comorbidities, including depression, anxiety, bipolar disorder, substance abuse, learning disorders, borderline personality disorder and antisocial personality disorder (Katzman et al. 2017; Choi et al. 2022) [19,21], Some literature suggests an association with autism (Grimm et al. 2020), although I have not noticed much of a special connection in my office practice. ADHD has also been associated with medical problems such as pre-term birth, obesity, sleep disturbances, asthma, and autoimmune and inflammatory diseases (Da Silva et al. 2023) [22]. The obesity finding seems suspect because ADHD is not associated with cardiac risk – so something doesn't add up (Instanes et al. 2018) [23].

Genetics:

The heritability of ADHD is estimated to be 60-90% (Cortese and Coghill 2018) [18]. Genome-wide association studies (GWAS) implicate many thousand genes, each with relatively small effects – similar to other heritable psychiatric conditions (da Silva et al. 2023) [22] It is perhaps not a coincidence that the heritability of

schizophrenia, bipolar disorder, ADHD, and autism are all around 80%, which may have evolutionary implications. Candidate genes such as the dopamine D4 receptor gene have been inconsistently associated with ADHD (Cabral et al. 2020) [1]. Possible epigenetic or other gene x environment interactions have not been clarified.

Neurobiology:

ADHD has many varied cognitive/emotional effects, which will make the discovery of a gross singular pathology unlikely. The fact that methylphenidate and other stimulants will sometimes ameliorate ADHD could provide inroads toward a neurobiological understanding of ADHD.

Such stimulants seem to mostly block the reuptake of dopamine and norepinephrine. However, serotonergic, glutamatergic and GABAergic systems are also involved, which greatly expands the possible key neuroreceptors involved in ADHD (Patel et al. 2021, MacDonald et al. 2024) [24,25]. Some researchers - noticing that attentional lapses may involve frontoparietal, ventral or dorsal attention networks - have proposed a "default network hypothesis of ADHD" (Cortese and Coghill 2018) [18]; and although reasonable, it is also speculative. Similarly, neuroimaging studies have not shown notable or consistent findings in ADHD, especially in adult populations (da Silva et al. 2023) [22].

Animal Models:

Almost all animal models for ADHD have involved rats and mice. A perfect animal model would incorporate hyperactivity, impulsivity and attentional behaviours; however, most rodent studies have focused solely on hyperactivity, since impulsivity and attention are difficult to reliably measure in animal populations. In one mouse strain (coloboma mutant mouse strain), for example, hyperactivity could be reduced by d-amphetamine, but not methylphenidate (even though both are effective in human clinical populations) (Sontag et al. 2010) [26]. Such discrepancies highlight the imperfections of animal models. It should also be kept in mind that animal models may have the ability to elucidate gross behavioural patterns with a long phylogenetic history, such as libido, appetite or hyperactivity, but they may not be as helpful for complex human phenotypes that have perhaps only evolved over the last few hundred thousand years (Russell 2011) [27].

History of ADHD Evolutionary Theories

The idea that ADHD could have perhaps once been adaptive seems to have first been suggested by Thom Hartmann in 1993 [28]. He made the intuitive suggestion that ADHD could have once been useful for ancient hunters; however, the advent of agriculture around 8000 years ago reduced the selection pressure on the ADHD phenotype. This is not an unreasonable notion; evolutionary theorists would specifically describe this dynamic as directional evolution in transit – meaning that we are catching evolution in progress.

In 1996, Shelley-Tremblay et al. [29] proposed that many ADHD traits would be useful for a hominid warrior. They put forward a general suggestion, "As the African sapiens pushed northward, they would have been in direct competition with the Neanderthals for access to food supplies. As has often been the case, this conflict over resources could have led to war. In this case, a fierce, energetic human with reduced inhibitions would have been ideal".

In 1997, Jensen et al. [30] suggested that both hunting and warfare could be related to ADHD – hyperactivity to facilitate hunting behaviours and impulsivity to foster war. Moreover, they clearly articulated the need to solve the boundary between "disorder" and "health" (which perhaps could be more accurately described as "disease" versus "phenotype").

In 2005, Williams and Taylor [31] were the first to explicitly notice a specific evolutionary conundrum with ADHD. Why was ADHD only present in a minority of individuals? They invoked a group selection argument, which is appropriate in this context. They suggested that hominid groups could benefit if risk-taking were partitioned into only one segment of society. Although not explicitly stated, they must have assumed that ADHD is a categorical phenotype rather than a dimensional one.

In 2008, the eminent evolutionary psychiatrist Martin Brune (2008) [32] proposed that a 7-repeat variant of the dopamine D4 receptor gene may have resulted in increased novelty-seeking behaviour (and risk-taking) during a previous period of environmental uncertainty in hominid history.

Le Cunff (2024) [33] proposed that "high trait curiosity" is the core ADHD phenotype, which was previously more adaptive in our ancestral environment compared to the modern industrialized world.

Annie Swanepoel and co-authors (Swanepoel et al. 2017 and Swanepoel et al. 2022) [34,35] have recently advocated for evolutionary theories to be incorporated into clinical practice, and that "an evolutionary view can help both professionals and patients understand ADHD in a broader sense, where it can be thought of as both a liability and a strength and where attempts are made to adapt the environment before resorting to medication to adapt the individual."

Evolutionary Analysis of ADHD

Many prior claims that certain behaviours could be evolutionary advantageous have often lacked scientific rigour. This has made such ideas susceptible to being called "just-so stories". However, this criticism has sometimes been unfair, especially when researchers have acknowledged the hypothetical nature of their assertions. One way to elevate a hypothesis (i.e. just-so story) into a proper theory is by ensuring that all of the propositions (and ramifications) are consistent with all components of evolutionary theory (Stevens and Price 1996, McGuire and Troisi 1998) [36,37]. For a biological supposition to approach scientific validity, it must integrate nicely into all of nature's operations, from genes to physiology

to population dynamics. Accordingly, any discussion about potentially evolutionarily advantageous traits must consider the environment of ancient hominid societies, commonly described as the environment of evolutionary adaptiveness (EEA). This is where the candidate selection pressure can be found. Although debatable, we can reasonably propose that ADHD is not a disease because of its high heritability and high prevalence in fecund populations – meaning that it is reasonable to frame ADHD as a phenotypic trait. The propagation and maintenance of any phenotypic trait requires absolute consistency with evolutionary theory.

The theory of evolution involves three broad components: replication, variation and competition. Genes are imperfectly replicated, resulting in variation, and followed by competition between these varied traits. Then, those "best" traits are preferentially replicated, round by round (through many generations), resulting in a gradual transition towards ideal forms. Darwin called this process "descent with modification" (Darwin 1859) [38], but it was later termed evolution (or expressed as "survival of the fittest"). These evolutionary processes (replication, genotypic/phenotypic variation, and competition) propagate favoured phenotypes but also tolerate hindrances (i.e. medical diseases). Let's examine how ADHD may interact with these fundamental components of evolution.

Replication

The basic mechanisms of genetic replication were worked out by theoretical contributions from Gregory Mendel and microbiological analyses by Watson and Crick. There is no evidence to suggest that ADHD is caused by a specific pathology during genetic replication. Instead, ADHD appears to be a polygenic phenotype.

Variation (genotypic)

Genotypic variation can often, but not always, lead to phenotypic variation. Nature has many ways to introduce genetic variation (Wool 2006; Mousseau et al. 2000; Jablonka and Lamb 2006; Hallgrimsson and Hall 2005) [39-42]. In addition to singular mutations, another method is, for example, copy number variation. This is a genetic mechanism where copies of small segments of the genome are added or subtracted, sometimes resulting in enhancing a phenotype but, at other times, introducing disadvantages (interpreted as a disease). There is yet no evidence of any genetic pathology in ADHD.

Variation (phenotypic)

If evolutionary processes were simple, one would expect nature's most advantageous genes to be predominant; however, nature contains much more variation. A well-known example is the sickle-cell anemia balanced polymorphism, which maintains a disadvantageous sickle-cell gene because heterozygotes demonstrate the greatest survivability in malaria-infested environments (Allison 1954) [43]. Several evolutionary mechanisms can support the maintenance of more than one gene at a specific allele, with the major ones being 1) genetic drift, 2) directional evolution in transit, 3) mutation-selection balance, 4) negative frequency-dependent selection, 5) temporal-spatial variation selection, 6) antagonistic pleiotropy, and 7) heterozygote advantage (Keller and Miller 2006, Polimeni 2012) [12,13]. If ADHD is a categorical diagnosis (i.e. a phenotype distinct from others), it should ideally be explained by one of these mechanisms. It is not yet known whether any of these evolutionary mechanisms applies to the ADHD phenomenon.

Competition:

It is widely accepted that organisms typically compete on an individual basis; however, organisms can also compete alongside their kin against others (i.e. kin selection) or between groups (i.e. group selection) (Wilson and Sober 1994) [44]. The clustering of any phenotype within a species, such as sterile worker bees or primate hierarchies (e.g. alpha males), could be reinforced through group selection (also known as multilevel selection). The evolutionary raison d'etre of a phenotype can sometimes implicate one of these mechanisms.

Dimensional versus Categorical Diagnoses:

When it comes to psychiatric diagnoses, researchers have generally considered either dimensional or categorical approaches (Kraemer et al. 2004) [45]. Because of the relatively indistinct nature of most psychiatric conditions, it is not obvious whether a dimensional or categorical approach is more appropriate. Psychiatrists normally view psychiatric conditions as categorical conditions; either you have it, or you don't - while acknowledging a small minority of borderline cases. Even dimensional approaches tend to only consider a small subset of the population. However, in his book The Pattern Seekers, Simon Baron-Cohen introduces the idea that perhaps everyone has autistic traits around a Gaussian mean (Baron-Cohen 2020) [46]. This may sound sensationalistic, but to some degree, all humans are mechanically minded - a core feature of autism. The least mechanically-minded individuals may seem to lack the trait, but only compared to the rest of us. Perhaps, if compared to some other hominid species, it would be more obvious that everyone had some autistic traits around a Gaussian distribution.

Similarly, all humans can be distractible, active or impulsive. Is it possible that we all have the ADHD phenotype? However, those of us with the least of these traits are seen as having no ADHD. The fact that genetic research is beginning to show that hundreds of genes (or more) are associated with certain highly heritable psychiatric conditions (i.e. ADHD, autism, schizophrenia, and bipolar disorder) is consistent with such a dimensional concept. There is also a distinct possibility that stimulants may improve concentration in all individuals, although the precise magnitude of cognitive enhancement has not yet been clarified (Wood et al. 2014; Weyandt et al., 2018; Ilieva et al. 2012) [47-49].

It is also possible that both dimensional and categorical mechanisms could apply. Take schizophrenia, where increasing schizophrenic liability genes could make someone more genotypically schizophrenic, but the triggering of a psychotic episode may represent a categorical phenotype. Could there be similar thresholding effects that have the potential to amplify the ADHD phenotype in a subset of the population? The ethological concept of fixed action patterns may very well apply to psychiatric conditions. A fixed action pattern is an instinctual series of complex behaviours, commonly found throughout the animal kingdom. Examples include beaver dam construction, dolphin acrobatics, squirrels methodically hiding nuts and the waggle dance of honeybees. Both genetic predisposition and environmental influences determine whether a fixed action pattern is triggered.

In psychiatry, researchers sometimes divide psychiatric conditions into state versus trait. For example, depression is usually temporary, and so it is generally considered a state-like condition - perhaps akin to a fixed action pattern (depression appears to be caused by a genetic vulnerability interacting with the environment, usually some type of loss). In contrast, psychiatric conditions like personality disorders or autism are ever-present and are considered to reflect persistent traits. It appears that ADHD behaves more like a trait, but it is also conceivable that certain environments could intensify ADHD-like behaviours. For example, could the anticipation of battle enhance environmental vigilance, and increase distractibility and other ADHD traits?

Hypothesis: ADHD Could Enhance Warring Capabilities for Both Individuals and the Group

A critical question is, what evolutionary problem did ADHD solve around the time the behaviour spread through our species (acknowledging we have no solid evidence about possible phylogenetic timelines)? In surveying the anthropological literature, there appears to be some evidence supporting previous researchers who have suggested ADHD makes better warriors. I would specifically add, frontline warriors associated with ritual wars conducted by Homo sapiens. To fully appreciate how powerful ADHD may have been, it will be helpful to examine ancient warfare and the purported huge selection pressures that made Homo sapiens better at war.

Zoologists believe that the primary reason animals form herds is for protection from predators (Hamilton 1971; Miller 2002, 96) [50,51]. The protective features of herding may allow more members to watch for threats or mob attacking predators. Predatory animals, in contrast, tend to be solitary or prowl around in small groups. Hominid evolution is accompanied by a transition from being a prey species to being one of a more predatory disposition, although experts do not agree precisely when our ancestors graduated from scavenger to hunter. Contemporary human tribes (sometimes consisting of several clans) typically have about 150 individuals. So, who could have been the primary predator of ancient hominids? Humankind's greatest predator seems to have been other humans. Over the last few decades, archeologists have discovered that ancient violence may have produced death rates approaching 30% (Keeley 1996, 91; Maschner 1997, 273) [52,53], especially as a result of inter-tribal warfare. Even if such death rates were half that figure, it would still mean selection pressures through warfare were significant for ancient man.

There is much converging evidence to suggest that humans are behaviorally "designed" for war (Bowles 2009, Kissel and Kim 2018, Glowacki et al. 2020) [54-56]. Homo sapiens often experience three distinct positive emotions that seem to be especially associated with the act of war: excitement (the adrenaline rush of battle), camaraderie and glory. These agreeable feelings are also the cornerstones of team sports (as well as shooter video games). If an alien were to observe team sports with a dispassionate eye, that alien might wonder why earthlings bother with such spectacles. It is our instinctual predilection to side with our group and battle with others (with all the associated emotional highs and lows) that perhaps best explains everyone's passion for team sports. In fact, so powerfully attractive are these emotions that they not only imbue the participants with them but also the spectators. This kind of evolutionary pre-wired disposition to battle would have been immensely helpful in the face of tribal threats.

Military theorists of ancient warring identify two types of war - ritualistic war (perhaps to test the strength of an opposing tribe) and ambushes (Van Crevald 1991) [57]. Ritualistic war was almost always conducted by males although females could on occasion be warriors (Keeley 1996,35) [52]. It would seem that distractibility would allow frontline warriors to scan the war front and contend with the chaos of it. Impulsivity and hyperactivity could help in decisively attacking others, and allow certain groups of warriors to gain the upper hand. ADHD could have been particularly helpful as Homo sapiens tribes grew, and war became more chaotic. Notice that frontline warriors would have typically represented a small subset of male warriors, which may explain ADHD's distribution through the population (keeping in mind that due to evolutionary constraints, females would have also had ADHD to pass these traits to their sons).

Not all ADHD traits have to necessarily support warring functions. Nature is an old pro at recycling traits for alternate purposes. Take the tongue, for example; its original function was for digestive purposes, but it was later co-opted for language functions in hominids (this common evolutionary process is called exaptation). Similarly, certain ADHD traits, like creativity, could have been intensified at phylogenetically later dates.

Conclusion

The author proposes that evolutionary principles must be applied to any psychiatric condition that is both highly heritable and common (in fecund populations), such as ADHD. Prior hypotheses proposing that the phenomenon of ADHD augmented war-like behaviours appear to be consistent with evolutionary theory and the anthropological record. Moreover, it is not yet known

whether ADHD represents a categorical diagnosis or a dimensional phenotype; but if ADHD represents a dimensional phenotype, it could mean that some degree of ADHD expression resides in all of humanity.

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