

RESEARCH PAPER

Entrepreneurship, mental disorders and the saga of the adventure gene: debunking the fad of hardwired entrepreneurship

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ABSTRACT

This paper disputes the thesis that a self-reported mental condition of apparent genetic origin makes its carriers entertain entrepreneurial intentions and assesses its implications for entrepreneurship-driven economic development. The findings are that the research in question mistakes true causative agents for superficial cause-hiding vectors, thus confusing a self-reported attention-deficit/hyperactivity disorder (ADHD) condition with the cause of entrepreneurial intentions. This is all the more so because ADHD was not properly accounted for, relying instead on the Global University Entrepreneurial Spirit Students Survey (GUESSS), self-administered without diagnostic evidence. Thus, entrepreneurship has been turned on its head, being misrepresented as a hardwired, innate human trait instead of a course of action that hinges on subjective preferences that can be influenced by the structure of rewards shaped by public policy. This misrepresentation may dwell in the so-called ‘hardness bias’, which underestimates the softer but crucial test of the quality of conjectures. The originality of this paper lies in the use of the harmful-dysfunction analysis, showing that the entrepreneurship research in question is based on a bogus notion of disorder.

KEYWORDS

entrepreneurship, causality, ADHD, GUESSS, entrepreneurship policy, fad, debunking

Introduction

[I]nduction – the formation of a belief by repetition – is a myth. It was first in animals and children, but later also in adults, that I observed the immensely powerful *need for regularity* – the need which makes them seek for regularities; which makes them sometimes experience regularities even where there are none; which makes them cling to their expectations dogmatically; and which makes them unhappy and may drive them to despair and to the verge of madness if certain assumed regularities break down. (Popper, 1971, pp.189–90)

Few scientific fields can match the development of genetics, where the mysteries of procreation were puzzled out in the mid-twentieth century by Watson and Crick’s (1953) discovery of a

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molecular mechanism for the replication of DNA, eventually leading to the mapping of all human genes, or genome. The breadth and depth of knowledge about the human genome have brought genetics very close to the ideal of deductive nomological science, where solutions to puzzling phenomena can be deduced from an extant body of knowledge, thus tumbling out of the conceptual framework used to make observations (Hempel, 1966; Hanson, 1967). As put by Hood and Galas (2003, pp.445–6):

[I]t illustrates the concept of ‘discovery science’ – the idea that all the elements of the system (that is, the complete genome sequence and the entire RNA and protein output encoded by the genome) can be defined, archived in a database and made available to facilitate hypothesis-driven science and global analysis.

The genome and the way it lends itself to hypothesis-driven science have stirred up hopes for a predictive biological science, stimulating a short-lived exchange about the discovery of an ‘adventure gene’ and the complex behaviours it apparently determined, which is recounted in the next section. The exchange was short-lived because determinism, however desirable for a hypothesis-driven science, does not follow a one-direction pathway from molecules to phenotypes. After all, it has been a merit of biological science to shed light on the interactions of multiple levels of biological organisation, where biochemical processes are determined from downstream by multiple genes acting together, and from upstream by lifestyles, known as ‘epigenetic mechanisms’ (Simmons, 2008). It is thus not surprising that the saga of the adventure gene has sagged in the natural sciences (Baron, 1998).

Against the grain, recent research has turned entrepreneurship on its head, from a course of action that hinges on the subjective preferences of individuals and is influenced by the structure of rewards shaped by public policy (Baumol, 1993; Baumol *et al.*, 2011), contingent on institutional settings (Boettke and Coyne, 2007), to a hardwired, innate hypothetical human trait attributed to individuals apparently suffering from attention-deficit/hyperactivity disorder (ADHD), a condition characterised by a relentless pattern of inattention and/or hyperactivity-impulsivity that hinders an individual’s functioning (American Psychiatric Association, 2013).

Such a hypothetical hardwired trait is pre-strategic venturing without intentionality, which has been concocted to make sense of a statistically significant link between a self-reported ADHD condition and entrepreneurial intentions found in data collected by the Netherlands chapter of the Global University Entrepreneurial Spirit Students Survey (GUESSS) (e.g., Verheul *et al.*, 2015, 2016; Lerner *et al.*, 2017, 2018a, 2018b, 2019; Wismans *et al.*, 2020). Highly penetrant so-called monogenic phenotypes (i.e., a single gene whose strong influence is expressed in physical or behavioural traits notwithstanding epigenetic mechanisms) are, however, known for being rare (Chial, 2008). It remains to be explained why ADHD-afflicted individuals would not find equally adaptive environments in roles as diverse as, say, a videogame tester, an extreme sports athlete or a seasoned leisure traveller.

Drawing lessons from epidemiology, psychiatry and the philosophy of science, this paper points out serious gaps in the way such a link has been conjectured and tested, as well as its implications for public policy. Although the apparent link in question falls within the conceptual stage of the public policy cycle, it is not far from the very end of the cycle where a course of action is enacted by governments to bear upon entrepreneurship-driven economic development. It is thus crucial to anticipate such a stage by assessing findings that have been based on self-administered surveys without diagnostic evidence.

In a nutshell, this paper is composed of two parts that posit, on the one hand, that the entrepreneurship research in question mistakes true causative agents for superficial cause-hiding vectors with the unintended public-policy consequences of playing down both the harmful effects of ADHD and the potential consequences of pathological levels of risk-taking. On the other hand, ADHD was not properly accounted for, relying instead on self-administered surveys without

diagnostic evidence. The latter is, anyway, hard to come by in the case of syndromally-defined disorders that lack an indisputable origin in biological or brain algorithmic dysfunctions.

The sagging saga of the adventure gene

With the genome project underway in 1997, a call for a moratorium on case-control genetic association studies – employed to probe the influence of a gene variant, or polymorphism, on temperament – was issued by Paterson (1997). What could have motivated such a call in the era of the genome? It was the enthusiasm generated by the apparent association between a variant of the D4DR gene and human behaviour. In fact, such an association was thought to have been found between a gene variant and a certain temperament, instead of overt behaviour properly speaking.

The difference between temperament and overt behaviour is important because the personality traits thought to originate in crude temperamental biases do not invariably manifest in pre-specified behaviours (i.e., the relationship between temperament and behaviour is far from being linear). Thus, Cloninger *et al.* (1996) posited that: ‘even weak correlations among a network of multiple heritable dimensions produces complex non-linear dynamics because each person responds to experience with multiple motivations, which sometimes conflict’ (p.3), claiming instead that:

it may be more fruitful to map genes contributing to temperament, which has a relatively simple genetic architecture and can be quantified easily and reliably by questionnaires. Later susceptibility to complex disorders like schizophrenia and alcoholism can be evaluated in terms of risk from heritable personality traits and possible disease-specific factors. (p.4)

Their view, however, may have come across as too optimistic according to Baron (1998). This is because shortly after the publication of Cloninger *et al.* (1996), the systematic review of Ebstein and Belmaker (1997) revealed that the search for an association between the L-D4DR gene variant and novelty-seeking (including population samples of alcoholics and heroin addicts, whose personality traits are prominent in novelty-seeking) had produced inconsistent results. Some studies found an association while others did not. Ebstein and Belmaker (1997, p.381) concluded that:

the signal generated by L-D4DR on temperament is modest enough (effect size: $\eta^2 = 5\%$) such that the noise generated by demographic or methodological differences between studies ... may be sufficient to obscure the weak effect of this gene on personality. It should also be kept in mind that the nature of small gene effects on complex traits is such that any particular polymorphism like L-D4DR may be neither necessary nor sufficient in the determination of the NS [novelty-seeking] phenotype.

Thus, the saga of the adventure gene – as dubbed by Ebstein and Belmaker (1997) – was sagging according to Baron (1998). The enthusiasm for a tentative association between the L-D4DR gene variant and novelty-seeking is difficult to explain in the light of extant knowledge about genetic influence on phenotypes. For the L-D4DR variant to be highly penetrant (i.e., to strongly influence behaviour), it must code for a single and distinctive personality trait, similar to the shapes and colours of Gregor Mendel’s pea seeds. These so-called monogenic or single-gene phenotypes are rare (Chial, 2008), however. Even phenotypes like rooster combs, petal colours, and horse coat colours are known to be the product of the interaction of at least two genes (Miko, 2008), known as epistasis (Frankel and Schork, 1996). If that were not enough, gene expression is known for being either switchable, turning on and off, or rheostatic (Beaudet and Jiang, 2002), with varying degrees between two possible extreme values, acting through epigenetic mechanisms (Simmons, 2008) which comprise biochemical processes that can be triggered by environmental factors brought about, for instance, by lifestyles (Egger *et al.*, 2004).

Against this backdrop, it is surprising to see how a link between a self-reported ADHD condition and entrepreneurial intentions in GUESSS has given rise to the idea of hardwired entrepreneurial intentions or ‘pre-strategic venturing without intentionality’ (Lerner *et al.*, 2017, p.5; 2018a, p.64). Thus, the entrepreneurship research in question claims to have confirmed that ADHD-afflicted individuals are significantly more likely (Verheul *et al.*, 2015) or almost twice as likely (Lerner *et al.*, 2019) to espouse entrepreneurial intentions, as well as to be self-employed (Verheul *et al.*, 2016). While the causal language in Verheul *et al.* (2016) is less salient, Verheul *et al.* (2015, pp.85, 89), in contrast, claim to ‘predict’ the aforementioned link and, perceiving a good fit between entrepreneurship and ADHD-like behaviour, assume that it is because of ADHD that subjects are likely to have entrepreneurial intentions.

The claims of Lerner *et al.* (2019) are even stronger, despite initially denying a causal pathway. Setting out to examine if the link is ‘true’ and ‘veridical’, they conclude that ‘the results provide a straightforward test and clear support for a positive link between ADHD and entrepreneurship’ (p.386), and that their quantitative examination ‘squarely tests the effect of *attention deficit/hyperactivity disorder*’ (p.390). Taking stock, Lerner *et al.* (2018b) submit that ‘not only do individuals with ADHD think about entrepreneurship more often than do individuals without ADHD, but also that they are likely to have greater intrinsic interest in entrepreneurial ideas’ (p.273).

Later, hopping on the bandwagon, Peltonen *et al.* (2020) posited that a positive relationship between ADHD and entrepreneurship is supported by the inverse correlation they claim to have found between ADHD medication intake and the probability of launching startups. They concede, however, that their dataset is blind as to medication consumption being conditional on the diagnosis of ADHD. The latter point disputes Peltonen *et al.*’s (2020) findings because ADHD medication is overprescribed to treat an array of other conditions (Sinita and Coghill, 2014; Sibley, 2018).

ADHD is, however, a mental disorder whose status is syndromal only (i.e., without clear-cut etiological evidence). Although a certain gene variant, the seven-repeat allele of the DRD4 gene (DRD4/7R), has been tentatively found to be associated with ADHD-like personality traits (Ding *et al.*, 2002), it also happens to be significantly correlated with people migrating over long periods, from 1,000 to 30,000 years in the past, like the indigenous populations of the Americas (Chen *et al.*, 1999), as well as with alcoholism and heroin addiction more generally (Ebstein and Belmaker, 1997). Most recently, 12 loci (i.e., the location of genes on a chromosome) have been proposed as candidates for influencing ADHD (Demontis *et al.*, 2019), making this personality trait a moving target.

Positing the existence of hardwired entrepreneurial intentions is problematic because even Alzheimer’s, a disorder whose etiological origin counts on more solid grounds, may manifest neither when the individual has only the appropriate genetical makeup (i.e., the gene APOE4) nor when the individual has only been exposed to the appropriate environmental factors (i.e., the herpes simplex virus) (Robson, 2021). Moreover, it remains to be seen whether a given gene is dysfunctional (i.e., the product of the breakdown of a natural evolutionary mechanism) (Wakefield, 1992, 2010; Cosmides and Tooby, 1999; Agafonow and Perez, 2020).

Methodological underpinnings

In the midst of academic management systems that misguidedly adjudicate rewards based on imperfect proxy measures (Agafonow and Perez, 2023, 2024), it may not come as a surprise that systematic reviews are being added to the social scientist’s toolkit, for it has long since been noted that review articles tend to attract more citations (Seglen, 1997; DORA, 2020). Systematic reviews, however, have a very narrow range of applicability, namely, they are of service only when the papers reviewed share qualities that are homogenous and, thus, additive. This is a precondition fulfilled by papers that focus on the same questions while relying on the correlational analysis of samples whose underlying variation is small. Only then can the ‘uncertainty baked into the definitions of units’ (Bernhard, 2018) be overcome to make the comparison of effect size possible and the conclusions about the reliability of findings meaningful (Uman, 2011).

On the contrary, the present paper trades off breadth (more observations) for depth (greater detail), analyzing the inferential ramifications of a small number of articles which, owing to their empirical methods and the large samples they tap, have attracted a sizeable number of citations from researchers interested in the apparent implications of mental disorders for entrepreneurship. Thus, these papers (i.e., Verheul *et al.*, 2015, 2016; Lerner *et al.*, 2019; Wismans *et al.*, 2020), which in Figure 1 appear in green, are the baseline of my critique and their findings are exhaustively analyzed herein along with the arguments of a relevant subset of follow-up papers.

Figure 1, which is a network generated by an online citation mapping tool, is meant to give a taste of the reception of the baseline papers which, since their publication and until December 2023, count on citations involving 49 other papers dealing with neighbouring subjects. Verheul *et al.*'s (2016) paper is the only one that appears detached from the network, perhaps because it was published in an epidemiology journal and, therefore, it may have escaped entrepreneurship academics' attention. Also, the baseline papers include a total of 13 co-authors with only three of them (Joern Block, Roy Thurik and Ingrid Verheul) co-authoring two or more such papers.

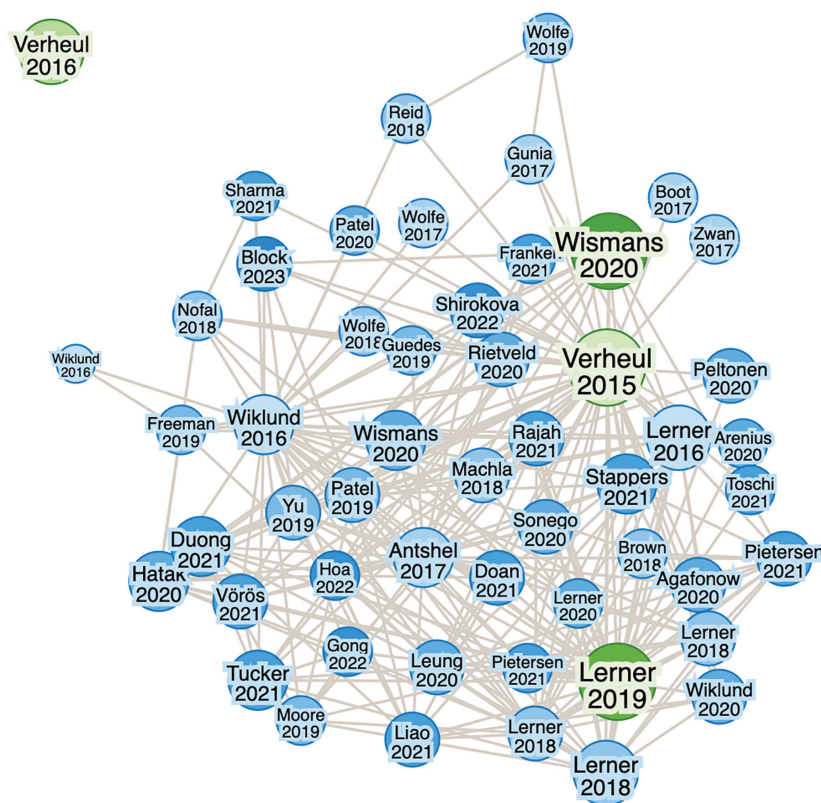


Figure 1. Citation network of the baseline articles (in green)

Source: Research Rabbit.

The hypothesis here is that the reception of the baseline papers may have been favoured by the so-called 'hardness bias' (Akerlof, 2020), which, in the mistaken belief that quantitative methods always lead to more precision, rejects 'softer tests of theories, such as those that evaluate models based upon the quality of their assumptions as well as the quality of their conclusions' (Akerlof, 2020, p.408). As the present paper shows, data and the methods to treat them are subsidiary to the quality of conjectures (McMullin, 1992; Popper, 2002), which, despite being softer, is a crucial test which both the baseline papers and their follow-up papers have failed.

The dangers of formulating public policies based on cause-less correlations

The economic burden of ADHD on public coffers has long since been recognised (Matza *et al.*, 2005). Thus, it is just a matter of time until governments connect the dots to formulate public policies that go beyond health concerns only. For instance, in 2018 the Australian government's Productivity Commission inquired into mental health and studied ways to enhance the country's productivity by supporting the economic participation of ADHD-afflicted individuals. On that occasion, a report commissioned from the consulting firm Deloitte concluded that 'There are likely to be substantial opportunities for targeted policy interventions to help mitigate this costly condition' (Deloitte, 2019, p.47).

A pervading problem in public policy is how to hit the sweet spot that improves aggregate net benefits while making no one worse off, known as 'Pareto optimality' (Dunn, 1994; Weimer and Vining, 1999) after the early twentieth-century Italian sociologist, Vilfredo Pareto. Most public policies targeted at entrepreneurship are not Pareto optimal, however, because achieving the desired effect on the subjective preferences of people to make them venture involves a change in the structure of rewards that inherently worsens someone. For instance, subsidies or tax breaks that make launching ventures more enticing impose liabilities on taxpayers.

However uncommon, Pareto optimal public policies could be within reach if only the people made worse off could be compensated by the policy winners, thanks to a tweak known as the 'Kaldor–Hicks efficiency criterion' (Dunn, 1994; Weimer and Vining, 1999) after the eponymous economists. If worse-off people were compensated, the feasibility of public policies would increase because aggrieved people would have no reason to raise objections. It is known, however, that this tweak opens a can of worms because aggrieved people would have a reason for lobbying; that is, trying to capture policymakers to pre-empt public policies in the first place (Buchanan and Tullock, 1965).

The holy grail of public policy is one that overcomes the limits of both Pareto optimal policies and Kaldor–Hicks compatible policies; that is, a policy that brings about both aggregate net benefits and public money savings, in practice making everyone better off while giving no reason for lobbying policymakers (Buchanan and Tullock, 1965). Finding a true link between ADHD and entrepreneurial intentions would, *ex hypothesi*, hold the key to such a supreme public policy, and it is already being toyed with.

Finding inspiration in the work of the authors under scrutiny, Peltonen *et al.* (2020) inquire whether increasing the use of ADHD medication abates the formation of new businesses, thus weakening entrepreneurship-driven economic development. If such a link were confirmed, a public policy that aims at reducing the intake of ADHD medication would both save public money and enhance economic development, which is very close to the above-mentioned policy holy grail. Despite the strong confirmation bias of Peltonen *et al.* (2020), they stop short of making such a recommendation for the reasons that will be clear next.

As expected, Peltonen *et al.* (2020) report a statistically significant inverse correlation between patterns of ADHD medication consumption and the probability of launching startups. This statistical finding is, however, reported alongside a cursory disclaimer, for data were not sifted to capture medication consumption based on ADHD diagnosis only. This point is crucial because the stimulants that make up the cocktail of drugs for the treatment of ADHD are being overprescribed:

as a first resort for underperforming schoolchildren, to adolescents and young adults seeking cognitive enhancement, and to women interested in weight loss ... A related concern is that stimulant medications are becoming default treatments for a broad range of cognitive-behavioural symptoms, which might prevent patients from receiving optimal treatment (e.g., detoxification, behavioural sleep therapy, or antidepressant medications) on the basis of the cause of their difficulties. (Sibley, 2018, p.1)

Time and effort are being channelled into seeking a statistical fit between ADHD and entrepreneurial intentions that, ignoring the true mechanisms at work, aims to have a say in public policy

matters. Next, this paper expounds on the perils of formulating public policy of relevance for entrepreneurship based on black box accounts; that is, a statistical fit between prediction and response variables that ignores the true mechanisms at work. These perils are two-fold.

First, there are countless fortuitous and cause-less correlations that can put one's research off the trail. For instance, the more Nicolas Cage appears in films, the higher the number of people who drown by falling into a pool. Also, the per capita consumption of cheese is highly correlated to the number of deaths by getting tangled in one's own bedsheets. These are but two spurious correlations compiled by Tyler Vigen as part of a project to see the funny side of correlations (Vigen, 2019), lending credence to the argument that it takes far more than surveying large samples to rule out spurious correlations.

Second, a statistical fit alone can at best point out superficial cause-hiding vectors that disregard the true causative agents behind the onset of a phenomenon. For instance, pregnant women who went into labour in the streets after failing to reach the Vienna General Hospital in the nineteenth century were spared from childbed fever transmitted in the hospital. Yet a public policy aimed at compelling childbirths in the streets to prevent deaths from childbed fever, at a time when the causes of infectious diseases were unknown, would have raised eyebrows even in the nineteenth century. Thus, it will be shown next that the apparent link between self-reported ADHD and entrepreneurial intentions is likely to play down the harmful effects of ADHD suffered by individuals, as well as to underestimate the public effects of pathological levels of risk-taking.

The problem of conjecturing and testing black box hypotheses

A correlation between a self-reported ADHD condition and entrepreneurial intentions falls short of pinning down what part ADHD plays in the formation of entrepreneurial intentions, or if ADHD plays a part at all. The problem lies in the way the association between ADHD and entrepreneurial intentions has been conjectured and tested, which parallels problems long recognised and tackled in the field of epidemiology. To start with, trying 'to squarely test' – as Lerner *et al.* (2019, p.390) say – the effect of ADHD on entrepreneurial intentions is like trying to explain hypertension based on the interaction of quarks (Kendler, 2005), which begs the question of the suitability of the level of explanation chosen (Agafonow, 2018).

The level of explanation refers to nature's hierarchic or nested structure. Nature is organised in structures with different levels or layers, which have been compared to nesting dolls or Chinese boxes (Simon, 1996; Susser and Susser, 1996a; Agafonow, 2018); that is, wooden structures of graduated size that encapsulate each other from the outermost box to the innermost, smallest box. Being a social phenomenon, entrepreneurship is located at the outermost boxes, trying to establish a true correlation with an innermost phenomenon like ADHD. This may be self-defeating if epigenetic mechanisms (Simmons, 2008) are overlooked; that is, it remains to be explained why ADHD-afflicted individuals would not find equally adaptive environments in roles as diverse as a videogame tester, an extreme sports athlete or a seasoned leisure traveller. If too many levels of explanations are jumbled, almost any response variable located at a given box may be linked to prediction variables located at a distant different box.

This was the problem with the miasma theory in the nineteenth century, which explained mortality and morbidity with recourse to foul emanations from the soil, air and water, instead of specific diseases (Susser and Susser, 1996b). It was also the crux of the apparent link between street births and puerperal or childbed fever mortality in the nineteenth-century Vienna General Hospital (Hempel, 2000; Scholl, 2013). In Vienna, Ignaz Semmelweis was confronted with the puzzle of an abnormally high rate of mortality caused by childbed fever among women in labour admitted to the hospital's first maternity division, compared to the second division.

There was, however, a subgroup of women in the first division that was spared by childbed fever, namely, women who gave birth in the street because they lived far away and went into labour on their way to the hospital. Hypothesising giving birth in the street as the reason why this group of

mothers had a lower incidence of childbed fever is indeed a test implication borne out by the correlation between street births and lower childbed fever mortality (Hempel, 2000; Scholl, 2013). Yet Semmelweis questioned this hypothesis – among many others – when he realised that these women were not being examined by doctors upon admission because they arrived at the hospital with their babies already in their arms.

That there was evidence to credit street births with the lack of childbed fever mortality (i.e., there was a lower mortality rate among women who did not reach the hospital on time to give birth) does not mean that childbed fever is caused by giving birth in the hospital's first maternity division as such (Hempel, 2000). By the same token, that a self-reported ADHD condition is to be found mostly among students entertaining entrepreneurial intentions does not mean that entrepreneurial intentions are necessarily caused by ADHD. The latter is, however, what the entrepreneurship research in question predicates.

Epidemiologists have long recognised the problems of fitting statistical models to epidemiological data. A fit may be found between prediction and response variables even when a different function underlies the real mechanisms at work. Were not street births and childbed fever mortality correlated after all, even if the true cause of childbed fever was to be found in a different pathway that involved a yet unknown bacterium?

This property may delight the mathematician but should terrify the scientist; for it means that a purely 'black box' description of a system by regression analysis, using some function for no better reason than that it gives an optimal fit, may tell us nothing at all scientifically ... It is obvious, then, that in practice, excellence of fit of some function does not guarantee insight. (Murphy, 1978, p.8)

Thus, to posit that individuals who self-report to be ADHD-afflicted are intrinsically more likely to entertain entrepreneurial ideas based on the fitness of a statistical model alone (Lerner *et al.*, 2018b, 2017) is unwarranted, which undermines the reliability of public policies that thus claim to heighten entrepreneurship-driven economic development.

Superficial cause-hiding vectors

Public policies of dubious benefit would follow if only a statistical fit between prediction and response variables were enough to inform policy interventions. For instance, should the apparent negative correlation between childbed fever mortality and street births be taken seriously, a public policy that let pregnant women make do without medical assistance, leaving them to their fate in the streets, would have brought about a reduction in childbed fever mortality (Hempel, 2000; Scholl, 2013). The reason for this is to be found in the paucity of the explanatory power of black box descriptions inherent in the goodness of fit of a statistical model. By itself, such a statistical fit can only point out superficial cause-hiding vectors that are often far removed from the true causative agents that give rise to a phenomenon. In the case of childbed fever mortality, giving birth in the street and, therefore, skipping medical examination was just a vector that incidentally carried the benefit of preventing the contracting of childbed fever, with the true causative agent being present in a yet unknown bacterium (*streptococcus pyogenes*).

Thus, it is only reasonable to ask where the causative agent of entrepreneurial intentions lies and whether the statistical fit between self-reported ADHD and entrepreneurial intentions can reasonably be deemed to signal the presence of such an agent. The answer given by the entrepreneurship research in question is undoubtedly affirmative; that is, the authors posit that ADHD is a distinctive disorder that can be considered a causative agent of entrepreneurial intentions, or to recall once again the assessment of their own research made in hindsight, 'not only do individuals with ADHD think about entrepreneurship more often than do individuals without ADHD, but also that they are likely to have greater intrinsic interest in entrepreneurial ideas' (Lerner *et al.*, 2018b, p.273).

To put the authors' assessment in context, ADHD is, in their account, a disorder that strongly predisposes afflicted individuals to entertain entrepreneurial intentions. Given this strong effect on the human behavioural phenotype, should ADHD be genetically determined after all, it would make entrepreneurship, or at least its weak form of entrepreneurial intentions, a monogenic or single-gene phenotype (Chial, 2008) equivalent to the shapes and colours of Gregor Mendel's pea seeds, able to offset the manifold influence of environmental factors acting through epigenetic mechanisms (Egger *et al.*, 2004; Simmons, 2008).

A problem with this account is that it may inform public policy of dubious benefit, tantamount to leaving pregnant women to their fates in the streets without medical assistance, as discussed above. For instance, ADHD-afflicted individuals could become the targets of policies aimed at encouraging the creation of new ventures while playing down the harmful effects of the condition on these individuals (Agafonow and Perez, 2020). Recall that Peltonen *et al.* (2020) were just a step away from recommending the reduction of ADHD medication intake based on a cause-less inverse correlation with the probability of launching startups. Despite the apparent benefits conferred by ADHD in the narrow occupational contexts where entrepreneurial intentions may be brought to fruition, the overlooked suffering of the individuals concerned may offset the benefits.

Besides the suffering of afflicted individuals, the capital they would be entrusted with to launch ventures may require far more than a venturesome personality to pay off. Evidence is emerging that when risk-seeking is distinguished from suboptimal decision-making, ADHD-afflicted individuals do not score differently from control groups in risk-seeking. This suggests that ADHD, instead of involving a higher tolerance for risk, perhaps 'involves some disruption in the perception of the choice outcomes, which may lead to non-optimal choices' (Shoham *et al.*, 2016, p.5). In fact, unreasonable risk-taking has been linked to the cognitive effects suffered by traders and asset managers from chronic exposure to steroid hormones (Coates *et al.*, 2010), which begs the question: Is a venturesome behaviour enough to engage in entrepreneurial activities? Thus, the obvious problem is whether the statistical fit between self-reported ADHD and entrepreneurial intentions is the product of a superficial cause-hiding vector, like the link between childhood fever mortality and street births, or the outcome of true causative agents.

Obstacles to accepting the link between ADHD and entrepreneurial intentions

Even if the research in question survived the problems raised by the level of explanation chosen and the causal mechanisms ignored, it is necessary to delve into how the authors measured ADHD. Should the authors have failed to properly account for ADHD, their research would have been pulled out of thin air after all. It is thus no coincidence that the prediction variable of their research is termed 'self-reported ADHD'. There are three obstacles to accepting that the link in question is plausible, including (1) the putative nature of ADHD, which, unlike other disorders, remains syndromally defined and without a clear origin in biological or brain algorithmic dysfunctions; (2) the limitations of self-administered questionnaires that were never meant to substitute for clinical judgment in the diagnosis of disorders; and (3) the likely presence of a Hawthorne Effect (Hindle, 2008) on the sampled population.

Putative versus true disorders

First, there is a paucity of etiological knowledge about ADHD's underlying dysfunctions, which makes it a putative disorder, only syndromally defined. That is, there is no failure of designed function (Wakefield, 1992, 2010) or part dysfunction (Klein, 1999) widely recognised as the cause of the disorder or, in the term used by the American Psychiatric Association (2013), there is no 'biological marker' associated with ADHD, notwithstanding candidate markers being proposed for further research.

The opposite happens, for instance, with depressive disorders which are characterised by abnormal levels of steroids in blood or urine originating in the pituitary gland (American Psychiatric Association, 2013). Thus, ADHD fulfils only one of the preconditions to be considered a true disorder; that is, individuals may suffer because of a seeming maladaptation to their environment, but there is not as yet a natural evolutionary mechanism – either physiological or algorithmic (Cosmides and Tooby, 1999) – whose breakdown is recognised as the condition's ultimate cause (Agafonow and Perez, 2020).

Why does the medical community need these two preconditions for the imputation of a disorder? Or, put otherwise, why is not a maladaptation to an environment enough for the imputation of a disorder? Because maladaptations alone have been co-opted in the past by partisan interests to discriminate against socially deviant behaviour (Agafonow and Perez, 2020). Remember, for instance, drapetomania, a bogus mental disorder attributed to runaway slaves to justify their captivity (Szasz, 1971) or, again, the incarceration of political dissidents in the Soviet Union for alleged mental health reasons (Szasz, 1984). There was, in both instances, a maladaptation to the environment, yet no failure of natural evolutionary mechanisms could possibly be discovered. Thus, pinning down whether a condition, whose disorderly nature is just putative or presumptive, plays a role in the formation of entrepreneurial intentions is far too problematic.

The perils of quantifying complex behaviours

Second, Verheul *et al.* (2015, p.20) and Verheul *et al.* (2016) arrive at the apparent link between ADHD and entrepreneurial intentions based on Kessler *et al.*'s (2005) symptom checklist, which was self-administered. It is not, however, a complex behaviour like ADHD which can be, in principle, quantified by questionnaires, but crude temperamental biases provided that there exists a relatively simple genetic architecture, according to Cloninger *et al.* (1996). Yet even Cloninger's tridimensional personality questionnaire (TPQ), which is designed to capture temperamental biases, faces the challenge of properly interpreting adjectives to describe traits and then assorting individuals into personality dimensions. Thus, a successful assortment hinges on expert opinion about pattern recognition (Cloninger, 1987), and the latter is a challenge for the proper application of the TPQ. One can only expect that this problem is magnified when one moves from crude temperament to a complex behaviour like ADHD, let alone if the checklist is self-administered.

Dimensional epidemiological surveys are symptom checklists usually administered in one contact with the sampled subjects by a layperson without medical training (Wakefield and Schmitz, 2010, 2011), or self-administered through a computer terminal, which is the case with the entrepreneurship research in question. As a consequence, it is impossible that the cause of a disorder – that is, a failure of designed function (Wakefield, 1992, 2010) or part dysfunction (Klein, 1999) – is identified because it is common for several disorders to share the same symptoms. For instance, ADHD shares many symptoms, but should not be confused with (a) intermittent explosive disorder, (b) stereotypic movement disorder, (c) specific learning disorder, (d) intellectual disability, (e) autism spectrum disorder, (f) reactive attachment disorder, (g) anxiety disorder, (h) depressive disorder, (i) bipolar disorder, (j) disruptive mood dysregulation disorder, (k) substance use disorder, (l) psychotic disorder, and (m) neurocognitive disorders, among others (American Psychiatric Association, 2013). If a symptom checklist succeeds in identifying a disorder, it is more a matter of chance than causation, although Kessler *et al.* (2005) used clinical judgments in combination with stepwise logistic regression to help pin down symptoms with a high probability of being associated with ADHD. This was not meant, however, to substitute for clinical judgment, but to be used as a tool to screen likely cases that require further medical evaluation to rule out false positives.

This brings us to the limitations of how Verheul *et al.* (2015) and Verheul *et al.* (2016) employ Kessler *et al.*'s (2005) symptom checklist, namely, that its predictive value considerably diminishes when it is applied to 'segments of the population that cannot be considered representative of the total USA population' (p.254). Let us remember that the GUESSS Dutch sample

is a university student population, probably with a non-negligible cultural admixture. Such a caveat is important because it has been recognised that the interpretation of behaviour can vary from one culture to another, with clinical identification rates varying even within a diverse country like the United States, where incidence of ADHD tends to be lower among African Americans and Latinos than among Caucasians (American Psychiatric Association, 1994, 2013; Bussing *et al.*, 1998).

Diagnostic evidence versus self-administered surveys

Unlike Verheul *et al.* (2015) and Verheul *et al.* (2016), the aim of Lerner *et al.* (2019) is to reveal the ADHD clinical prevalence in the GUESSS Dutch sample, meaning by ‘clinical’ the diagnosed disorder. However, such prevalence is again the product of a self-administered survey, with students answering ‘yes’ or ‘no’ to ‘whether the individual had the diagnosed condition of ADHD’ (p.385). There are indeed good reasons to second-guess such answers. For a start, the public visibility of tycoons attributing their success to ADHD, including the founder of Virgin Group, Sir Richard Branson, the founder of Ikea, Ingvar Kamprad, and JetBlue Airways’ founder, David Neeleman (*Economist*, 2012; Archer, 2014; Vila, 2018), may have motivated some students to answer in the positive to such a question. Lerner *et al.* (2019) do not report any attempt to double-check such answers against certified diagnostic evidence.

Dutch tertiary education institutions have participated in the GUESSS project since 2011, including the attempts of Verheul *et al.* (2015) and Verheul *et al.* (2016) to spot ADHD-like behaviour, joining several data collection waves. It is plausible that subsequent cohorts of students know already what to expect from the GUESSS initiative thanks to word of mouth, particularly because they have been enticed to participate with the raffle of iPads among those who completed the survey. It is thus reasonable to expect a form of Hawthorne Effect (Hindle, 2008); that is, the individuals studied change their behaviour as a result of being observed.

In addition, and as noted by the American Psychiatric Association (2013), ‘ADHD often persists into adulthood, with resultant impairments of social, *academic* [my italics] and occupational functioning’ (p.32). This is because these individuals have their attention and organisation impaired, such that the disorder ‘is associated with reduced school performance and academic attainment’. Hence these individuals may be less likely to pursue higher education and it is, therefore, reasonable to conjecture a lower incidence of ADHD in university students, which makes suspect the 4.2% of self-reported diagnosed conditions in Lerner *et al.*’s (2019) sample, which (according to them) is comparable to 5% in the Netherlands overall.

Discussion: economics, organisation capabilities and ADHD

Efforts to integrate entrepreneurship into economics have often construed entrepreneurship as a dependent variable that can be adjusted by making changes in the structure of rewards at policy-makers’ disposal: ‘It restores the relevance of the massive body of theory that devotes itself to the analysis of resource allocation, treating entrepreneurship, in this respect, as yet another resource subject to the allocating influences of the price mechanism’ (Baumol, 1993, p.203; Baumol *et al.*, 2011), as well as a subjective course of action contingent on institutional settings (Boettke and Coyne, 2007; Lucas *et al.*, 2018). The proposition that entrepreneurship is a resource to be allocated by public policies needs substantial qualification if, in the end, it is proved that entrepreneurship happens to be the product of a hardwired, innate human trait.

Hardwired or not, an ADHD-like personality may find a place within evolutionary economics and, in particular, the organisational capability approach, where the modes of storage or the repositories of knowledge have played an important role in shedding light on the firm’s decision-making process (Nelson and Winter, 1982; Winter, 2012). Here the firm is modelled as an array of capabilities that facilitates the smooth sequence of coordinated behaviour, allowing both the

replication of routines and their modification to adapt to novel circumstances (Nelson and Winter, 1982; Zollo and Winter, 2002; Winter, 2003).

The crucial aspect of an ADHD-like personality is that the vast array of routines and their repositories consist of the most explicit and codified knowledge contained in, for instance, manuals and spreadsheets, to the most tacit knowledge, such as ‘semiautomatic stimulus-response processes’ which are sheltered in the nervous system of individuals (Nelson and Winter, 1982; Zollo and Winter, 2002). Thus, the routines and their repositories that form the capabilities of organisations are arranged in a hierarchic or nested fashion, comparable to the aforementioned Chinese boxes, (i.e., tacit knowledge embedded in the nervous system forms the innermost boxes, while explicit knowledge that allows inter-subjective deliberation, as well as codification, forms the outermost, visible boxes).

The outermost boxes are exemplified by macro-routines that require a high degree of deliberation and cognitive effort to be executed (Winter, 2003, 2012), such as the managing of the most recent crises at Tesla, for instance. The US Securities and Exchange Commission (SEC) opened a fraud investigation after Elon Musk (Tesla’s CEO and chairman) posted on Twitter a deal to take the company private. While Musk’s Twitter message sent the value of the company shares soaring, the deal never materialised. Tesla’s board of directors had to devote a battery of higher-order capabilities toward the conclusion of an agreement with the SEC, which included the stepping down of Musk as the company’s chairman; the addition of two independent directors to the board; the payment of a \$20-million fine for lack of control over Musk’s social media communications; and another \$20 million to settle the case (Disparte, 2018).

Buried deep in the hierarchic structure of an organisation lie the micro-routines, which are more akin to stimulus-response processes. Just think about the little cognitive effort that Tesla’s directors must have put in either drawing the board office’s curtains or turning the light on, yet without such a knee-jerk reaction, a poorly illuminated office could hardly have assisted the formulation of the strategy to deal with the SEC.

However, while the causal ambiguity between street births and childbed fever mortality in the nineteenth century was reduced when Ignaz Semmelweis postulated the putative variable of cadaveric matter (i.e., cadaveric matter was supposed or, more accurately, retroduced¹ without direct supporting evidence), entrepreneurship scholarship is far from identifying the putative variables that can dissipate the cloud of causal ambiguity engulfing the connection between distant Chinese boxes (i.e., ADHD and entrepreneurial intentions). Note that the explanatory efficacy of a putative variable does not lie in the evidence offered in support of its existence, for which there may not be any at the moment of its retroduction. On the contrary, its efficacy lies in the observation statements derived from the broader theory within which such a variable plays the role of a key piece of the jigsaw puzzle on which the coherent articulation of a theory hinges, and the test implications that such observation statements enable (Hempel, 1966; Hanson, 2010). In particular, the decrease in childbed fever mortality was a test implication of cadaveric matter as part of an emerging theoretical explanation of infectious diseases and the seeming antibacterial properties of chlorinate lime. Cadaveric matter was instrumental in understanding childbed fever and how to prevent it, despite subsequently being abandoned as a result of the discovery of bacteria and the development of the germ theory of disease (Broadbent, 2013).

In the case of ADHD, it is not clear what the explanatory efficacy of an apparent putative variable in the authors’ work is, namely ‘pre-strategic venturing without intentionality’ (Lerner *et al.*, 2017, p.5; 2018a, p.64). To begin with, they claim that the GUESSS Dutch sample offers significant

¹Retroduction, which is distinct from deduction and induction, is the only form of inference able to generate new knowledge in the context of fragmentary or incomplete evidence (Hanson, 2010; McMullin, 1992).

evidence to support such a variable, which this paper refutes. For pre-strategic venturing without intentionality to convey explanatory efficacy, it would have to be instrumental in accounting for why and how ADHD-afflicted individuals espouse entrepreneurial intentions instead of entertaining other adaptive and maladaptive alternatives. How does one know that an ADHD-afflicted individual entertaining entrepreneurial intentions did not simply stumble into entrepreneurship by accident?

When it is posited that ADHD has pre-strategic venturing attributes, one cannot be blamed for expecting that ADHD necessarily leads to venturing, otherwise it could equally be pre-video-game testing, pre-extreme sport practising, pre-leisure travelling, etc. That is, there cannot be such a thing as pre-strategic venturing if ADHD serves to play any role that offers novelty-seeking opportunities outside the realm of entrepreneurship. Thus, the mechanisms that tie ADHD to entrepreneurship are missing in the authors' work.

The causal ambiguity between ADHD and entrepreneurial intentions is, more generally, intrinsic to organisation capabilities, where it is notoriously difficult to pinpoint a clear causal relationship between the routines rehearsed and the outcomes obtained (Zollo and Winter, 2002). Otherwise put, the micro-routines of stimulus-response-like processes arguably facilitated by ADHD are simply too far removed from the macro-routines of entrepreneurial intentions, which calls instead for an informative level of explanation for entrepreneurship.

Conclusion

Because of the authors' uninformative level of explanation, their model specification leaves between ADHD and entrepreneurship a black box that makes the correlation identified the likely product of accident instead of real causality. This is all the more so because the authors relied on a self-administered survey instead of diagnostic evidence, which would anyway be problematic in the case of syndromally-defined disorders. Not long ago, the authors claimed to have replicated the results of the entrepreneurship research in question (i.e., Wismans *et al.*, 2020), yet it is known that: 'If the results of a study are erroneous due to some flaw in the design, then the same error is apt to occur again if the study is repeated in identically the same way' (Hammond, 1958, pp.335–6; Munafò and Davey Smith, 2018).

Much as nineteenth-century bystanders who mistook street births for the cause of a reduction in childbed fever mortality, the research in question mistakes a self-reported ADHD condition for the cause of entrepreneurial intentions, with potentially negative consequences for public policies fostering entrepreneurship. If entrepreneurship is seen as the product of a hardwired human trait linked to ADHD instead of a variable dependent on the structure of rewards that is within policymakers' reach (Baumol, 1993; Boettke and Coyne, 2007; Baumol *et al.*, 2011; Lucas *et al.*, 2018), the harmful effects suffered by afflicted individuals can be played down and the consequences of pathological levels of risk-taking underestimated.

That brings us to what to make of an ADHD psychological phenotype in the twenty-first century. The answer runs against the folk notion of evolution, with ADHD being far from the expression of a gene for entrepreneurship. It may be instead a potential exaptation to be capitalised on by ADHD-afflicted individuals, depending on the demands posed by the modern environments they inhabit. A typical example of an exaptation is feathers in birds, which were by hypothesis selected at the beginning for temperature regulation whereas their current use for flight is recent on an evolutionary scale (Gould and Vrba, 1982).

Similarly, an ADHD psychological phenotype is arguably not an adaptation proper to entrepreneurship or any other potential novelty-seeking outlet, but an exaptation which more generally refers to any trait co-opted into functions other than the ones initially determined by natural selection (Gould and Vrba, 1982). Thus, the right research question the authors missed is: What makes ADHD be co-opted into entrepreneurship instead of any other adaptive or maladaptive role offering novelty-seeking opportunities?

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