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ADHD and Brain Anatomy: What Do Academic Textbooks Used in the Netherlands Tell Students?

Sanne te Meerman*, Laura Batstra†, Justin E. Freedman‡, Rink Hoekstra§ and Hans Grietens†
*Integrated Youth Policy, Hanze University of Applied Sciences, Groningen, The Netherlands
†Department of Special Needs Education and Child Care, University of Groningen, Groningen, The Netherlands
‡Interdisciplinary and Inclusive Education, Rowan University, Glassboro, NJ, USA
§Department of Educational Sciences, learning & Instruction, University of Groningen, Groningen, The Netherlands

Studies of brain size of children classified with ADHD appear to reveal smaller brains when compared to 'normal' children. Yet, what does this mean? Even with the use of rigorously screened case and control groups, these studies show only small, average group differences between children with and without an ADHD classification. However, academic textbooks used in the Netherlands often portray individual children with an ADHD classification as having a different, malfunctioning brain that necessitates medical intervention. This conceptualisation of ADHD might serve professional interests, but not necessarily the interests of children. © 2019 The Authors. Children & Society *published by National Children's Bureau and John Wiley & Sons Ltd*

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Introduction

ADHD is one of 400+ disorders that are defined and described in the DSM-5, the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). A 'diagnosis' is possible if a child meets six out of nine criteria for hyperactivity/impulsivity such as 'often fidgets with or taps hands or feet', and/or six out of nine criteria for inattention, including 'often loses things necessary for task or activities'. Furthermore, children have to meet additional criteria, such as exhibiting these behaviours before age 12 and for a duration of at least 6 months (American Psychiatric Association, 2013). Additionally, the behaviours must interfere with social and academic functioning.

In the research agenda for the development of the DSM-5, the authors stated that 'the field of psychiatry has thus far failed to indicate a single neurobiological cause for any of the mental disorders' (Kupfer, First and Regier, 2002, p. 33). After many years of expensive brain studies, ADHD has proven to be no exception; 'No single risk factor explains ADHD. Both inherited and non-inherited factors contribute and their effects are interdependent' (Thapar *et al.*, 2013, p. 3). Despite this and despite the clear cultural underpinnings of what are perceived as (ab)normal behaviours (Freedman and Honkasilta, 2017; Kriegler, 2015), the authors of the DSM-5 decided to classify ADHD as a 'neurodevelopmental disorder'.

Many scholars promote the image of ADHD as a discrete brain disorder based on the questionable claims. For example, Russel Barkley, in a 2.5-hour video addressing parents of children classified with ADHD, states that in those who have inherited the disorder (about 2/3 according to Barkley) several brain regions are smaller (3–10%), 'causing' the disorder.

However, these differences are 'not enough to use brain imaging to diagnose this disorder' (Barkley, 2013, 56th minute). Barkley's explanation is peculiar: how could they have discovered these differences other than by brain imaging? The differences are in fact not too small, but rather too inconsistent as the effect size between brain size and behaviour is low, denoting the overlap between the case and the control groups. Such 'lack of group-to-individual generalisability' (Fisher Aaron et al., 2018) is the actual reason that, as Barkley later acknowledges, 'no brain imaging technique (. . .) is of any value for diagnosis'.

In a similar vein, much attention has been given to the largest ADHD case–control study to date by Hoogman et al. (Hoogman *et al.*, 2017). The study indicated, on average, a small delayed growth in five subcortical brain areas, while one area appeared to be slightly bigger in children diagnosed with ADHD. These mean differences disappeared in adulthood. However, far-reaching claims were made from the results of this study, for example: 'the data from our highly powered analysis confirm that patients with ADHD do have altered brains and therefore that ADHD is a disorder of the brain. This message is clear for clinicians to convey to parents and patients' (Hoogman *et al.*, 2017, p. 316). Their conclusion incited several criticisms (Batstra *et al.*, 2017; Bejerot, Nilsonne and Humble, 2017; Dehue *et al.*, 2017), and a petition to withdraw the study altogether was initiated by the website *Mad In America*. The study was neither withdrawn nor were changes made to the conclusions, which were reported in many media outlets worldwide, including CNN and Newsweek (https://www.madinamerica.com/2017/04/lancet-psychiatry-needs-to-retract-the-adhd-enigma-study/).

Readers, and particularly laypersons, may have difficulty in understanding the limitations of the study. First, the lack of group-to-individual generalisability means that many in the group of those classified with ADHD have larger brains than average, and many in the control group have smaller-than-average brains. Second, both case and control groups often use rigorously screened individuals, called 'refined phenotypes' (Holmes *et al.*, 2000) and extremely 'well-controls' (Horga, Kaur and Peterson, 2014), who hardly represent their respective populations (for a discussion see also te Meerman *et al.*, 2017). Third, brain maturation is a 'moving developmental target' (Hyman, 2010) as children develop at their own rate. Mean differences disappear because brain growth can catch up later in life (Hoogman *et al.*, 2017; Shaw *et al.*, 2007). Fourth, a correlation between ADHD-related behaviours and any (brain) measure does not necessarily imply causality. Neuroplasticity, or the fact that brain structure and function change as a result of interaction with the environment is a well-known phenomenon, seen in musicians for instance (Hyde *et al.*, 2009; Münte, Altenmüller and Jäncke, 2002). On the downside, poverty (Noble *et al.*, 2015) and maltreatment in children (Riem *et al.*, 2015) are negatively associated with brain size. Finally, even if differences were found on the individual level, these would not necessarily imply a disorder. If a particular brain characteristic is discovered for homosexuality, does this mean we should add it to the DSM again after it was removed in the DSM-III? (Dehue, 2014). Brain scans might help studying behaviour, but it is ultimately human beings that decide which behaviours are considered (in)appropriate and characteristic of illness (Altermark, 2014; Dehue *et al.*, 2017).

Journalists (Gonon *et al.*, 2012) and researchers (Gonon, Bezard and Boraud, 2011) may have mutual benefits for simplifying and overstating findings. These benefits include standing out amidst the fierce competition for publications and readership among authors, scientific journals and newspapers. However, such dissemination has important implications for influencing how audiences, such as parents, teachers and (health care) professionals, perceive and respond to children's behaviour.

The present study analyses how authors of academic textbooks used in the Netherlands write about case–control studies of brain anatomy of those classified with ADHD. We aim to answer the following two questions relating to group-to-individual and sample-to-population

generalisations: first, are group outcomes rightly presented as mere averages with little predictive value for individuals? And second, are readers of these textbooks presented with additional information regarding well-controls and refined phenotypes? This paper concludes by relating generalisations to 'reification' of perceived issues concerning childhood. We also consider possible motives for projecting problems inside (the brains of) children, a practice that can conveniently distract from broader societal problems and interests (Stryker, 2013).

Method

Data selection

The Netherlands has 18 public universities, eleven of which have a wide academic orientation including medical and behavioural scientific bachelor's and master's programs. These are included in this study. The remaining seven universities are theological (4), (primarily) technical (2), and agricultural (1). (Source: Association of universities in the Netherlands http://www.vsnu.nl).

Using purposeful sampling (Coyne, 1997), we attained up to date academic textbooks used by universities to educate future health care professionals. Information about medical and behavioural science programs was searched for the following subjects: psychopathology, (biological, cognitive, clinical, biological) psychology, psychiatry, psychiatric disorders, diagnostics and behavioural problems. The glossaries of the prescribed textbooks for individual courses were then searched for the keywords 'Attention Deficit Hyperactivity Disorder' or ADHD. If the keyword was found, the book was checked to identify if it contained a (sub)-section, paragraph or chapter on ADHD. Forty-three books were selected for analysis. We searched for the most recent versions up to January 2016.

Analytic framework and coding

Every section on ADHD was scanned and imported to Atlas TI, a software program for qualitative data analysis. Next, all claims relating to empirical research concerned with volumetric measures of brains of subjects with ADHD were selected and classified into five categories A through E:

A. A non-generalising claim, containing 'hedged' positions on the generalisations

Hedging can be considered as the 'expression of tentativeness' (Hyland, 1996, p. 433). Although hedges can bring unwanted 'fuzziness' (Crompton, 1997, p. 272), in the case of group-to-individual generalisations we argue hedges are necessary to avoid logical errors. A 'classic' way to hedge a generalisation is by using a qualifying term such as 'tend to' (Black *et al.*, 2015, p. 30), other possibilities are for instance 'to contribute' and 'to cause', the latter being more definite (Black *et al.*, 2015). So-called 'vague quantifiers' (Bradburn and Miles, 1979), for example 'a number of, a minority, a few, several, often', etc. (Hamp-Lyons and Heasley, 2006, p. 65), and research outcomes referred to as 'group findings' or 'means/averages' are also considered as hedges. Other hedges relevant to generalisations are words used to express 'a component of tentativeness or possibility', for instance *may, argue, believe,* or *hypothesis*. Particularly, words like hypothesis, or theory, express 'epistemic modality' (Varttala, 1999, p. 183).

B. A generalising claim, stating that all classified with ADHD share a certain anatomical feature without hedges to clarify this

A neuroanatomical finding without a hedge was classified as generalising. An example of this is: 'several brain regions, including the prefrontal cortex, are smaller in children with

ADHD than in children without the diagnosis' (Bukatko and Daehler, 2012, p. 300). Note that these claims may contain hedges that do not relate to the generalisation but to something else such as the number of researchers that allegedly support the claim. For instance: 'All structural magnetic resonance imaging (MRI) studies that measured frontal regions reported reduced volume in PFC, and several studies reported volume reductions in the caudate nucleus of the basal ganglia, a structure with close connections to dorsolateral and ventrolateral PFC' (Willcutt, 2010, p. 397). This sentence is classified as generalising, and the reference to 'all', versus 'several' studies is not considered in this decision as it does not refer to brain anatomy.

C. An ambiguous claim, in case of uncertainty about the claim being a generalisation, or uncertainty about the claim referring to anatomy

These aforementioned hedges and quantifiers served as 'sensitising concepts', as it is 'probably impossible to form an exhaustive taxonomy of potential lexical hedging devices in English' (Varttala, 1999, p. 183). There are phrases in which no such words are used, yet the phrases still do not seem to be full out generalisations. For example: 'volume of the frontal, striatal, and temporal lobe region has been found to be directly related to inhibition' (Wicks-Nelson, 2015, p. 232). 'Directly related' is considered ambiguous because it seems to describe a stronger relation than the mere statistical association it actually refers to. Other phrases we considered ambiguous are, for instance, those that are clearly generalising, yet the authors do not make explicit whether brain anatomy, physiology or chemistry is implied.

D. Disclaimers hedging the generalisation

We further coded for the presence of 'disclaimers'. We define disclaimers as a passage in which the authors discuss the heterogeneity of neuroanatomical findings by, for instance, mentioning the overlap between the research groups or explaining that not every person with ADHD has smaller brain structures. The difference between a disclaimer and a non-generalising claim is that in the latter, the generalisation of the claim itself is hedged while a disclaimer reflects on several or all of the neuroanatomical findings.

E. Disclaimers regarding refined phenotypes and super healthy controls

Finally, we analysed the sections addressing ADHD for the presence of other critical considerations regarding the exact value of the average scores: the use of refined phenotypes and super healthy controls. Any consideration regarding the selection of case or control groups was coded. For both type of disclaimers mentioned under section D/E we also analysed external chapters, if these were cross-referenced in the ADHD section.

Consolidation, analysis and reporting

The claims were selected and coded as (non-)generalising or ambiguous by the first author, after which the second author coded the same claims. This resulted in an inter-rater reliability (IRR) of 0.76 (Cohens Kappa). The disagreements were then discussed until full agreement was reached and the criteria were clarified when needed. Then, the third author coded the claims that resulted in an IRR of .94 with the first and second author. After a final discussion, consensus was reached about all the codes.

Next, the (co)occurrence of the coded claims with disclaimers, and references to other chapters with disclaimers was determined. On the basis of these (co)occurrences, we classified the sections on ADHD in four categories. These categories ranged from sections that did not discuss brain anatomy at all, sections that included additional disclaimers and those that contained only generalising claims on brain anatomy.

Results

In the 43 academic textbooks, a total of 94 claims about brain anatomy were identified. Of those, 48 (51%) were classified as generalising. Thirty-seven claims (39%) were classified as non-generalising. Nine claims (10%) were classified as ambiguous. An additional 19 disclaimers were found relating to anatomy. These disclaimers clarify if claims represent theories (n = 9), relate to group level (n = 3), do not apply to each individual (n = 3), refer to the differences not being 'reliable' (n = 1) or refer to brain size being a mere association (n = 3).

Note that a single disclaimer can compensate for all generalising claims. The most important information is therefore revealed when the co-occurrences of disclaimers and (non)generalising claims are established at the level of book chapters/sections. Figure 1 gives an overview of the co-occurrence in our data.

Chapters without disclaimers (n = 21 [49%])

We begin by discussing co-occurring statements at the level of chapters/sections, starting with sections that contain only generalising/ambiguous statements without any disclaimer (n = 17). In other instances (n = 4) they co-occur with non-generalising statements. We will exemplify some of the generalising statements and elaborate on their differences and similarities, and on the co-occurrence with non-generalising or ambiguous claims. Appendix S1 displays how often the (non-)generalising and ambiguous statements co-occur in these chapters without disclaimers.

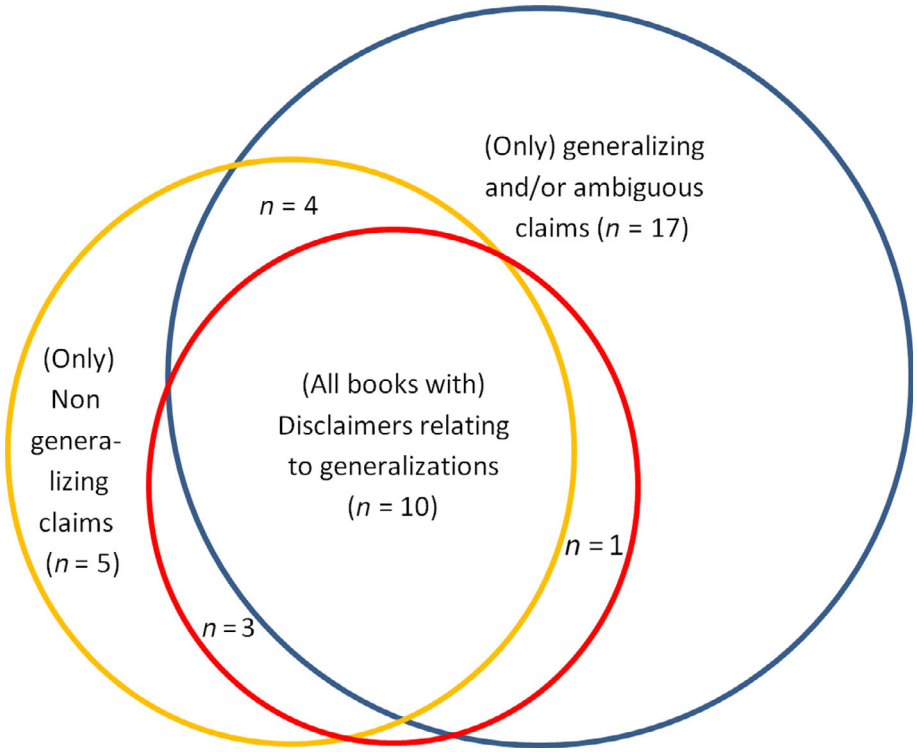

Figure 1. Co-occurence of claims in the data. [Colour figure can be viewed at wileyonlinelibrary.com]

A chapter by Buitelaar and van der Gaag (2009) includes a generalisation in its simplest form; without any disclaimers, non-generalising or ambiguous claims present.

> Volumetric MRI-research with children and adults shows smaller volumes of grey matter (ibid., p. 536)

The statement suggests that all children with an ADHD classification have smaller volumes of grey matter. Similarly, Kerig, Ludlow and Wenar (2012, p. 222) state:

> Magnetic resonance imaging (MRI) techniques have revealed that children with ADHD have a smaller splenium, which is the posterior portion of the corpus callosum (the structure connecting the two hemispheres of the brain).

In addition to the generalisation, it is interesting to note that these excerpts contain no references to research studies and there is no mention of actual researchers who conducted the MRI study. Latour (1987, p. 23) refers to a statements in which the original study and relevant information is stripped down so it becomes 'devoid of any trace of ownership, construction, time and place', as a 'positive modality'. The phrasing suggests objectivity, as if the MRI-technology itself has shown the anatomical differences.

In the next paragraph, Kerig, Ludlow and Wenar add a statement that we classified as non-generalising:

> It has even been suggested that the ADHD brain is wired differently (. . .). For example it has been proposed that ADHD may be dysfunction of or disconnection between brain regions that support the 'default network'.

Because in this case the epistemological status is explicated, as it is 'suggested' that it 'may be dysfunction (. . .) or disconnection', we classified this phrase as non-generalising. However, the phrase does not place the earlier generalisation about the splenium into perspective. Rather, it extrapolates that the brain might even be wired differently.

The next example shows a similar generalisation, although actual researchers and not MRI-scans are now the subject of the sentence. Barlow and Durand (2015, p. 517) state:

> In general, researchers now know that the overall volume of the brain in those with this disorder is slightly smaller (3% to 4%) than in children without this disorder (. . .)

The authors do express some tentativeness ("In general)", but this relates only to how many researchers know of the alleged fact, not to the certainty of the fact, that the overall brain volume is 3% to 4% smaller in children with ADHD. The authors follow this statement with:

> A number of areas in the brains of those with ADHD appear affected, especially those involved in self-organizational abilities

The epistemological status of the latter sentence is somewhat dubious; certain areas 'appear affected'. Therefore, we classified this sentence as ambiguous. Note that the ambiguity does not seem to have the intention to cast doubt on the findings in any way. Rather, it leads logically into the next sentence with which the paragraph ends:

> These changes seem less pronounced in persons who received medication

As the effects of medication are not the subject of this study, this sentence was not coded. However, this statement reveals what Latour (1987, p. 23) explains as the purpose of positive modalities: they 'lead a statement away from its conditions of production, making it solid enough to render some consequences necessary'. In this case, the merits of medication are now suggested; those who receive medication have less 'pronounced changes'.

Carlson (2013, p. 601) generalises research findings in a similar fashion, but depicts more agency on the part of the researchers:

> A study by Shaw et al. (2007) found differences in the development of the brains of children with ADHD as well. The investigators found that cortical growth was delayed in children with ADHD.

The author adds:

> Ultimately, the growth of the brains of the children with ADHD caught up with those of unaffected children

Although this statement provides perspective about the relevance of brain size, it is still generalising. This is problematic as it might obfuscate the fact that many children with an ADHD classification did not have 'slower developing brains' that needed 'catching up'. Mentioning group findings and discussing correlations seem to provide a way out of this conundrum. For instance, Higgins and George (2013, p. 247) state:

> Both groups showed the usual pruning of the total gray matter as they grew through adolescence (Figure 20.8). However, two regions were unique when correlated with clinical outcome:
>
> 1. Children who remained impaired at follow-up had thinner grey matter in the medial PFC at the beginning of the study
> 2. Children who grew out of the disorder showed a normalisation of the grey matter thickness in the right parietal cortex

These statements show how the use of correlations and mentioning groups can avoid generalisations. Furthermore, the example demonstrates how generalising sentences can be nuanced by other non-generalising sentences. The first two sentences are non-generalising: *both groups showed the usual pruning*, and *two regions were unique when correlated*. The next two sentences (numbered 1 and 2) are generalising in themselves, but they are rectified because the author addresses correlations.

Four chapters in this first category of chapters without disclaimers contain an ambiguous claim. In two cases, it was unclear whether statements referred to neuroanatomy or only to neurophysiology or chemistry. For instance, van der Oord and ten Brink (2014, p. 295) state:

> Using uncomplicated language, the therapist explains the characteristics of ADHD to parents, comorbidity, the subtypes, potential genetic predisposition, the deficits in the brain (. . .)

Possibly, the authors refer to neurochemical findings. This is not the focus of the current study, but it is important to mention as it is an even more varying characteristic than anatomy, with inconsistencies between studies and intra-personal differences from day to day (Weyandt, Swentosky and Gudmundsdottir, 2013). Therefore, this generalisation is arguably even more poignant than those involving neuroanatomy.

Chapters with (non-)generalising/ambiguous claims in combination with hedges/disclaimers (n = 10)

In this second category we will discuss sections that contain disclaimers. Disclaimers occur alongside generalising claims, in addition to non-generalising claims or, in some cases, in addition to both. We identified 10 sections in which all generalisations were hedged, or when extra caution was expressed by either a hedge or what we refer to as a 'disclaimer'. Appendix S2 displays how all (non-)generalising, ambiguous, claims co-occur with hedges/disclaimers.

One example of hedging is by beginning a paragraph with a *topic sentence* (van Loon *et al.*, 2013) that frames all subsequent generalising claims in the paragraph. For instance,

Sonuga-Barke and Taylor (2015, p. 744) begin a section about ADHD and brain structure as follows:

> Structural alterations in multiple brain systems have been implicated in ADHD (Sonuga-Barke & Fairchild, 2012). Group comparisons with controls find significantly smaller brains in ADHD (Castellanos et al., 2002) with cerebellum, corpus callosum, striatal—for example, caudate nucleus, putamen and globus pallidus (Ellison-Wright et al., 2008) — and frontal regions—for example, dorsolateral prefrontal cortex (DLPFC) (Valera et al, 2007) especially affected.

Although the first sentence is classified as generalising, it introduces the topic. In the next sentence the authors discuss several case–control studies and indicate that those are 'group comparisons'. We have interpreted this second sentence as a hedge, as the authors seem to have intended to indicate that all subsequent studies mentioned in the paragraph are group comparisons.

Carr (2016, p. 376) uses a similar construction opening a paragraph about neuroimaging studies:

> Neuroimaging studies have shown that ADHD is associated with a range of structural and functional neuroanatomical abnormalities (. . .). The best established of these is lobar volume loss of around 3-4%, and abnormalities of the frontostriatal circuitry which underpins executive function.

Because the authors mention that neuroimaging studies are 'associated' with abnormalities, we have classified this particular sentence as non-generalising: association studies typically work with comparing averages, and hopefully, readers are able to view the results as relating to group differences only.

Disclaimer in (preliminary) conclusions/reflections/cross-reference

An interesting disclaimer is found in Sonuga-Barke and Taylor (2015, p. 746).

> While it is helpful to think of ADHD as having a characteristic developmental phenotype that can be charted across the life span, the idea that there is a single trajectory runs counter to what we know of the complexity of ADHD developmental psychopathology (. . .) and the heterogeneity within disorders (see Chapter 3).

The excerpt does not entirely and unequivocally hedge the generalisations, but it does explain that *the idea of characteristic developmental phenotype/trajectory contradicts with what we know of ADHD*. Furthermore — using a cross-reference — this is the only section on ADHD that addresses sample-to-population issues by discussing the use of refined phenotypes (Horga *et al.*, 2014) in two famous studies by Shaw *et al.*, 2006, 2007) about ADHD and brain development:

> The ADHD participants were atypical in their high IQ, socially advantaged background and lack of comorbidities (Thapar et al. 2015, p. 33)

This is crucial information as it is likely that less explained variance from environmental factors is found in these youths from socially advantaged background.

The following disclaimers all relate to the fact that group outcomes cannot be generalised to individuals. For instance, Carr (2016, p. 376) ends a paragraph on anatomy with:

> Not all cases of ADHD show all abnormalities. It is likely that the symptom patterns of different sub-groups of cases are associated with different neurophysiological abnormalities.

The authors seem to have used this passage to avoid generalisations by referring to an 'association'. Furthermore, they explain that not all with a diagnosis have 'abnormalities' in their brains. The authors do seem to suggest that, at least *some* abnormalities are present, a claim that is not supported by existing scientific knowledge.

Orobio de Castro, van der Oord, Raaijmakers and Prins (2009, p. 593) more clearly explain that those with an ADHD classification do not necessarily have those abnormalities:

> Those who do a groupwise comparison of children with ADHD and children without ADHD, see differences (. . .) on brain scans and genetic make-up. However, it concerns different abnormalities in different children with ADHD, and there are consistently children with ADHD that do not differ from other children. It is not possible to diagnose ADHD in this fashion.

In this statement, the authors provide a more thorough and less pathologising disclaimer. First, they refer to *differences*, and not *abnormalities*. Second, they make clear that there are children diagnosed with ADHD 'that do not differ' from other children in terms of brain anatomy. Wicks-Nelson (2015, p. 234) adds the following additional information:

> The finding that non-diagnosed, typically developing youths exhibited brain changes similar to youths with the syndrome of ADHD lends neurobiological support to the dimensional view of ADHD.

These authors do not refer to the 'case' group, but to the control group to make a similar point about the overlap between the groups: non-diagnosed children sometimes also have 'brain changes' comparable to those clasified with ADHD.

These types of disclaimers are functional in the epistemological clarity they provide, and are examples of what Bruno Latour (1987) calls 'negative modalities', as opposed to 'positive modalities' discussed earlier. Negative modalities 'lead a statement (. . .) towards its conditions of production and (. . .) explain in detail why it is solid or weak'. Orobio de Castro et al. clearly exemplify how authors may reveal such weaknesses by explaining that no abnormality sets those with ADHD apart so it is not possible to diagnose ADHD with a brain scan or based on genetics.

In sum, each of the above disclaimers adds some additional perspective to the problem that group findings do not apply to individuals. Willcutt, 2010, p. 411) , in Yeates *et al.* (2010) mention the following in their conclusion:

> The neuropsychology of ADHD is clearly complex and multifactorial, with no single deficit that is necessary or sufficient to explain all cases of ADHD

This disclaimer nuances all previous generalisations by the author. Although factors associated with ADHD, such as brain size, do not necessarily 'explain' ADHD, it still illustrates how simple and effective a disclaimer can be.

Chapters with only non-generalising claims (n = 5)

Several sections contain only non-generalising claims. An example comes from Prins and Van der Oord (2014, p. 288), whose section mentions the group level of the findings.

> MRI-research shows differences between groups with and without ADHD in three areas of the brain: the dorsolateral cortex, components of the basal ganglia and the cerebellum

Another example is Gazzaniga, Heatherton and Halpern (2016, p. 648), who state:

> Researchers have also demonstrated differences in the basal ganglia in the brains of some ADHD patients

The authors use the 'vague quantifier' (Schaeffer, 1991) *some* that gives a partial 'degree of certainty' (Hamp-Lyons and Heasley, 2006, p. 65). Such phrasing might be easier to interpret than Van der Oord and ten Brink's group findings, which still leave the option open that all people in the group have a different brain. Gazzaniga et al. do seem to suggest that the differences set some ADHD 'patients' apart. The overlap in research groups, however, indicates that subjects in the control group could also have smaller brains.

Comer (2014, p. 453) hedges the generalisation by using the vague quantifier 'many'.

> Biological factors have been identified in many cases, particularly abnormal activity of the neurotransmitter dopamine and abnormalities in the frontal-striatal regions of the brain.

It is dubious if 'many' is an appropriate term, as neuroanatomical (as well as neurochemical) studies typically show only low effect sizes and abnormalities are not a necessary or sufficient condition for a classification. However, it is not a complete generalisation.

Chapters with no reference to anatomical brain studies and ADHD (n = 7)

Seven books made no reference to case–control studies relating to ADHD brain anatomy. In six cases, these sections were small (<3 pages). In one case, an older book was used (1999), while many anatomical studies were conducted after 1999. One book (Tak *et al.*, 2014) did have a relatively large section on ADHD (18 pages), yet with no reference to brain anatomical studies. This exclusion is a defiant, but defensible choice as anatomy does not correlate strongly with ADHD. As these chapters do not contain information on ADHD in relation to anatomy, we do not discuss them further.

Discussion

The introduction of this paper began with the generalising notion of Hoogman and colleagues (2017) that 'patients with ADHD (. . .) have altered brains'. Motivated by such claims, this study investigated how authors of academic textbooks used in the Netherlands write about case–control studies of brain anatomy of those classified with ADHD, specifically how group outcomes are presented to readers. Findings of this study indicate that generalising claims about brain anatomy are common within the academic discourse on ADHD. In academic textbooks used at universities in the Netherlands, group-to-individual generalisations appear to be the rule rather than the exception.

Twenty-one out of 36 textbooks (58%) that discuss brain anatomy have sections on ADHD with only generalisations and/or ambiguous claims that are not qualified by disclaimers. Ten chapters (28%) did include a hedge or disclaimer. Two chapters (6%) discussed that a smaller brain is not a necessary condition and one chapter (3%) mentioned that detecting smaller regions is not a sufficient condition for an ADHD classification. Only one chapter (3%) mentioned that smaller brain areas are neither necessary nor sufficient. None of the chapters placed the average findings of case–control studies in perspective, for instance by referring to the use of refined phenotypes or super healthy controls in studies. Although four chapters contained a cross-reference, only one of them, by Sonuga-Barke and Taylor (2015) in Thapar *et al.* (2015), cross-referenced a chapter that (implicitly) discussed refined phenotypes.

An important implication of the analysis of textbooks is how the use of generalisations reify ADHD, and childhood more broadly. Reification literally means 'to make a thing' out of something — such as referring to the construct of ADHD as a concrete disorder or even as a disease. Generalisations suggest that those with an ADHD classification share certain suggested flaws, such as smaller areas of the brain. In fact, these findings only indicate that — on average — this trait is a non-persistent developmental difference (Hoogman *et al.*, 2017) and occurs slightly more often in a group compared to 'normal' populations. Generalisations thereby have a reifying effect of creating an unduly definitive impression that ADHD is a discrete entity. Reification is acknowledged as a fundamental problem in mental health care. Hyman (2010) warns that as reified entities, DSM classifications such as ADHD can create 'epistemic blinders that impede scientific progress' (ibid., p. 58). They may cause health care research and practice to become overdependent on these classifications by creating a

threshold for reimbursement for health care. Likewise, grants for research and developments must be linked to the 'conditions' described in the DSM like ADHD.

In the larger picture, the reification of problems with young people might also help to establish 'childhood as a historical stage of development and a general form of behaviour' (Foucault *et al.*, 2003, p 304) which in turn helps to target (subgroups) of young people for health care intervention. Reifying problems of childhood, and childhood itself, for the purpose of facilitating health care intervention, however, can have negative consequences. For example, Stryker (2013) argues in relation to children labelled as 'RAD' kids (Reactive Attachment Disorder), that such a label and the surrounding narratives 'reflect a tendency to over-value dispositional or pathological explanations for the observed behaviours of children diagnosed (. . .) while under-valuing structural explanations'. Classifications such as RAD and ADHD might thereby serve to represent (or cover up) larger societal problems (Miller and Leger, 2003) , such as divorce, poverty, large classrooms, lack of opportunities for play, etc. Indeed, ADHD has been correlated with many such societal issues that are at risk of being obscured by the catch-all umbrella of ADHD (te Meerman *et al.*, 2017; Richards, 2013). In other words, generalising the small brain-anatomical group differences effectively places the pathology directly inside the child and helps to avoid such broader issues. A narrow medically oriented focus might also result in the unnecessary prescription of psychotropic substances which might have serious adverse physical effects such as brain atrophy and growth inhibition as research indicates (Breggin, 2014; Curtin *et al.*, 2018; Swanson *et al.*, 2017; Van Den Eeden, 2011) and limited positive outcomes in terms of academic success (Kortekaas-Rijlaarsdam *et al.*, 2019).

There are also competing interests and there likely is much 'economic, cultural, and political capital bound up' (Stryker, 2013, p. 1186) in the superficial framing of complex problems. For instance, some of the criteria for an ADHD 'diagnosis' directly relate to school. Criterion 1f under 'Inattention' declares that disliking activities that require sustained mental effort like schoolwork or homework as a possible 'symptom' for ADHD. The connection between ADHD and the economic and political interests of schooling are further demonstrated through several internationally replicated studies finding that the youngest students in a class are roughly twice as likely to receive an ADHD diagnosis and prescription of psychotropic drugs. These studies illustrate how educational structures, such as combining children who differ in age up to a year into groups, can contribute to interpreting age-related variation of behaviours as indicative of a disorder, which often result in them receiving potentially dangerous psychotropic drugs (Whitely *et al.*, 2018).

Limitations

Perhaps much like the difficulty of 'diagnosing' children based on a limited selection of behavioural criteria, our 'diagnosis' of chapters/sections in textbooks was a challenge. Some ambiguity was still present after discussion even if our criteria were clear, and texts do not change from one moment to the next, like behaviour. Second, our findings cannot speak to how readers might interpret the textbook chapters analysed in this study. Other designs with generalising and non-generalising texts given to experimental and control groups, followed by questionnaires or interviews, could provide valuable additional information about how generalisations are perceived. Third, we have analysed segments about brain anatomy in chapters/sections on ADHD and included only cross-referenced chapters as contextual information. It is possible that other non-referenced chapters within those textbooks discuss the conceptual considerations of which we are critical. Fourth, our selection of textbooks is not random, and might not be representative of textbooks used globally. International replication studies are warranted.

Conclusion

In — and likely outside of — academic textbooks, generalisations inflate neuroanatomical findings and reify ADHD. Whatever the causes or motives behind this reification, it may have serious downfalls as reification can serve many interests that may not overlap with those of the child. Among these competing interests, safeguarding the voice of the child seems necessary. As Breggin (2014) proposes, the Convention on the Rights of the Child (United Nations, 1989) might fulfil this role. For instance, respect for the views of the child (article 12.1) means we should respect the *aspiring agents'* own voice (Singh, 2013).

This also means we should acknowledge, as Singh's findings suggest, that for some children the use of stimulants gives them a sense of agency and self-esteem. On the other hand, Singh does asserts that self-esteem is related to children being able to follow disputable societal norms and the pharmaceutical industry has contributed to popularising 'normal' behaviours and emotions as part of the ongoing construction of childhood. Furthermore, 'through their direct to consumer advertising, industry may promote models of achievement and success, happy families and good parenting' (Singh, 2013, p. 364).

Besides safeguarding the voice of the child, such conflict of interest emphasise that we should take 'all appropriate measures, including legislative, administrative, social and *educational* measures, to protect children from the illicit use of narcotic drugs and psychotropic substances' (United Nations, 1989, article 33; italics added). This study has clarified that one of the premises of the worldwide prescription of psychostimulants and medical treatment of ADHD — abnormally developing brains — is far too simplistic, yet it is often presented in this fashion in academic textbooks. Therefore, improving the discourse on brain-anatomical studies and placing these small brain correlates among the many other cultural and environmental correlates is imperative to protect the rights of children.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Correspondence to: Sanne te Meerman, Researcher, Integrated Youth Policy, Hanze University of Applied Sciences, Zernikeplein 9, 9747 AS Groningen, The Netherlands, Tel.: +31 6 14829390. E-mail: s.te.meerman@pl.hanze.nl

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