university of groningen

GUIDELINES

PSYCHO-EDUCATIONAL LITERATURE ON ADHD

edited by Sanne te Meerman

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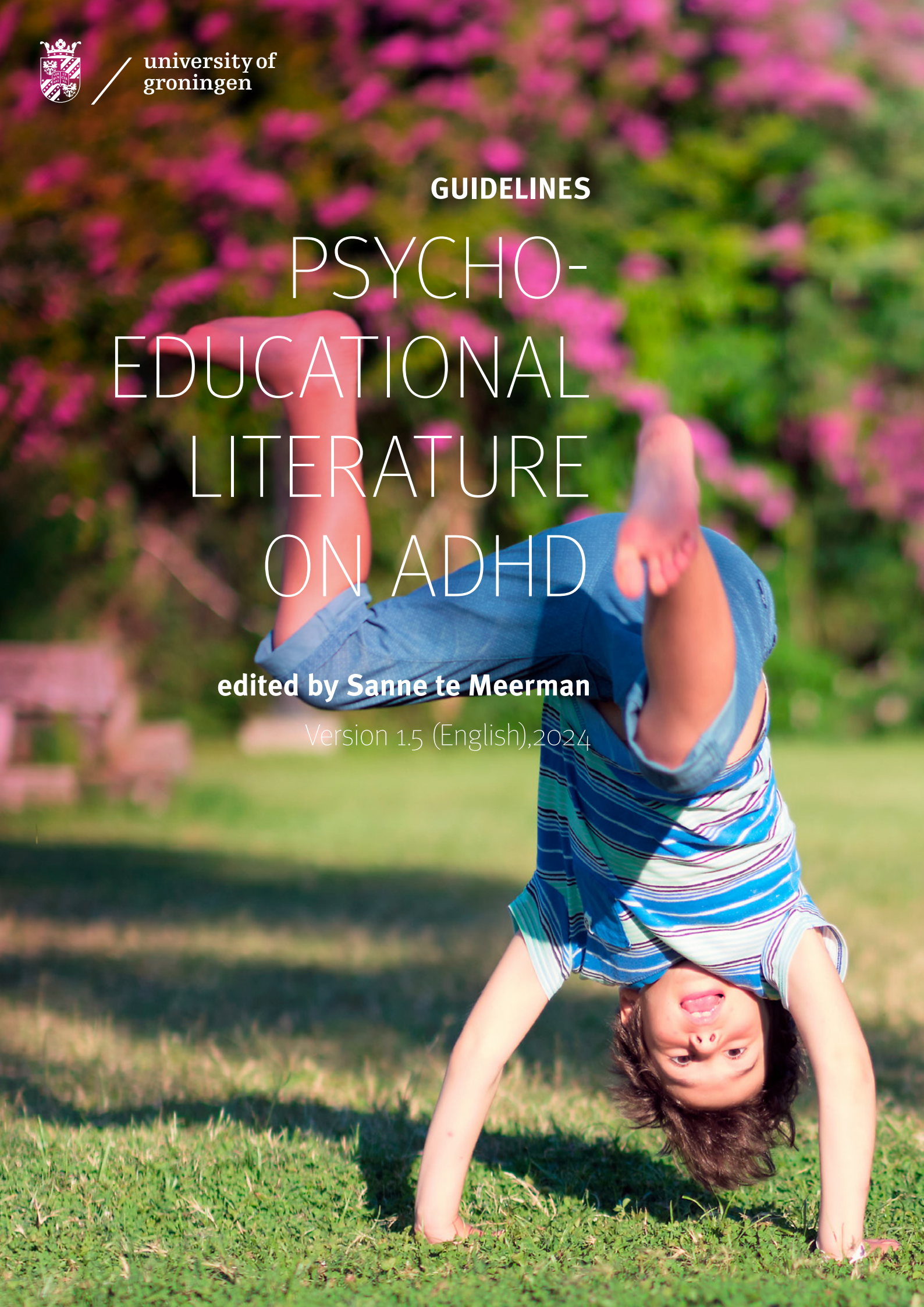

SUMMARY

ADHD (Attention Deficit Hyperactivity Disorder) is a descriptive classification. It places individual behaviors that seem indicative of a particular kind—particularly in terms of perceived challenges and experienced problems—under a single type or category. However, the behaviors that may bring about a classification under the label ADHD have widely varying motives and causes. ADHD is therefore best understood as a multifactorial descriptive classification. Moreover, the dispositional and environmental factors that are in play typically interact, and this pattern likely varies by context; the mix of factors involved in an ADHD classification is therefore highly dynamic and varies by individual and context. Although in past decennia a fairly singular biomedical view on ADHD was dominant, there is now increasing attention to individual difference, environmental factors, societal norms and contexts—why are certain behaviors experienced as negative or disordered? Despite this more nuanced view of behavior as individually determined and context- and situation-dependent, in the topical literature the descriptions of ADHD often remain one-sided or confusing. This set of guidelines gives evidence-based suggestions for understanding ADHD beyond such a narrow and one-sided biomedical view. It is an (updated) translation of guidelines on this subject published in the Netherlands in 2021.

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CONTENTS

ABOUT THE GUIDELINES

These guidelines are intended for care professionals who write about ADHD, for instance on websites, in brochures or in books. The guidelines may furthermore prove useful to psychiatrists, psychologists, social workers, and so on, who explain to parents and children what ADHD is and what it is not; and what we do know and do not know about ADHD in terms of reliable research findings. Aside from addressing care professionals, this document can also be of great interest to those with experience of restless, unruly, impulsive or inattentive behavior and ADHD and who seek to understand how research in this domain can best be interpreted. Lastly, journalists who write about ADHD are strongly advised to study these guidelines.

Some information contained in the guidelines can be complicated, even for those with a professional or scientific background. Passages that primarily serve as background information are indicated with '**[i]**'. Despite the careful crafting of this text, it is possible that some information is missing or needs to be improved. Please contact us with questions or suggestions for improvements via email to s.te.meerman@rug.nl or l.batstra@rug.nl.

INTRODUCTION

ADHD (Attention Deficit Hyperactivity Disorder) is defined in the Diagnostic Statistical Manual of Mental Disorders, currently in its fifth edition (see Appendix B). Like all disorders listed in the Manual, the entry for ADHD is a descriptive classification. It places individual behaviors that seem indicative of a particular kind—particularly in terms of perceived challenges and experienced problems—under a single type or category. However, the behaviors that may bring about a classification under the label ADHD have widely varying motives and causes. ADHD is therefore best understood as a multifactorial descriptive classification. Moreover, the dispositional and environmental factors that are in play typically interact, and this pattern likely varies by context; the mix of factors involved in an ADHD classification is therefore highly dynamic and varies by individual and context. Although in past decennia a fairly singular biomedical view on ADHD was dominant, there is now increasing attention to individual difference, environmental factors, societal norms and contexts – why are certain behaviors experienced as negative or disordered?[1]

Despite this more nuanced view of behavior as individually determined and context- and situation-dependent, in the psycho-educational literature the descriptions of ADHD often remain one-sided or confusing[2,3,4]. This makes it challenging to communicate the outcomes of ADHD-related research to the general public. In particular, it is difficult to explain that the group outcomes that are typically reported in scientific studies are often very limited in enabling predictions to be safely made about whether an individual meets the ADHD criteria. This commonplace gap between group findings and individual classification can challenge especially those without a scientific background. What is meant for example by research outcomes being statistically significant; and do children with an ADHD-classification have different genes? These guidelines answer such questions and aim to facilitate communication on the outcomes of ADHD studies amongst professionals and others.

The guidelines are primarily based on examples taken from academic textbooks as well as websites addressed to the general public. Discussed are examples that are likely to confuse readers, or that may give a distorted view of what ADHD ‘is’. The examples do not necessarily suggest that their authors lack understanding of the research they describe. More often, confusion can arise because authors may assume that their readers have prior knowledge that they may in fact not have.

Examples of better descriptions are of course also given. They are again taken from the psycho-educational literature, and they are clearly contrasted with information we consider to be of lesser quality. In those rare cases where there are no good examples ready to hand for information that seems important to get right, we constructed such an example from existing information.

These guidelines focus above all on scientific research aimed at identifying the causes of restless, impulsive, inattentive behaviors. The guiding assumption in research of that sort is that prior to effective support and care, or prior to contextual adjustments to be made, a thorough understanding of the background and course of behavioral problems is needed. Chapter 1 of the guidelines discusses ADHD and brain studies; chapter 2 discusses ADHD and environmental influences, and chapter 3 discusses ADHD and genetics. In chapter 4, several common challenges to presenting clear research results are discussed. Finally, Chapter 5 discusses key choices that inevitably need to be made in writing about ADHD research outcomes. Full references and further information about the guidelines can be found in the appendices.

CHAPTER 1

ADHD AND THE BRAIN

In this chapter we discuss psycho-educational literature on brain size, brain anatomy and brain activity of groups of children and adults meeting the ADHD criteria, compared with control groups: groups of people without an ADHD classification. This research is mostly concerned with identifying the cause of impulsive behaviors and inattentiveness and looks for that cause in the brain. Unfortunately, both the causal explanation and by consequence the interpretation of such studies are problematic. Here are some important points of consideration.

THE INTERPRETATION OF RESEARCH EVIDENCE

- Research often examines an **average difference** between a research group of people with an ADHD classification and a control group of people not meeting ADHD criteria. Researchers may for instance compare the average brain size of a group of people with an ADHD classification—the case group—to the average brain size of a control group. When there is less than 5% chance that the average difference found between the case group and the control group is coincidental, the result is called **'statistically significant'**. However, this does not mean that the difference is useful in daily practice, or **clinically relevant.** The clinical relevance of statistically significant findings depends, amongst other things, on effect size: how big or small is the actual difference found between, say, the average brain size of case group and control group?

- For example, it may be reported that the average size of a particular brain area is smaller across case group individuals (those with an ADHD classification) than that found across control group individuals. While it may be statistically significant, the effect size of such a finding is considered small if there are also many people in the control group in which this area of the brain is smaller. If on the other hand a smaller area of the brain would typically be found in the case group while being rarely found in control group individuals, then this would be indicated by a large effect size. As a rule, brain studies of ADHD report very small effect sizes; it seems there is inevitably a lot of overlap between the case groups and the control groups. This large overlap explains why brain tests and brain scans are uninformative and lack clinical relevance: the very small effect size may tell us something about groups of individuals, but they tell us next to nothing about individuals as such.

- Correlation is not the same as causality. While an attribute such as smaller size of a brain area may happen to correlate with ADHD classification, this does not by itself force the conclusion that a smaller brain area causes restless or inattentive behaviors. If, purely hypothetically speaking, a correlation was found between skirt length and gross national product, then it would be unwise to suppose a causal connection: the correlation might well be a spurious one. A related issue concerns the direction of causality. Brain plasticity means that the brain develops in response to stimuli of various kinds. Studies of musicians' brains show that areas of the brain associated with fine motor skills are larger, on average, among well-practiced musicians. These brain differences are most likely acquired through diligent music practice. In this example brain structure has not caused the behavior (being able to play an instrument well), but the behavior—a lot of musical practice—has most likely influenced brain development and structure.

- It is also important to consider limitations concerning the extent to which case- and control groups are truly representative of cases and non-cases in the general population. There are often problems in the selection of studies' participants. For example, the children that researchers select for the case group often display the restless/inattentive behaviors of the ADHD criteria in a relatively severe manner. At the same time, the children that researchers select for the control group are often what could be called hypernormal. They are selected because neither they themselves, nor their immediate or wider family members have ever used psychologic/psychiatric services. While there is strong empirical invitation for selecting such extreme samples—selection at the extremes is likely to bring about more pronounced statistical results—such extreme samples do not represent the actual population very well. The results of studies with these relatively extreme samples can therefore not automatically be transferred to other groups and individuals with/without an ADHD classification.

The examples we list below highlight some of the challenges in communicating the outcomes of group studies clearly[5]. The reader may interpret small effect sizes as absolute effects, or infer causality where only correlation is found. Or the reader can think that an average difference found between case group and control group applies to all children. We discuss several desirable and less desirable ways of describing the outcomes of studies in neuroanatomy, neurophysiology and neurochemistry.

ANATOMY

The example below can easily confuse readers:

> **Example 1: less desirable**
> *'In children with ADHD, there is a general reduction of volume in certain brain structures, with a proportionally greater decrease in the volume in the left-sided prefrontal cortex.'* [6]

This example does not mention that case groups overlap with control groups. Many children with an ADHD classification do not have a general reduction of volume in certain brain structures, or any decrease in the volume in the left-sided prefrontal cortex. Research indicates[7] that readers may get the impression that all people with an ADHD classification have different brains[8][i]. The next example is clearer in this respect.

> **Example 2: more desirable**
> *'When groups of children with ADHD are compared to groups of children without ADHD, there are performance differences in computertasks and measurements of higher cognitive functions, brain scans and genetic make-up (...). However, there are different anomalies in different children with ADHD, and there are consistently children with ADHD that do not deviate from other children. It is not possible to diagnose ADHD in this fashion, and for diagnoses we depend on observable behavior as defined in the DSM-IV-TR9.*[9]

The above example clarifies that children who meet the ADHD criteria do not necessarily have smaller areas of the brain. In logical terms, smaller brains are not a *necessary condition* for ADHD classification. The next passage (example 3) emphasizes that when someone does have a smaller brain area, this does not mean s/he has ADHD. In logical terms, smaller brain areas are *not sufficient* for ADHD classification.

> **Example 3: more desirable**
> *'... non-diagnosed, typically developing youths exhibited brain changes similar to youths with the syndrome of ADHD....'* [10]

Examples 2 and 3 combined indicate that smaller brains are neither a necessary (example 2) nor a sufficient condition (example 3) for ADHD. That brain size fails to meet the dual

[i] The erroneous suggestion that average group outcomes, such as smaller brain size in a group of people who meet the criteria for ADHD, apply to everyone in the group is known as the ecological fallacy[8]. Group outcomes predict very little about individuals in the group.

explanation criteria of necessity and of sufficiency helps to explain why it is not possible to identify those who meet the criteria for ADHD classification by looking at brain size. Smaller brain size might or might not explain deviant behavior, since brain size differences are found among children showing normal behavior too.
A simple example may help to clarify the point. Like brain size, parental divorce has also been found to be a risk factor for ADHD classification, though of course not every child with an ADHD classification has experienced a divorce—divorce is not a necessary condition. Furthermore, divorce does not inevitably bring about an ADHD classification in children—divorce is not a sufficient condition, either. In general, critically submitting correlational studies to a test of necessary and sufficient conditions can be helpful in considering risk factors involved in almost all disorders defined in the Diagnostic Statistical Manual and can help avoid the 'ecological fallacy' mentioned in the footnote on page 9.

Persistence and anatomy

Research has shown that the small average differences in brain anatomy between groups of those with and without an ADHD classification are neither permanent nor persistent. A large meta-analysis from 2017 [11] shows there are no statistically significant differences in brains of adults with an ADHD classification compared to control groups. Often this key finding is not mentioned, as in the example below.

> **Example 4: less desirable**
> *'…reduced brain volume has been revealed for several brain structures, with reductions in total volume estimated at 3 to 5%.'* [12]

A simple textual clarification, such as the one below can remind readers that a low brain volume is not necessarily a persistent attribute of the brain.

> **Example 5: more desirable**
> *'Ultimately, the growth of the brains of the children with ADHD caught up with those of unaffected children.'* [13]

We emphasize that these outcomes too are based on group studies. Some children with an ADHD classification do retain smaller areas of the brain, as do some children without an ADHD classification. In both groups, parts of the brains can also become larger than average. With respect to the variation found again here too both across groups and at individual level, the conclusion is that findings of this sort lack clinical relevance.

'Normal' brain development

Finally, it is generally speaking undesirable to make strong normative claims about ADHD and brain anatomy. By normative claims we mean in this case overly confident but scientifically questionable assertions about what is to be considered normal. Science does not lend itself terribly well to determining what is to be considered normal, since that judgment tends to rest in social ethics—typically involving a mix of moral and cultural considerations and values—rather than merely being indicated by largest number. This means that claims about which brain anatomical features are considered (ab)normal are best avoided. Research can therefore determine group differences, but cannot thereby determine which trajectory of brain development is better. For instance, on average, men have larger brains than women. This does not mean all men have larger brains and it certainly does not mean that women have a brain developmental disorder. Even less does the finding mean that it is better to be a man; smaller brains could perhaps be more efficient. Moreover, when women's smaller average height is taken into account, their brains are not smaller.
However, the next quote precisely suggests that differences in group averages imply brain-related problems.

> **Example 6: Less desirable.**
> *'In general, researchers now assume that these active, restless children suffer from a disorder in the development of the central nervous system.'* [14]

In keeping with our discussion above, slower than average brain does not imply a brain disorder, nor does it need to be a problem, at all.
The next example discusses slower than average brain development while avoiding the claim that faster brain development is better. At the same time, the example points to overlap between research groups and the absence of anatomical differences in adult research samples.

> **Example 7: more desirable**
> *'the development of the brain, which can be slower in some children who meet the criteria for ADHD, has in general caught up by the time children reach adulthood. Brain development does not always fully catch up in every child with an ADHD classification, but the same is true for children without an ADHD classification: they can have similar lag in growth that does not completely catch up in adulthood. In both groups a more or less permanent lag in brain development does not necessarily affect behavior in a negative way, as brains simply differ from one person to the next.' (example by taskforce).*

Table 1: base ingredients for good communication on brain anatomy:

	Less desirable	More desirable
Clearly assess whether or not there are necessary and sufficient conditions of risk factors	Suggesting that all those with ADHD have smaller brains (ex. 1,2)	Emphasizing that there is no consistent relation between brain anatomy and ADHD (ex. 3 & 4)
Inform about the non-persistence of brain growth	Failing to mention that slower than average brain development is not necessarily permanent (ex. 1 & 6)	Emphasizing that brain development varies and in ADHD it is only slower than average during childhood in some children; emphasizing brain development is variable from one person to the next (ex. 5)
Avoid normative claims	Suggesting that brain development that is not average implies disorder or illness (ex. 6)	Emphasizing that differences in brain development or shape does not necessarily imply a disorder or hampered brain development (ex. 7)

ADHD AND NEUROCHEMISTRY/PHYSIOLOGY

Besides the study of brain anatomy, research on ADHD also includes the study of brain activity and the presence of certain neurotransmitters in the brain. A well-known neurotransmitter is dopamine. In research that seeks to clarify the functioning of the brain and the role of neurotransmitters, subjects typically perform certain tasks while researchers examine, for instance, brain activity. Research into neurophysiology and chemistry is similar to research on neuroanatomy in design. Again, groups of children with an ADHD classification are often compared with control groups, using techniques such as magnetic resonance imaging (MRI).

The outcomes of studies of neurophysiology and neurochemistry also show many similarities with studies of anatomy. At the group level, small mean differences are found but again there is much overlap between the research groups. So again, no unique brain characteristic is found, i.e. no particular blood flow or neurotransmitter is present to such a categorically greater or lesser degree that it enables researchers to predict whether a person will meet the behavioral criteria for ADHD. An additional problem relative to brain anatomy research is that brain

physiology and chemistry are much more variable. While performing a particular task blood flow may be lower or higher at any time of measurement without this necessarily indicating a revealing pattern. Furthermore, there are no calibration values that can tell researchers what is normal or abnormal with respect to blood flow or the amount of neurotransmitters present in the brain at any one time. Since it is clearly important to communicate this to readers, the following excerpt is less desirable.

> **Example 1: less desirable**
> *'In a healthy brain, concentration causes blood flow to increase appropriately in certain regions, especially the prefrontal cortex. This helps us to focus, plan ahead, stay organized, and follow through on tasks. However, when people with ADD/ADHD try to concentrate, blood flow decreases in the prefrontal cortex, making it more difficult for them to focus and filter out distractions. In fact, the harder they try to concentrate, the harder it can get.'* [15]

This excerpt does not mention that these are group findings, and this omission suggests dysfunction in anyone with an ADHD classification. There is also no basis for the claim that blood flow to the prefrontal cortex actually decreases when people with an ADHD classification try to concentrate on a task. Furthermore, this excerpt suggests that there is clarity about the distinction between normal and abnormal functioning. However, there are no known calibration values about what, for example, constitutes a normal or abnormal amount of blood flow[16]. Partly because none of the values found for variables like blood flow only occur in people with ADHD, it is difficult to say what can be considered a normal or abnormal value. And vice versa, in many people with ADHD there is no higher or lower than average blood flow. Hence the following excerpt is more desirable.

> **Example 2: more desirable**
> *'ADHD is probably not a single neurobiological entity, but rather an umbrella term covering a variety of pathophysiological profiles. Each deficit (...) affects only a minority of cases.'* [17]

This example highlights that there are no biological differences that apply to the ADHD group as a whole, as differences may affect the behavior of only a minority of the group. However, the excerpt retains an undesirable normative claim in its suggestive reference to deficit. The following excerpt omits normatively charged jargon and is therefore preferable.

> **Example 3: more desirable**
> *'... when performing more complex tasks, children with ADHD use brain regions associated with more basic (motor, visual and spatial) processes, whereas children without ADHD are inclined to use brain regions associated with the planning and organization of behavior, i.e. the higher cognitive functions.'* [18]

The authors avoid the suggestion that the tendency to use certain brain areas is better or worse than using other areas. The authors also partly avoid generalization. They write that children without ADHD are inclined to use other brain areas; this acknowledges that this is a tendency that does not apply to all children. However, some information is still missing. For example, children with ADHD do not always, but at best more often, use brain areas associated with more basic processes. Also, it would be good to clarify that children without an ADHD classification may also use these brain areas in complex tasks. A better example would be the following.

> **Example 4: most desirable**
> *'When performing more complex tasks, groups of children who meet the ADHD criteria are on average slightly more likely to use brain regions associated with more basic (motor, visual and spatial) processes, whereas groups of children without an ADHD classification are on average more likely to use brain regions mostly associated with the planning and organization of behavior, i.e. the higher cognitive functions. These are group level findings that do not tell us about individual children with and without an ADHD classification' (example taskforce).*

Neurochemistry and medication

Research on neurochemistry can often address the effects of medication. Because we are in fact unable to measure concentrations of neurotransmitters such as dopamine and norepinephrine directly, researchers look instead at the amount of receptors for these substances in the brain. There is evidence that adults with an ADHD classification have on average more of these brain receptors, and the assumption is that a higher number of receptors indicates there being less dopamine and/or norepinephrine available in the brain. Active substances in medication such as methylphenidate make these neurotransmitters more available. Literature can be unclear on such matters, as in the following example:

> **Example 5: less desirable**
> *'ADHD is a neurobiological disorder. Something is not going well in the brain; there is deficiency in so-called neurotransmitters (dopamine and norepinephrine). These neurotransmitters ensure that information between one nerve cell is passed on quickly and properly to another nerve cell. Because of the deficiency, that process does not go well or does not go fast enough, with all the consequences that entails.'* [19]

Firstly, this excerpt generalizes: the results are at group level so it is not clear whether there is an alleged deficit at individual level. The normative aspect is undesirable: there are no known values of dopamine or norepinephrine that are too high or too low. Furthermore, these kinds of values are not known for children. For ethical reasons, PET scans have not been used to study the amount of receptors in the brain of children. Even if the assumption that the presence of more receptors means that less dopamine is present in the brain is at all correct, we therefore still do not know whether this is also the case in children. The following example is more desirable.

Example 6: desirable

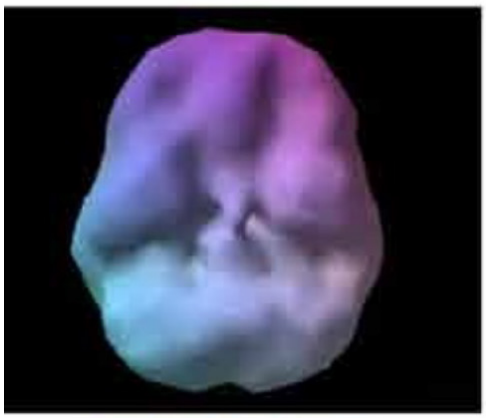
'... it would behoove us in the scientific community to avoid describing findings as 'abnormal' (i.e., abnormal blood flow, abnormal circuitry, abnormal connectivity, abnormal activation) and instead to use more accurate descriptive terms such as 'statistically less activity' or 'statistically less glucose metabolism' or 'different' when comparing neuroimaging findings between participants with ADHD and control subjects.' [20]

In this statement, normative statements are avoided, and with the explicit reference to findings being statistical findings it is made clearer, at least to many scientists, that these are group findings. This might be described in a more accessible way by stressing that the findings concern only group level findings, not individual level findings. It would furthermore also help to remind readers that in neurochemistry research less is known about children and that findings from data collected on adults do not necessarily apply to children.

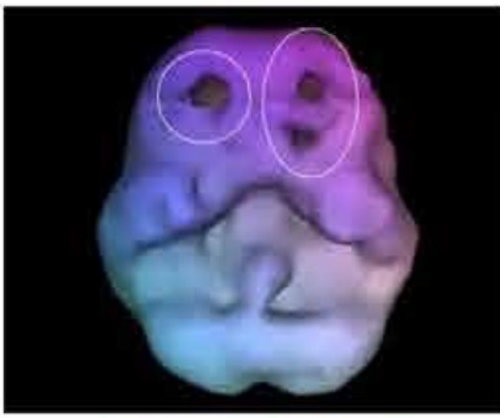
The use of images

Information about ADHD and brain research regularly uses images of brain scans. This can help to clarify at what locations in the brain average differences in structure, activity or levels of a particular neurotransmitter were found. However, images are sometimes used of individuals in the research or control group that are not necessarily representative of the average group difference that is observed. A more extreme and striking example from both the study and control group may be used instead, to suggest substantial anomalies in those with an ADHD classification. It may then in addition be suggested, for example in the image's caption, that each person in the group shows such a pattern, as in the following example:

Example 7: less desirable:



Healthy Brain Scan



ADD / ADHD Brain Scan

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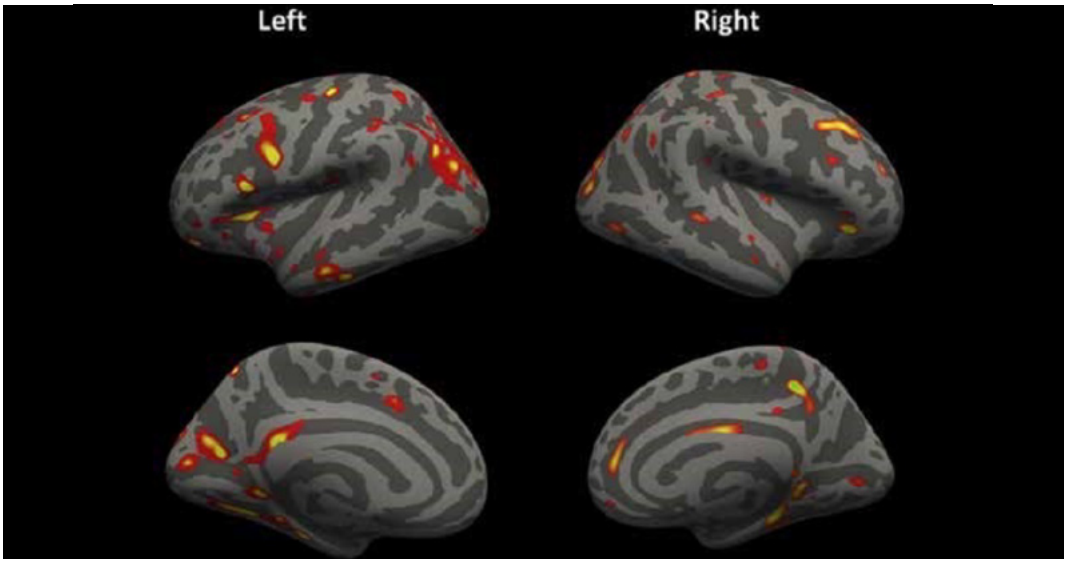
'Spect (Single photon emission computed tomography) is a nuclear medicine study that evaluates blood flow and activity in the brain. Basically, it shows three things: healthy activity (blood flow), too little activity, or too much activity. The healthy surface brain SPECT scan on the left show full, even symmetrical activity. The ADD/ ADHD scan on the right, taken during a concentration task, reveals decreased blood flow (the areas that look like 'holes') in the prefrontal cortex.'

By generalizing group averages to individuals, the caption further contributes to likely misunderstandings and misinterpretation of such scans. Instead, no pattern of blood flow activity is unique to those with an ADHD classification. The excerpt is also highly normative, suggesting that one level of blood flow activity is healthy, while others are not. To –again- avoid unwarranted normative judgment and avoid the suggestion that this pattern of brain activation is unique to everyone in the group with an ADHD classification, it is preferable to speak of group differences in which the pictures can serve to make clear where the group differences occur. The following example is therefore more desirable, and fortunately much more common:

Example 8: More desirable



'Brain areas for which smaller volumes were found in the groups with ADHD, with or without ODD, relative to the control group, based on whole-brain analysis (not corrected for multiple comparisons; p < 0.0001)'

'Colored areas: significant group differences; yellow color: center of area; darker areas: sulci; lighter areas: gyri [22]

This example is preferable because it speaks of group differences.

Table 2: Base ingredients for good communication on neurophysiology and chemistry.

	Less desirable	More desirable
Clearly assess whether or not there are necessary and sufficient conditions of risk factors	Suggesting that all children with ADHD have higher or lower activity or presence of certain neurotransmitters in the brain (ex. 1)	Making it clear that children with ADHD do not necessarily have a higher or lower activity or presence of certain neurotransmitters in the brain, and that these are very small differences at the group level (ex. 4)
Explicitly recognize variability of brain activity and/or neurotransmitters present within both case groups and control groups	Leaving unmentioned that there are no standard values for the presence of neurotransmitters, brain activity or blood flow (ex.1, 5)	Making it clear that brain activity and the presence of neurotransmitters not only varies within the group, but also varies greatly within the individual from moment to moment (ex. 6)
Avoid normative claims	Normatively charged jargon that suggests that there are certain values of brain activity or the presence of neurotransmitters that are 'too high' or 'too low' (ex. 5)	Speaking of differences rather than 'dysfunction' or 'deficiency' and making it clear that no certain desirable values are known (ex. 3, 6)
Use illustrations carefully	Suggesting that scans of individuals apply to everyone in the control/research group (ex. 7)	Use illustrations explicitly to explain that the differences found apply only at the group level and have little predictive value for individuals (ex. 8)

CHAPTER 2

ADHD AND ENVIRONMENTAL INFLUENCES

> The same principles apply in writing clearly on environmental influences on ADHD as in writing about findings in neuroanatomy, physiology and chemistry. Generalizations should be avoided and clarity about the interpretation and limitations of the research are just as important here. In the literature on environmental influences it is actually difficult to find less desirable examples of writing. Hypothetical statements such as: 'Persons with ADHD have less money or suffer from poverty' have fortunately not been encountered, unlike statements such as: 'Persons with ADHD have smaller/differently functioning brains'.

This is not without interest, considering that poverty and social deprivation have a stronger connection to ADHD than do brain attributes[23,24]. Yet, when describing environmental influences, hedging expressions (ifs and buts) are more frequently added. Still, in this context too there is room for improvement.

> **Example 1: less desirable**
> '*....no attention to other possible causes of ADHD such as poverty, overburdened parents and teachers, and the performance society.*' [25]

Although it is important that factors such as poverty receive attention, this passage suggests that these factors have a direct causal relationship with ADHD. As we have noted, even if a correlation between two variables is found (for example, poverty and ADHD), it does not necessarily follow that one causes the other. A third variable or a combination of other variables can also cause the two variables to show a correlation. In the case of poverty in relation to ADHD, this may include unstable family circumstances, domestic violence and attachment problems, that may be the root cause rather than poverty per se.

> **Example 2: more desirable**
> *'Adverse social and family environments such as low parental education, social class, poverty, bullying/peer victimisation, negative parenting, maltreatment and family discord are associated with ADHD. However, the designs used so far have not been able to show that these are definite causes of ADHD.'* [26]

As this passage notes, the research designs that have been used to date cannot yet be conclusive about causality. It is also important to consider that overarching concepts such as poverty can be examined in different ways. For example, the neighborhood in which one lives, housing, the means to participate in sport and other activities, etcetera, could each exert different influence. Thereby, poverty may itself have causes in the social domain but may also have to do with individual capabilities.

Although seeing factors as poverty in perspective is important, environmental problems are regularly downplayed, while biological triggers are emphasized. This is demonstrated in the following example.

> **Example 3: less desirable**
> *'... children with ADHD are more likely to come from homes in which marriages are unhappy and family stress is high. But a stressful home life rarely causes ADHD. Rather, these children's behavior can contribute to family problems, which intensifies the child's pre-existing problems.'* [27]

In this excerpt, the associations that exist between family stress and the underlying causes for that stress are almost entirely set aside under the claim that stressful home life rarely causes ADHD, and child behavior is presented instead as a trigger, arising from nowhere, for experienced problems. Although children's behavior may certainly contribute to family stress, the excerpt hardly does justice to the considerable effect that family stress and unhappy marriages are known to have on child behavior. The next example still somewhat downplays the links to environmental influences, but is more nuanced.

> **Example 4: more desirable**
> *'Environmental factors relate to birth, prenatal exposure to nicotine and alcohol, environmental toxins, sensitivity to certain color and nutrients and the psychosocial influence of upbringing and family. These factors could include low socioeconomic status and/or pathology present in the educators -such as depression, alcoholism and antisocial behavior- issues that can increase the individual vulnerability. These risk factors are based on correlational research—indicating that there is some relationship—but this says nothing about causation.'* [28]

It should be remembered that a link or correlation may not necessarily imply causation, but correlation is a prerequisite for causation and thus may well indicate that one thing partly causes another. Correlation and causation are discussed again in chapter 4 because of their importance[i].

[i] It is quite possible that both brain development and environmental factors explain some of the behavior for some individuals with an ADHD classification, but not all behavior can be explained by these factors.

The following example gives some more perspective on the multifactorial side by showing that parenting style itself is a risk factor. The one-sided approach in which the child is the main cause of problems is also put into perspective:

> **Example 5: more desirable**
> *'The contemporary view is that disturbed parenting behavior or disrupted parent-child interactions are the result of child temperament and impulsive and oppositional behavior rather than that they cause the behavior in the child. In short, problematic parent-child relationships may maintain or intensify the child's problematic behavior but do not cause these behaviors. However, (...) a number of studies, also show that a deficient parenting style, e.g. lack of responsiveness and overstimulation on the part of the parent, is a risk factor for the development of attention problems and hyperactivity in early childhood.'* [29]

This excerpt gives perspective to the one-sided view that biological causes are the basis of problematic behavior and the environment only perpetuates or intensifies them. As discussed, a strong basis does not stem from brain research because of the small effect sizes. Genetic studies also cannot lead to such a conclusion, as the next chapter will discuss.

The foregoing is again summarized in a table:

Table 3: Base ingredients for good writing about environmental influences.

	Less desirable	More desirable
Clearly assess whether or not there are necessary and sufficient conditions of risk factors	Suggesting that a relationship with adverse conditions automatically implies causality (ex. 1)	To make clear that correlation does not imply causation (ex. 2, see also chapter 4)
Clearly distinguish nature from nurture and give due consideration to both	To suggest that the environment only contributes to an organic disorder that was already present at birth (ex. 3)	Clarifying the interaction between predisposition and environment (ex. 4, 5, see chapter 3)

CHAPTER 3

ADHD, HEREDITY AND THE ENVIRONMENT

There are many studies available on the heritability of ADHD. The first heritability studies looked at similarities and differences in ADHD characteristics between family members and other relatives. In this area, twin studies in particular are salient. Contemporary research focuses on finding the genes that play a role in people with an ADHD classification. These are the well-known 'case-control' studies, that study the higher or lower incidence of certain genetic variants in groups with an ADHD classification (the case group) or without (the control group). Both types of research will be explained, starting with twin studies. More and less desirable examples of literature will follow.

TWIN STUDIES

Twin/family and adoption studies look at similarities and differences in behaviors between children and adults who are more or less biologically related to each other, such as parents and children, siblings, and so on. If it turns out that people often exhibit the same behaviors within families, it may indicate that heredity plays a role. The tricky thing is that people within a family also live in one and the same environment, making it difficult to distinguish the relative influence of heredity and environment on behavior.

Twin studies in particular are therefore considered a better method than family studies to estimate the relative contribution of genetics and environment to behavior. The similarities in behavior of identical twin pairs, who are genetically similar, are then compared with the similarities in behavior of fraternal twin pairs, which are genetically similar by about 50% (on average). If identical twins are much more similar in their respective behavior than fraternal twins, then genes are concluded to play an important role; in these studies it is often assumed that both identical and fraternal twin pairs live in much similar environments.

MOLECULAR-GENETIC STUDIES

The second type of research, molecular-genetic studies, looks directly at particular genetic variants, specifically so-called Single Nucleotide Polymorphisms (SNPs). For the purpose of these guidelines it would take too long to explain genetic variants in detail, but the point is that in the case of ADHD, the genes of groups of people with and without an ADHD classification are compared. If certain genetic variants are relatively more or less prevalent in the different groups, these genetic variants may be involved in the behavior.

THE ADDED VALUE OF TWIN STUDIES AND MOLECULAR GENETIC STUDIES

Both types of research have provided useful knowledge. For example, twin studies have demonstrated that many character traits such as temperament and (hyper)activity have a substantial heritable basis. This has implications for the extent to which we can expect to change that temperament, should it be considered at all desirable to do so. Certain genetic variants appear to be somewhat more common in ADHD-classified children, although the results are not yet very consistent; and again there is considerable overlap between the case groups and the control groups. Thus, ADHD-related genetic variants do also occur almost as often in the control group, while these genetic variants do not necessarily occur in children with an ADHD classification.

In molecular-genetic research, recent studies have combined all the genetic variants that have been found and combined their effects into a single genetic risk score. This means that the total effect of multiple genetic 'risk variants' in relation to ADHD can be investigated. Although the effects are larger than for single genetic variants, this research also shows only small effects. As is the case for all the previous group studies we have reported thus far, there is considerable overlap between groups: people with an ADHD classification do not necessarily have the genetic variants, and people without an ADHD classification have them almost as often. However, these studies are still in their infancy stage, and the outcomes have yet to be examined more closely.

Interpretation of outcomes and limitations of twin studies

It is frequently suggested that, based on twin studies, a simple heritability score can be calculated for ADHD as a disorder entity. This is regularly a very exact number, as in the following example:

> **Example 1: less desirable**
> *Genetic factors play an important role; ADHD tends to run in families and has a heritability rate of 74%.*[30]

Mentions of this sort fail to add that the seemingly exact figure, confidently reported, is in fact a calculated average of different outcome figures reported across many studies. The outcome figures differ between studies because different populations bring about different heritability estimates. Heritability coefficients describe behavioral traits in a given group of people/population at a given time, as is reflected in rather different figures being reported across research studies. However, reporting a single value of an assumed 'heritability coefficient' suggests that there is one value that applies to ADHD behavioral traits.

More in general, heritability is a complicated concept for many people. It is in fact a 'population' parameter, and not a 'trait parameter'. An example might clarify what this means. In a population in which social problems, such as divorce or poverty play a major role, the contribution of genetic factors may differ from their contribution in populations in which such problems are much less prominent. This is precisely the case with ADHD. Studies have shown that ADHD is much more often diagnosed in areas with lower SES (Social Economic Status[i]). Poverty - per definition-, alcohol abuse, divorce, attachment problems and many other disadvantaging situations are more typical of areas with lower SES. In these areas, such social disadvantage factors will contribute more strongly to concentration problems, impulsivity and hyperactivity.

Given the considerable presence of social risk factors in areas with lower SES, the influence of heredity may therefore be relatively less prominent in such areas than in socially secure areas where social disadvantage is more rare and children enjoy better life conditions. In such a more favorable environment their genetic predisposition is also more likely to flourish and less likely to run up against developmental barriers. In short, the influence of predisposition/environment depends on the group of people studied in heritability research. Because the relative influence of genetic predisposition varies along this and other dimensions, it is therefore desirable to speak of a 'range' or an interval of heritability, not a fixed value:

> **Example 2: more desirable**
> *'According to twin studies, ADHD is among the most heritable disorders with estimates between 60 and 90%'.* [31]

Expressing heritability as an interval rather than a fixed number hopefully also has the advantage of highlighting that heritability studies—like studies of brain anatomy, brain physiology, et cetera—are group studies. Twin studies or molecular genetic studies, being based on group statistics, cannot predict the influence of genetics for an individual person, and so their clinical relevance is modest. The following example helps to make this clear.

> **Example 3: more desirable**
> *'The nature and extent of the contribution made by genetic and environmental factors varies from case to case.''* [32]

[i] Social Economic Status (SES) is widely used, mostly by social scientists, to discuss the extent to which input variables such as income, education, housing and the like affect outcomes such as income in later life, health, children's educational performance, and so on.

The fallacy of heritability and environment as contrast

What also complicates twin studies is that a given estimate of heritability, such as 50-80%, does not mean that the remaining percentage (20-50%) of the variation in behavior can be explained by the environment. Indeed, estimates of heritability themselves already and inevitably include environmental influences woven into the heritability estimate, since the individuals in the research groups that were studied inevitably lived in social settings and in situations relevant to behavior. A high estimate of heritability only means that in a given population, more of the found *variation* was due to heritability. This may then also be because the environment to which the estimate applies was fairly uniform. A different example can help to clarify this point. Consider the notion of reading ability. If good reading instruction is offered in a population, then any differences in reading ability that remain will be mainly due to heredity. This can result in twin studies showing high heritability. However, the influence of the environment on overall reading ability is known to be far greater than heritability. Hence a high heritability score for reading ability does not mean that reading education has no effect on the reading skills of children. In fact, consistently good and evenly spread educational input will give hereditary factors far more opportunity to manifest statistically. Translated to ADHD behavior, high heritability estimates do not mean that environmental factors have little influence on such behaviors being manifest. The following statement is therefore less desirable.

> **Example 4: less desirable**
> *'Problems in parenting or parenting styles may make ADHD better or worse, but these do not cause the disorder.'* [33]

In the next example, it is claimed even more categorically that specific child factors matter while parenting does not.

> **Example 5: less desirable**
> *'Sometimes, you still hear that it is because of parenting that a child is so restless. That is absolutely not true. [...] therefore, ADHD is not a disease, it is not due to parenting. However, ADHD is hereditary. It can, so-called, 'run in the family.'"* [34]

Because genetic predisposition and the environment always interact and an ADHD classification is based entirely on behavior manifestations and not, for example, on physical factors such as brain activity and brain size, it can quite simply not be claimed that environment and situation (including also parenting) do not affect the behaviors in question. After all, both genetic predisposition and the environment have in combination exerted their influence when a person exhibited the behaviors that are the basis of the ADHD classification. In a favorable environment and with good education, children with a predisposition for impulsiveness or busy behavior can learn to control themselves better so that the criteria

for ADHD are no longer met, although they will certainly not succeed equally well at every moment in time. The following statement makes clear that it is more difficult to dichotomize genetic predisposition and the environment.

> **Example 6: more desirable**
> *'Genetic and environmental influences are profoundly intertwined for ADHD and need to be considered jointly.'* [35]

A more elaborate way of making this clear is the following example:

> **Example 7: more desirable**
> *'In contrast to the high heritability estimates, the effects of specific genes are small. When aggregated, they account for only a fraction of variance in symptom expression. How can this gap be explained? First, twin studies, although a potentially powerful tool for dissecting genetic and environmental effects, need to be interpreted with caution for a number of reasons, as they may overestimate genetic main effects (...). For instance, heritability estimates subsume the effects of gene x environment interactions so that subtler environmental effects can be missed.*
>
> *Second, it remains possible that a large number of genes, some of at least moderate effect, exist but have yet to be identified. The results from linkage studies, if further replicated, provide support for this although genes of major effect are unlikely (...)*
>
> *Third, genes may interact with each other (...) and with environmental risk factors (...) to increase the risk of ADHD in a non-linear manner so that genes of small main effect have disproportionate power when acting together or with environmental factors.'* [36]

This last example shows how authors can and should express the pitfalls of heritability studies. It is moreover important to note that studies of 'gene x environment' interaction and of other genes involved in 'GWAS' (Genome Wide Association Studies), have not yet produced results that can explain much of the behavior associated with ADHD. The effects found are small. No genes with a sizable average effect have been found either: only genetic variants with small size effects have so far been found[i].

[i] Previously found gene variations were based on candidate gene studies. In these studies, certain gene variations were examined that were 'candidates' to display an effect. For example, they were selected because they were related to the brain or to dopamine. As yet, the outcomes of these studies are not reproduced in genome-wide studies. This means that the outcomes of these candidate gene studies were most likely the result of findings by chance due to the use of research samples that were too small. Combined with publication bias, the scientific phenomenon that positive findings are more likely to be published, this has led to these 'false positive' findings not having been filtered out in published meta-analyses. After all, if positive findings are published more often, those effects will also be found in meta-studies because studies that show no effect are insufficiently included in meta-analyses. It is therefore important to stop relying on (meta-analyses of) candidate gene studies and sooner take genome-wide studies into consideration, provided they have been conducted with large research samples.

Molecular genetic studies

In the previous example, the strong effects of twin studies are contrasted to the low effects shown by molecular genetic studies. Thus, the genetic variants found seem to explain very little of the behavior. The contrast discussed by the authors provides an opportunity to highlight limitations of twin studies and explain so-called interaction effects between genetic predisposition and the environment. In textbooks, this 'gene x environment' interaction is not often highlighted and it may be the same in other forms of education[37]. Explicitly mentioning 'phantom heredity' or the 'problem of missing heredity', as this phenomenon is known, occurs even less.

By not discussing the contrast between twin and molecular genetic studies, the false impression may arise that children with an ADHD classification have genetic variants that 'normal' children do not have, as in the following example.

> **Example 8: less desirable**
> *'From twin, adoption and family studies, genetic influences are known to play an important role in the etiology of ADHD; the disorder might be determined for about 60 - 80% by genetic factors. The risk of ADHD in brothers and sisters of a child with ADHD appears to be about three times greater than in the general population (Biederman, 2005). Furthermore, clear indications have been found for the involvement of genes that influence the dopaminergic systems (DRD4, DRD5, dat-1). So-called 'genomewide linkage studies' have shown possible ADHD associated regions on chromosomes 16pi3, i5q, 9q and yp and i7pn.'* [38]

First of all, the involvement of the gene variations discussed is insufficiently specified in this excerpt. After all, the effect sizes are small and there is much overlap in case groups and control groups: many people with an ADHD classification do not have these gene variations, and the gene variations are almost as common in the control groups with people without an ADHD classification. The following excerpt clearly explains the small influence of genetic and other influences:

> **Example 9: more desirable**
> *'Finally, it is emphasized that the (...) etiological factors each make only a small contribution to the development of ADHD. It is the combination of various genetic and environmental factors that increases the risk of developing ADHD. In addition, there are different combinations of factors and different developmental pathways that can ultimately lead to ADHD.'* [39]

It is important to re-emphasize that most genetic variations that were found in candidate gene studies were not found as a possible risk factor in Genome Wide Association Studies. These GWAS did find other genetic variations, but even these can only explain little of the behavior associated with ADHD. 'Normal' children have these genetic variants almost as often in relative terms, and even more often in absolute terms (because the group of children without an ADHD classification is obviously far larger). Many children with an ADHD classification do not show the genetic variants associated with ADHD. Precisely as was also the case with the neuroanatomical, neurophysiological and neurochemical attributes discussed earlier, genetic variants are thus neither a necessary nor a sufficient condition for ADHD classification.

Table 4: Base ingredients for good explanation of genetics.

	Less desirable	More desirable
Clearly set out heritability as a fluctuating estimate of the contribution of genetic factors to a trait or set of behaviors in a certain population at a certain time and not as a fixed value belonging to a disorder	A specific value as if ADHD has one value of genetic influence that is the same for each population and each individual (ex. 1)	A range of heritability from different studies (ex. 2, 3)
Highlight differences between the effect sizes of twin studies vs. molecular genetic studies and their possible explanations	Only the large effects of twin studies, in combination with 'associated' genetic variants without reporting low effect sizes (ex. 8)	Effect sizes of both twins and molecular genetic studies, and reflect on discrepancies (ex. 7)
Acknowledge the shared contribution and interaction of genetic and environmental influences	That environment alone contributes to the pre-existing disorder (ex. 4,5)	That genes and environment interact leading to the disruptive behavior (ex. 6,7,9)

CHAPTER 4

PROBLEMS IN THE INTERPRETATION OF ADHD RESEARCH

This chapter addresses some of the challenges in clearly discussing and interpreting research on ADHD, which need to be addressed in good communicating about ADHD. Some of these issues have already been touched upon in earlier chapters, but they are brought together here in overview.

CORRELATION AND CAUSATION

ADHD has been associated with several environmental as well as personal predisposing factors. These factors often interact, making causal pathways difficult to identify and variable from one person to the next. Attention problems, restlessness and impulsivity can themselves be a root cause of problems at school and in later life, but underlying adverse environmental circumstances may also directly or indirectly cause unfavorable behavior development in life. In writing about ADHD, sufficient attention must therefore be paid to the complexity of all these interacting risk factors together. Often, attention problems, hyperactivity and impulsiveness are mentioned as risk factors, but the underlying relations of these behaviors with other factors and possible confounders are often left unmentioned. The following example illustrates this point well.

> **Example 1: less desirable.**
> *'Several longitudinal studies leave no doubt that this disorder puts children at risk for problems in adolescence. This includes poor school performance, reading problems, internalizing problems, conduct disorder, anti-social behavior, drug use or abuse, social problems, accidents, symptoms of eating disorders and teenage pregnancy.'* [40]

Longitudinal studies are studies that look at long-term relationships between several factors. The section cited above suggests that ADHD is at the root of a variety of problems later in life. However, these problems may interact and be the outcome of underlying causes that have little to do with ADHD in various ways that are left unmentioned. For example, reading problems may contribute to internalizing problems and poor school performance; social problems can contribute to the development of substance abuse and antisocial behavior, and so on. Moreover, in many cases, underlying social problems, such as poverty,

discrimination, divorce and neglect are present, which are also associated with and can contribute to development of hyperactive behavior and attentional problems. Most important is this message: ADHD does not explain any of the behavioral problems, but only gives a name to these problems. With regard to smoking during pregnancy—as undesirable and harmful as this is—doubt has been cast on the causal link of smoking during pregnancy and ADHD, because there is an underlying correlation between parents who smoke and lower SES (Social Economic Status) and the latter relates far stronger to having an ADHD classification. In addition, parents who themselves are classified with ADHD are more likely to smoke cigarettes[i]. As such, the association between smoking during pregnancy and ADHD is not as clear cut as it is often represented. The following excerpt reflects this complexity.

> **Example 2: more desirable**
> *'According to some brain researchers, including Dick Swaab, children are much more likely to develop ADHD when the mother smokes during pregnancy. However, this does not necessarily imply a cause-and-effect relationship, but denotes an observed statistical correlation.'* [41]

CIRCULAR REASONING AND TAUTOLOGIES

Several scholars have warned about confusing 'naming & explaining', such as the former chair of the 4th edition of the DSM, the psychiatric diagnostic manual. The ADHD classification is no exception. When we classify attentional problems, hyperactive and impulsive behavior as ADHD, we have not yet explained any of these behaviors: we have merely named (identified) them. The following example shows how the false suggestion is made that ADHD causes the behaviors it merely describes, with the authors leaning towards circular reasoning:

> **Example 3: less desirable**
> *'ADHD affects not only a child's ability to pay attention or sit still at school, it also affects relationships with family and other children.'* [42]

The ADHD definition consists of several criteria pertaining to psychological/social dysfunction in education (or in relation to work). Children would not receive an ADHD classification if we did not deem their behavior in educational settings and in contact with others as problematic. ADHD does not cause these problems, but names them; after all, the causes and motives for the behavior can vary greatly, as we have discussed throughout in relation to evidence. The following statement is another example of circular reasoning:

[i] This is an example of gene – environment correlation.

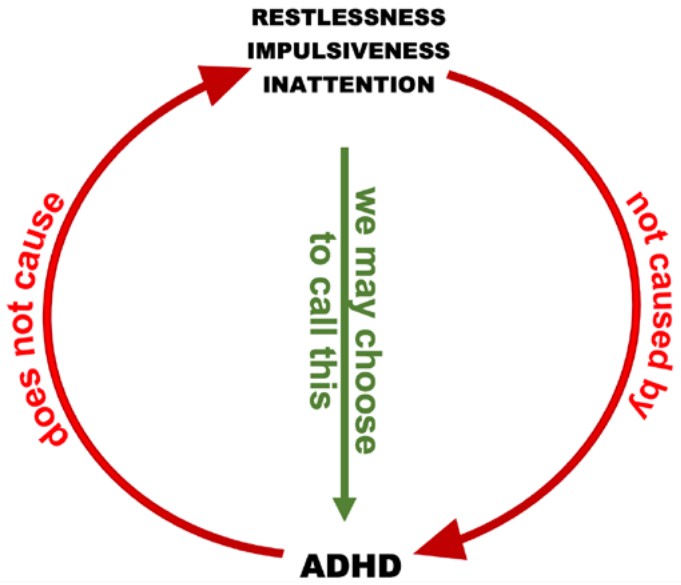
Example 4: less desirable

'ADHD is an explanation of behaviors, not an excuse for them.' [43]

Again, ADHD does not explain behaviors, it merely names them. To prevent naming and explaining from being confused and becoming apt to circular reasoning, it can be helpful to name existing problems as clearly as possible and to clarify how the causal paths can run in different ways, such as in the following example:

Example 5: more desirable

'Concentration problems, impulsivity and hyperactivity can have different and mutually interfering causes and these behaviors can by themselves also contribute to problems in education or in a work situation. However, using the term ADHD to indicate the presence of these problems does not explain them.' [44]



Based on Pameijer, Kramer & Draaisma (2024), Handelingsgerichte Diagnostiek in de Jeugdzorg en JGGZ. Leuven: Accolearn.

THE LIFE COURSE OF ADHD

Research findings on the life course of ADHD vary. According to some estimates, the majority of those with an ADHD classification no longer meet the criteria later in life, while according to other studies, it is a minority. It is not for this guideline to make a final judgment on which research outcome is the most valid; the point sooner is that where scientific agreement is lacking, this should be clearly indicated in writing about ADHD. The differences found in studies also depend on definitions used. Is it about being not meeting any of the behavioral criteria for ADHD? Or is it about a change in behavior such that one can no longer speak

of ADHD according to the DSM definition? In both cases, however, it is important to avoid determinism and generalization. To suggest that children with an ADHD classification always grow out of the problems is contrary to research outcomes, but to suggest that ADHD is lifelong is equally incorrect, potentially stigmatizing and burdensome for children. In addition, research also shows that lower expectations of the environment can actually result in lower performance; this is called a self-fulfilling prophecy, or a self-affirming prediction. The following examples are obviously less desirable:

Example 6: less desirable
'A person does not 'grow out of' ADHD.' [45]

Example 7: less desirable
'ADHD is a lifelong brain disorder.' [46]

It is more desirable to make it clear that there are individual differences regarding the life course of behavior problems, as in the following example.

Example 8: more desirable
'Attention problems are more persistent than hyperactivity and impulsivity. In children with more severe problems these problems tend to be more persevering than in children with less severe problems, but it is not the case that children with more severe problems by definition do not grow over them and children with milder problems do.' [47]

Table 5: Base ingredients for good writing about research findings

	Less desirable	More desirable
Distinguish between correlation and causality	Emphasizing ADHD behaviors as a risk factor without considering underlying connections (ex. 1)	Naming underlying connections and/or warn against correlation-causality confusion (ex. 2)
Do not confuse naming and explaining	ADHD, a term for social problems and issues in e.g. educational situations as an explanation for those problems (ex. 3,4)	Warn against the confusing of naming and explaining (ex. 5)
Be explicit about lack of consensus, different findings and individual variation	Suggesting that ADHD symptoms are always permanent (ex. 6, 7)	Making it clear that the course may vary, depending on the severity of symptoms and many other variables (ex. 8)

CHAPTER 5

DECISIONS IN THE WORDING AND SELECTION OF TOPICS

These guidelines have so far discussed the interpretation of research findings and ways to write about these as clearly as possible. By doing so, stigmatization and a suction effect of classifications can possibly be prevented; for example, by avoiding telling children they are medically 'ill', merely on the basis of their behavior. Decisions that are made in the course of writing information about ADHD are also important: which topics do I cover, and which tricky discussions are best avoided? Which words should I use in the information, and what terminology is best avoided? A biased selection of topics or an unbalanced attention to evidence can create an incorrect or one-sided picture of ADHD, and decisions in wording as well as uncritically following terms and expressions that researchers or clinicians may use, can further contribute to one-sidedness.

WORDING

Wording is not so much about choosing the wrong words, but about being aware that lexical choices, cutting corners for the sake of clarity, and overly strong assertions and claims can contribute to stigmatization and to a one-sided, often unduly medicalizing and excessively reductive or even categorical view of ADHD behavior. The following recommendations are therefore proposed to authors who write about ADHD and who care about factual accuracy, justified nuance and a good balance of perspectives in their writing. The goal, we believe, should be writing that:

- well reflects the greatly diverse findings found across the scientific literature (across science and social science disciplines),
- provides a nuanced grasp of the rich array of experiences of those with an ADHD classification and the very versatile combinations of factors, different for each person, that give rise to it, and
- explicitly recognizes that ADHD names, but does not explain, the wealth of behaviors and experience it classifies.

Neurodevelopmental?

The DSM 5 has categorized ADHD as a neurodevelopmental disorder. Although the authors of the DSM themselves do not state that neurodevelopment refers to slower or problematic brain development, we fear that 'neurodevelopmental' might easily be understood as such (for example in being often used in conjunction with the term disorder, see below)

and we advise not to use it to avoid confusion. For example, Wikipedia wrongly explains neurodevelopmental as: 'disorders that affect the development of the nervous system'[6]. Behavior classifications have helped give structural coherence to the wealth of behavioral research that is being done across various research disciplines, and this was exactly what the 'valuable heuristic constructs' of the DSM intended to do[48]. However, the finding that ADHD behavior correlates with brain development at group level is merely one of many associations that may be cited, and as we have discussed extensively in these guidelines:

- The effect sizes that are found are small: the neurobiology of an individual child with an ADHD classification does not necessarily differ from a child without an ADHD classification as there is much overlap between those with and without an ADHD classification.
- Differences like slower brain development are not necessarily permanent and in general differences do not necessarily imply disorder.
- ADHD also correlates with many environmental factors and correlation does not imply causality.

Disease/Illness/disorder?

There are no biological, physiological, or medical tests for ADHD. It is not visible in the brains or genes of individuals and there are no other physical characteristics which are directly linked to ADHD, with the exception of children with rare genetic disorders, in whom ADHD behavior is part of multiple developmental problems. The classification is solely based on broadly defined and pragmatically considered behavioral criteria, behaviors that many children and adults display to a greater or lesser extent. These criteria depend on social norms: as is also explicitly recognized in the DSM-IV, it is often very hard to distinguish disorders from other disorders, or from behaviors that are not disorderly at all. It is therefore considered unnecessarily stigmatizing and undesirable to speak of a disease or illness. Some experts are advocating to refrain from 'disorder' as well and speak only of 'attention deficit and hyperactivity'. The authors of these guidelines take no definite stance in relation to the use of 'disorder', although we do advocate against the use of 'disease' and 'illness' [49].

Symptom versus Criterion

The behavioral characteristics of the disorders defined in the DSM are often referred to as 'symptoms', although the DSM also uses the term 'criteria'. The problem with 'symptom' is its common meaning; according to the Merriam Webster dictionary[50]: 'subjective evidence of disease or physical disturbance'. In the case of ADHD however, although classification is certainly subjective the behavioral criteria do not provide evidence for a disease or physical disturbance. The behaviors themselves, if they occur in combination and to a severe degree, are the problem. There can certainly be underlying causes, but ADHD is not the cause that is recognized, but the name for the combination of problems. Unlike, for example, fever, blood in the stool, skin rash or weight loss, the criteria for ADHD cannot be determined objectively. Compared with 'symptom', a word like 'criterion' does more justice to the subjective aspect and the decision-making process that is necessary to speak of ADHD.

Diagnosis

The DSM is a system of classifications that can promote good communication between care providers and facilitate research. ADHD can therefore best be described as a DSM classification—we have ourselves tried to consistently refer to those with or without an ADHD classification in this text. However, ADHD is also regularly referred to as a 'diagnosis'. According to the Oxford Learner's Dictionary[51], a diagnosis is 'the act of discovering or identifying the exact cause of an illness or a problem'. However, because no cause is identified with ADHD, and the word illness is also inappropriate, we suggest to describe ADHD as a (behavioral) classification rather than as diagnosis. An additional reason is that in mental health care, diagnosis often refers to a more extensive description of the problem analysis that does look—but then via individual-centred consultation and often extensive investigation—at possible causes of or motives for behavior. By clearly separating the classification from the search for causes, we hope to prevent confusion in the definition and understanding of ADHD, and in particular we hope to prevent ADHD from being perceived as the cause of the problems it names.

Patient versus client

The term 'patient' is sometimes used when it comes to children or adults with an ADHD classification. According to the Merriam Webster dictionary[52] the term 'patient' is used in connection with someone submitting to medical care and treatment. As these guidelines have stressed throughout, biological attributes are by no means necessary or sufficient and are anyway not part of an ADHD classification. The Merriam Webster definition also defines patient as 'one that is acted upon'. These guidelines instead follow wider guidelines about ADHD care that point to safeguarding the autonomy and self-esteem of people with an ADHD classification. For this reason, authors sometimes prefer the word 'client' when discussing those with an ADHD classification in care contexts. The term client however does have some connotations associated with care as a product and people as consumers, and these connotations may in particular contexts give the term client a politically charged meaning that is perhaps also less welcome. We recommend avoiding healthcare terms such as 'patient' and 'client', and even 'person with ADHD', and sooner refer to persons who experience certain clearly identified problems. We advise to be as descriptive as possible in the latter identification of problems, for example by referring to someone who has feelings of restlessness or has trouble focusing while performing certain activities.

CHOICE OF TOPIC

A particularly single-sided selection of topics can also greatly contribute to confusion and a one-sided view of ADHD. Writing only or mainly about biological research or mainly about environmental influences and society can create an information imbalance or bias. We discuss a number of important topics that do not always receive the attention they deserve in information about ADHD.

Societal influences

In Chapter 3 we discussed some examples in which the influence of the direct environment, such as the family, was strongly downplayed, while the influence of, for example, genetics, was emphasized. Information about risk and protective factors in the social environment are regularly lacking texts about ADHD. Nevertheless, in 2014 and 2017, the Dutch Health Council[53] wrote about the importance of society and the environment in relation to ADHD. In 2014, the Health Council described a number of societal factors that may be related to ADHD, such as:

- changing family situations due to changes in family size, shape and forms of cohabitation
- changing forms of childcare due to changes in parents' work patterns and financing of childcare
- changing parenting styles due to changes in pedagogical insights
- changes in media due to the rise of the internet and mobile media
- changes in education due to changes in teacher education and emphasis on specific skills in children.

The report states that it is difficult to establish this societal influence empirically, but some effect mechanisms are nevertheless quite convincing. As an example, the report shows the influence of diagnosis-treatment combinations (DBCs), in which insurance companies only reimburse mental health care in the case of a DSM classification. This introduced an incentive to classify behavior according to the DSM because doing so was a precondition for subsequent financial support and care. Currently in the Netherlands, however, it is increasingly accepted to seek and provide mental healthcare without a DSM classification.

Birth month studies

Research indicates that there are strong systemic incentives in healthcare and education that have contributed to the increase in ADHD classifications. For example, birth month studies have shown that the behaviors of early learners are more likely than those of late learners to be classified as ADHD. These associations have been found in various countries. The effect sizes were considerable, with youngest in class being between 20 and 80% more likely to receive an ADHD classification, with youngest in class being 20 and 80% more likely to receive an ADHD classification, and outliers of over 150% increase and 2 studies in Denmark finding no effect[54]. It should be noted that other influences than early/late school attendance could in theory also contribute to this association.

Given the considerable effect size and the fact that it is very easy for care providers to check whether relatively young age plays a role, the inclusion of information about these birth month studies in information is considered highly desirable.

Should ADHD still be used in practice?

The taskforce acknowledges that there are individuals who are more active, more impulsive and who have more difficulties than others when focusing on tasks, in particular if they do not find these interesting. These behaviors can primarily be related to circumstances or societal demands but can also be mainly related to a person's disposition. We also agree that some of these people may benefit from professional psycho-social interventions and/ or (preferably temporary) treatment with psycho-stimulants. Additionally, we agree that research based on the ADHD construct has provided useful insights into the possible benefits of such interventions and origins of e.g. differences in disposition between children. However, the authors of these guidelines hold differing views on whether and when an ADHD classification should be used in practice.

On the one hand there are those members of the taskforce who are more inclined to use an ADHD classification as a starting point. They do so mainly because they believe this does most justice to, and takes most advantage of, the research that has been done and practice guidelines that have thus far been developed. On the other hand, some members of the taskforce believe that an ADHD classification should be the last resort. They aim to avoid individualized psychological/ psychiatric 'treatment' for issues that are often rooted in, or related to, the social context of the child such as overburdened parents, teachers in overcrowded classrooms or society's difficulty in dealing with temperamental children.

However, regardless of our position on this continuum, all of us believe that widespread misinformation about ADHD should be kept well away from children in particular, and preferably be absent altogether. Additionally, all of us believe that for the ADHD construct to remain useful, dedicated collective effort is needed to improve public discourse on ADHD. We hope that these guidelines are a starting point for doing so.

Table 6: choices when making information: subject and choice of words

	Les desirable	More desirable
Wording	Excessive use of medical jargon and overly healthcare oriented terms such as patient	Avoid medical jargon, use normalizing language as much as possible, and credit all involved with agency
Balance in topic selection	Omitting the influence of norms and society	Discuss nurture attributes in as much topical detail as nature (predisposition) attributes

FURTHER READING

For those who are interested in the discourse on ADHD this is a brief selection of articles that are related to this.

Batstra, L., Foget, L., van Haeringen, C., Te Meerman, S., & Thoutenhoofd, E. D. (2020). What children and young people learn about ADHD from youth information books: A text analysis of nine books on ADHD available in Dutch. Scandinavian journal of child and adolescent psychiatry and psychology.

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APPENDIX A: PARTICIPANTS AND BACKGROUND

These guidelines were created by the Taskforce on psychoeducational literature, coordinated by Sanne te Meerman (sociologist, Druk & Dwars). The taskforce is part of the Academic Workshop on ADHD and unruly behavior and held its first meeting in July 2018. Since then, work on these guidelines has been done through phone conferences, (online) meetings and email. With several discussion papers, participants have provided substantive feedback on desirable and less desirable examples related to education. Additional professionals have contributed in later stages of the development of these guidelines. The participants are (in alphabetical order):

Laura Batstra (Druk & Dwars, Professor Child & Family Welfare, University of Groningen)
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David Con (Psychiatrist, private practice).
Tycho Dekkers (Senior Researcher/GZ-psychologist, Accare/Levvel)
Jeannette Doornenbal† (lector Youth, Education & Society, Hanze University of Applied Sciences)
Justin E. Freedman (Assistant Professor, Rowan University)
Annabeth Groenman (Senior Researcher, Accare)
Maria Groen-Blokhuis (Psychiatrist).
Pieter Hoekstra (Professor Child & Adolescent psychiatry, University Medical Center Groningen).
Maya Hofhuis-van den Brink (Pedagogue, KOOS Utrecht)
Rudi Hofstede (Youth policy, Heerenveen)
Elin Hondebrink (Druk & Dwars, Lay-out and website, University of Groningen).
Branko van Hulst (Child & Adolescent Psychiatrist, LUMC-Curium) Colin Janssen (Clinical Psychologist, Team manager, Dimence).
Geja Jol-Rikkers (Youth Health Care Physician, KNMG)
Richard Jonkers (Parent, experience expert)
Ewout Kattouw (Chairman of Foundation Pill, Experience expert psychiatry & psychotropic drugs)
Nanda Lambregts-Rommelse (Professor of Neuroscience, Radboud University)
Anke van der Landen (Program Manager Youth, VNG)
Birgit Levelink (Paediatrician, Maastricht UMC+)
Sanne te Meerman (Druk & Dwars, Senior Researcher, University of Groningen)
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Ankie Schevers (Teacher Primary education, Heeswijk-Dinther)
Liesbeth Singor (Balans association)
Monique Schweitz (Manager Youth affairs, Zaanstad)

Ernst Thoutenhoofd (Senior Lecturer, University of Göteborg)
Betty Veenman (coordinator Academic Workshop ADHD & Unruly Behavior, GZ psychologist Accare)
Monique Verburg (Child & Adolescent Psychiatrist, Licht-r)
Karin Verheijen (director Primary Education, Onze Toekomst)
Bert Wienen (Druk & Dwars, Associate lector Youth, Windesheim, University of Applied Sciences)
Patrick de Zeeuw (Clinical Psychologist, Altrecht)

APPENDIX B: ADHD AND THE DSM

What is ADHD?

These guidelines are based on the definition of ADHD as defined in the DSM-5[55] and (the Dutch version of these guidelines) the Dutch ADHD care standard. In this standard, ADHD is referred to as an extreme on the continuum from concentrated, calm and controlled behavior to unconcentrated and/or busy and impulsive behavior.

Definition and criteria

In DSM 5-TR, in order to speak of ADHD, a person must meet 6 out of 9 behavioral criteria for: 1. **Inattention** and 2. **Hyperactivity/impulsivity**.

The criteria for **inattention** include behavioral criteria like:

- Often fails to give close attention to details or makes careless mistakes in e.g. schoolwork.
- Often has difficulty sustaining attention in tasks or play activities.
- Often does not seem to listen when spoken to directly.

The criteria for **hyperactivity/impulsivity** include behavioral criteria like a.

- Often fidgets with or taps hands or feet or squirms in seat.
- Often leaves seat in situations when remaining seated is expected
- Often runs about or climbs in situations where it is inappropriate

Furthermore, there are several criteria that must be met in addition to these.

- The behavioral criteria must be present for at least 6 months.
- Several inattentive or hyperactive-impulsive criteria are met in two or more settings like home or school.
- There is clear evidence that the behaviors interfere with, or reduce the quality of, social, academic, or occupational functioning.

The DSM also makes a distinction between 3 types of 'presentations' of ADHD:
The **Combined presentation**: if 6 out of 9 behavioral criteria are met for inattention as well as hyperactivity/impulsivity.
The **Predominantly inattentive presentation**: if 6 out of 9 behavioral criteria are met for inattention but not for hyperactivity/impulsivity
The Predominantly hyperactive/impulsive presentation: if 6 out of 9 behavioral criteria are met for hyperactivity/impulsivity but not for inattention.
The DSM also specifies severity as follows:
Mild: Few, if any, criteria in excess of those required to make the diagnosis are present.
Moderate: criteria are met or functional impairment between 'mild' and 'severe' is present.
Severe: Many criteria in excess of those required to make the diagnosis are present.

Please note that this overview gives only an impression of the DSM section on ADHD. Always refer to the DSM-5 for exact specifications.

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