‘Braining’ psychiatry: an investigation into how complexity is managed in the practice of neuropsychiatric research

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Abstract Neuropsychiatry searches to understand mental disorders in terms of underlying brain activity by using brain imaging technologies. The field promises to offer a more objective foundation for diagnostic processes and to help developing forms of treatment that target the symptoms of a specific mental disorder. However, brain imaging technologies also reveal the brain as a complex network, suggesting that mental disorders cannot be easily linked to specific brain areas. In this paper, we analyze a case study conducted at a neuropsychiatry laboratory to explore how the complexity of the human brain is managed in light of the project of explaining mental disorders in terms of their neurological substrates. We use a combination of ethnomethodology and conversation analysis to show how previously assigned diagnostic labels are constitutive of interpretations of experimental data and, therefore, remain unchallenged. Furthermore, we show how diagnostic labels become materialized in experimental design, in that the linking of symptoms of mental disorders to specific brain areas is treated as indicative of successfully designed experimental stimuli. In conclusion, we argue that while researchers acknowledge the complexity of the brain on a generic level, they do not grant this complexity to the brains of individuals diagnosed with a mental disorder.

Keywords Neuropsychiatry · Complexity · Technological mediation theory · Ethnomethodology · Conversation analysis · Diagnostic categories

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Introduction

The widespread introduction of functional Magnetic Resonance Imaging (fMRI) into psychology in the 1990s initiated a great optimism in the capability to unravel the mysteries of the human mind through the visualization of brain functioning. Also, in psychiatry, brain imaging technologies were initially met with great optimism, in that they finally allowed researchers to establish with rigorous exactness the cause(s) of patient's symptoms by tracing mental disorders back to disorders of brain functioning. In the 4th version of the Diagnostic Manual of Mental Disorders (DSM-IV), for example, it was suggested that the transformation from psychiatry into neuropsychiatry would grant psychiatry its desired 'objectivity' by making obsolete the distinction between 'mental' and 'physical' disorders (APA 1994, p. xxi).

Initially, the use of brain imaging technologies in neuropsychiatry was guided by the assumption that specific cognitive functions could be mapped onto specific brain areas. From this perspective, the cause of a mental disorder could be found in a brain area that was not functioning 'normally' (Vidal 2009). Accordingly, the brain started to appear as a potential candidate for simplifying the process of psychiatric diagnosis by explaining mental disorders in terms of the malfunctioning of a specific brain area.

However, a more recent consensus in the neurosciences leans towards the idea that human cognitive functions are realized within large networks of neurons spread across the brain, which challenges the idea that these originate in a specific part of the brain (see, for example, the *Human Connectome Project*). Accordingly, many neuroscientists believe the relationship between brain activity and specific cognitive functions to be more complex than previously thought, as the workings of cognitive functions can no longer be explained with reference to a particular brain area. This also has consequences for neuropsychiatry: mental disorders can no longer be clearly localized because, based on the recent consensus, as they cannot be linked to a specific 'malfunctioning' area of the brain.

Abend (2017) has recently argued that a crucial question for neuroscientific research is how to develop demarcation criteria when investigating a specific phenomenon. Examples that he gives about how difficult it is to demarcate a specific cognitive function include the way in which neuroscience of love distinguishes love from non-love and how the neuroscience of morality can distinguish morality from non-morality (416). Research in neuropsychiatry faces a similar issue: a core business is attempting to ensure that the patterns of brain activity being measured and observed, correlate with a mental disorder rather than with something within the range of 'normality.' In addition, neuropsychiatrists are confronted with the problem of demarcating one mental disorder (e.g., Attention Deficit Hyperactivity Disorder [ADHD]) from another one (e.g., Autism Spectrum Disorder [ASD]).

Neuroscientific optimists are convinced that brain activity itself will offer relevant demarcation criteria and argue that 'abnormal' brain functioning can be clearly observed. On the basis of such observations, so it is posited, the distinction

between different mental disorders, as well as the one between ‘healthy’ individuals and individuals suffering from a mental disorder, becomes intelligible (e.g., Insel and Cuthbert 2015). As we show in this paper, the complexity of the human brain revealed through brain imaging technologies is treated as making it increasingly difficult to localize mental disorders in the human body (cf. Pickersgill 2009). However, neuropsychiatrists do so not by challenging the nature or desirability of this ‘localization project’ altogether. Rather, they treat the complexity of the brain as something that makes localization more difficult: instead of tying mental disorders to one specific location, they localize their cause within complex networks responsible for specific cognitive functions by discovering patterns of ‘aberrant’ organization within these networks (e.g., Buckner et al. 2013; Menon 2011; Sporns 2014).

Of course, the conception of the brain as a complex network is not new. Throughout history, complexity has often been singled out as one of the central features of the human brain. For example, the famous Spanish histologist Santiago Ramón y Cajal developed an understanding of the brain as a complex organ on the basis of microscopic observations of the neural structure of brain cells at the end of the nineteenth century. However, as the French epistemologist George Canguilhem has argued, one should not be tricked into “thinking that persistent use of a particular term indicates an invariant underlying concept” (Canguilhem 1994, p. 32 op. cit. Rees 2016, p. 92). A variety of different forms of complexity can be attributed to the human brain, urging us to ask how exactly the use of brain imaging technologies in neuropsychiatry makes the complexity of the human brain present.

There seems to be both a cultural and clinical desire to understand the brain in terms of complexity. The idea that unraveling this complexity will help mitigating the sufferings of people with mental disorders (e.g., Borck 2012) is reflected in current discussions about how classification systems such as the DSM might impede progress in understanding mental disorders. A typical concern here is that the classification systems presented in diagnostic manuals insufficiently align with neurobiological evidence that suggests that there is a continuum between different mental disorders, as well as between individuals suffering from a mental disorder and ‘healthy’ individuals (e.g., Cuthbert and Insel 2013). Unraveling the complexity of the brain is offered as a solution to the inadequacies of diagnostic manuals by revealing the neurobiological mechanisms underlying mental disorders.

Recent insights from philosophy of technology can shed a new light on the role of brain imaging technologies in this discussion. In the ‘postphenomenological’ approach, which has developed around the work of Don Ihde, the way technologies shape how scientific objects become present is conceptualized as the *technological mediation* of scientific knowledge (e.g., Ihde 2009; Rosenberger and Verbeek 2015; Verbeek 2005). The hypothesis that can be derived from this idea is that brain imaging technologies mediate how the human brain is made present *as* complex. Following this line of reasoning, this paper draws on ethnomethodology and conversation analysis to explore *how* brain imaging technologies mediate the way(s) in which neuropsychiatrists both conceive of the complexity of the human brain

and manage it practically. We do so through a detailed analysis of the interactions between a group of neuropsychiatrists who interpret data obtained by psychological experiments.¹

The paper is structured as follows: First, we introduce the idea that technologies mediate scientific knowledge, thereby shaping how the human brain is disclosed *as* complex (section "[The complexity of the brain as technologically mediated](#)"). Second, we introduce the theoretical framework used for analyzing our data (section "[Data and method](#)"). Third, we briefly discuss how the issue of complexity is framed in the neuropsychiatric laboratory under study (section "[Neuropsychiatry: complexity and simplicity](#)"). Then, we show how diagnostic labels (e.g., ADHD) are actively oriented to when interpreting experimental data (section "[The role of diagnostic labels in the interpretation of experimental data](#)"), and how these labels become materialized in experimental design by developing experimental stimuli that are tailor made to test previously established diagnostic labels (section "[‘Braining’ psychiatric experiments](#)"). In conclusion, we discuss the wider implications of our findings for how the complexity of the brain is made present in neuropsychiatric practice (section "[Discussion and conclusion](#)").

The complexity of the brain as technologically mediated

Neuropsychiatric research searches to understand mental disorders in terms of underlying brain activity (e.g., Greicius 2008; Poldrack et al. 2012). Investigations into the underlying neurological substrate(s) of mental disorders are crucially dependent on the possibility to observe brain activity in vivo. Without the presence of brain imaging technologies allowing for the visualization of functional brain activity, such as Electroencephalography (EEG) or functional Magnetic Resonance Imaging (fMRI), this cannot be done.

In postphenomenology, technologies in scientific practice are understood as mediating intentional relations between scientists and the world (e.g., Ihde 1991, 2009; Verbeek 2005). Technologies help to shape the objects that scientists investigate, instead of being mere instruments that allow the observation of objects ‘out there’; they mediate how scientists can perceive what they study, and in doing so they also shape the interpretative frameworks to make sense of these perceptions. That is, in the developing relationship between scientists and the technologies they use, simultaneously a specific relationship between scientist and reality is being constituted. Within such a relation, specific interpretative categories and options for action and manipulation come into being that allow scientists to understand the object under study in a variety of ways (Ihde 1991, p. 137). Technological mediations are always accompanied by human appropriations of those, such that they are integrated into particular schemes of interpretation and action (Verbeek 2016). The way in which technologies mediate how scientists understand their objects of study does not only

¹ We use the term ‘neuropsychiatrist’ to refer to researchers using neuroscientific methods to investigate mental disorders, not to clinical psychiatrists.

depend on the characteristics of these technologies but also on the ways in which scientists make sense of the technologies and give them a place in their relations to the object of study.

The perspective of technological mediation allows us to study how the objects that neuroscientists investigate (e.g., cognitive functions, mental disorders) are shaped by how brain imaging technologies help to disclose them (De Boer et al. 2020). Accordingly, also how neuropsychiatrists relate to (symptoms of) mental disorders is contingent on how they relate to the technologies they use to investigate those disorders. In this paper, we use this perspective to study how these technologies mediate the way(s) in which neuropsychiatrists understand mental disorders by making brain activity perceptually available *in vivo*. Specifically, we focus on how this technological mediation constitutes the human brain *as* complex, and on how researchers appropriate this complexity.

As was indicated before, the promise of integrating brain imaging technologies into neuropsychiatry is that these allow to measure brain activity, which will lead to more fine-grained diagnostics and treatment in the long run. In the laboratory in which the data of this study was obtained, brain imaging technologies such as fMRI are primarily used to investigate how mental disorders such as ASD, Obsessive Compulsive Disorder (OCD) or ADHD correlate with specific patterns of brain activity.

Diagnosing someone with ADHD, OCD, or ASD is not to point to a singular entity, but to categorize a multiplicity of different symptoms under a single header. For example, the DSM-V lists the following symptoms as diagnostic criteria for ASD: (i) Persistent deficits in social communication and social interaction, (ii) restricted, repetitive patterns of behavior, interest, or activities, (iii) symptoms must be present in early developmental period, (iv) symptoms cause clinically significant impairment, (v) disturbances are not better explained by intellectual disability (APA 2013, pp. 49–50). As will become clear throughout this paper, the neuropsychiatrists under study tend to follow this symptom-based understanding of mental disorders. They are specifically interested in developing causal explanations of symptoms of mental disorders, rather than targeting mental disorders as a whole.

The promise of neuropsychiatry is (i) to offer an objective foundation grounding diagnostic processes, and (ii) to prescribe forms of clinical (pharmacological) treatment that specifically target the symptoms of a mental disorder. Accordingly, neurological activity is theoretically posited as an explanatory cause of symptoms of specific mental disorders, such that the observation of brain activity is treated as potentially *simplifying* the process of psychiatric diagnosis and treatment by revealing clear causal pathways that are constitutive of mental disorders (Insel and Cuthbert 2015).

Such expectations show that the technologically mediated way in which the complexity of the brain and the way it helps realizing specific forms of cognitive (dis) functioning does not occur in a vacuum, but is relative to existing knowledge of mental disorders, and neurobiological knowledge of brain functioning. It is against this background that the mediated complexity of the brain is integrated into the existing goals and aims of neuropsychiatrists. Accordingly, when brain imaging technologies present the brain as a complex organ, neuropsychiatrists are not simply

confronted with a *fait accompli*. Rather, they need to develop new interpretative strategies and plans of action against the background from which they understand mental disorders. In the appropriation of the complexity of the brain, new ways open up to hypothesize how (symptoms of) mental disorders are realized and to develop experimental circumstances in which such hypotheses can be tested.

However, as Pickersgill (2011) has argued, there is no clear consensus in mental health research and practice about what mental disorders are and what is the best way to investigate them (cf. Rüppel and Voigt 2019). However, there is a clear consensus on another issue (hence the prefix “neuro-”): research into mental disorders *should* involve research into their neurological substrates, and experiments *should* be developed to make the link between cognitive functions and brain functions empirically testable (Cohn 2008; Fitzgerald 2014), even in the absence of direct clinical merit (Brosnan and Michael 2014). Building on these earlier studies, we intend to show in this paper that both in the ways in which neuropsychiatrists understand mental disorders and in their experimental designs, there seems to be a strong demand to ‘brain’ neuropsychiatric experiments. This practice of ‘braining’ requires the brain’s perceived complexity to be managed in light of the aim of neuropsychiatry, which is to reveal causal pathways that are constitutive of mental disorders. Thus, even though that the complexity of the brain as mediated through brain imaging technologies must be accounted for, it must be done in such a way that experimental set-ups allow for the development of causal explanations.

The management of complexity is of course not unique to the neurosciences. In the context of molecular biology, Rheinberger (1997) has argued that in the history of biology, crucial experiments typically developed parameters for simplification in order to be explanatory successful, while at the same time retaining the complexity of the research object (e.g., genes). Similarly, based on a critical reading of how pioneering synthetic biologists describe their work, Dan-Cohen (2016) points to the fact that many synthetic biologists were originally trained as computer scientists and electrical engineers, and largely ignored the biological complexity of life when developing models for designing and constructing novel organisms. These researchers proclaimed that a certain degree of ignorance of complexity was necessary for synthetic biology to develop as a field. Recently, in the context of neurocriminology, it has been shown that researchers orient to the neurological origins of allegedly criminal behavior at the expense of focusing on the complex social factors influencing it (Fallin et al. 2019). Based on an analysis of historical case studies or of written documents (scientific articles or autobiographical accounts of scientific work), these studies show that (neuro-)scientists need to manage complexity to make it a workable element within their research.

Our study aims to add to the body of literature that focuses on how complexity is managed in two ways. On the one hand, drawing from the perspective of technological mediation, we intend to augment existing social studies of neuroscience by focusing on how brain imaging technologies shape the objects that neuropsychiatrists study to become present *as* complex. This requires, on the other hand, to provide a detailed analysis of how scientists manage complexity in *practice*. To this purpose, we use a combination of ethnomethodology (EM) and conversation analysis (CA). We thereby study how technological mediations are *appropriated* and

made fit into the interpretative frameworks that neuropsychiatrists use to understand mental disorders. Through these two additions we want to turn attention not only to the fact *that* managing complexity is an integral part of science, but also to *how* scientists do so in their research practices and the implications thereof.

Data and method

In this paper, we draw on a case study conducted at a Dutch neuropsychiatry laboratory to explore how the complexity of the human brain is managed in light of the specific project of explaining mental disorders in terms of their neurological substrates. We use a combination of EM and CA for analyzing our data. EM (Garfinkel 1967) and CA (Sacks 1992; Te Molder and Potter 2005) define practices as inherently normative, because the shared reality within a practice is constituted by the fact that people orient towards certain norms and expectations *as* being shared. Instead of conceiving of norms as rules that are external to the practices in which people engage, EM and CA hold that, when describing their actions, people actively *display their orientation* towards some norm or expectation, thereby also making these norms and expectations available for the analyst (Heritage 1984, pp. 115–120). Through a detailed analysis of conversations between researchers, these approaches make it possible to reveal the norms which these participants draw on when investigating mental disorders. This, in turn, allows us to investigate *how* the complexity of the human brain—as mediated by brain imaging technologies—is appropriated in neuropsychiatric practice.

Michael Lynch (1985) used CA and EM to study how neurobiologists maintained a shared reality by orienting to specific projects. He argues that in their everyday practice, scientists explicitly or implicitly orient to projects, making that certain features of the obtained data are treated as reliable and/or relevant, while others do not (Lynch 1985, pp. 85–87). In his work, Lynch investigated to which norms neurobiologists orient when classifying parts of their experimental data as artifacts (i.e., as distortions of data caused by the experimental set-up, instead of ‘truthful’ observations of nature), and therefore as being irrelevant to the project they are engaged in. The goal of establishing (causal) relationships between brain activity and specific mental disorders can be considered as an example of a project in Lynch’s sense. Analogously to his work, our analysis focuses on the norms that researchers in neuropsychiatry orient to when making the complexity of the human brain workable in light of their specific project. The excerpts of the recorded conversations discussed in this paper serve to show how brain imaging technologies mediate the complexity of the brain in light of the project that the researchers in our study engage in.

Our data consists of approximately 7 h of recorded conversations. One author of this article was present during these recordings but did not interfere in the conversations in any manner. Two types of conversations were recorded: (1) monthly group meetings in which one of the members of the laboratory presents his or her research, followed by a discussion, and (2) meetings between members of the laboratory with the specific purpose of discussing how to interpret the experimental data obtained by brain imaging technologies. We chose to specifically record these events, because

they are the primary occasions in which the obtained data is explicitly linked to the project of establishing a (causal) relationship between brain activity and specific mental disorders.

The recorded conversations were transcribed using ELAN.² The main part of the data corpus was transcribed to a words-only level, but relevant parts (i.e., parts in which speakers displayed an orientation to the central project) were transcribed in more detail using a simplified Jeffersonian transcription notation (Jefferson 2004; see Online Appendix 1). We have refrained from a more detailed transcription level often used in CA because our intention is not to analyze the structure of conversations as such, but instead to investigate how complexity—as mediated by brain imaging technologies—is managed practically in the interactions between researchers. The conversations were held both in Dutch and English language. The Dutch fragments were translated into English by the authors. In our translation, we have maintained the word order of the participants themselves to remain as close to the real-life conversations as possible. As a result, our translations occasionally contain grammatically incorrect sentences reflecting the word order present in our recordings. We have transcribed overlaps between speakers and pauses in order to be able to analyze how the research project that neuropsychiatrists engage in is interactionally oriented to and made relevant.

Neuropsychiatry: complexity and simplicity

The laboratory in which the conversations were recorded has a strong focus on “restricted, repetitive patterns of behavior, interest, or activities,” which is considered a symptom of either ADHD, OCD, or ACD. Accordingly, brain imaging technologies were primarily used to target aspects of the brain that were thought to be relevant for this specific kind of behavior. However, at the same time, the neuropsychiatrists treat this use of brain imaging technologies as being in conflict with the alleged complexity of the brain. The following fragment of a conversation between Sally (a postdoctoral researcher) and Mike (the vice-director of the laboratory) is indicative of this tension:

² ELAN is developed by the Max Planck Institute for Psycholinguistics, The Language Archive, Nijmegen, The Netherlands (url: <http://tla.mpi.nl/tools/tla-tools/elan/>). For an explanation of ELAN, see Wittenburg et al. (2006).

Excerpt 1^{3,4}

1 **Sally** it has been very helpful for my biological background
 2 of the behavior that [eh we see
 3 **Mike** [it's not so straightforward
 4 [eh=
 5 **Sally** [no::
 6 **Mike** [=and that again and that's what makes doing research
 7 e::h terribly complex
 8 (3 lines omitted)
 9 **Sally** yes and that makes that you cannot say the putamen is
 10 my region of interest and because it is different we
 11 understand how autism works e::h because it is so
 12 extremely interwoven
 13 **Mike** yes yes yes I mean these loops remain [important=
 14 **Sally** [yes
 15 **Mike** [=of course and
 16 it might be possible that one loop gets in one way or
 17 another more attention than another loop that's
 18 possible regardless of whether they are more
 19 integrated into another system, it can still be the
 20 case that one gets suppressed and that has to be (0.5)
 21 otherwise the behavior can never be explained
 22 (3 lines omitted)
 23 **Sally** yes but it gives me now primarily the feeling that
 24 e::h you get the tendency to pull in more and more and
 25 that eventually:: you end with yes the brain is a large
 26 network and everything is [connected=
 27 **Mike** [hmhm
 28 **Sally** [=you need everything
 29 du:::h [that's
 30 **Mike** [yeah
 31 **Sally** [of course true but it's e::h incredibly
 32 difficult to e::h consider regions in isolation
 33 **Mike** t- that's simply the case

In this interaction, the brain is being built as a complex organ that evades simple explanatory models of mental disorders. Sally was asked to write a review on how the neural networks associated with repetitive and stereotyped behavior map onto the anatomical regions of the brain. She says that doing research for this review was a “good way to see the biological background of the observed behavior” (lines 1–2). This is treated by Mike as indicating that the origins of behavior in the brain are not “so straightforward” (line 3), which makes doing research “terribly complex” (lines 6–7). Sally responds affirmatively to Mike, and treats the complexity of the brain as preventing researchers from focusing on specific regions of the brain (the putamen, line 9) and use deviances in specific regions as explanatory for autistic behavior

³ The names of the participants have been altered.

⁴ Excerpts 1, 2, and 3 are parts of a conversation that originally took place in Dutch. The excerpts are translated by the authors. Original fragments can be delivered on request.

(lines 10–11). Instead, she orients to the complexity of the brain as indicative of the fact that autism is “extremely interwoven” (line 12) and resists a single-cause explanation. Accordingly, complexity is oriented to as a norm that forces one to move beyond the mapping of brain functions to specific brain regions.

In response to Sally, Mike suggests that the loops of activity observed in the brain may have an important explanatory function (line 13). ‘Loops’ are patterns of brain activity moving across different areas of the brain, and specific patterns of loops are thought to be indicative of specific cognitive functions (Amso and Scerif 2015). When invoking loops, Mike proposes a candidate concept for explaining autistic behavior that does not treat it as originating from a particular brain area (lines 18–20). Subsequently, he points to the need for such alternative concepts since the behavior observed in autism “can never be explained” otherwise (line 21). Sally demonstrates an insecurity with regard to the explanatory force of such new concepts when stating that such an approach only leads to the “tendency to invoke more,” and eventually leads to a view that “the brain is a large network and everything is connected” (lines 25–26). Her consecutive “you need everything du:::h” (line 28–29) displays the self-evidence of such networked complexity, yet also clearly demonstrates dissatisfaction with regard to the lack of explanatory potential of such a perspective. In this case, the desired simplicity of using the brain as an explanation of symptoms of ASD is oriented to as a norm conflicting with the complexity of the brain.

This fragment makes clear *how* the researchers orient to complexity in their conversational practice. They do so by saying that specific mental disorders and the symptoms of those cannot be easily tied to specific brain areas but also present the brain as large network that makes research into a specific disorder terribly complex. As a members’ category, complexity thus serves to display the difficulties that researchers face when trying to understand (symptoms of) mental disorders in terms of their underlying neurological substrates (cf. Fitzgerald 2014; Maung 2016). On the other hand, they treat complexity as a potential threat to developing causal hypotheses about the nature of mental disorders that needs to be carefully managed to fit the practical purposes of neuropsychiatry. This indicates that also simplicity is a members’ category, which functions as a norm in the sense that causal explanations of mental disorders are treated as desirable. This becomes clear in the end of the conversation where Mike presents complexity as a fact that “is simply the case” (line 33), indicating that even though that the complexity of the brain must be theoretically acknowledged, pragmatic ways of aligning the complexity of the brain with the goal of searching for explanations of mental disorders remain possible (e.g., by using new concepts such as ‘loops’). This suggests that the complexity of the brain is not uncritically accepted but appropriated and integrated into the existing aims of neuropsychiatry.

It is important to note that how the researchers orient to complexity does not make it principally irreconcilable with simplicity. Hence, they do not treat mental disorders as fundamentally evading every possibility of being reduced to brain activity, as would have been the case when the norms of complexity and reductionism would be for example oriented to in a contrastive manner. Rather, the tension between the two norms lies in the difficulties that researchers

reportedly face when interpreting the brain as a complex organ, while at the same time remaining to treat it as bearing the potential that it reveals how mental disorders are caused.

The role of diagnostic labels in the interpretation of experimental data

The complexity of the human brain and its role in the realization of cognitive functioning is not only a matter of theoretical concern as was shown in *Excerpt 1*, but also shapes how visualizations of brain activity are used to obtain relevant knowledge about the causes of mental disorders. In this section, we discuss how this is done by analyzing two excerpts of a conversation in which the data of an experiment that aims to target ‘repetitive and stereotyped behavior’ in individuals diagnosed with either ADHD, OCD, or ASD, is being discussed.

At the laboratory under study, the perceived complexity of the brain mediates what is understood by ‘repetitive and stereotyped behavior’ as a symptom of ADHD, OCD, and ASD. The symptom ‘repetitive and stereotyped behavior’ encompasses a wide range of heterogeneous instances of behavior. For example, in the DSM-V, examples of this symptom in the context of ASD range from “extreme distress at small changes” to “greeting rituals” (APA 2013, p. 50). However, these different types of behavior are treated as homogeneous in the sense that they are different instances of repetitive and/or stereotyped behavior. This type of behavior is treated a symptom of ADHD, OCD, as well as of ASD. Hence, neuropsychiatrists often stipulate that there is, in one way or another, a continuum between different mental disorders in terms of this symptom (e.g., Hyman 2010; Lichtenstein et al. 2010). At the same time, the different diagnostic categories constitute a normative expectation that different patterns of brain activity are underlying the behavior in the different patient groups. Researchers thus have to orient to the complexity of the human brain such that it becomes relevant in terms of the simplicity of existing diagnostic categories. How the complexity of the brain is oriented to in this context, will become clear from our analysis of two excerpts in which visualizations of brain activity are interpreted.

The first of these excerpts is taken from a conversation between Mary (the head of the research group), Sally (the postdoctoral researcher also included in Excerpt 1), and John (a PhD student). The conversation focuses on how to interpret the data that John obtained from a recent experiment on impulsivity. Using a ‘GO-NOGO’ task paradigm, individuals were asked to react as quickly as possible when presented with certain stimuli by pushing on a button (GO), while these individuals should refrain from pushing the button when being confronted with other stimuli (NOGO). In doing so, they aimed to study to what extent presenting deviant stimuli influences the participant’s ability to continue the task as instructed. It was hypothesized that individuals diagnosed with OCD, ASD or ADHD have an increased reaction time or push the button in cases when they were instructed not to do so.

Excerpt 2

1 **Mary** I estimate that this will simply be (.) in terms of
 2 diagnostic differences in brain activation in this go
 3 no go task eh between these patient groups simply a
 4 negative study
 5 **Sally** yes
 6 **Mary** at this point we can no longer deny [this
 7 **John** [no
 8 (9 lines omitted)
 9 **Mary** I would still like to know how these two groups are
 10 divided among the three diagnostic groups ADHD ASD and
 11 OCD and is this something
 12 **John** yes this is the first part of the ROI analysis in which
 13 I e::h actually these three and there are some more
 14 in the other groups that I tested but e::h in the first
 15 contrast nothing came out but I want to do it on
 16 contrast three because on a whole brain level
 17 [something
 18 **Mary** [that something was there
 19 (4.0)
 20 **Mary** that sounds like a good idea
 21 (2.0)
 22 **Mary** but thus far (.) the eventual conclusion is thus
 23 [primarily
 24 **John** [yes haha that one
 25 **Sally** stupidly this would fit in into my impression of the
 26 literature at this point and then it is really a pity
 27 that eh yes it is not the nicest things to write a
 28 negative [study=
 29 **Mary** [no but is not unimportant
 30 **Sally** [=but it is not a real surprise that you do
 31 not find anything in this group with this task a::nd
 32 maybe it makes sort of sense to (.) mention that just
 33 very explicitly

In this excerpt, the researchers demonstrate an expectation that when experimental participants would demonstrate a similar task performance, the underlying patterns of brain activity would be different between different patient groups. The excerpt starts with Mary saying that a conducted experiment does not show differences in brain activation patterns across the different patient groups (lines 1–3), which makes the experiment “a negative study” (line 4). Both Sally (line 5) and John (line 7) display agreement with this assessment. The adjective ‘negative’ indicates that earlier psychiatric diagnostic labels function as norms that the conversational participants orient to when developing experimental hypotheses.

Later, however, in response to Mary, John proposes differences on a whole-brain level as a potential way of differentiating between different patient groups (lines 15–16). Again, this assumption is guided by previous diagnostic labels, because it suggests that even though the analysis focusing on a particular region of interest in the brain might not display differences in brain activation, these differences might be present on the level of the brain as a whole. Mary’s response “that sounds like

a good idea” (line 18) suggests that a further investigation of differences in brain activity between different patient groups might be worthwhile, but after a long pause (line 21) she continues that the brain activity observed does not warrant such a further investigation at this point (lines 22–23), which John affirms (line 24). Even though the complex interactions on a whole-brain level might display differences across patient groups, this is not treated as potentially challenging the earlier conclusion. Hence, the complexity of brain activity on a whole-brain level is surpassed as a reliable source to detect differences between different patient groups.

In the last part of the discussion, Sally orients to a broader consensus in the scientific community when saying that this conclusion “stupidly fits her impression of the literature” (lines 25–26), making the results not “a real surprise” (line 30). However, during Sally’s conversational turn, Mary displays a positive stance towards the conducted study when saying that the negative outcome is “not unimportant” (line 29). Sally demonstrates a similar stance by invoking the particular task design used as an explanation for that no differences in brain activity could be observed, and that it is worthwhile to “mention that very explicitly” (lines 32–33). Hence, on the one hand, the observed brain activity is treated as objectively showing that no differences can be made across patient groups, yet on the other hand this is not treated as a feature of the (individual) participant’s brain activity as such but made relative to the experimental circumstances in which brain activity was measured. The previous diagnosis of a specific mental disorder is thus invoked as a norm allowing to make sense of the experimental data, in that the measured brain activity is not taken to objectively indicate a similarity across mental disorders, but instead prompts a discussion about whether specific experimental circumstances can still ‘pull out’ differences in brain activity. Accordingly, the complexity of the human brain is not taken for granted but made relative to the expectations constituted by the type of mental disorder that is already attributed to the brain under discussion.

An example of this can be noted in Excerpt 3, where Sally and Marry (who were introduced in the previous excerpt) continue to discuss the conceptual possibilities that might help reveal experimentally under what circumstances differences in brain activity can be detected:

Excerpt 3

1 **Sally** e::h what what the data from America would say is that
2 almost the only way to e::h get this type of behavior
3 using this task is to put something that is soo::
4 incredibly [salient=
5 **Mary** [salient yes yes
6 **Sally** =that it immediately sort of disturbs all behavior (.)
7 yes and that that then they can do task slightly worse
8 and then you speak about really slightly
9 (6 lines omitted)
10 **Mary** .hhhhhh yes and and and then the salience does
11 something and you get some movement and and then still
12 there are no enormous differences as far as I can see
13 (7 lines omitted)
14 **Sally** you can e:h create some little movement for example by
15 showing social stimuli but then it might still be
16 rather the [limbic system that is e::h=
17 **Mary** [that something's wrong with that
18 **Sally** [=causing a very litte disbalance e:h in
19 the cognitive control system then that the cognitive
20 control system itself is not properly functioning
21 **Mary** but how does this e::h fit when we look at the
22 behavioral [data=
23 **Sally** [yes
24 **Mary** [=all the data from questionnaires suggests
25 that oh it seems that [actually=
26 **Sally** [.hhhhh yes:::
27 **Mary** the three mental disorders are not really [discrete=
28 **Sally** [no
29 **Mary** [=but that
30 there is a continuum but then we do these sort of
31 studies and take an ADHD-task and we push and pull and
32 we actually=
33 ((all laugh))
34 **Mary** get no movement in the ASD group that was supposed to
35 be in the same continuum and that is just:::

Sally suggests that on the basis of earlier obtained data that it might be helpful to include stimuli that are “incredibly salient” (line 4) in order to show behavioral differences across patient groups in this particular experimental design (lines 2–3). Earlier experiments showed that in terms of task performance, groups diagnosed with a specific (yet up to now unspecified) mental disorder, had a slightly worse task performance, but Sally admits that this difference was really minor (lines 6–8). In response, Mary displays doubt with regard to Sally’s proposal, when stating that integrating more salient stimuli causes a “little movement,” but still does not display “enormous differences” between different mental disorders (lines 11–12). Again, previous diagnoses have a strong normative input: the expectation of the outcome of the experiment is that there are enormous differences that are really clear.

In response, Sally remains orienting to the task design as having the potential to reveal differences in brain activity in people diagnosed with ASD: presenting more

social stimuli might cause some more movement because it points to the possibility that something is wrong in the limbic system of the individuals diagnosed with a mental disorder (line 16). She treats these different reactions to different types of stimuli as potential new ways to understand the underlying causes of deviant behavior. They namely point to the possibility that this limbic system “brings the cognitive control system out of balance,” rather than that something is wrong in the “cognitive control system itself” (lines 18–20). This indicates that different experimental circumstances reflect different hypotheses about the ways in which certain types of behavior are realized in the brain. Instead of orienting to brain activity in order to find the root of deviant behavior, brain activity is treated as a malleable phenomenon reflecting the specificity of certain experimental circumstances.

After Sally has proposed a difference in terms of brain activity, Mary returns to the question of how the experimental data relates to the behavioral data obtained by questionnaires (lines 21–22). Mary treats the questionnaire data as relevant because these suggest that there is a continuum between the three mental disorders that are clinically manifest (lines 29–32). However, as she points out, when conducting their studies and using an “ADHD-task” (line 31), there is no movement in the ASD-group, even after they “push and pull” (line 31) the data. She suggests that this is peculiar because on the basis of questionnaires, ASD and ADHD would be in “the same continuum” (line 35). Again, rather than treating the experimental outcome and the observed brain activity as reflecting ‘facts of nature,’ they are oriented to relative to earlier clinical diagnoses.

Our analysis of these fragments shows that diagnostic labels have a strong normative function in processes of data interpretation. Researchers actively attempt to interpret visualizations of brain activity in terms of existing diagnostic labels, even when this appears to be difficult. Because of this, the visualized brain activity is attributed an objective status to the extent that it can be aligned with earlier diagnoses (Cohn 2012). However, the absence of relevant differences in brain activity in different diagnostic groups is not treated as an ‘objective’ fact, but rather functions as a reason for researchers to develop new experiments that allow for aligning the observed brain activity with previous diagnostic labels. In the next section, we will explore how the relation between these two aspects is managed in the development of new experiments.

‘Braining’ psychiatric experiments

In the previous section, we showed that researchers orient to the complexity of the human brain in terms of previous diagnostics labels, and that only in the light of these labels, it becomes possible to make sense of the observed brain activity. Because of this, the researchers need to develop experiments that are able to tie brain activity to specific symptoms of mental disorders. As we will see in this section, researchers develop a practice of ‘braining’ psychiatric experiments to do so. That is, they develop experimental set-ups in which brain imaging technologies are appropriated in such a way that they can be understood in terms of earlier psychiatric diagnoses. When doing so, researchers treat fMRI as allowing to link symptoms

of mental disorders—in this case, impulsivity—to specific brain regions. Orienting to fMRI in this way assumes that human cognitive functions originate from particular brain areas, and that fMRI allows researchers to objectively localize specific cognitive acts in the brain *in vivo*. This seems to be in conflict with the scientific consensus that the human brain should be understood as a complex network, such that mental disorders *cannot* be tied to particular brain areas.

In the excerpts analyzed in this section, it is discussed how ‘repetitive and stereotyped behavior’ can be experimentally investigated at the laboratory by linking it to the impulsive reactions of individuals when being confronted with something that is of their central interest. During a presentation at the monthly group meeting on the experimental study of repetitive patterns of behavior in children diagnosed with ASD, Karen (a postdoctoral researcher at the lab) explains why this link is made as follows:

Excerpt 4

1 **Karen** so e::h everyone has a hobby or an interest could be
2 sportcars
3 (5 lines omitted)
4 but in any case usually you should be able to function
5 around that interest so it should not really impair
6 you in your daily life you should be able to function
7 at a job without e::hm being busy with sport cars all
8 the time e::h but this is different in autism
9 (4 lines omitted)
10 and that interest e::h may be so intense that it does
11 impair them in their daily lives so:: e::h
12 (5 lines omitted)
13 they are rather all consuming and they do limit social
14 interactions when they interfere with daily activities
15 (3 lines omitted)
16 so: e:h what we wanted to know what is the behavioral
17 and neural pattern of impulse control to preferred
18 interests first in healthy adults that is a pilot study
19 we did (0.5) and then we wanted to know whether
20 children with autism would demonstrate a bias towards
21 their (.) restricted interests compared to typically
22 developing control children

Repetitive and stereotyped behavior is in this experimental context understood in terms of the specific interest of an individual diagnosed with ASD that “may be so intense that it does impair them in their daily lives” (lines 10–11). To a certain extent, the relation between individuals diagnosed with ASD and their interest is treated as being continuous with the hobbies and interests that healthy individuals have (lines 1–2). However, the behavior that individuals diagnosed with ASD display in relation to those interests, is treated as marking a difference because they “are rather all consuming [...] and interfere with daily activities” (lines 13–14). This behavioral difference is oriented to as a norm that constitutes

a break in the continuum between ‘healthy’ individuals and individuals diagnosed with ASD: the latter “demonstrate a bias towards their restricted interests” (line 20–21), which makes them deviate from the “behavioral and neural pattern of impulse control [...] in healthy adults” (lines 16–18). What is treated as the most relevant question in this context, is how this hypothesis can be tested in terms of both the behavioral and neural differences between healthy individuals and individuals diagnosed with ASD. Hence, the difference between ‘healthy individuals’ and ‘individuals with ASD’ is already assumed by orienting to how individuals were classified through earlier diagnostic processes.

Because the way in which scientific experiments are materialized is mediated by fMRI, the difference between healthy individuals and individuals that are diagnosed with ASD must be reflected on a neurological level. Given the complexity of neuronal interactions and their relation to cognitive and emotional functioning, experiments have to be designed in order to specifically target the neurological underpinnings of the ‘impairment’ in the brain that constitutes the deviant behavior in individuals diagnosed with ASD. The complexity of the human brain is thus made relative to specific interests of individuals and the associated impulsivity that is the consequence of those. Only when this situation is realized, the brain can have an explanatory function within this type of neuropsychiatric research. Ironically, this requires researchers to align the perceived ‘objectivity’ of the human brain as revealed through fMRI with the ‘subjective’ interests of individuals in order to become relevant *as* objective knowledge. Accordingly, ‘braining’ psychiatric experiments simultaneously requires them to personalize psychiatric experiments.

As Karen suggests in her presentation, because the impairments that are observable in individuals with ASD are relative to the behavior of ‘healthy people,’ it is first necessary to find a way to study impulsivity in this latter group. Prior to the experiment, individuals were asked to choose from 22 different potential interests (ranging from trains to SpongeBob Squarepants) what were the 3 interests that were most of their liking. Subsequently, these ‘healthy’ individuals performed a ‘GO-NOGO’ task and were asked to refrain from pushing a button when confronted with one of the interests of their choosing. As a control condition, participants were asked to perform the same task, but were respectively confronted with different facial expressions, and different colors instead. Behaviorally speaking, these healthy individuals were equally good in performing this task under the three different conditions. However, the researcher argues that in terms of brain activity, a difference can be detected. In Excerpt 5, it can be seen how the objectivity of fMRI is oriented to when substantiating this claim:

Excerpt 5

1 **Karen** e:~~~~h (.) it was an fmri study so::: e::h
 2 (4 lines omitted)
 3 basically the take away message here is that e:h areas
 4 were activated that you would expect during this type
 5 of task so e:h the visual cortex because they are
 6 looking at things the motor cortex because they're
 7 pressing buttons e:h and then e::h insula because you
 8 are looking at things that are salient to you or at
 9 least eh presu e:h presumably salient to you e::h and
 10 there was some inferial frontal gyrus cognitive
 11 control activation
 12 (11 lines omitted)
 13 which was somewhat makes sense because probably that
 14 what is your hobby is salient to you and e:h the insula
 15 e:h e:h is activated when seeing things that are
 16 salient to you e::h which is why e::h e:h left insula
 17 activity was higher but even though e:h there was a
 18 slight increase in brain activation in e:h healthy
 19 individuals are just able to inhibit themselves
 20 regardless

Interestingly, the fMRI results are not treated as relevant because they display something new and/or exciting, but because they show that the “areas [of the brain] were activated that you would expect during this type of task” (lines 4–5), which is further specified in lines 4–9. Only the potential reason for the “interior frontal gyrus cognitive control activation” (lines 10–11) is explicitly mentioned, indicating the presumed relevance of this brain area for this specific task. Activity in the insula is treated as making sense because it “is activated when seeing things that are salient to you” (line 14), yet not constitutive of deviant behavior because “healthy individuals are just able to inhibit themselves regardless” (lines 19–20). Instead of providing new information of the way behavior is neurologically constituted, fMRI is oriented to in this experiment as to validate whether the stimuli used in the experiment trigger activity in the brain areas they are supposed to trigger. fMRI is thus treated as offering objective certainty in the sense that it allows to validate whether the experimental stimuli can be mapped onto brain regions associated with certain cognitive functions (e.g., looking is linked to the visual cortex, etc.). Thus, rather than being invoked as introducing complexity, fMRI is oriented to as allowing to causally link specific brain areas to specific cognitive functions.

A similar function is ascribed to fMRI when it is discussed how the supposed ‘impairments’ of individuals diagnosed with ASD can be made present experimentally:

Excerpt 6

1 **Karen** e::h these sort of relatively neutral stimuli or
 2 stimuli that are just not of interest to an individual
 3 with autism e:h do not capture that change in impulse
 4 control (.) e:::h what does capture in individuals
 5 with autism's interest is what they like
 6 (3 lines omitted)
 7 and two recent studies have also shown e::h that again
 8 e:h individuals with autism show increased (.)
 9 activity in left insula when viewing e:h images of
 10 their special interests .hhh e:h and they also show
 11 increased activation in the fusiform area which
 12 traditionally is an area that is associated with face
 13 recognition or f e::h and it it indicates some form of
 14 high (.) visual expertise basically
 15 (5 lines omitted)
 16 somehow they have great visual expertise in watching
 17 what is very interesting to them thereby activating
 18 those regions that for us are activated for faces (0.5)
 19 and for them activated when watching what they like

Karen identifies as a potential problem that “relatively neutral stimuli [...] do not capture the change in impulse control” (lines 1–4). Rather, experimental stimuli should reflect “what they like” (line 5) (i.e., target impulsivity in relation to the specific restricted interests of the participants). She refers to other studies that showed evidence that “increased activity in left insula” was shown “when viewing images of their special interests” (lines 9–10). This resonates with the activation in healthy individuals when confronted with images of their interest (cf. Excerpt 5, lines 13–16). However, in addition, they display “increased activation in the fusiform area” (line 11), an area that neuroscientists associate with face recognition, and indicates high visual expertise (because healthy individuals are thought of as being relatively good in recognizing faces). Accordingly, a parallel is drawn between the visual expertise that healthy individuals display in face recognition, and the visual expertise of individuals with ASD “when watching what they like” (line 19). This suggests that *if* a GO-NO paradigm would be used to investigate, the stimuli used in it should at least trigger brain activity in the ‘left insula’ and in the ‘fusiform area.’ So, by linking specific cognitive functions to specific brain regions, fMRI is treated as introducing a norm to which experimental stimuli must conform: Only when activity is present in the identified brain areas, the development of experimental stimuli is treated as successful.

In sum, fMRI is treated as introducing a normative framework against which experimental stimuli are evaluated, eventually giving rise to the idea that these stimuli must be ‘tailor-made’ (i.e., relative to the subjective personal interests of individuals). Only in this way, prior distinctions between ‘healthy’ individuals and individuals diagnosed with a mental disorder can be maintained. The human brain is not oriented to in terms of its complexity in this context (as we would for example expect on the basis of Excerpt 1). Instead, fMRI is treated as an

incentive to develop experiments that are tailored to meet previously established diagnostic categories.

Discussion and conclusion

Our case study shows that the researchers at the laboratory under study present themselves as being thoroughly aware that the complexity of the human brain cannot be neglected in neuropsychiatric practice. Our analysis focused on how brain imaging technologies such as fMRI are appropriated in neuropsychiatric practice and suggests that the ability to visualize brain activity *in vivo* mediates how the brain is understood *as* complex. Building on postphenomenology, we suggested that brain imaging technologies such as fMRI do not straightforwardly provide a new understanding of human cognition but are actively appropriated by neuroscientists: They function against an existing background of pragmatic aims and existing theoretical knowledge.

We have shown that brain imaging technologies mediate the complexity of the human brain in a particular way: researchers conceive of the brain *as* complex *in terms of* existing diagnostic labels. These diagnostic labels, in turn, become materialized in experimental design, because the linking of symptoms of mental disorders to specific brain areas is treated as indicative of successfully designed experimental stimuli. Diagnostic labels thus give rise to a set of assumptions about how the brains of individuals diagnosed with a certain mental disorder should work. These assumptions need to be confirmed in neuropsychiatric experiments to check the validity of experimental stimuli. Because of this, the 'braining' of neuropsychiatric experiments that promises to make diagnostic processes more objective and challenge existing diagnostic labels, actually reinforces these labels by giving rise to experimental set-ups tailor-made to individuals diagnosed with a certain mental disorder.

By revealing the normative influence of diagnostic labels in both the handling of experimental data and the design of future experiments, we have shown that visualizations of brain activity only become meaningful to researchers because of their embeddedness in the larger context of psychiatry. Previous diagnostic processes as well as psychological tests are oriented to by researchers because it is only when visualizations of brain activity are situated into a meaningful past that they are treated as interpretable. This past, then, is also constitutive of the future ways in which mental disorders are experimentally investigated. This is not so much a form of bias towards existing psychiatric labels but rather a collective practice of making visualizations of brain activity relevant to the project of neuropsychiatry.

In what sense, then, does the complexity of the human brain as disclosed through brain imaging technologies inform neuropsychiatry, when considered in light of the crucial importance attributed to previously assigned diagnostic labels? In the context of autism research, Des Fitzgerald has argued that the complexity of the human brain as revealed through the neurosciences puts autism research in what he calls an "awkward place": "unable to tack autism to an emerging project of 'neuroreductive' certainty [...] neuroscientists are equally unable to simply ignore both the evidence and the desire for a neurogenetic basis to the disorder" (2014, p. 247). On the one

hand, the disclosed complexity of the brain prevents neuroscientists from developing straightforward reductionist explanations of mental disorders, while on the other hand, they are put into a position in which they cannot deny the biological evidence that suggests a difference between ‘normal’ and ‘pathological’ brains. In one way or another, the complexity attributed to the human brain needs to be dealt with within neuropsychiatry to come to explanations of mental disorders.

The focus on how brain imaging technologies are appropriated in neuropsychiatric practice reveals another ‘awkward place’: the very project of *challenging* the diagnostic labels presented in the DSM thus simultaneously involves orienting to their *reliability* when interpreting experimental data. When experiments do not conform to the expectations constituted by diagnostic labels, this is treated as a failure in the experimental design. The complexity of the human brain is treated by researchers as being relative to the experimental stimuli used. How the neuropsychiatrists under study ‘brain’ and personalize neuropsychiatric experiments, can be considered an instance of how this ‘awkward place’ is managed in practice.

One of the questions arising when neuropsychiatric experiments—precisely because of how brain imaging technologies are appropriated—become entangled with the behavior and interests of specific experimental participants is to what extent such personalized ‘brained’ experiments can be extrapolated beyond the (medical) biographies of specific participants. This is a problem Karen (the researcher discussing her experimental approach in Excerpts 4–6) reportedly often encounters when presenting her research: aligning experiments to personal interests was often treated as making the experiments ‘highly subjective’ and ‘difficult to interpret.’ Hence, the promise of objectivity associated with the introduction of brain imaging technologies in psychiatry constitutes an uneasy relationship between the complexity of the brain and the desired simplicity of psychiatric explanatory models.

When aligning experiments explicitly to the specific interests of individuals, so it seems to be suggested, complexity can be integrated into neuropsychiatric research. However, this complexity becomes located in the variety of interests that individuals might have, and of which it is assumed that these have a shared neurobiological origin on the basis of previous diagnostic classifications. As a result, there might be a discrepancy between the theoretical appeal of the idea that there is a continuum between different mental disorders as well as between the ‘normal’ and the ‘healthy’ and its practical implementation in neuropsychiatry, in which the discontinuity introduced by diagnostic classifications remains largely uncontested. As a result, researchers acknowledge the complexity of the human brain on a generic level but do not grant this complexity to the brains of individuals diagnosed with a mental disorder. Individuals diagnosed with a mental disorder might become treated as increasingly homogeneous: although they might be heterogeneous when it concerns their biographies, they are treated as homogeneous in terms of their neurobiology.

It is an open question whether our findings are representative of the field of neuropsychiatry at large. Future research building on our findings could explore if it is more generally the case that research in neuropsychiatry tends to privilege previously established diagnostic categories when interpreting experimental data and designing experiments, such that neuropsychiatric investigations are grounded in the very classificatory systems that they intend to challenge. Furthermore, it should be

investigated if and how, also beyond the field of neuropsychiatry, there is a tendency to assume that individual differences can be explained away by tracing them back to specific neurobiological pathways. And, given the presence of numerous different (imaging) technologies in the neurosciences, a key issue to understand is whether and how each of these mediate the complexity of the brain differently, and to which standards of ‘normality’ researchers orient when appropriating these potentially different technological mediations.

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Declarations

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical approval The research conducted in the paper was granted ethical submission from the ethical committee at the University of Twente, The Netherlands.

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