Compulsive Body Spaces

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Chapter 2

Complications: Neuropsychiatric rationalisations

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2 Complications

Neuropsychiatric rationalisations

As the experiential rationalities that govern other-than-compulsive bodily action are insufficient to understand compulsivity and compulsive interactions, the current-day neuropsychiatric and current-day non-psychoanalytical clinical sciences present an alternative framework for understanding this aspect of people's life. In effect, this medicalisation of compulsion is another effort to rationalise the phenomenon. Rather than centring experiential systems of logic, this effort is built on biological and neurocognitive arguments. By no means can the neuropsychiatric, biomedical, and clinical sciences be seen as putting forward one undisputed understanding of compulsions, and the these sciences of compulsivity form a complex landscape filled with differences and internal disagreements, as will become clear in this chapter. However, as these sciences are underpinned by positivism, they share principles of what counts as valuable knowledge and how valuable knowledge should be created, and for that reason they are discussed in combination. Whilst these principles have led these sciences to produce extensive insight into compulsivity, as this chapter points out, they have also led to neglect certain dimensions, a dismissal of particular sources of knowledge, and a discouragement of other kinds of knowledge creation. As a result, confusions surrounding compulsive interactions partially resolve and partially shift into new explanatory territories.

In order for the neuropsychiatric, biomedical, and clinical rationalisations to formulate explanations as to why compulsivity can be part of human life and why compulsive interactions take place, the phenomenon has to go through a number of transformations. These transformations present another set of origins and set of problem definitions, many of which are not immediately, or at all, derived from the experiences of the people who perform compulsions. Rather, they become derivatives of diagnostic and neurological categorisations and different analytical epistemes. The onus of this rationalisation then does not fall on *why certain compulsive acts* take place, but on *how compulsivity is an abnormality* and how *this body* is *affected*. Indeed, this reframing reflects the research philosophy underpinning the primarily deductive approaches that produce 'objective', 'outsider' knowledge of compulsive acts in quantitative terms of difference in *degree*,

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but not *kind*, nor do they necessarily engage with the *socio-spatial circumstances* under which compulsions are performed.

This chapter traces the neuropsychiatric rationalisation of compulsive interactions and considers how it affects the confusions that people with Tourette syndrome who perform compulsive acts experience. It does so by examining four interconnected transformations and the onto-epistemological philosophies that underpin them as well as their implications for the ways in which the phenomenon has been rendered and treatment has been mapped onto it. This brings to light how certain confusions surrounding these compulsions have, at best, been lifted or shifted, have been neglected, or, at worst, have been exacerbated.

Transformation 1: Pathologisation

The neuropsychiatric scientific rationalisation of compulsive realities involves clinical diagnosis and starts at the point that compulsions are so problematic that professional help is required to cope with them. For many people, this rendition becomes an important – if not the most important – framework of understanding when they are diagnosed with Tourette syndrome, or as Joe puts it "I had it my entire life of course, but officially I know it for only four years". In lieu of failing to find sufficient answers to their compulsive experiences and the desperation that accompanies it, this new formal rendition offers a persuasive new understanding that could dissolve many confusions, or at least bring (partial) answers. Its comprehensive conceptualisation offers an explanation of many aspects of people's lives that extend beyond compulsive acts. The process of diagnostisation and eventually receiving the diagnosis of Tourette syndrome radically changes how they consider compulsivity, and how compulsive acts are perceived and even how these are experienced.

The pathologisation of a particular aspect of their lives introduces a normative division between healthy or normal engagements and unhealthy or abnormal ones. All compulsive interactions are thus grouped by virtue of it not adhering to the behavioural rationalities that govern what is defined as 'healthy', 'intended', and 'rational' behaviours. Indeed, for many research participants the Tourette's diagnosis had become a shorthand for alluding to their compulsive bodily movements and engagements. After his first mobile eye-tracking session, Dylan and I watch the recordings, and he considers his compulsions: "it all doesn't look very Touretty, I think", but he cannot quite put his finger on what it is that makes movements themselves particularly 'Touretty'. In line with Dylan and many others when examining their compulsivity, Tomos invokes his compulsive sensibilities as entirely self-contained psychological process when he explains his daily struggle: "then Tourette's really takes power over me I'm afraid". Whilst the clinical and experiential vocabularies differ in constitution and purpose, as well as remit and scope, in capturing compulsivity, the participants often merged these vocabularies in the expression of their experiences. Hence, with the

diagnosis, a new priority is placed on the shared seemingly unintended and irrational dimension of compulsions, which reduces the need for people to consider all compulsive acts as individual acts in the adoption of the clinical rationalisation of compulsive acts. The new diagnostic framing of these acts then helps to reduce their own confusions about the phenomenon and provides them with an appealing 'way out'; something that sits in between themselves and the acts.

The transformation of lived experience and knowledge of one's body to the clinical rendering of one's compulsive movements can be clarifying. Rhys demonstrates how it takes away confusions. For instance, Rhys' incessant sniff he had from a young age, and what he believed had been the sign of him having a year-round cold, was actually deemed to be a compulsion; a 'vocal tic' in the medical terminology. This refiguration of his body lifted worries over his immune system and helped him better understand his bodily movements. However, understanding the pathologisation of one's compulsions does not always go smoothly. The pathologisation of Lowri's acts left her struggling to understand which ones were problematised. She had expected to better understand her unwanted touching and ordering tendencies through acquainting herself with the clinical vocabulary after her diagnosis. However, she found it to be very confusing because of its narrow and rigid definitions and "was unsure if [she] was relieved at that point". It led her to closely examine all her movements, habits, routines, rituals, and preferences that she could not quite explain, and consequently feeling ashamed for "not knowing" that a particular movement she used to do "was a tic". In effect, the clinical rendition of her movements that she needed to adhere to led her to question her knowledge of her body as gathered throughout her life.

The pathologisation and diagnostisation of compulsive interactions change people's understanding of compulsive interactions because both processes change how they relate to these acts, their body and themselves. Their compulsive interactions – or tics to be more precise – were pathologised, but like in other illnesses, *they as individuals* were diagnosed. In the interview Joe remarks that he struggled with this transformation, but Sage in her interview welcomed it:

> It was rather difficult to take that first step, to ask for help, you know. You get a stamp 'he's faulty, so he's probably not right in the head'
>
> (Joe)

> I recall feeling incredibly relieved, because I knew already that I didn't do things on purpose (…) like an 'I told you so!' confirmation: 'I can't help it', so that was all good really.
>
> (Sage)

Joe's fear reflects a fundamental issue that is well developed in health sociology, disability studies, and the critical medical humanities; pathologisation

of a person and diagnostisation of a body risks the reduction of people as victims of their own biology; it renders them more predictable and takes away agency that is granted to undiagnosed others. Also, it opens the door to a stigma-induced overclaim of people's incapacities, for instance in terms of self-organisation. Subsequently, it lowers the standards to which undiagnosed others are held, how they can be treated, and what merits intervention.

Tying her diagnosis to her social relations, Sage reflects on ongoing disputes with her mother about the nature of her compulsions, as her mother would not believe Sage could not help but performing them. The diagnosis then changes individuals having to take responsibility for their compulsive bodily movements to the admission that there was a problem in their body, thereby shifting the location of the problem away from themselves, and onto their diagnosed body, and/or Tourette's (Schroeder 2005, Sandle 2012, Bervoets forthcoming, Bervoets and Beljaars, in review). This transforms compulsivity as confusing acts – 'why do I perform this act?' – to a problem of the individual – 'something is wrong with *me*' – and to a biological problem of their brain – 'my brain is faulty'. Paradoxically perhaps, the focus of the diagnosis on the individual and its biologisation of the compulsive interactions that it incites does help to lift confusions rooted in questions about the self and one's desires.

Transformation 2: Biologisation and neuroscientific logic

Another transformation that builds on the pathological one entails the biologisation of the compulsive interactions. The compulsive tendencies and the interactions that had puzzled Joe and others continue to shift from a personal crisis of intention and meaning to a normative behavioural enigma and then to a biological defect. In particular, the neuropsychiatric sciences formulate compulsivity as arising from a malfunctioning brain and the distorted processes that make up the nervous system. Following Robbins et al. (2012: 81), the combination of the broad set of involved neuropsychiatric perspectives entail a biological approach that is based on 'neurocognitive endophenotypes', "whereby changes in behavioural or cognitive processes are associated with discrete deficits in defined neural systems". In other words, this reflects the notion that what bodies do is a direct expression of the functionality of the brain, so neuroscientific logic holds that if people do things that are considered abnormal, there must be something wrong with their brain. This logic reflects a conceptualisation of the problem fundamentally as a deficit.

Tics and compulsions were considered to having a distinct neurological cause already in the late 1800s. As part of the eugenicist ideology that drove many sciences (Dowbiggin 1991), Georges Gilles De la Tourette (1885) and Jean-Martin Charcot (1887–1888, c.f. Kushner 1999), saw them as manifesting problems with the nervous system that signified a degenerative inheritance that was caused by alcoholism and immoral behaviour by past

generations (Kushner 1999). Currently, compulsive interactions are considered to be "caused by a defective metabolism of the neurotransmitters in the brain" (2020 ICD-10-CM Diagnosis Code F95.2): particularly structurally and functionally involving the basal ganglia, thalamus, prefrontal cortex and the cerebellum (for an overview see Ramkiran et al. 2019). This clear premise for the organic rooting of compulsions has been arrived at through different methods of disrupting their occurrence.

Understanding compulsive interactions as emerging from a problem in the brain thus permits the neurosciences a variety of interventions, notably physically, electronically, and chemically. Any kind of reduction in tics and compulsive interactions is deemed as a successful interference. Chemical interference has developed from the anti-psychotic drug Haloperidol. Kushner (1999) explains that this is a tranquilliser originally utilised to handle 'unmanageable' psychiatric patients that can induce Parkinson-like movements if taken in doses that are too high. The reduction in compulsions were considered to break the neurobiological chain in the production of the urge to perform them, but not cure the brain, as stopping taking the medication would increase the tics again. This is the same for current-day medication that includes neuroleptic and a-typical antipsychotics (e.g. aripiprazole, risperidone, olanzapine, and ziprasidone), dopamine receptor blockers or first-line antipsychotics (e.g. haloperidol, pimozide, and fluphenazine). Whereas atypical antipsychotics are preferred, what medicine is prescribed depends on the kind and multitude of diagnoses, as well as the severity as established with clinical tests, such as the Yale Global Tic Severity Scale (YGTSS) (Leckman et al. 1989).

No specific medicine has been developed for urge-driven conditions, therefore these drugs have pervasive effects (see Lombroso and Scahill 2008, Shprecher and Kurlan 2009), which also tend to cause an increase of many unwanted and other harmful phenomena that have been rendered as 'side effects'. They included muscle spasms, zombiism, restlessness, lethargy, phobias, suicidal thoughts, and can be so powerful that many have to stop treatment. Arguably, this treatment option is like the method of a shotgun: when shot and one shell hits the target, it could be claimed as a success, independent of the other elements shot by the other shells. Although the workings of antidepressants are related to different serotonin systems in the brain, it is not entirely clear why certain this type and other types of medication have certain effect on one person's tics and compulsions, whilst another person can have very different experiences.

In the 1970s neurosurgery entailed lobotomy in which lesions were caused in the thalamus to permanently disrupt brain structures and was found to reduce tics, and probably compulsive interactions, for most people (Kushner 1999). Since the 2000s, the invasive, but ostensibly reversible, Deep Brain Stimulation (DBS) seems to reduce the 'superfluous' movements for at least up to three years (Kimura et al. 2021), probably also including urge-driven compulsive interactions through electrodes implanted in the brain offering

variable electric *stimulation* (see Xu et al. 2020). Despite the research that seeks to map the production of tics in great detail, Kimura et al. (2021) argue that the electrodes can be placed in a wide variety of brain parts and yield the same results. The new physically non-invasive brain oscillation interference technique reduces tics by stimulates the cortical motor areas in the brain with an altered pattern of electric stimuli that is introduced to the Median Nerve via an electrified wristband (Morera Maiquez et al. 2020). It is important to keep in mind that any treatment that targets the brain is deemed successful when it reduces tics, including compulsive interactions and the plethora of severe side effects[1]. Lobotomy was found to reduce tics for less than 2 years but returned more severely afterward (Asam and Karrass 1981). For DBS, long-term effects are still largely unknown (but see Smeets et al. 2018) although it does tend to reduce tics for the overwhelming majority of those treated in the short term. For the new brain wave interference technique both long-term effects on compulsions and other effects are also as yet unknown (Morera Maiquez et al. 2020)

The effects of antipsychotics and neurosurgery on the manifestation of compulsions does not necessarily prove that they emerge from damage to the brain, rather that the chemicals, electrodes, and wristband interfere with their appearance through their alteration of the nerve stimulations or dopaminic regimes in the nervous system. Neither does it mean that the workings on tics necessarily indicate that there is something wrong with the brain. However, framing compulsivity as a neurobiological 'defect' in 'inhibitory' structures does not only open up the possibility to fix it, it also favours such fixture to be of the neurobiological kind. This reinforces and expands the importance of the roles of neuroscientific knowledge in understanding the phenomenon. In effect, it implies that a reduction in compulsive interactions equals a better functioning brain, and that an interference achieving the absence of compulsions for at least a year (in accordance to the diagnosis) signifies an optimal outcome for Tourette's as a disordered condition.

As with any science organised around positivist knowledge construction and mobilising platonic essentialism in its conceptualisation of phenomena, the life sciences of Tourette syndrome understand compulsive interactions as symptoms or signifiers of an underlying problem that causes them to manifest. As explained elsewhere, compulsions as conceived as biological entities grants compulsivity and the Tourette's diagnoses an ongoing ontological presence, rather than an intermittent one if the compulsive acts had been given ontic essence (Beljaars 2020). This onto-epistemology that guides these sciences thus has a strong transformative power on the conceptualisation of compulsive interactions. It follows that such understandings are built on dimensions of the phenomena that are, or otherwise become, measurable and quantifiable, brought into relations that are correlative – and not causal – in nature. The studies, as well as the DSM, prioritise observational methodology, assuming a universality of the human body, and reinforce findings through 'evidence-based' regimes of knowledge creation that only

recognise similar kinds of data as valuable input for new research. These sciences therefore exclude apprehending compulsive interactions in all their complexity (Clegg et al. 2013) and take greater experiential dimensions into account (Bankey 2004, Greenhough 2011).

In lieu of Tourette's having been declared a neurodevelopmental disorder, compulsive interactions have been included on a scale of symptom 'complexity' of lesser to greater extent. In this transformation, compulsive interactions are recast as *movements*, centring the involvement of the bones, muscles, and tendons[2]. Involving multiple muscle groups, compulsive interactions are then considered the most complex and are placed at the extreme end of the scale. People diagnosed with Tourette syndrome are considered to perform 'simple tics' in childhood and start to perform more complex compulsions when they age into adolescence (Bloch et al. 2006, Bloch & Leckman 2009, Groth et al. 2017). However, simple tics remain the hallmark symptom of the diagnosis from which it follows that the Tourette's condition is considered to go into remission (see Bloch et al. 2006, Rothenberger and Roessner 2019), and that it might well be possible that compulsive interactions are too complex to be recognised as symptoms. As they may take longer than the very brief instance of a tic, look like, or be made to look like 'normal' movements, or occur outside medical and clinical diagnostic, treatment, and research spaces, no research effort into these relatively extremely complex acts would be mandated according to the model's theoretical logic, despite its capacity for such research. In practice, the life scientific epistemological tendency to create and utilise stable constructs of phenomena, such as through disinhibition, cannot account for the acknowledged inherent slipperiness of the symptom expression that is used to allude to compulsive interactions.

The transformation that the neuropsychiatric, biomedical, and clinical rationalisation of compulsive interactions induces through their biologisation reconceptualises them as complex in biomechanical, rather than psychological, terms, and it obscures them by conceiving them as merely an effect of a brain problem. Whilst this strongly reductive account of compulsive acts does not offer intricate answers about compulsive interactions, it can offer a helpful way to make sense of inexplicable elements of their occurrence. Dylan's sense-making processes of having to do acts he does not intend to do are strongly based on his knowledge about their biological dimension. Exploring how compulsions feel he brings our discussion to the workings of neurotransmitters and how they are a reality for him:

> Dylan: You kind of feel that there's something not right in your nervous system. The only reason it's not right is of course because there are too many neurotransmitters, and not enough here and there … Even that's not even known, ehm … the argument at the moment is that you have too much dopamine, yeah okay … then that's how I shall put it; I have too much dopamine in my ass[3] … but if it actually works like that, if that's

> really true … and seratonin, noradrenalin are coming back, so they're included. But those are the most well-known ones … ehm … it goes a lot deeper and, actually, if that's what you have, you could medicate it.
>
> DB: yeah, but if you then exactly know if it … is secreting too much neurotransmitters …
>
> Dylan: You feel something is not right, ehm … if you transmit a signal over a particular nerve, then you can make it right in a way, so it's often a movement stimulus ehm … A movement stimulus thus means that you flex a muscle until the sensation has passed through the nerve, which makes it more right again.

The biologisation of compulsion and compulsive processes that precede the acts is then positioned as whole explanation of the phenomenon or as partial explanation that fit the gaps that are left by the incomplete sense-making exercises. In other words, the focus on inhibition puts forward the idea that acts can be good or bad, and that therefore the problem just is a lack of neurotransmitter-mechanics to impair the 'bad'[4]. It is a poignant reality that takes away the need to ask questions about *this* act, because it effectively shifts the focus to *how* any compulsion happens in the first place.

Transformation 3: Erasure of performative difference

Another transformation of considering compulsive interactions that stems from the rationalisation of the neuropsychiatric, biomedical, and clinical sciences involves a change in the possibility for certain questions that can be asked. As a result of the biologisation of compulsivity analytically situating the brain and further nervous system as the causal focal point, bodily movements are understood as little more than effects of the brain problem. Their rendition as 'symptom' is more important than the intricacies of the compulsions themselves. What these movements look like does not necessarily matter in the neuropsychiatric scientific analysis: the difference between Siôn having to compulsively reorder the dishwasher and Cai having to compulsively pick up his parents' cats has no analytical meaning or value in this rationalisation. Therefore, these understandings offer very little insight into the kinds of compulsive acts that people do. This deindividualisation transformation of compulsive interactions emerges from the biologisation in accordance with its positivist onto-epistemological principles and diagnostic procedures and their related categorisation exercises.

As positivist epistemologies do not require a sensitivity towards difference in kind and as there seems to be no limit to the variation between tics and compulsions, medical and clinical literature understand the instability of the symptom group as a given. With the exception of a short period in the 1930s, neurobiological sciences have not endeavoured to find patterns of the recurrence of particular compulsions (Kushner 2008). Therefore, tics and

compulsions are conceived of as 'highly idiosyncratic' (e.g. O'Connor et al. 1994, Verdellen 2007) which remains unchallenged. On this basis, research participants in positivist life scientific research – with the exception of those partaking in phenotype studies – are often requested to quantify their experience of having to do a compulsion as a singular experiential entity. According to the participants to this study, this quantification is one of the most profound transformations their compulsive interactions go through to fit the life scientific episteme.

Mina's professional medical knowledge and commensurate familiarity with clinical vocabularies and diagnostics had led her to consider how compulsivity should be studied based on her own experiences. She had been keen to talk through the clinical construction of her experiences during all our meetings, and during her second eye-tracking session, we discuss the implications of the genetic overlap between 'compulsive disorders' and 'schizophrenia' which she had read an article about. She had proposed to make a drawing as it would bring out her compulsive tendencies in great detail. Getting increasingly frustrated with how the charcoal lion emerged on the paper in front of her, she speaks slowly and deliberately relating her compulsions to the concepts of psychosis and addiction:

> Why it didn't really surprise me, but that would – it's quite unscientific what I'm saying – because it's so persistent and because these psychotic disorders seem so organic, I thought it really wouldn't surprise me; it's rather psychotic, I think, those compulsions. (…) It's something that drives you. I mean, it's outside your control. If you have a psychosis, and without having any say over it's like when you have compulsions … And that it's also something that you almost can't have a say over. Then addiction is also something that just gets worse all the time … So it's something – I would say – that it's outside yourself and controls you, and that's also in psychiatry. Maybe that's why I keep thinking about it. I think it's really just a kind of psychosis, maybe they should see it more like that.

In suggesting how the fluidity of the unfolding and the oppressive feeling of psychosis and addiction and compulsivity are very similar in her experience, she paints an intricate picture of how compulsivity has an ongoing complex presence. She depicts a phenomenon that is difficult to even capture in words, let alone in the rigid and categorical neuropsychiatric description that requires it to have a traceable, universal, and consistent numerical existence. The fact that so much is lost in this transformation renders the neuropsychiatric conception unhelpful, which makes her doubt the usefulness of the treatment options on offer.

In addition to the necessity for rendering compulsive acts quantifiable, the neuropsychiatric conceptualisation requires diagnosed people to unify all their sensations and actions and discuss them on the same terms. Indeed,

the life scientific 'unit of calculation' is a diagnosed person or the presence of symptom, not an act itself, which makes the compelled movements entirely a function of the individual. Therefore, where not further specified, blanket statements are made about different movements and acts (in addition to different sensations) under the heading of 'symptoms' because it is the same person who performs them (e.g. van der Salm et al. 2012). In turn, this confirms and emphasises the requirement for the person to be diagnosed with Tourette syndrome.

Diagnosis is premised on a deductive method of determining what disease a patient has; criteria forming one diagnosis are therefore always different from another diagnoses. Individual symptoms can occur with other diagnoses; pain for instance, but the combination of the symptom collection is unique to a diagnosis. The diagnostic criteria for Tourette syndrome consist of two motor tics, for example eye-blinking, shoulder shrugging, or nose scrunching, and one vocal tic, such as sniffing, coughing, or uttering a sound or word, for at least a year with an onset prior to the age of 18, and not as a result of medicine or other drugs (DSM5). The complex and intricate issues that have led people like Mina to seek diagnosis, including compulsive interactions, nonetheless, the diagnosis shifts motor and vocal tics into the central focus. As these bodily movements are most strongly problematised and foregrounded in treatment options, it incites a diagnosed person to prioritise the experience of these particular bodily movements over others (see also Kushner 1999). In effect, the clinical rationalisation of bodily movement introduces a hierarchy of more and less important acts that background compulsive interactions.

Compulsive interaction is not an official symptom category of Tourette syndrome, as the movements are regarded as acts, which reflects the ontic focus on the universal biological body. The clinical capture of compulsive interactions emerges from 'phenotype' studies that render visible and examine the relations between bodily movements and brain divergence by (re)producing movement categories (e.g. Cath et al. 2001, Worbe et al. 2010, Ferrao et al. 2013). These studies address the demand for practical distinction possibilities between the symptomatologies of Tourette syndrome, Obsessive Compulsive Disorder (OCD), and Attention Deficit Hyperactivity Distorder (ADHD) diagnoses to improve the capture of these diagnoses, reduce misdiagnosis, and signal 'comorbidities' – the clinical recognition of people having multiple diagnoses. Unclear how these categories are produced exactly, they are reified in deductive (clinical) tests, such as the most widely employed Yale Global Tic Severity Scale (YGTSS) (Leckman et al. 1989).

The clinical categories that capture compulsive interactions involve 'touching' (e.g. pressing one's finger into the corner of a table, or clasping a mug), and include 'tapping', 'rubbing', 'clapping', and 'picking' (e.g. removing a flower from its bed, or a leaf from its stalk), 'ordering' (e.g. grouping similar objects, or repositioning objects into a new composition) that includes positioning, arranging, 'symmetry behaviour', and 'evening-up performances' (Cath et al. 1992, 2001, Rosario-Campos et al. 2001, Alsobrook and Pauls 2002,

Mansueto and Keuler 2005, Palumbo and Kurlan 2007, Robertson and Cavanna 2007, Worbe et al. 2010, Ferrao et al. 2013, Neal and Cavanna 2013, Huisman-van Dijk et al. 2016, Sambrani et al. 2016). The clinical capture of compulsive interactions also extends to 'paliphenomena', describing acts that involve repetition of actions (palipraxia) or sounds (palilalia): their own or those from other people, animals, and objects. Mina explains that for her this includes having to reread a paragraph in a book "because it was not read properly", and for Lowri this is typified by her having to step through a doorframe again if the first time was not done in the 'right' way.

Compulsive interactions that involve seeking or creating some kind of pattern in the broadly conceived bodily environment have been clinically denoted under the headings of 'mental play' (Cath et al. 2001, Worbe et al. 2010) or 'mental compulsions' (Williams et al. 2011). They include counting a variety of things and seeking (un)even amounts of things in a place, finger tapping on musical rhythms, as well as aligning objects and people visually (Cath et al. 2001, Alsobrook and Pauls 2002, Worbe et al. 2010). This latter kind resonates with Alan, who had to move his head to visually align a strand of my hair with a lamppost outside whilst we were having lunch after the interview. Another example of these interactions that demonstrate an astoundingly complex spatial imaginary is artist and karate instructor Shane Fistell compulsively blowing air close to Oliver Sacks' mouth because he had to 'touch' Sacks' breath with his own. This compulsion and many others figure on *The Mind Traveller* (1996)[5], a documentary following Dr Oliver Sacks engaging with some of his patients.

Compulsive interactions that have painful consequences to the person are denoted as 'self-injurious behaviours'. In contrast to the other categories, these compulsions do not pinpoint a particular kind of movement; rather they are used to indicate clinical severity of Tourette syndrome. Distinctly different from acts of self-harm, the harmful element is an *unwanted consequence*, not the purpose, of the act, according to people who have to perform them. Examples include hitting oneself in the chest, violently dropping oneself on the floor, burning or cutting parts of one's own body (Robertson and Cavanna 2007, Robertson et al. 2008). Despite these particular clinical categorisations of compulsive interactions and other acts as part of the Tourette's symptomatology, they are not treated differently[6], nor do they incite a diversification of clinical explanations as to why people compulsively touch rather than compulsively arrange, for instance.

The limited clinical capture of compulsive interactions incites a reduced acknowledgement or ill-recognition of others. In addition to the quantification of their sensations, many participants to this study affirmed that this transformation that is imposed from the outside is not always particularly welcomed. Having aligned his expressions of his compulsions in strong alignment with the biological conception, during our meetings Dylan often questioned his movements and sensations on their accuracy, and purpose. Even the way he reached for the tap during an eye-tracking session was

assessed in detailed clinical categorical language. He had initially been content with the capacity of the Tourette's diagnosis to capture his experiences, but after a while I noticed how some acts and sensations could not be related to the diagnosis. As he found this absence unacceptable, he had identified a gap in the scientific literature where his experiences should be fitted:

> I actually did make up a new category that makes me think like, yeah, these are indeed motor tics. Only it doesn't consist of the movements that we know of, and I miss a category with a normal description which I named 'passive tics'. Passive motor tics … Can something like that exist? Yes of course it can exist! I mean, I am a patient, I feel that, therefore that is what it is.

We discuss what he means by that and he demonstrates what he deems a normal versus a compulsive way of sitting on the sofa:

> Passive tics at tics that in one way or another cause a pain or a pressure that you want to get rid of … That you want to get rid of by, for instance, immobilising a body part; you pull your arm very far behind your head so that you can't feel your arm anymore after a while.

The discomfort that this way of sitting invokes precludes the requirement to move his arm compulsively. Dylan expected and needed the clinical vocabulary to provide answers to, or even recognition of, his experiences to lift confusions around the phenomena, but it did not. Therefore, he needed the current set to be expanded. Nonetheless, in addition to sense-making exercises, the neurobiological and clinical rationalisations cannot explain why *this* compulsion takes place and only offer a device for description. Indeed, the erasure of differences between individual compulsive acts through the process of diagnosis is amplified by the biologisation of the acts. Hence the neuropsychiatric, biomedical, and clinical rationalisation of compulsive interactions invites people who have to perform them to discard denoting differences and accept them as idiosyncratic. This, therefore, more of less lifts the confusion, as there seem to be no answers. In a similar way, the compulsions as rationalised within the life sciences does not encourage querying if any contextual aspects could be informative of how they take place as and when they take place.

Transformation 4: Erasure of circumstances

In addition to compulsions and tics being regarded as idiosyncratic, they are deemed to be 'waxing and waning', which alludes to both the kind of compulsion performed by a diagnosed person, and the temporal variations of compulsivity in a given period. Similar to idiosyncracy of kind, the denotation of these variations in the clinical sciences have been made in the exploratory, descriptive mode serving as acknowledgement of social worlds

and activities as having salience (Cohen and Leckman 1992, Conelea and Woods 2008, Woods et al. 2009, Cavanna and Nani 2013). However, as per the natural scientific pursuit of universal truths, these relations have not been subjected to a rigorous examination.

Studies do register differences in the frequency and severity of simple tics that can be observed by clinicians and close others (e.g. partners, parents) between the home and the doctors office (Goetz et al. 2001), "reading a book", "spending time with friends", and "moving to a new home" (Christenson et al. 1993, Silva et al. 1995, Miltenberg et al. 1998). Nonetheless, these studies set up activity categories that suggest the idea of certain general life situations but are too vaguely defined and lack acknowledgement of the materiality, micro-dynamics, and social fabric of situations. These are therefore too abstract to start understanding why this compulsive interaction happens now, here, and under these circumstances. Indeed, studies that do focus on 'environmental influences', such as those related to the social world, utilise questionnaires of broad categories that do not allow for nuance, nor differentiation between situations on the basis of which participants to these studies choose their answers (Leckman et al. 1993, Woods et al. 2005, Conelea and Woods 2008, Conelea et al. 2011, Wang et al. 2011, Capriotti et al. 2013).

There are exceptions in which the circumstances under which compulsions take place are acknowledged more fully. For instance, Karp and Hallett's (1996) study that is based on experiential accounts from other studies, argue that the bodily surroundings locate the starting point of the sensations that lead a person to act compulsively outside the body. These studies are almost exclusively studies that allow for more nuance through case studies (Eapen et al. 1994, Cohen et al. 2013), first person narratives (Bliss 1980, Kane 1994, Hollenbeck 2003, Turtle and Robertson 2008), and non-academic autobiographies (Wilensky 1999, Van Bloss 2006). Joseph Bliss (1980: 1347) explains this as "a mental projection of sensory impressions to other persons and to inanimate or even non-existent objects". For example, he would perceive a "firm cord running down the center line of the sheet. A need appears to apply pressure to this phantom cord by pulling" (ibid.). Bliss also describes *feeling*, not *touching* an object:

> At times there is a recurring need while writing to press the pencil point hard against the surface of the paper. A 'feel' is perceived at the end of the pencil; in my mind, the point becomes an extension of the body, and the 'feel' at the point is translated into a TS-sensitised body site that demands even greater pressure until the point is broken.
>
> (Bliss, Sensory experiences of Gilles de la Tourette syndrome, 1980. p. 1347)

These intricate and complex experiences seem to strongly resonate by many people with Touretteic sensibilities, including the research participants of this study. There is, therefore, no lack of evidence that the circumstances under which compulsive interactions take place are crucial in their performance.

Nonetheless, in the scientific onto-epistemology, bodily surroundings are understood as inert and passive, and differences between places are treated as a given but have no ontological power. In fact, the context of compulsions is rather understood as an analytical nuisance that needs erasure from empirical research, hence the necessity of laboratory conditions under which neuropsychiatric, biomedical, and clinical knowledge is created. These laboratory conditions that underpin life scientific studies with a focus on frequency, which is often used to qualify improvement of a mode of interference such as behavioural therapies, render the person mostly static in a seated position facing a camera and/or observation booth in an otherwise unfamiliar, white, and mostly empty room. This includes studies that measure the impact of exercise on tic frequency, such as a study by Jackson et al. (2020) that asked children aged 10–12 to do Kick Boxing and Tai Chi exercises as mediated through an X-Box 360 Kinect, whilst wearing a heart rate monitor and with a camera pointed at them at two meters distance in a laboratory space. Similar to Morera Maiquez et al.'s (2020) study that tested the tic-reducing wrist band mentioned earlier, the analytical focus was only on simple tics in the face and upper body, which reinforces the hallmark position of these tics at the detriment of other movements, and, in the process, dismisses possibilities for a more holistic consideration of compulsions as subset of Tourettic motions.

Methodologies with positivist underpinnings do not only measure frequency in highly artificial circumstances but also forego more complex compulsions, including all interactions. These studies and other medical encounters thus cannot account for what happens outside the broader medical spaces. Nonetheless, the claims these studies make sustain the life scientific rationalisation of compulsive interactions, which can lead to confusions about certain situations, but can also directly oppose the experience of those diagnosed. The 'rebound effect' is one such ongoing dispute. It is the observation of people with Tourette's experiencing a strong increase of the need to do tics and compulsions after they 'suppress'[7] them for a period that cannot be registered following clinical methodology (Verdellen et al. 2008). People who perform compulsions almost unanimously disagree that it does not exist, as Dylan explains:

> Dylan: Actually, everything you catch[8] speaking of tics, you get that. Scientifically, it is not proven yet; it's not confirmed that the rebound effect exists … But it exists. Why? Because it does.
>
> DB: Well … yeah …
>
> Dylan: That's why we don't need more supporting arguments, missus scientist!

Aside from a discrepancy between the two knowledge systems, it also highlights how compulsivity remains analytically entirely unaccounted for in the spaces beyond the direct gaze of the life sciences (see Beljaars 2020). The implications of this fourth transformation thus entail that the life sciences

encourage to only consider compulsions as contextualised in quantitative measures, and through the way the person conceives of situations, rather than the situations themselves. This analytical conceptualisation reinforces clinical treatment as decontextualised as well; through biomedicine as well as through behavioural therapies. These push the message that compulsions are performed because the person cannot inhibit themselves, which transforms compulsivity into a matter of personal control in which the body is positioned as enemy which needs to be kept under control; regardless of the circumstances (see Hollenbeck 2003).

Moving forward

There are, broadly defined, four elements to the neuropsychiatric, biomedical, and clinical rationalisation of compulsive interaction that create a new set of confusions on top of those created in the frameworks that are currently available for making sense of compulsions (see Chapter 1). They entail discouragements to see differences in kind as well as in context of compulsion and encouragements to see compulsive interactions as always and only problematic, both from a personal and from socio-normative point of view. These four elements represent the transformations but do not provide complete answers to people's own experiences and conceptions of why they feel compelled to interact with their surroundings in a very particular way without wanting to, without knowing why, and often without anticipating they have to. Addressing them presents us with a requirement of a radically new ethics of analysing compulsivity.

The neuropsychiatric rationalisation of the determination that compulsive acts are a sign of a faulty brain leads to a reluctance to say which acts through the accepted idiosyncracy makes this rationalisation a dangerous one. It also takes away the possibility to express the idiosyncracy of compulsion. This rationalisation, however much currently felt as helpful answer by people with a diagnosis, is dangerous in that it effectively blocks the consideration of the compulsive phenomenon as performance. As such, we remain stuck in Lowri's frustration in which she cannot tell what movement she makes is compulsive. Indeed, rather than an understanding of what compulsion is, the biological narrative describes the boundaries between the 'healthy' and this version of the 'unhealthy' in conjunction with the 'abnormal' derivation of the 'normal' through the production of signs of subversion. Compulsion can never become considered on its own merit because it is precisely defined as pathological behaviour; the becoming abnormal; it is a colonisation of human movement and bodily expression. The life sciences can only ever locate the frontier and allow encroachment on unrationalised bodily performance. Indeed, we are reminded of Deleuze and Guattari's (2004 [1980]: 275) thoughts on the value of heterogeneity:

> The histories of ideas should never be continuous; it should be wary of resemblances, but also of descents of filiations; it should be content to

> mark the thresholds through which an idea passes, the journeys it takes that change its nature or object.
>
> (Deleuze & Guattari, *Thousand Plateaus. Capitalism and Schizophrenia*, 2004 [1980], p. 275)

Confusions, in part, emerge because of the immediate problematisation of compulsions, which is the only explanation the neuropsychiatric sciences offer, and their biologication confirms it. Hence all analytical effort is focused on understanding this problem and then solving it. It does not formulate, nor question the conditions under which compulsive interactions are understood as a problem; hence, it does not examine these acts beyond their framing as a problem. The problem is a given that is decontextualised beyond the body; in other words, the problem is emerging solely from the person who needs help and seeks it through diagnostisation (one of the few options available) not from the broader socio-cultural and political contexts. Indeed, through diagnostisation that transforms a set of performances to a problematic person, these people acquire a new ontic essence that sweeps the person up in political processes of exclusion that makes them vulnerable to processes of dehumanisation and permits acts of colonisation; of legal interference with the person through their body to make them more 'fully human' again. Therefore, an episteme that emancipates the individual person and their circumstances by ontologically centring compulsive interactions can help mediate the vulnerability of these people to such processes.

Many participants discussed their struggles and difficulties with compulsivity and the part of their lives that they and other around them associate with Tourette syndrome. However, when discussing and performing the individual acts – with some exceptions – the problematic dimension was largely unimportant. Discussing compulsive interactions with Ginny and her manners of coping with having to do them, she explains how completion of some compulsions makes her feel:

> You know, if you were to remove it all, you'd become unhappy *laughs* That's true, you shouldn't want to remove it all with those tics and that, because you'd remove happiness. I really believe that, and you do have to make it work for yourself, but that you can really be in that moment of happiness! Yeah, you know, it doesn't have any function, and it doesn't have any content, but that just doesn't matter!

She was not alone in remarking on the liveliness of some of the experiences emerging from aesthetically pleasing results of compulsive interactions. Such slight and brief exhilaration might sprout from such an act precisely because they are a-personal and unprecedented, and can thus retain an element of surprise to the person performing them. The latter might especially be the case if the compulsion follows seamlessly on an other-than-compulsive act. Nonetheless, Ginny and all other participants contend that having

to perform compulsive interactions remains stressful. This paradox highlights the internal intricacies of compulsive interactions, which does not only produce experiential confusions for the person performing them. It also presents us with a slippage on favourability, functionality, and enjoyability, thereby conjuring up analytical confusions of compulsive interactions. In turn, they bring up questions of the justness of dismissing certain aspects of compulsive interactions in analysis. Indeed, rather than retaining analytical difference between compulsive and other-than-compulsive, or medicalised or not medicalised acts, these questions suggest that in order to understand compulsive interactions, they demand further individuation on both experiential and analytical levels.

Suspending the problematisation of compulsions does *not* encourage the rendering of these interactions as unproblematic; what it does is denote how the narrow definition of compulsion as a problem may have stifled further exploration and examination, and thus a broader understanding of the phenomenon. I argue that the confusions surrounding this phenomenon are at the heart of this, in particular the experiential ones, because they reflect the necessity of a thorough re-examination of explanatory power of the structures that Western sciences have in place to understand human behaviour, and shed light on the blind spots they leave.

Notes

1 For lobotomy, these working include problems with keeping balance, walking, swallowing, and speaking, as well as suffering from brain infections, spasticity and paralysis of all four limbs, and cognitive functioning (see Mukhida et al. 2008 for an overview). Other effects of DBS include seizures, problems with vision, and headaches (Testini et al. 2016, Marano et al. 2019), problems performing small movements (Huys et al. 2016), apathy, paraesthesia, erectile dysfunction, problems with emotions, weight gain, (Balderman et al. 2019).
2 This is a deliberate move away from psychoanalytical approaches to Tourette syndrome that had conceptualised them as *acts* to emphasise as a deliberately meaningful understanding of bodily action.
3 Original word: "donder" which is difficult to translate.
4 With thanks to Jo Bervoets.
5 Directed by Christopher Rawlence.
6 Some behavioural therapies, such as Habit Reversal Therapy, do differentiate between compulsions and tics to the extent that they target those acts that are most problematic for these people.
7 The possibility of tic suppression is currently (summer 2021) being challenged by many people with a Tourette syndrome diagnosis, as accurate description of what is experienced to happen. It suggests that not performing tics or compulsions – suppressing them – is the end of it. However, with the attempt to hold in tics, the pressure to perform them increases, so there might not be a 'net gain'. Also, many people experience having to perform many more tics after a period of holding them in, so instead of tics and compulsions disappearing, it constitutes a displacement over time. Therefore, indicating this phenomenon in the remainder of the book, the term appears in quotation marks.
8 Dylan uses 'catching' (original: 'opvangen') to indicate 'suppressing' the need to perform compulsions and consequently not performing them.

References

Alsobrook, J.P. II and Pauls, D.L. 2002. A factor analysis of tic symptoms in Gilles de la Tourette syndrome. *American Journal of Psychiatry* 159, pp. 291–296.

Asam, U. and Karrass, W. 1981. Gilles de la Tourette syndrome and psychosurgery. *Acta Paedopsychiatrica* 47, pp. 39–48.

Baldermann, J., Melzer, C., Zapf, A., Kohl, S., Timmermann, L., Tittgemeyer, M., Huys, D., Visser-Vandewalle, V., Kühn, A., Horn, A. and Kuhn, J., 2019. Connectivity profile predictive of effective deep brain stimulation in obsessive-compulsive disorder. *Biological Psychiatry* 85(9), pp. 735–743.

Bankey, R. 2004. The agoraphobic condition. *Cultural Geographies* 11, pp. 347–355.

Beljaars, D. 2020. Towards compulsive geographies. *Transactions of the Institute of British Geographers* 45, pp. 284–298.

Bervoets, J. forthcoming. Tourette Syndrome and Dynamic Moral Responsibility: A Healthier Look at Tourette's?. PhD Thesis. University of Antwerp.

Bervoets, J. and Beljaars, D. in review. From deficit to surplus models of mental illness. Tourette syndrome: A case study. *Social Science & Medicine*.

Bliss, J. 1980. Sensory experiences of Gilles de la Tourette syndrome. *Archives of General Psychiatry* 37, pp. 1343–1347.

Bloch, M.H. and Leckman, J.F. 2009. Clinical course of Tourette syndrome. *Journal of Psychosomatic Research* 67(6), pp. 497–501.

Bloch, M.H., Peterson, B.S., Scahill, L., Otka, J., Katsovich, L., Zhang, H. and Leckman, J.F. 2006. Adulthood outcome of tic and obsessive-compulsive symptom severity in children with Tourette syndrome. *Archives of Pediatrics and Adolescent Medicine* 160(1), pp. 65–69.

Capriotti, M.R. et al. 2013. Environmental factors as potential determinants of premonitory urge severity in youth with Tourette syndrome. *Journal of Obsessive-Compulsive and Related Disorders* 2, pp. 37–42.

Cath, D.C. et al. 1992. Mental play in Gilles de la Tourette's syndrome and obsessive-compulsive disorder. *British Journal of Psychiatry* 161, pp. 542–545.

Cath, D.C. et al. 2001. Repetitive behaviors in Tourette's syndrome and OCD with and without tics: What are the differences? *Psychiatry Research* 101, pp. 171–185.

Cavanna, A.E. and Nani, A. 2013. Tourette syndrome and consciousness of action. *Tremor and Other Hyperkinetic Movements* 3, pp. 1–8.

Christenson, G.A., Ristvedt, S.L. and Mackenzie, T.B. 1993. Identification of trichotillomania cue profiles. *Behavioral Research and Therapy* 31, pp. 315–320.

Clegg, J., Gillott, A. and Jones, J. 2013. Conceptual issues in neurodevelopmental disorders: lives out of sync. *Current Opinion in Psychiatry* 26, pp. 289–294.

Cohen, A.J. and Leckman, J.F. 1992. Sensory phenomena associated with Gilles de la Tourette's syndrome. *Journal of Clinical Psychiatry* 53, pp. 319–323.

Cohen, S., Leckman, J.F. and Bloch, M.H. 2013. Clinical assessment of Tourette syndrome and Tic disorders. *Neuroscience and Biobehavioral Reviews* 376, pp. 997–1007.

Conelea, C.A. and Woods, D.W. 2008. The influence of contextual factors on tic expression in Tourette syndrome: a review. *Journal of Psychosomatic Research* 65, pp. 487–496.

Conelea, C.A., Woods, D.W. and Zinner, S.H. 2011. Exploring the impact of chronic tic disorders on youth: results from the Tourette syndrome impact survey. *Child Psychiatry and Human Development* 42, pp. 219–242.

De la Tourette, G.G. 1885. Étude sur une affection nerveuse caractérisée par de l'Incoordination motrice accompagnée d'Écholalie et de coprolalie (Jumping, latah, myriachit). *Archives de Neurologie* 9(19–42), pp. 158–200.

Deleuze, G. and Guattari, F. 2004 [1980]. *A Thousand Plateaus. Capitalism and Schizophrenia*. London: Continuum.

Dowbiggin, I.R. 1991. *Inheriting Madness: Professionalization and Psychiatric Knowledge in Nineteenth Century France*. Berkeley: University of California Press.

Eapen, V., Mortiarty, J. and Robertson, M.M. 1994. Stimulus induced behaviours in Tourette's syndrome. *Journal of Neurology, Neurosurgery, and Psychiatry* 57, pp. 853–855.

Ferrao, Y.A., de Alvarenga, P.G., Hounie, A.G., de Mathis, M.A., de Rosario, M.C. and Miguel, E. 2013. The phenomenology of obsessive-compulsive symptoms in Tourette syndrome. In: Martino, D. and Leckman J.F., eds. *Tourette Syndrome*. Oxford, New York: Oxford University Press, pp. 50–73.

Goetz, C.G., Leurgans, S. and Chmura, T.A. 2001. Home alone: Methods to maximize tic expression for objective videotape assessments in Gilles de la Tourette syndrome. *Movement Disorders* 16(4), pp. 693–697.

Greenhough, B. 2011. Citizenship, care and companionship: Approaching geographies of health and bioscience. *Progress in Human Geography* 35(2), pp. 153–171.

Groth, C., Mol Debes, N., Rask, C.U., Lange, T. and Skov, L. 2017. Course of Tourette syndrome and comorbidities in a large prospective clinical study. *Journal of the American Academy of Child and Adolescent Psychiatry* 56(4), pp. 304–312.

Hollenbeck, P.J. 2003. A jangling journey: Life with Tourette syndrome. *Cerebrum* 53, pp. 47–61.

Huisman-van Dijk, H.M., Schoot, R., Rijkeboer, M.M., Mathews, C.A. and Cath, D.C. 2016. The relationship between tics, OC, ADHD and autism symptoms: A cross- disorder symptom analysis in Gilles de la Tourette syndrome patients and family-members. *Psychiatry Research* 237, pp. 138–146.

Huys, D., Bartsch, C., Koester, P., Lenartz, D., Maarouf, M., Daumann, J., Mai, J., Klosterkötter, J., Hunsche, S., Visser-Vandewalle, V., Woopen, C., Timmermann, L., Sturm, V. and Kuhn, J., 2016. Motor improvement and emotional stabilization in patients with Tourette Syndrome after deep brain stimulation of the ventral anterior and ventrolateral motor part of the thalamus. *Biological Psychiatry* 79(5), pp. 392–401.

Jackson, G.M., Nixon, E. and Jackson, S.R., 2020. Tic frequency and behavioural measures of cognitive control are improved in individuals with Tourette syndrome by aerobic exercise training. *Cortex* 129, pp. 188–198.

Kane, M.J. 1994. Premonitory urges as 'attentional tics' in Tourette's syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry* 33, pp. 805–808.

Karp, B.I. and Hallett, M. 1996. Extracorporeal 'phantom' tics in Tourette's syndrome. *Neurology* 46, pp. 38–40.

Kimura, Y., Iijima, K., Takayama, Y., Yokosako, S., Kaneko, Y., Omori, M., Kaido, T., Kano, Y. and Iwasaki, M. 2021. Deep brain stimulation for refractory Tourette syndrome: Electrode position and clinical outcome. *Neurologia Medico-Chirurgica* 61(1), pp. 33–39.

Kushner, H.I. 1999. *A Cursing Brain? The Histories of Tourette Syndrome*. Cambridge, MA: Harvard University Press.

Kushner, H.I. 2008. History as a medical tool. *Lancet (London, England)* 371(9612), pp. 552–553.

Leckman, J.F., Riddle, M.A., Hardin, M.T., Ort, S.I., Swartz, K.L., Stevenson, J. and Cohen, D.J. 1989. The Yale global tic severity scale: Initial testing of a clinician-rated scale of tic severity. *Journal of the American Academy of Child and Adolescent Psychiatry* 28, pp. 566–573.

Leckman, J.F., Walker, D.A. and Cohen, D.J. 1993, Premonitory urges in Tourette's syndrome. *Journal of American Psychiatry* 150, pp. 98–102.

Lombroso, P.J. and Scahill, L. 2008. Tourette syndrome and obsessive-compulsive disorder. *Brain & Development* 30(4), pp. 231–237.

Mansueto, C.S. and Keuler, D.J. 2005. Tic or compulsion?: It's Tourettic OCD. *Behavior Modification* 29(5), pp. 784–799.

Marano, M., Migliore, S., Squitieri, F., Insola, A., Scarnati, E. and Mazzone, P., 2019. CM-Pf deep brain stimulation and the long term management of motor and psychiatric symptoms in a case of Tourette syndrome. *Journal of Clinical Neuroscience* 62, pp. 269–272.

Miltenberger, R.G., Fuqua, R.W. and Woods, D.W. 1998. Applying behavior analysis to clinical problems: Review and analysis of habit reversal. *Journal of Applied Behavior Analysis* 31, pp. 447–469.

Morera Maiquez, B. et al. 2020. Entraining movement-related brain oscillations to suppress tics in Tourette syndrome. *Current Biology* 30 (12), pp. 2334–2342.e3.

Mukhida K., Bishop M., Hong M., and Mendez I. 2008. Neurosurgical strategies for Gilles de la Tourette's syndrome. *Neuropsychiatric Disease and Treatment* 4(6), pp. 1111–1128

Neal, M. and Cavanna, A.E. 2013. Not just right experiences in patients with Tourette syndrome: Complex motor tics or compulsions? *Psychiatry Research* 210, pp. 559–563.

O'Connor, K.P., Gareau, D. and Blowers, G.H. 1994. Personal constructs associated with tics. *British Journal of Clinical Psychology* 33, pp. 151–158.

Palumbo, D. and Kurlan, R. 2007. Complex obsessive compulsive and impulsive symptoms in Tourette's syndrome. *Neuropsychiatric disease and treatment* 3(5), pp. 687–693.

Ramkiran, S., Heidemeyer, L., Gaebler, A., Shah, N.J. and Neuner, I. (2019) Alterations in basal ganglia-cerebello-thalamo-cortical connectivity and whole brain functional network topology in Tourette's syndrome. *NeuroImage: Clinical* 24, p. 101998

Robbins, T.W., Gillan, C.M., Smith, D.G., de Wit, S. and Ersche, K.D. 2012. Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends in Cognitive Sciences* 16(1), pp. 81–91.

Robertson, M.M. and Cavanna, A.E., 2007. The Gilles de la Tourette syndrome: a principle component factor analytic study of a large pedigree. *Psychiatric Genetics* 17, pp. 143–152.

Robertson, M.M. et al. 2008. Principal components analysis of a large cohort with Tourette syndrome. *British Journal of Psychiatry* 193, pp. 31–36.

Rosario-Campos, M., Leckman, J., Mercadante, M., Shavitt, R., Prado, H., Sada, P., Zamignani, D. and Miguel, E. 2001. Adults with early-onset obsessive-compulsive disorder. *American Journal of Psychiatry* 158(11), pp. 1899–1903.

Rothenberger, A. and Roessner, V. 2019. Psychopharmacotherapy of obsessive-compulsive symptoms within the framework of Tourette syndrome. *Current Neuropharmacology* 17(8), pp. 703–709.

Sambrani, T., Jakubovski, E. and Müller-Vahl, K.R. 2016. New insights into clinical characteristics of Gilles de la Tourette syndrome: findings in 1032 patients from a single German center. *Frontiers in Neuroscience* 10, p. 415.

Sandle, R.V. 2012. The deconstruction of Gilles de la Tourette's syndrome. *Journal of European Psychology Students* 3(1), pp. 68–77.

Schroeder, T. 2005. Moral responsibility and Tourette syndrome. *Philosophy and Phenomenological Research* 71(1), pp. 106–123.

Shprecher, D. and Kurlan, R. 2009. The management of tics. *Movement Disorders* 24(1), pp. 15–24.

Silva, R.R. et al. 1995. Environmental factors and related fluctuation of symptoms in children and adolescents with Tourette's disorder. *Journal of Child Psychology and Psychiatry* 36, pp. 305–312.

Smeets, A. et al. 2018. Thalamic deep brain stimulation for refractory Tourette syndrome: clinical evidence for increasing disbalance of therapeutic effects and side effects at long-term follow-up. *Neuromodulation* 21(2), pp. 197–202.

Testini, P., Min, H., Bashir, A. and Lee, K., 2016. Deep brain stimulation for Tourette's syndrome: The case for targeting the thalamic centromedian–parafascicular complex. *Frontiers in Neurology* 7, pp. 193.

Turtle, L. and Robertson, M.M. 2008. Tics, twitches, tales: The experiences of Gilles de la Tourette's syndrome. *American Journal of Orthopsychiatry* 78(4), pp. 449–455.

van Bloss, N. 2006. *Busy Body. My Life With Tourette Syndrome*. London: Fusion Press.

van der Salm, S.M., Tijssen, M.A., Koelman, J.H. and van Rootselaar, A.F. 2012. The bereitschaftspotential in jerky movement disorders. *Journal of Neurology, Neurosurgery, and Psychiatry* 83(12), pp. 1162–1167.

Verdellen, C.W.J. 2007. *Exposure and Response Prevention in the treatment of Tourette syndrome*. PhD Thesis, Radboud University.

Verdellen, C.W.J. et al. 2008. Habituation of premonitory sensations during exposure and response prevention treatment in Tourette's syndrome. *Behavior Modification* 322, pp. 215–227.

Wang, Z. et al. 2011. The neural circuits that generate tics in Tourette syndrome. *The American Journal of Psychiatry* 168, pp. 1326–1337.

Wilensky, A. 1999. *Passing for Normal: A Memoir of Compulsion*. New York: Broadway Books.

Williams, M.T. et al. 2011. Myth of the pure obsessional type in obsessive–compulsive disorder. *Depression and Anxiety* 28(6), pp. 495–500.

Woods, D.W. et al. 2005. The premonitory urge for tics scale PUTS: Initial psychometric results and examination of the premonitory urge phenomenon in youths with tic disorders. *Journal of Developmental and Behavioral Pediatrics* 26, pp. 397–403.

Woods, D.W. et al. 2009. The development of stimulus control over tics: a potential explanation for contextually-based variability in the symptoms of Tourette syndrome. *Behaviour Research and Therapy* 47, pp. 41–47.

Worbe, Y. et al. 2010. Repetitive behaviours in patients with Gilles de la Tourette syndrome: tics, compulsions, or both? *PLoS ONE* 5, p. e12959.

Xu, W., Zhang, C., Deeb, W., Patel, B., Wu, Y., Voon, V., Okun, M.S. and Sun, B. 2020. Deep brain stimulation for Tourette's syndrome. *Translational Neurodegeneration* 9, p. 4.