

Executive Function, Disability, and Agency

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Abstract

This paper considers how a number of particular disabilities can impact agency primarily by affecting what psychologists refer to as ‘executive function’. Some disabilities, I argue, could decrease agency even without fully undermining it. I see this argument as contributing to the growing literature that sees agency as coming in degrees. The first section gives a broad outline of a fairly standard approach to agency. The second section relates that framework to the existing literature which suggests that agency comes in degrees. The third section considers the psychological literature on executive function with a particular focus on how aspects of executive function contribute to agency. I then consider, in sections 4 and 5, two disabilities that have an impact on an agent’s executive function. Other disabilities will likely involve comparable impacts, although I don’t have time to explore additional disabilities in the present paper.

Introduction

Elsewhere, I’ve given an overview of a myriad of ways that disabilities impact moral agency, and have argued that different types of disabilities can impact agency in different ways (see Timpe (forthcoming)). In the present paper, I consider how a number of particular disabilities can impact agency primarily by affecting what psychologists refer to as ‘executive function’. In doing so, I hope to contribute to the growing literature that sees agency as coming in degrees. Some disabilities, I will argue, could decrease agency even without fully undermining it. I proceed as follows. In section 1, I give a broad outline of a fairly standard approach to agency. I don’t want to be too wedded to the details of any particular understanding of agency in the remainder of the paper, but I think that having this framework help anchor the subsequent discussion. Section 2 relates that framework to the existing literature which suggests that agency comes in degrees. In section 3, I briefly consider the psychological literature on executive function with a particular focus on how aspects of executive function contribute to agency. I then consider, in sections 4 and 5, two disabilities that have an impact on an agent’s executive function. Other disabilities will likely involve comparable impacts, although I don’t have time to explore more conditions in the present paper.

In approaching these issues, I want to meet what Manuel Vargas calls ‘the standard of naturalistic plausibility’: “on a standard of naturalistic

plausibility the account requires something that speaks in its favor beyond mere coherence with the known facts.... We seek a theory that has something to be said for it, in light of what we know about the natural world” (Vargas2013a, 58). Even though I’m not a naturalist (see Timpe and Jacobs (2015)), I still think that we ought to proceed with an eye toward the available evidence we have about how human agency works. Elsewhere I’ve endorsed what I call the the *Principle of Minimal Agential Realism*:

Make sure, when constructing a theory of agency, that the kinds of powers, capacities, and outputs posited by that theory could, for all we know, be had by us.

Given that my interest in the present paper is with the impact a number of disabilities have on agency, living up to the Principle of Minimal Agential Realism will require significant interaction with the relevant psychological and biological literatures on the disabilities in question. Each of the last two sections, therefore, begins with a summary of the current scientific understanding of the disability in question. I realize that the present scientific understanding of these conditions is fallible and might change, even in significant ways, as a result of further empirical work. If that were to happen, then some of the details of my argument would need to change as well. Given the nature of this project, that’s an inherent risk that I don’t think can be avoided.

As already mentioned, I want to stress at the outset that that different disabilities will impact agency in different ways and to different degrees. My goal is not to be exhaustive, but merely illustrative of how disabilities can impair agency in a way that might have broader application than considered here. One might wonder why *these* two particular disabilities were picked, rather than some others. I have picked them, in part, because they are diverse and, as I show below, impact agency in different ways. This same point could, of course, be illustrated by considering different disabilities. But given that there is no way to treat the complete range of disabilities that affect agency at present, a limited focus is required. In the conclusion, I mention a number of other disabilities that also involve executive dysfunction. The relevant impairments involved in them will overlap but not be identical with those discussed in sections 4 and 5 below. Future work will hopefully extend the present work to include a wider range of conditions.

Before beginning, let me also recognize a significant worry that accounts of disabled agency can go wrong in important ways. Prior to the rise of the self-advocacy movement in the late 1960s,

psychologists argued that people with retardation could have no sense of self and therefore were incapable of making decisions. They were automatically placed in institutions, which were set up so that doctors and other staffers could make all decisions. Those that lived at home relied on their mothers and father to make most decisions for them. [Only later was it widely] realized that with support and teaching people with mental retardation could make decisions of their own. (Shapiro (1994), 1956)

Even a cursory look at the history of how the disabled have been treated in the United States will show that significant harm has been done to those with disabilities by others who claim to speak for them. (See Shapiro (1994) for a discussion of a number of historical examples of this phenomenon.) I have tried very hard to be sensitive to this concern and to avoid contributing to that harm. It is important to note, for instance, that I'm not claiming that the disabilities considered here completely undermine agency, only that they can impair it or lessen its degree. This conclusion should not be used to deny anyone's agency.

1 An Outline of Agency

My goal in this section it is to give an overview of a theory of agency; I do not intend this section to provide a fully worked out or systematic account of agency. In part because of space constraints, and in part because I want what follows to be compatible with as wide a range of detailed theories of agency as possible, I'm going to speak largely in fairly broad strokes. Since later sections focus on how particular disabilities can impact executive function, the overview in the present section will focus particularly on those aspects of agency that will be most relevant for these discussions.

Agency is typically taken to involve the ability to respond to reasons and to guide or control one's behaviors in light of those reasons.¹ My use of 'behaviors' should be construed broadly, including not only bodily movements (e.g., reading a book aloud) but also mental acts (e.g., deciding how to get dressed). Bodily movements and mental acts are both behaviors in the relevant since and will be "agentive when ... sensitive to reasons, that is, able to adjust flexibly its means and goals to varying constraints or opportunities" (Proust (2013), 209f). Agency also involves an intellectual component given the need to be able to consider, evaluate, and weigh reasons. But, as the quotation above shows, intellectual activities will not be sufficient for agency—the individual must also be capable of 'adjusting her means and goals' in light of both the reasons she considers and the relevant details of the situation she finds herself in.

Volitions, which many take to be necessary for agency (see Ekstrom (2013) and Timpe (2013), chapter 2), will be sensitive to the reasons an agent has. Vargas, for instance writes of volitions as involving "means-end reasoning, the ability to formulate and execute action plans, and the presence of ordinary epistemic abilities, including a general capacity for some degree of foresight regarding the consequences of actions" (2013a, 201). According to leading neuropsychologist Muriel D. Lezak, "*Volition* refers to the complex process of determining what one needs or wants and conceptualizing some kind of future realization of that need or want. In

¹For specifically moral agency, a closely related claim is that responsible agency requires the powers of reflective self-control. As Kennett and Matthews describe it, reflective self-control "has two parts. First, the agent must have acquired moral concepts and be able to apply them to deliberate about what to do. Second, the agent must have the capacity to control their behavior in accordance with their deliberative conclusion" (Kennett and Matthews (2009), 328).

short, it is the capacity to formulate a goal or, at a lower conceptual level, to form an intention” (2004, 612). When an agent forms an intention to pursue some goal via some particular means, and executes that intention, we can say that the agent wills. While some philosophers are skeptical of faculties talk of the will, and the medieval moral psychology it brings to mind, one need not think that the will is a separate faculty. Rather, it may simply be that set of capacities that allows an agent, when successful, to act on the basis of those reasons and goals that she has. (See also Kane (1996), particularly chapter 2.) On this understanding, the will may simply be the capacity to form intentions that, when allowed to carry through to completion, ultimately move the agent to act in various ways. I exercise agency, for instance, when I form the intention to take a drink of water in order to both quench my thirst and collect my thoughts.

While there may be various exercises of agency that can be unintentional, my particular interest in this paper is with intentional agency. Intentions can be understood, at a general level, to involve plans for actions; they are executive attitudes toward planned agency. Alfred Mele differentiates a ‘standing intention’ from an ‘occurrent intention’. Occurrent intentions are those intentions which are “suitably at work at that time in producing relevant intentional actions or in producing items appropriate for the production of relevant intentional actions,” while standing intentions are general dispositions of a certain kind to form corresponding occurrent intentions (2009, 4; see also Mele (2007)). An agent, for example, may have the standing intention of being disposed toward intentionally drinking coffee in the afternoon; but that general disposition doesn’t always get triggered. It may be that the agent’s becoming aware of the smell of some richly roasted Arabica beans leads to an executive triggering of that standing intention in the formation of an occurrent intention: “I intend to make an Americano as soon as I finish typing the present sentence.”

It is possible for an agent to desire to perform a particular action without intending to do that action, though on some accounts one cannot intend to do an action without desiring to do it in some way.² Intentions can be “acquired without having been actively formed” (Mele (2013), 109) as in the case of intending to unlock one’s office door when one steps out of it momentarily. Particularly if the agent regularly unlocks her office door in similar circumstances, she might not have conscious awareness of forming this intention. As Bill Pollard has shown in his work on habitual action, “just because we don’t *consciously* intend, or *consciously* deliberate about, an action before we do it, it does not follow that we don’t intend, or don’t have reasons, to do it: intentions and reasons can be entertained unconsciously” (2013, 75).³

²The most obvious connection between desire and action is perhaps the ‘guise of the good’ thesis, which I discuss in Timpe (2013), chapter 2. Even though that discussion is primarily about volitions rather than intentions, much of what I say there about the connection with desire can also be seen as applying to intention.

³Later in the same chapter, Pollard argues that habit formation, like both intention and volition, can also be unconscious, but that this often still involves control over the formation of the habit (2013, 79). Mele also rejects that intentions need to be conscious; see 2009, chapter 2.

Attempts at agency are not always successful. There are a number of ways to fail to do something. One might refrain from doing some particular behavior, of course, as would be the case when an agent actively considers but restrains a standing disposition, preventing her from forming an occurrent disposition to do it. Or she might form the occurrent intention but later withdraw (or overrule) it before she's able to carry through with its execution (Bach (2013)). But one may also try (or attempt) to do it and still fail to succeed. This can happen, for instance, in cases where difficulty prevents an agent from doing what she thinks she should do and is, in fact, trying to do. (See, for instance, Kennett's discussion of 'orthonomous self-control' in 2001, 132.) In such a case, an agent may try to do something, and fail due to issues involving fatigue; ego-depletion; paralysis;⁴ or a failure in motor-planning to execute the needed bodily movements, particularly for complicated behaviors.

As already indicated, this account of agency is intended to be schematic rather than exhaustive.⁵ And while various scholars working in the philosophy of agency would certainly reject aspects of it, I don't think that any part of the above overview is particularly controversial. Even though the focus so far has been on agency in general and not morally responsible in general, the former is a precondition for the latter. Despite their extensive divergence, most extant accounts of morally responsible agency hold that whatever other conditions are also required for moral responsibility, agents must satisfy at least two conditions to be morally responsible: a control condition and an epistemic condition. The control condition is sometimes called the 'freedom-relevant condition' for moral responsibility or simply 'free will'. Elsewhere, I've suggested⁶ that we should think of free will as "the capacity or set of capacities of an agent to control her choices or volitions, the exercise of which is necessary for the agent to be morally responsible for those choices or volitions" (Timpe and Jacobs (2015), 320; see also Timpe (2013), particularly chapter 2, for my account of free will). In other words, an agent acts freely when he controls his actions in the way needed for him to be morally responsible for that action. And, via the epistemic condition, responsible agency will also require that the agent have the cognitive capacities required to meet the epistemic condition.⁷ Whatever the exact understanding of these conditions for morally

⁴One might will to move one's arm, for instance, but not be able to due to paralysis; see Hornsby (2013). The mismatch here is also related to the distinction between freedom of action and freedom of will; see Timpe (2012), 12-3.

⁵This picture is incomplete, in part, because it doesn't yet address the role that the emotions have to play in moral agency. That there is such a role is held by most contemporary accounts of moral agency, though here too the exact nature of that role depends upon the account in question. One standard view, roughly Aristotelian in nature, holds that the proper training of the emotions is crucial for responsible agency given the ability of the emotions to shape voluntary behavior. See, for instance, the discussion in King (2012), Adams (2009), and Roberts (2013). There will be numerous interactions between executive function abilities and emotional regulation, such as an agent's ability to prevent a strong emotion from causing inappropriate behavior in a social setting. I address these issues more fully in a companion paper.

⁶That is, I've suggested it along with my co-author, Jon Jacobs.

⁷See Timpe (2011) for my account of the epistemic condition. Though I don't explore it explicitly, the account I develop there should make it clear that the epistemic cognition also

responsible agency, they beyond what is required for mere agency.

2 Degrees of Agency

Recently, a number of philosophers have argued that agency comes in degrees.⁸ My hope is that the discussion in sections 4 and 5 will contribute further reasons for endorsing a graduated notion of agency.

Discussions of the degrees of agency typically come up in discussions of children, with a focus on responsible agency.⁹ Consider, for instance, this representative passage from Al Mele:

Normal parents eventually come to view their children as having some degree of moral responsibility for what they do. The word degree is important here. Normal four-year-olds are not as well equipped for impulse control as normal eight-year-olds, and they have less developed capacity for anticipating and understanding the effects of their actions. . . . Moral responsibility is very commonly and very plausibly regarded as a matter of degree. If young children and adults are morally responsible for some of what they do, it is plausible . . . that young children are not as nearly *as responsible* for any of their deeds as some adults are for some of their adult deeds. (2008, 271-4; emphasis added)

Jeanette Kennett also endorses a degreed concept of agency, with a particular focus on self-control:

Moral responsibility comes in degrees. The ordinary view implicitly recognizes both degrees of difficulty in the exercise of self-control (and indeed of judgement), and a distinction between those who are capable of synchronic self-control and those who must instead rely on diachronic techniques of control. Factors which impinge on the ease with which the capacity for self-control can be exercised mitigate responsibility. Some of those are obstacles to good judgement as well: for example, tiredness, emotional pressures, and lack of information. (2001, 182)

Though she doesn't make specific reference to children, contemporary developmental psychology would connect typical child development to an increase in self-control.

comes in degrees.

⁸For three recent papers examining and defending the claim that responsible agency does come in degrees, see Coates and Swenson (2013), Nelkin (2014), and Vargas (forthcoming). For present purposes, the differences between these views need not concern us. For accounts which reject degrees of responsibility, see Warmke (forthcoming) and Fischer (2006), 233. On Fischer's view, responsibility is a threshold concept, and it is blameworthiness and praiseworthiness that come in degrees. Much of what I say in the following paragraphs could be modified to fit Fischer's framework by talking about degrees of blameworthiness or praiseworthiness.

⁹There is empirical work that is relevant here. See, for instance, Baird and Fugelsang (2004), Blakemore and Robbins (2015).

Manuel Vargas suggests that cognitive impairments are one kind of paradigmatic case of less-than-full responsibility (forthcoming). An agent can be cognitively impaired either in her ability to recognize the relevant moral considerations or in terms of her ability to be properly motivated by those reasons that she does recognize. “The mitigating element in impairment cases is not the absence of the relevant faculty, but something like the diminution of the involved capacity, or perhaps, the difficulty in exercising the relevant capacity or power” (19). Though Vargas doesn’t examine disability in any detail, it should be easy to see how various disabilities—such as cognitive disabilities—could also impair responsible agency. Some individuals are able to consider a wider range of moral considerations than are others, and some individuals are more sensitive to the the relevance of moral considerations than are others. It is commonly held that responsible agency involves the ability to detect and weight reasons (see, among others, Vargas (2010) and Timpe (2013), 87-8). So if morally responsible agency depends on certain cognitive capacities and disabilities can impair those capacities—either by making it harder to recognize the reasons that the agent ought to be considering, or by making her unable to compare or weigh those reasons properly—then another way that cognitive and developmental disabilities could impact responsible agency is by lessening the degree of responsibility, even if it doesn’t entirely undermine it.¹⁰

D. Justin Coates and Philip Swenson argue in a recent paper that “the degree to which an agent is morally responsible depends on the degree to which she controls her action” (2013, 631). They ask us to consider two different agents—Marcia and Thomas—who each fail to pick you up from the airport after a trip after they have promised you that they would. The difference between the two cases, however, is the reason why they failed to fulfill their promise. Whereas Thomas failed to pick you up because he simply didn’t feel like doing it at the time since he would rather be watching television, Marcia was “suffering from serious but non-debilitating bouts of depression, and that she was having trouble getting off of the couch on the day she was supposed to pick you up” (629f). They suggest that we would (and should) treat the two agents differently, holding Marcia’s depression “to *mitigate* the degree to which she is responsible for that failure” (630). The idea seems to be this: whereas above we considered ways in which disability could impact an agent’s ability to recognize reasons, here we see a motivational failure. Maria is aware of the relevant factors but finds it difficult to form the intention to go to the airport because of her depression. (They also admit in passing that Marcia’s depression could also impact her cognitive ability to recognize and react to reasons as well; see 639). “A natural thought here is that Marcia is less responsible because it is more difficult (in some sense of ‘difficult’) for her to be motivated to get off the couch to come pick you up” (638).¹¹

¹⁰I address other ways that cognitive disabilities may impair agency in the section on ‘Intersectionality and Disability’ in Timpe (forthcoming).

¹¹Coates and Swenson understand the relevant sense of difficulty here to be one of ‘accessibility’, but the details of how they understand that relationship need not concern us at present. See Coates and Swenson (2013), 638-40. For a similar view, though one that critiques Coates and Swenson’s view on a number of points, see Nelkin (2014).

In a recent paper, Benjamin Kozuch and Michael McKenna argue that while it is commonly thought that mental illness often functions as a moral excuse, “the relation between mental illness and moral excuse is simply far more delicate than it is sometimes taken to be” (2015, 89). Rather than undermining morally responsible agency, they argue that in many cases, even when a mental illness plays a nontrivial causal role in an agent’s actions, mental illness instead diminishes the degree of responsibility. While I find much to agree with in their treatment of mental illness, I find their presentation of disability to manifest some of the oversimplifications that they argue against regarding mental illness.¹² Just as they claim that the focus on dramatic cases of mental illness skew the understanding of more mundane cases (103), so too with many discussions of disabled agency.

In the remainder of this paper, I hope to give reasons to think that many cases of disability that impact executive function should be seen as impairing rather than undermining agency. If, as those mentioned above have argued, responsible agency comes in degrees because responsibility comes in degrees, disabilities could impact responsibility by making it harder for an agent to do the morally right thing. This is, as we’ll see, one of the lessons that I think reflection on disabled agency can show us. The kinds of impairments of executive function that I explore in greater degree below might mitigate the degree of one’s responsibility. In a recent paper, Jesús Aguilar and Andrei Buckareff develop a model of agency that I think shows the possibility of a more direct impact of certain disabilities on agency. They argue for a “gradualist metaphysics of agency” according to which “lots of things can be truthfully described as agents by exercising varying degrees of agency” (2015, 31). Thus, on their view, agency itself—not just morally responsible agency—comes in degrees. In short, Aguilar and Buckareff develop a teleological account of agency. What makes a thing an *agent* is that its causal powers are *directed* to something beyond itself. While they are neutral in this paper about the kind of causal relations involved in free will, I think their view in the present paper can be combined with an agent-causal account of free will of the sort that I’ve endorsed elsewhere (see Timpe and Jacobs (2015)).

If one adopts such a gradualist account of agency, the connections between those capacities involved in agency and the ways they, or their operation, might be impaired by various disabilities will be even more obvious than I think they already are. One way of understanding the discussion of executive functioning in the next section is as giving support from current empirical research on executive function to further support the claim that agency is a degreed concept.

3 Executive Function and Agency

When one turns toward the scientific literature on agency, much of it focuses on what psychologists refer to as ‘executive function’, a concept which was first introduced into the psychological literature in the 1970s (Goldstein et al. (2013)). A difficulty which confronts anyone interested

¹²See, for instance, their discussion of Lenny from Steinbeck’s *Of Mice and Men* in Kozuch and McKenna (2015), 92.

in how disabilities can impact agency by exploring how disabilities impact executive function is the “failure to find consensus on a general definition” (Borkowski and Burke (1996), 244).¹³ For present purposes, I’ll take executive function to be an umbrella term that encompasses the following abilities:¹⁴

- agential planning
- initiation of action, particularly for goal-directed behavior
- working memory¹⁵
- self-monitoring¹⁶
- behavioral self-regulation (including restraint and inhibition)
- emotional self-regulation
- attention/focus
- selective attention¹⁷
- effective performance.

Even this list is incomplete, as over 30 particular abilities have been included within the scope of executive function (Goldstein et al. (2013), 4).¹⁸ Michael Lezak and colleagues write that “some techniques for examining executive functions involve so many of them that they defy classification

¹³Similarly, “There continues to be no consensus definition of executive functions” (Senn et al. (2004), 445); see also Brier (2015), 2 and Chung et al. (2014), 13. Chung et al. (2014) also contains a useful discussion of the physiology underlying many tasks associated with executive function. Weyandt et al. (2014) address how these various approaches to the nature of executive functioning make it difficult to evaluate executive function impairments in a clinical setting; see particularly 81-83. Some of the difficulties in measuring executive function competence are discussed in Gioio et al. (2000).

¹⁴For some of the relevant psychological literature on executive function, see Brier (2015), 2-3; Smidts et al. (2004), 386; Sample (2008), 75-6; Siegler (1991), Lezak et al. (2004), 611. As Weyandt et al. (2014) makes clear, “a universally accepted definition of EF does not exist, and many have criticized the broad definitions of the construct” (69).

¹⁵Working memory allows “information to be held after sensory input so that a course of action can be planned” (Williamson and Allman (2011), 55). According to Baddeley (1992), working memory is central to executive function in virtue of its role in controlling attention.

¹⁶Self-monitoring refers to the processes that individuals “use to check in and note if their in depended behavior is matching their actual behavior” (Brier (2015), 5). Williamson and Allman include the following under self-monitoring: “self-appraisal, agency, autobiographical memories, prospection, and theory of mind” (Williamson and Allman (2011), 123).

¹⁷Selective cognitive attention also illustrates the intersectional nature of disabilities, even with a focus restricted to those impacting executive function. See Timpe (forthcoming) for a discussion of the intersectional nature of disabilities in how they impact agency.

¹⁸On the above construal of executive functioning, it’s hard to delineate when cognitive capacities are and are not related to executive function. However, the intersectional analysis I give doesn’t require there to be to be a hard and fast line between executive dysfunction and cognitive impairments. Goldstein *et al’s* introduction also includes a useful review and comparison of useful executive function definitions and models in the scientific literature. I’m inclined to reject those theories of executive functioning, such as Baddeley *et al’s*, which view executive functioning as a single unified system; see Baddely et al. (1996). Finally, if—as some models of executive functioning suggest—management of the emotions also falls within the scope of executive functioning, the degree of disabilities’ intersectionality will increase. For relevant psychological research on the connections between the emotions and executive function, see Lezak et al. (2004), chapter 19.

under any one of the subdivisions” (2004, 636) and it seems from the literature that the same is often true of the functions themselves—it’s not always clear how precisely they can be delineated and differentiated.¹⁹ Even if the various abilities involved in executive function are separable, they will clump together in how they help ground agential capacities. This, of course, will mean that disabilities’ effects on executive function will also involve imprecise differentiation at times.

The exact contribution of these various abilities included in executive function to an account of agency will depend on the exact specifics of that account. While the present discussion is not intended to be exhaustive, and might need to be modified if one adopts a more detailed account of agency, let me note a number of connections between these abilities and the outline of agency in section 1. And the fact that these abilities are scalar further supports the connection to a gradualist account of agency as endorsed in section 2.

Self-monitoring and attention/focus could all affect an agent’s successful reasons-responsiveness and the means-end reasoning involved in volitions. Those abilities that involve or affect motivation will be relevant to the agent’s ability to be motivated by those reasons that she detects. Behavioral self-regulation (which is related to the agent’s abilities to engage in restraint and inhibition) could not only those aspects of agency already noted in this paragraph, but also the ability to form long-term intentions and to inhibit those actions that might get in the way of or undermine those intentions.

One reason that executive function impairment will have such wide-ranging impact is that the abilities involved in executive function are thought to be non-modular (see Hirstein (2011); Hirstein and Sifferd (2011), particularly section 3.2; and Levy (2014)). Since executive functions get their input from such a wide range of inputs (e.g., proprioception, perception, cognitive, memory) and a similar wide-reaching outputs (e.g., attention, memory formation, decision making, action planning), these abilities play a critical role in organizing complex actions and allow the agent to sort through various possibilities for behavior in a reflexive way. This helps with agential self-control across a wide range of domains, and well as over diachronically extended patterns of action (which is another reason that the self-monitoring role of executive function is important).

Difficulties with executive functioning issues are not considered by psychologists to be a disability in their own right; however, it should be obvious from this list that certain kinds of disability can significantly

¹⁹Peterson and Welsh (2014) suggests that there may be two kinds of executive tasks: cool (“the goal directed, future-oriented skills such as planning, inhibition, flexibility, working memory, and monitoring that are manifested under relatively decontextualized, nonemotional, and analytical testing conditions”) and hot (“goal-directed, future-oriented cognitive processes elected in contexts that engender emotion, motivation, and a tension between immediate gratification and long-term rewards” (45)). Research on the latter is only about a decade old, and Peterson and Welsh argue that it’s not clear from the existing data that whether the apparent difference between cold and hot executive functions are really explained by emotion or by some other factor; see 60f. If there is an important difference here between cool and hot executive functions, this would lead to another kind of intersectionality between executive functions and emotion. See section 4.3 below.

impair those abilities. According to some researchers, the most significant problems with executive functioning impairment are “impaired capacity to initiate activity, decreased or absent motivation (*anergia*), and defects in planning and carrying out the activity sequences that make up goal-directed behaviors” (Lezak et al. (2004), 36). They continue by noting that executive functions can break down at any step of agency (611). Executive function impairment, they argue, can even be more damaging for autonomous agency than are cognitive impairments:

So long as the executive functions are intact, a person can sustain considerable cognitive loss and still continue to be independent, constructively self-serving, and productive. When executive functions are impaired, the individual may no longer be capable of satisfactory self-care, or performing remunerative or useful work independently, or of maintaining normal social relationships regardless of how well-preserved the cognitive capacities are—or how high the person scores on tests of skills, knowledge, and abilities. Cognitive deficits usually involve specific functions or functional areas; impairments in executive functions tend to show up globally, affecting all aspects of behavior. However, executive disorders can affect cognitive functioning directly in compromised strategies to approaching, planning, or carrying out cognitive tasks, or in defective monitoring of the performance. (35)²⁰

It is not clear to me how one would go about evaluating the kind of comparative claim which suggests that executive defects *usually* impair agency more than do cognitive defects. But I think that Lezak *et al.*'s work is right that executive function impairment often have a profound and systematic influence on agency.²¹

In an earlier paper, I noted a number of such impacts—by causing cognitive or developmental disabilities; by hampering agential planning; by making it harder for the agent to engage in self-distraction, which is need for delaying gratification; etc. . . —though my discussion of them was fairly brief. The present section has attempted to expand on these impacts in general. However, in subsequent sections, I explore in greater detail how specific disabilities impact the abilities involved in executive function. But first, as indicated above, I must first spend some time with the nature of the disabilities I want to focus on.

²⁰Commenting on a previous draft, a referee inquired into what differentiates executive function abilities from cognitive abilities beyond what Lezak *et al.* indicate in this quotation. This is a question that, so far as I can tell, isn't settled among those psychologists working on executive function. Furthermore, given that I'm not trying to draw sharp boundaries upon the impact of disabilities on agency in this paper, porousness about the exact boundaries is acceptable. A complete discussion of disability and agency would treat both cognitive and executive function impairments (as well as their intersection), but such a comprehensive treatment goes beyond the present paper.

²¹The preceding quotation also gets at the issue of the intersectionality of disability, particularly at the end. More on this below.

4 Narcolepsy

My goal in this section is to outline a number of ways that narcolepsy can impact agency. However, before doing so, I begin by presenting the nature of and etiology of the illness.

4.1 Narcolepsy and its Symptoms

Narcolepsy defined by the International Classification of Sleep Disorders as a lifelong neurological disorder which involves “excessive sleepiness that typically is associated with cataplexy and other REM-sleep phenomena” (38). It is of unknown etiology and there is currently no cure. Narcolepsy is typically characterized by a tetrad of symptoms:

1. excessive daytime sleepiness (*ESD*) regardless of the amount or quality of nocturnal sleep,
2. cataplexy,
3. hypnagogic or hypnopompic hallucinations, and
4. sleep paralysis (which often accompanies hypnagogic hallucinations).²²

Of these, *EDS* is the most universal symptom and is rated as most severe of life-impacting by those who have narcolepsy (Alaia (1992), 11). Cataplexy is the second most common symptom (Kotagal and Paruthi, 56). Narcolepsy with and without cataplexy are typically differentiated (see Mignot (2010), 3 and Bayard et al. (2012), which claims that the two

²²The diagnostic criteria in the current revision of the *The International Classification of Sleep Disorders* are as follows:

- (a) The patient has a complaint of excessive sleepiness or sudden muscle weakness.
- (b) Recurrent daytime naps or lapses into sleep occur almost daily for at least 3 months.
- (c) Sudden bilateral loss of postural muscle tone occurs in association with intense emotion (cataplexy).
- (d) Associated features include:
 - i. Sleep paralysis
 - ii. Hypnagogic hallucinations
 - iii. Automatic behaviors
 - iv. Disrupted major sleep episode
- (e) Polysomnography demonstrates one or more of the following:
 - i. Sleep latency less than 10 minutes
 - ii. REM sleep latency less than 20 minutes and
 - iii. An MSLT that demonstrates a mean sleep latency of less than 5 minutes and
 - iv. Two or more sleep-onset REM periods
- (f) HLA typing demonstrates DQB1*0602 or DR2 positivity.
- (g) No medical or mental disorder accounts for the symptoms.
- (h) Other sleep disorders (e.g., periodic limb movement disorder or central sleep apnea syndrome) may be present but are not the primary cause of the symptoms.

A clinical diagnosis requires either (b) and (c), or (a) and (d) and (e) and (g). See *The International Classification of Sleep Disorders* (2001), 42. According to Hale, “given the variable phenotype for narcolepsy, there is no gold standard for diagnosis. Many of the symptoms overlap with non-narcoleptic patients” (Hale (2010), 47).

forms of narcolepsy have different etiologies), but even this is presently contested as “no consensus has yet been reached on whether cataplexy is an entirely separable and unique symptom or, alternatively, the intrusion of REM sleep muscle atonia into wakefulness” (Sinton (2010), 33). Most of my discussion will focus on narcolepsy with cataplexy, though some of what I say will also apply to non-cataplexic varieties if it turns out that they are distinct. Only 36% of clinically confirmed cases of narcolepsy exhibit the complete tetrad of symptoms (Zamarian et al. (2015), 45). Other symptoms may include disturbed nocturnal sleep, REM sleep behavior disorder, or automatic behavior (i.e., “automatic continuation of activity without memory of the event” (Dauvilliers and Plazzi (2010), 79)), and sexual dysfunction.

As mentioned above, cataplexy is not the most common symptom of narcolepsy, even though it occurs in approximately 60–90% of patients diagnosed with narcolepsy (Kotagal and Paruthi, 57). Nor is it the usually the first clinical symptom, as it sometimes doesn’t manifest until 30 years after the onset of *EDS* (Dauvilliers and Plazzi (2010), 78; The International Classification of Sleep Disorders (2001), 40). It is, however, more frequently observed close to the onset of the disease and, like hypnagogic hallucinations and sleep paralysis, usually decrease over time (Kotagal and Paruthi, 61). Cataplexy involves episodic bilateral muscle weakness without a loss of consciousness; during such episodes the individual is unable to move despite retaining consciousness. “The duration of each episode is generally less than 2–3 min, but sometimes a series of episodes may be clustered together in a sequence” (57). Cataplexic episodes usual occur in response to emotional triggers, especially positive ones like laughter or surprise, but also less commonly in response to negative emotions like fright or anger (Dauvilliers and Plazzi (2010), 78). During cataplexic episodes, the individual remains conscious, memory is not impaired, and respiration is normal (The International Classification of Sleep Disorders (2001), 38). The degree of muscle atonia ranges in severity from head droop, facial sagging, or jaw drop (which may not even be noticed by others) to buckling of the knees and, in extreme cases, a complete postural collapse (39).

Hypnagogic or hypnopompic hallucinations are experience by approximately 66% of narcoleptics. They are often auditory or visual in nature and most frequently occur when transitioning into our out of sleep. Finally, sleep paralysis refers to “an inability to move one’s head or limbs during the transition to sleep or wakefulness” (Hale (2010), 47) and, like hypnagogic or hypnopompic hallucinations, often accompany cateplexic episodes. Sleep paralysis occurs in approximately half of the cases of narcolepsy (Dauvilliers and Plazzi (2010), 79).

Narcolepsy is fairly rare, with a prevalence estimated to be approximately 0.05% of the global population (Kotagal and Paruthi, 55f; see also Zamarian et al. (2015), 45). It’s exact etiology is unknown, though “a combination of genetic predisposition and acquired stress seems to trigger most cases of narcolepsy” (Kotagal and Paruthi, 58; see also Mignot (2010)). Hale suggests that there is most likely a genetic susceptibility to narcolepsy which is then triggered by environmental factors, as indicated by the fact that there is over twice as high a prevalence among individuals

born between March and September in a study using data from France, Canada, and the US (Hale (2010), 51). In most cases, it has an early age of onset, typically during the second decade of life and with a median age of onset of 16 (Attarian (2010), 69).

4.2 Impact on Agency

How might narcolepsy impact agency? Cataplexic episodes are the most obvious way—the inability to move while conscious is a temporary impairment of agency. However, I think the impact of narcolepsy on agency will extend significantly beyond temporary periods of conscious immobility, in part because narcolepsy impacts all aspects of an individual's life—personal, social, education, vocational. As one leading narcolepsy researcher puts it, “people with narcolepsy are globally impaired often for much of the day and throughout their lifetimes” (Pollack (1992), 147). *Zambian et al.* refer to narcolepsy as “severely disabling” (2015, 45). What Pollak has in mind here is the inability to stay awake and alert due to the disease, which can impact not only cognitive performance but a number of facets of executive function as well. *Zambian et al.* point out the connection, for example, between EDS and “attention and executive function deficits” (45; see also Bayard et al. (2012), 1). In a study of children, narcoleptics aged 4–18 “showed significant problems in behavior, emotional state, quality of life, educational progress and family impact among children with narcolepsy when compared to healthy age and gender-matched controls” (Panossian and Avidan (2010), 111). Fatigue, especially chronic fatigue, can impair nearly every ability involved in executive function.

Current research also suggests an impact of narcolepsy on memory. Diminished working memory has been linked, in turn, to executive function problems via a number of different tests (see Lezak et al. (2004), 358ff). And narcolepsy researchers have connected working memory impairments with “difficulties in tasks that put high demands on monitoring and manipulation of information (i.e., in tasks that require a high degree of executive control)” (Zamarian et al. (2015), 46). These results are particularly associated with tasks requiring flexibility under time pressure. In another study, nearly 60% of narcoleptics reported that their memory had worsened since the onset of their disease (Ferrans et al. (1992), 30). Some studies have found conflicting impact of narcolepsy on both short- and long-term memory in laboratory settings. Broughton suggests that the artificial setting of some of these tests might explain why the memory effects aren't consistently found:

It appears that narcoleptics have an ability to ‘rally’ and overcome their pathological drowsiness for at least short periods of time in a laboratory setting. . . . These negative results do not indicate that no memory deficits are present in narcolepsy, but rather that they are entirely attributable to drowsiness and so are potentially reversible, rather than representing permanent affects. (Broughton (1992a), 34; see also Broughton (1992b), 40)

Despite this ambiguity with respect to tests in the lab, researchers have

consistently found memory deficits with regard to “prolonged, monotonous tasks requiring sustained attention” (Smith et al. (1992), 104). For my present purposes, even this more limited impairment shows that narcolepsy impacts agency. It is largely due to these significant affects on agency that approximately 15% of narcoleptics are permanently disabled by their disease (Alaia (1992), 6).

Working memory has also been correlated with decreased performance in complex agential tasks, such as problem solving (Carpenter et al. (2000), 195). Since, on some understandings of the nature of executive functioning, the abilities to construct and carry out cognitive strategies can be understood as falling within the bounds of executive functioning, the impact of both fatigue and impairments of working memory on successful cognitive strategies will also likely be related to narcoleptic impairment (see Carpenter et al. (2000), 197 and Zamarian et al. (2015), 50; for another discussion of working memory and ‘strategy choice’, though one focusing more on cognitive disability than executive function, see McLean and Hitch (1999)).

4.3 Intersectionality

Finally, before leaving behind narcolepsy, let me briefly consider ways that having narcolepsy could intersect with other challenges to agency. Here, I’m borrowing the concept of ‘intersectionality’ from feminist theory, though putting it to slightly different use (Carastathis (2014)). Ann Garry describes intersectionality as “a framework or strategy for thinking about issues . . . to consider their mutual construction or at least their intermeshing (if these are different)” (2012, 517) and it is precisely in this way that I shall use the term. Just as a number of dimensions of social oppression can intersect so the total oppression she experiences is greater than the sum of the individual oppressions, I want to suggest that various disabilities can intersect so that the total impact on agency is greater than the sum of the individual disabilities. In both contexts, intersectionality should be thought of as as multiplicative rather than merely additive. In a parallel way to how various kinds of oppression or privilege may intersect to amplify their individual magnitudes, I want to suggest that disabilities can also intersect in their impact on executive function’s role in agency.²³

Narcolepsy’s impact on agency might intersect with other disabilities and illnesses in a number of ways. First, the existing research shows a number of correlations between narcolepsy and other conditions that can also affect agency. For instance, narcolepsy is comorbid with, among other things, cognitive disorders and psychosocial impairment, various psychiatric disorders such as anxiety and schizophrenia (Panossian and Avidan (2010)). There is a higher frequency of depression, anxiety, and psychopathology among narcoleptics (Alaia (1992), 2-3; Zamarian et al.

²³A referee has suggested the ‘comorbidity’ might be a more appropriate framework than in intersectionality. Comorbidity is certainly important for disability; but the point being made at present isn’t just that there may be two or more disabilities that are simultaneous present. Rather, the point of the present section is the stronger claim that how two comorbid disabilities (or two comorbid impacts of disability) affect agency is more complicated than merely being additive.

(2015), 45; and Merritt et al. (1992)). In one study, 49% of narcoleptics experienced depressive symptoms, a percentage that is significantly higher than for the general population (Merritt et al. (1992), 56).

Some researchers have also linked narcolepsy with particular impairments in cognitive functions. Consider, for example, the following:

A study by Ohayon et al. has found significant impairments in attention and concentration, delayed recall, and difficulty with orientation to persons (recalling names or recognizing acquaintances), among narcolepsy patients younger than age 45 as compared to healthy controls. Narcolepsy patients older than age 45 exhibited cognitive difficulties that were significantly worse than age-matched healthy controls across multiple areas, including attention and concentration, praxis, delayed recall, orientation to persons, temporal orientation, and prospective memory. Because sleepiness alone may account for some degree of cognitive impairment, the authors subsequently controlled for sleepiness (as measured by the Epworth Sleepiness Scale scores) as well as for physical health, use of psychotropic medications, age and sleep apnea; they again found that narcolepsy was still associated with a significantly higher risk of attention and concentration deficits and difficulty with prospective memory. Thus, while the majority of cognitive dysfunction found in narcolepsy appears to be secondary to EDS, some degree of cognitive impairment seems directly related to the underlying disease pathophysiology and is independent of age or degree of sleepiness. Various investigators have had conflicting results in examining objective cognitive impairment in narcolepsy, and have found no evidence for specific cognitive deficits, although the majority of patients in these studies did have subjective cognitive complaints. . . . These results suggest that rather than a problem in a specific cognitive area, narcolepsy patients appear to exhibit a limitation or reduction in cognitive processing resources. (Panossian and Avidan (2010), 110.)

Finally, narcolepsy correlates with decreased social support, which in turn makes it harder for those suffering from executive function impairments to compensate for those impairments via social scaffolding. Narcoleptics tend to score very low in terms of social support. A third of narcoleptics report that their illness contributes “moderately or a great deal to social isolation” (Ferrans et al. (1992), 28) and that they have difficulty making or retaining close friends (Ferrans et al. (1992), 31). According to another scholar, “these individuals feel their situation is beyond the ability of their support system to comprehend. The failure to find a relationship between social support and any of the life-effect or affect measures suggest that social support, as a coping strategy for those aspects of their lives related to narcolepsy, is not available” (Alaia (1992), 17).²⁴

²⁴Alaia goes on to admit that it is possible that “the negative life effects of narcolepsy are such that perceptions of social support are distorted. This would support Barrera’s (1986)

Though I don't have the space to explore the interconnections at present, there's also some research connecting narcolepsy with depression, the emotions involve in which might also impact agency. (For a discussion of the connection between depression and narcolepsy, see Smith et al. (1992), 112.) There may also be intersectionality between narcolepsy and Parkinson's disease, discussed below. As Karacan *et al.* note, "one of the more widely accepted theories of the etiology of narcolepsy is that it results from widespread under-secretion of dopamine in the central nervous system with hypersensitivity to acetylcholine. . . . It is well known that patients with Parkinson's disease, who suffer from lack of dopamine in basal ganglia, frequently have sexual dysfunction" (1992, 87). More work, both philosophic and empirical, may need to be done to further explore these particular potential intersectionalities. But the present section should be sufficient to show how narcolepsy can have a negative impact on agency.

5 Chromosomal Abnormalities: Deletions

In this section, I consider a particular set of disabilities caused by chromosomal abnormalities and their affect on agency via executive functioning. Chromosomal abnormalities range greatly; entire chromosomes, or segments of them, may be duplicated, missing, translocated, or otherwise altered. Even if only a small amount of genetic information is impacted (or, in the case of a deletion, missing), there can be large impacts over a wide range of abilities in the agent since "the expression of a gene is altered ... in a complex way that cuts across multiple functional systems" (Swanson (2012), 273).²⁵ In fact, contemporary neuroscientists think "quite possibly there is no direct correspondence between the organization of functional systems in the brain and the regulatory networks that determine global patters of gene expression" (273). I cannot hope to do any justice to all chromosomal abnormalities in this section given their diversity. I shall focus my attention on deletions, and in particular one particular kind of deletion, that found in 2p15-16.1 Microdeletion Syndrome. But the kinds of considerations I raise will also have applicability to other chromosomal abnormalities as well.

One of the difficulties with examining the agency of individuals suffering from disabilities due to malformation syndromes is the range of effects among those who suffer from the same underlying genetic abnormality. As reported in *Smith's Recognizable Patterns of Human Malformation*, "except for such nonspecific general features as mental deficiency and small stature, it is unusual to find a given anomaly in 100% of patients with the same etiological syndrome" (Jones (2006), 3). This is true not only of various physical phenotypical characteristics, but also in terms of behaviors and abilities. Consider cognitive functioning. Most individuals

view that perceptions of social support decrease as life stresses increase. He also found that depression had a negative effect on perceived social support, and this was demonstrated by Prociano and Heller (1983)" (Alaia (1992), 17).

²⁵Since there is evidence to think that various executive functions are themselves depended on multiple brain regions (see Carpenter et al. (2000), 197), it should not be surprising that deletions can have a dramatic impact despite their comparatively small size.

with a number of deletion syndromes—such as 3p Deletion Syndrome, 4p Deletion Syndrome, 4q Deletion Syndrome—have moderate to profound mental retardation, depending on the syndrome. In contrast, individuals with 13q Deletion Syndrome range from mild to severe mental retardation (56) and those with 11q Deletion Syndrome vary from moderate mental retardation to normal intelligence (54).²⁶ Similarly, some individuals with 18q Deletion Syndrome have moderate mental retardation while others are “not obviously debilitated” (62).

One reason for this the range of impact is that “the expression of a particular gene typically will be complex and multifunctional” (Swanson (2012), 274); another is that the degree of impairment is a function of more than just genetics; it depends on epigenetics, early intervention, family dynamics, etc. . . . Even when considering a particular chromosomal abnormality, we ought not think it’s impact on all who have the same condition will be identical.

5.1 2p15-16.1 Microdeletion Syndrome

Of the various various deletion syndromes, I focus here on 2p15-16.1 Microdeletion Syndrome. This syndrome involves a deletion on the short (p) arm of chromosome 2; the chromosome has one breakpoint in band 15 of the p arm and another in band 16, and the genetic material usually contained is simply missing. The deletion usually occurs in only one copy of the chromosome (de Leeuw et al. (2008) and Piccione et al. (2012)). The deletion involved is so small that it can only be “found using a molecular technique such as multiplex ligation-dependent probe amplification (MLPA) and array comparative genomic hybridization (array-CGH) or by cytogenetic fluorescent in situ hybridization (FISH) techniques using fluorescent DNA probes targeted to gene markers within the involved chromosome 2p15p16.1 region” (*2p15p16.1 Microdeletion Syndrome* (2014), 2). Even though only a small amount of genetic information is missing, as with other deletion syndromes, there can be large impacts over a wide range of features in the agent. The exact etiology of 2p15-16.1 Microdeletion Syndrome is unknown, but in most cases it’s believed to be caused by a *de novo* mutation. The emerging phenotype of individuals with the syndrome includes a number of physical affects (e.g., microcephaly, vision problems, kidney abnormalities); speech impairments; gross and fine motor control issues; and cognitive and developmental disabilities (*2p15p16.1 Microdeletion Syndrome* (2014), 4 and 7).

Individuals with this syndrome are often not able to perform age-appropriate behaviors for one or more reasons relating to executive function. One problem is the dissociation between intention and action, which can fail due to the numerous steps needed in the planning process. This need not be a problem in intention formation but may instead be a defect “of translation from thought to action” (Lezak et al. (2004), 626).

²⁶In 11q Deletion Syndrome, the level of cognitive impairment correlates with the size and location of the deletion. In other syndromes, e.g. 4p Deletion Syndrome, “the phenotype does not differ on the size of the deletion, which can vary from almost one half of the short arm to so small as to be cytogenetically undetectable” (Jones (2006), 36).

Relatedly, they typically also experience ego-depletion at a greater rate than non-disabled peers, especially in contexts involving task-switching. Developmentally there is a delay in agential planning and behavioral self-monitoring and regulation. A particularly noteworthy effect of the syndrome in the present context involves agential focus. Many suffer from dyspraxia, a developmental condition that makes it hard to stay on task. While individuals with 2p15-16.1 Microdeletion Syndrome are sometimes capable of unusually extended periods of focus on preferred tasks, in general their decreased ability to focus on demanding tasks has systemic implications:

Although attention, concentration, and tracking can be differentiated theoretically, in practice they are difficult to separate. Purely attentional defects appear as distractibility or impaired ability for focused behavior, regardless of the patient's intention. Intact attention is a necessary precondition of both concentration and mental tracking activities. Concentration problems may be due to a simple attentional disturbance, or to inability to maintain a purposeful attentional focus or, as is often the case, to both problems. At the next level of complexity, conceptual tracking can be prevented or interrupted by attention or concentration problems and also by diminished ability to maintain focused attention on one's mental contents while solving problems or following a sequence of ideas. (Lezak et al. (2004), 349)

It should not be surprising that self-control and self-management are more difficult as a result.

5.2 Intersectionality

As seen earlier in the discussion of narcolepsy, various impairments caused by 2p15-16.1 Microdeletion Syndrome can intersect in ways that magnify the agential impact of the disability.

2p15-16.1 Microdeletion Syndrome typically involves development delay, and it also has an impact on cognitive functioning, with sufferers ranging from mild to severe cognitive impairment.²⁷ While cognitive impairment has its own direct impact on agency, it also impacts successful executive functioning. Individuals with a cognitive impairment might not

²⁷There are a number of different ways to differentiate degrees of cognitive impairment. According to one influential system developed by Grossman, the classification according to IQ is as follows:

- mild: from 50/55 through 70
- moderate: from 35/40 through 50/55
- severe: from 20/25 through 35/40
- profound: under 20/25

For a history and discussion of a number of such rankings, see Richards et al. (2015), chapter 2.

be able to understand the exact ways that they need to engage in self-distraction and self-determination strategies to overcome the impact their disability has on self-control. The planning required for these kinds of tasks come with a high cognitive demand. Such an impairment could also effect an agent’s ability to engage in self-monitoring to make sure that her actions are appropriate for the present situation. Consider, for example, the following passage from Lezak *et al*:

The identification and organization of the steps and elements (e.g., skills, materials, other persons) needed to carry out an intention or achieve a goal constitute planning and involve a number of capacities. In order to plan, one must be able to conceptualize changes from present circumstances (i.e., look ahead), deal objectively with oneself in relation to the environment, and view the environment objectively. . . . The planner must also be able to conceive of alternatives, weigh and make choices, and entertain both sequential and hierarchical ideas necessary for the development of a conceptual framework or structure that will give direction to the carrying out of a plan. Good impulse control and reasonably intact working memory are also necessary. (2004, 614)

If the difficulty or demandingness of a task can decrease one’s agency, we see another potential feature of the cognitive impairment involved in 2p15-16.2 Microdeletion Syndrome.

But Francis and Silvers point out another cognitive intersectionality for responsible agency: “Responsibility is about control in the sense of self-determination: agents must be in control of determining their thoughts about the good rather than allowing an idea of the good to take control of them. But impairment of executive capacity often is an element of cognitive disability. Some people with cognitive disabilities do not have this capacity of reviewing their idea of the good to assess whether it is a proper aim for them, and some others have the power only to an attenuated degree” (Francis and Silvers (2010), 237). If an agent is not capable of understanding moral categories like ‘good’ or ‘bad’, she won’t be able to control her actions to realize (or avoid) those categories.²⁸

According to a leading view of the nature of the human nervous system—the ‘four systems network model’—there can be no separation of the agent’s cognitive structure from her self-control of bodily actions. On this model,

behavior is a direct function of motor system input. The motor system is controlled in turn by three classes of inputs—from the sensory, behavioral state [which modulates between wakefulness and sleep processing], and cognitive systems. Direct sensory inputs to the motor system mediate reflex behaviors. . . , inputs from the cognitive system mediate voluntary behavior,

²⁸It’s not clear to me that the cognitive impairment involved with 2p15-16.1 Microdeletion Syndrome is sufficiently severe that the individual with this condition would not be able to grasp moral categories at all; thus, it doesn’t seem to amount to an exemption from moral agency. For a related discussion, see Shoemaker (2015).

and inputs from the third system mediate state control influences. (Swanson (2012), 125)

If something like this view is correct, we can further see how the deletion would impact fine- and gross-motor tasks, self-care, and other agential functioning.

Finally, there is a significant correlation between 2p15-16.1 Microdeletion Syndrome and Autism Spectrum Disorder, perhaps greater than 20% (*2p15p16.1 Microdeletion Syndrome* (2014)).²⁹ One frequent consequence of ASD is social isolation; but social impairments themselves can have a large impact on agency. Lavoie is one psychologist who’s documented how impairments of executive function can have “a profound negative impact on a child’s social skill development” (2005, 9). Executive function impairments have also been linked directly with difficulty forming and maintaining social relationships (Lezak et al. (2004), 37). These difficulties will make it harder for certain disabled individuals to get the kind of social assistance that could help with offset or overcome their impairments.

According to Levy, if a person “is to successfully bring themselves to the point where they have a sufficiently unified self to engage in the project of further self-making, they will require help. At minimum, the child requires that caregivers ensure that there is the opportunity successfully to deploy basic self-unification strategies (2007, 217). Such self-unification involves cognitive, self-direction, and social abilities. And the kind of social-scaffolding that can improve our agency require a moral ecology that often those with social impairments struggle with:

If environmental manipulations can be used to weaken self-control, they can also be used, by ourselves or others, to strengthen it. We saw above that self-control depends, in important part, upon a set of skills that children acquire as they mature; for instance, skills of self-distraction. . . . We must each learn to delay gratification, and to sacrifice immediate, smaller rewards for later, larger, rewards. In doing so, we unify ourselves. But successfully achieving this self-unification requires that the child’s environment be structured properly. (Levy (2007), 216)

The environmental structure that Levy refers to here can be understood as one important part of what Manuel Vargas refers to as ‘moral ecology’ in *Building Better Beings*. In setting up the idea of moral ecology, Vargas rejects both atomism—the view that “free will is a non-relational property of agents, that is, it is characterizable in isolation from broader social and physical contexts” (2013a, 204)—and monism, the view that “there is only one natural power or arrangement of agential feature that constitutes free will or the control condition on moral responsibility” (205). I think Vargas is right that at least one, and often both, of these is assumed by many philosophical accounts of agency. Vargas argues that both of these assumptions are “at odds with the emerging picture of agency in

²⁹Many also suffer from ADHD or ADD. The shortened attention span characteristic of ADHD will likely have an impact on those with cognitive disability, since it will increase the difficulty of individuals to focus in the way that much deep learning requires.

the social, cognitive, and neurosciences” (204) and I think he makes a persuasive case for this claim.

Given my earlier endorsements of the Principle of Minimal Agential Realism, the present discussion of disabled agency ought to take seriously the relevant scientific literature. Given that my primary interest here isn’t free will, I’m going to set aside monism for present purposes. But the point above about the impact of the agent’s disability on her social environment is related to Vargas’ rejection of atomism. In opposition to atomism, Vargas favors circumstantialism, the idea that “the powers that matter for whether an agent is responsible are best characterized non-intrinsically, as functions of agents in circumstances” (Vargas (2013a), 3; see also Vargas (2013b)). On this view, both self-directed agency and free will depend not just on intrinsic factors of the agent, but upon features of both the agent and the larger circumstances she is in. Executive function impairments often have a “profound negative impact on a child’s social skill development” (Lavoie (2005), 9), an implication that needs to be taken seriously.³⁰

Finally, there’s some reason to think that 2p15-16.1 Microdeletion Syndrome can also impact agency via the emotions; often those disorders that impair executive function can also lead to “emotional lability or flattening” (Lezak et al. (2004), 36). However, an exploration of the impact of emotional disabilities, either from 2p15-16.1 Microdeletion Syndrome or in general, cannot be undertaken at present.

6 Conclusion

In this paper, I’ve examined a number of different ways that various disabilities or diseases can impact an agent’s executive function. As mentioned earlier, I do not take these kinds of effects to be exhaustive. There are a number of other conditions—including but not limited to Parkinson’s syndrome, alien hand syndrome,³¹ attention-deficit/hyperactivity

³⁰Consider here too the social dimension of autism:

A number of social behavioral deficits have been observed in children with autism. Compared to healthy volunteers and mentally retarded children without autism, children with autism are less likely to engage in pretend play, to imitate others, and to follow the gaze and pointing of others. . . . Children with autism . . . have deficits in executive function. However, these deficits tend to be more related to flexibility and planning rather than inhibition (Williamson and Allman (2011), 90).

³¹Alien hand syndrome is an extremely rare movement disorder where individuals “experience one of their limbs as alien, which acts autonomously and performs movements without being guided by the intention of the patient. The patients find themselves unable to stop the alien hand from reaching and grabbing objects without using their other hand” (Schaefer et al. (2010), 1). They suggest that a likely cause of alien hand syndrome is a “release of the primary motor cortex from conscious control by intentional planning systems” and that the areas of the brain involved are “related to movement execution and planning as well as with areas that have been linked to inhibition control (IFG) and experience of agency (pre-cuneus)” (Schaefer et al. (2010), 1 and 5). Individuals with alien hand syndrome are still able to elicit movements of the alien hand in a controlled way, but are not able to control the alien movements directly. It is also sometimes called ‘anarchic hand syndrome; see Eilan (2013),

disorder (Weyandt et al. (2014) 69f), conduct disorder (Weyandt et al. (2014), 70f) or any disability that might cause apraxia—that should also be explored.

Above in section 1, I gave a general account of agency and considered a number of arguments, in section 2, that agency in general, and morally responsible agency in particular, may come in degrees. The description in sections 4 and 5 of how narcolepsy and 2p16-16.1 Microdeletion Syndrome can impair various aspects of executive function can be seen as further evidence for such a degreed notion. The greater, both in terms of impact and breadth of scope, the executive dysfunction is, the less the individual will be capable of exercising agential control. The degree to which agency is impacted will depend on the specifics of the account of agency adopted, the effect of the disability in question on executive function, and how the impairment caused by the disability in general effects the particular individual in question. Because of the third factor in particular, the exact nature of the impact of a particular disability on agency across the board can't be pinned down to a specified degree. However, that disabilities can impair the various capacities of executive function is, I believe, hereby established. And impairment of those executive functions gives further support for thinking of agency as coming in degrees.³²

182. Interestingly, one of the patients in Schaefer *et al's* study also suffered from Parkinson's disease, and their research suggests an interaction between the two diseases.

³²Thanks to Claire and Oliver Crisp for help in making sure that I accurately represent the nature and impact of narcolepsy. Some of the materials in this paper were presented at the Third International Symposium on Agency, Responsibility, and Character, held jointly at the Universidad del Rosario and Universidad de los Andes, where I benefited greatly by the input of Jesus Aguilar, Santiago Amaya, Andrei Buckareff, Luca Ferrero, Carlos Patarroyo, Maria Lucia Rivera, and Carolina Sartorio. Meghan Griffith and Aaron Cobb provided me with very useful comments on earlier drafts of this paper. I would also like to thank four anonymous referees for the journal whose careful comments greatly improved this paper, as well as the editor for securing me such useful and extensive feedback via those referees.

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