THREE

Cerebralizing Distress

From our exploration of the neurodisciplines that deal with culture and cultural productions, we concluded that "cortex without context won't do." By this we meant to sum up the observation that the methodologies that require leaving out or are incapable of taking into account contextual factors turn out to miss the objects and processes they claim to be studying—objects and processes that are intrinsically contextual. But if there is an area where the role of context has been the focus of debate, it is the understanding and management of mental distress in all its forms. (We shall retain the term "distress" even though, as we shall see below in connection with neurodiversity, not all diagnosed people agree that they suffer or that their suffering can be attributed to the diagnosed condition.) Contested contexts here include the entire range from the genetic to the biographical and the familial to the ethnic, economic, and sociopolitical. While the role of these environments in mental distress is widely recognized, the discussion hinges on their relative weight and on how best to understand their interactions. The

"cerebralizing" of psychological suffering has long been at the heart of that discussion. While disagreements have frequently involved stark dichotomies between nature and culture or reductionistic views of different types (for reduction can be as much culturalist as genetic or neurobiological), we have here chosen to examine the ambivalent modes in which the *neuro* serves a variety of often contrary claims and purposes.

The Engines of Cerebralization

In an article published in *Nature* in 2008, Steven Hyman, a Harvard professor of neurobiology and NIMH's director from 1996 to 2001, acknowledged that "despite the disease burden attributable to neuropsychiatric disorders, and despite significant research, their mechanisms of pathogenesis and precise genetic and non-genetic risk factors have remained stubbornly out of reach" (Hyman 2008, 890). Immediately following his rather bleak assessment, Hyman claimed that "this parlous state of affairs is finally beginning to improve, in part through the application of new genomic technologies coupled to advances in neuroscience." This "glimmer of light" announced a "new dawn" in the diagnosis and treatment of "neuropsychiatric disorders" (893). We could quote dozens of claims characterized by the same structure: first comes a strongly pessimistic observation about the "current" situation, then a declaration of hope in future breakthroughs in understanding pathogenesis. These breakthroughs depend on the underlying belief that psychological distress is essentially a state of the brain and must be ultimately understood and explained as such, a belief also expressed by the common use of *brain disorder* and *neuropsychiatry* to refer to what used to be called *mental disorder* and *psychiatry*.

Hyman defines "mental disorders" as a "diverse group of brain disorders" principally affecting "emotion, higher cognition and executive function." In fact, for him, the expression "mental disorders" is an "unfortunate anachronism" dating back to an era when the conditions thus named "were not universally understood to reflect abnormalities of brain structure, connectivity or function." Widespread as it is, such conviction is, as just mentioned, invariably accompanied by the equally general acknowledgment that identifying precise neural abnormalities underlying those disorders

has "stubbornly defied" research efforts (Hyman 2007, 725). At least since the 1990s, such ambivalence has been a central feature of the cerebralization of distress and, therefore, too, of how it has impinged on self-identity and self-understanding.

There is by now considerable anthropological and sociological work on these matters. Some of it deals with how neuroimaging, as a major vector of cerebralization, has contributed to shape subjectivities and has been integrated into the discourses and practices not only of patients but also of parent groups and health professionals (e.g., Borgelt et al. 2012; Buchman et al. 2013; Cohn 2010, 2012; Dumit 2003, 2004; Eijkholt, Andersson, and Illes 2012; Illes et al. 2008). In her groundbreaking ethnographic exploration of mania and depression in American culture, the anthropologist Emily Martin examined the spread of brain-based vocabularies in psychiatry and their impact upon issues of self-identity and self-identification (Martin 2007, 2010). The case of alcoholism (and we shall mention others) illustrates how the tendency to map personhood and illnesses onto the brain by way of a certain "folk neurology" coexists with and may preserve rather than upset older notions (Vrecko 2006).

The subjectivation processes at work in the area of mental disorders instantiate a phenomenon we noted when discussing the notion of the *cerebral subject* in Chapter 1: Neuroscientific ideas do not necessarily transform self-understandings in any radical manner but combine with existing perceptions and sometimes reinforce current norms. Thus, addiction understood as a brain disorder turns out to strengthen rather than weaken the appeal to individual responsibility. Maintaining a healthy brain implies a "way of life characterized by autonomous, responsible citizenship," for whose attainment an actively exerted willpower is more important than passively taken medication (Netherland 2011, 172).

In short, the cerebralization of psychological distress is no straightforward affair, and ambivalence is one of its central features. At the level of individual and group experience, interpreting mental illness as a brain condition can be liberating, but it can also generate new stereotypes and mechanisms of exclusion; it can inspire new socialities but also erect identitarian barriers. At the scientific level, it promises to be the source of progress in diagnosis and treatment, yet even its protagonists acknowledge over half a century of few advances and many failures. In this chapter, we shall

explore these dynamics by way of two cases, one focused on scientific research, the other on the making of collective and individual subjectivity: the neuroimaging of depression and the claims for autism as a form of "neurodiversity." Before this, however, we must sketch some important elements of the broader contexts to which both belong, namely the emergence of the "pharma-psych nexus," the globalization of mental health, the logic of biomarkers, and the crisis of the biological model.

Pharma-Psych

The expression *pharma-psych nexus* (Williams, Katz, and Martin 2011) has been used to capture the spread of psychopharmaceutical products that target the brain's chemistry. This includes, among others, the commercialization of selective serotonin reuptake inhibitors (SSRIs) for depression and anxiety-related disorders and of psychostimulants like methylphenidate (well known under the trade name Ritalin) for ADHD as well as the use of these and other substances, such as modafinil (indicated for the treatment of narcolepsy), for recreational and enhancement purposes. In his books *The Antidepressant Era* (1997), *The Creation of Psychopharmacology* (2002), *Let Them Eat Prozac* (2004), *Mania: A Short History of Bipolar Disorder* (2008), and *Pharmaggedon* (2013), the psychiatrist and historian David Healy has critically examined the collusion between medicine and the pharmaceutical industry, particularly in the domain of mental health, with depression as a major case (see also Bentall 2009, Greenberg 2010, Kirsch 2009). Healy and others have demonstrated how extensively the production of evidence in psychiatry has been co-opted by economic and marketing considerations. Pharmaceutical companies largely draw on biased ghostwriting, make sure that only positive results are published while reframing or concealing the negative outcomes of clinical trials, and exaggerate the effectiveness of medications (Angel 2004; Dumit 2012; Goldacre 2013; Gupta 2014; Healy 2004, 2008; Kirmayer and Raikhel 2009). Insofar as the drug-based approach has fueled the expansion of mental illness to its current epidemic proportions, the system sustains itself (Whitaker 2010).

The pharma-psych drive is not merely a matter of economics and medicine but also of professional ethics. The pharmaceutical industry's funding

of biomedical research and education generates conflicts of interest that medical doctors and researchers frequently prefer not to disclose. Companies use material incentives to increase prescription rates and encourage the adoption of new drugs over available generics; most American physicians have accepted gifts yet tend to downplay their influence (Armstrong 2012; Gibbons et al. 1998; Grande 2010; Green et al. 2012; Grande, Shea, and Mitchell 2009; Hodges 1995; Wazana 2000). This situation has led to a significant weakening of public trust and to intense discussions about how best to regulate this area of the medical profession (Grande 2010).

As reflected in hundreds of online forums, trust has also been shaken by other factors. One is increasing awareness that the spread in the use of a particular medication enlarges diagnostic boundaries and even generates new diagnostic categories. Depression, for example, has expanded to encompass sorrow, sadness, and shyness—states that, even when intense or prolonged, do not necessarily signal mental illness (Frances 2013, Horwitz and Wakefield 2007, Lane 2007). Another concerns medication. The discovery in the 1950s of the antipsychotic and antidepressant effect of certain synthetic compounds (chlorpromazine was the first) and the subsequent introduction of prescription psychotropic drugs gave rise to the claim that mental illness is caused by a "chemical imbalance" in the brain (Whitaker 2010). As far as depression is concerned, the "imbalance" view received support from the fact that SSRIs have antidepressant effects in some patients. In fact, neither the cause of those effects nor the modes of action of those drugs are known. Many call the imbalance theory a "myth," and it is clear that it should be seen at the minimum as a metaphor (Moncrieff 2008). Yet it has been uncritically conveyed by the media and successfully promoted by both psychiatrists and the pharmaceutical industry, for whom it has had a huge marketing value (Lacasse and Leo 2005, Leo and Lacasse 2008).

Contrary to the assertions of pharmaceutical advertising about the action of particular medications, psychiatric drugs lack specificity and have overlapping effects that do not correspond neatly to specific symptoms, disorders, or neurotransmitters. Pharmaceutical companies, however, have covertly and intentionally committed the "therapeutic fallacy," suggesting that the drugs they advertise are supported by a causal theory about the targeted psychopathology. The theory, however, looks valid mainly because

the marketed drug improves certain symptoms. Recent debates about antidepressants probe three possible explanations for the effectiveness of these medications: They are effective because their active component has specific and targeted psychodynamic action (this is most commercially interesting claim), the placebo effect is responsible for the medications' effectiveness, or the drugs involve some unknown mechanism of action that provokes a nonspecific altered mental state alongside with a placebo effect (Gupta 2014, 59).

The psychiatrist and bioethicist Mona Gupta notes, "All three interpretations are plausible, but none is self-evidently true or false" (59). Now, if that is the case, then the psychiatric community has the prerogative to determine which is most likely. Professional and financial interests tend to balance the choice toward the first explanation, which presupposes specific antidepressant effectiveness. Hence, as the historian Edward Shorter (2013, 4–5) sharply put it,

> Today, with the ubiquity of the diagnosis of depression, we have the idea that low mood and an inability to experience pleasure are our main problems; we see ourselves as having a mood disorder situated solely in the brain and mind that antidepressants can correct. But this is not science; it is pharmaceutical advertising.

Globalization

Psychopharmaceutical marketing has also contributed to the globalization of psychiatry and the high prevalence of depression, as documented by research in India (Ecks 2013, Ecks and Basu 2009, Sumeet and Jadhav 2009), Japan (Applbaum 2006, Kirmayer 2002, Kitanaka 2011), Brazil (Béhague 2009; Biehl 2005, 2006; Leibing 2009) and Argentina (Lakoff 2005, 2006). While ethnographic studies tend to corroborate the existence of a global psychopharmaceutical hegemony (Good 2010), the distribution of spending on pharmaceuticals is strongly asymmetric and is determined by economic incentives (Petryna and Kleinman 2006). In the area of mental health, the result is overdiagnosis and overmedication in the richest countries and dismal negligence in the poorer ones (Kleinman 2012).

Such an imbalance in the distribution of resources must be placed in the framework of the discussion concerning the contribution of mental disorders to the global burden of disease (GBD) as measured in Disability-Adjusted Life Years (DALYs, or number of years lost due to ill health, disability, or early death). Neuropsychiatric conditions, including common ones such as depression and anxiety, addiction disorders (alcohol and substance abuse), and psychoses and dementia, account for up to a quarter of all DALYs and up to a third of those credited to noncommunicable diseases, with high variability among countries and income levels (Prince et al. 2007, 2014). Depression is considered to be the major contributor to the GBD and, together with anxiety disorder, accounts for between one-quarter and one-third of all primary healthcare visits worldwide (Prince et al. 2014, 103). The high burden imposed by mental disorders according to epidemiological estimates coexists with the secondary place of mental health in global health agendas and policies. The Global Mental Health (GMH) movement, which was most visibly launched by the British medical journal *The Lancet* in 2007, highlights the "treatment gap" between the need for and the availability of mental health services, especially in low- and middle-income countries. To overcome this gap, the World Health Organization (WHO) initiated in 2008 the "Mental Health Gap Action Programme" (mhGAP) (Cohen, Patel, and Minas 2014; Hanlon, Fekadu, and Patel 2014; Patel 2012; WHO 2008).

These proposals have been accompanied by controversy, for example over the validity of diagnostic instruments across different countries and the reliability of epidemiological estimates of the global prevalence of mental disorders (Mills 2014; Summerfield 2008, 2012; Watters 2010). However, disputes about technical aspects ultimately concern the conceptual framework that merges mental distress with neurological disorder, the fundamental assumption that mental illnesses are essentially disorders of the brain. Inherent in the cerebral localization of mental distress is the epistemic hierarchy we noticed in the previous chapter: It is believed that only the discovery of neurobiological causes will satisfy the ambition to "define true madness" and that thereby will the "real" contribution of mental disorder to the GBD be established (Rose and Abi-Rached 2013, 130).

Such convictions about causality (to which we shall return) are relevant for the quest, present in the GMH movement, to reconcile biological universality and cultural particularity. Different cultures have different beliefs

about the meaning of *mind* and mind-body relationships, but it is accepted that brains are basically the same across the entire human species. The 2001 World Health Report states: "Mental disorders are not the exclusive preserve of any social group; they are truly universal. Mental and behavioural disorders are found in people of all regions, all countries and all societies" (WHO 2001, 23). The universality of disease is in this case sustained by a universal neurobiology, which justifies introducing in different cultural contexts transcultural intervention packages and modes of diagnosis while at the same time acknowledging variation at the level of the expression and triggers of psychopathology (Cohen, Patel, and Minas 2014; Patel 2012; WHO 2013).

Since the mid-1990s, however, and at least within the GMH movement, the universality of disease has become dissociated from the globalization of nosologies and even from the global use of the very notion of "mental disorder." Primary care workers in developing countries are uncomfortable with that notion and "contend that the use of symptoms to diagnose mental disorders, without consideration of context . . . essentially flags non-clinically significant distress" (Jacob and Patel 2014, 1433). Thus, noticing, for example, that in low- and middle-income countries "very few patients report feeling depressed" and that most interventions targeting depression avoid the use of the label, two major actors of GMH have advocated not just dimensional approaches to distress but the abandonment of prevalent international classifications in favor of new bottom-up taxonomies that would be elaborated independently of specialist perspectives (Jacob and Patel 2014; compare with Patel and Winston 1994).

Biomarkers

At the research level the assumption that mental distress involves brain anomalies fuels the quest for biomarkers that can distinguish better between normality and pathology, grasp etiological factors, and aid in the development of treatments that will be effective because they hit the targeted abnormalities. Yet even the staunchest advocates of the neurobiological approach acknowledge that biomarkers remain "stubbornly out of reach" (Hyman 2008, 890). In 2002, in a contribution to the preparation of the fifth edition of the *DSM*, the American Psychiatric Association's *Diagnostic and*

Statistical Manual of Mental Disorders (released in 2013), a group of prominent biological psychiatrists remarked that psychiatry had "thus far failed to identify a single neurobiological phenotypic marker or gene that is useful in making a diagnosis of a major psychiatric disorder or for predicting response to psychopharmacologic treatment" (Charney et al. 2002, 33). Over a decade later the situation has not changed.

Thus, an article from 2011 on the challenges involved in searching for autism biomarkers concluded that "despite huge advances in the basic scientific understanding of autism, comparatively little has been achieved to date with regard to translating the resulting evidence into clinically useful biomarkers" (Walsh et al. 2011, 609–610). And in 2014, the Emory University neuropsychiatrist Helen S. Mayberg, a major figure in the field of depression neuroimaging, pessimistically admitted that "the claims of clinics that they can reliably use structural or functional brain scans" for diagnostic and treatments goals "is without medical or scientific support." Even worse, such claims are "beyond the scope of current research and give false hope to patients and their families" (Mayberg 2014, S34).

The reason for such failures reside, partly at least, in the categories for which the biomarkers are being searched, which are those provided by the *DSM* and the *ICD* (the WHO's *International Classification of Diseases*).[1] Possibly there are no biomarkers for the symptom clusters these classifications identify as diagnostic categories. Hence the NIMH initiative, launched in 2011, to turn away from *DSM* categories and develop "Research Domain Criteria" (RDoC) aimed at transforming psychiatric diagnosis via the convergence of genetics, neuroimaging, and cognitive science (Insel et al. 2010; Insel 2013; Kapur, Phillips, and Insel 2012).

RDoC represent a new angle in the search for neurobiological markers but not a radical new departure. In fact, they maintain intact the established neurobiological view of mental disorder, with its focus on discrete biological mechanisms at the expense of a more integrated "ecosocial" approach (Kirmayer and Crafa 2014). In RDoC, biomarkers will no longer be coupled with *DSM* categories, but mental illnesses will remain defined as "biological disorders involving brain circuits that implicate specific domains of cognition, emotion, or behavior" (Insel 2013). The problem is that elucidating the etiology of those disorders demands "trust in the brain, not in DSM" (Rose 2013b, 10), yet trust in the brain has so far produced virtually

no results of clinical or diagnostic utility. Various neuroimaging findings have been said to correlate with learning and performance in children and adults, criminality, health-related behaviors, and responses to treatments, and it has been claimed that insofar as they may function as neuromarkers, they may contribute to personalize practices in those domains (Gabrieli, Ghosh, and Whifield-Gabrieli 2015). Yet the situation remains as Nikolas Rose and Joelle Abi-Rached (2013, 138) described it, namely, "Each of the pathways that neuropsychiatry has attempted to trace through the brain seems to run, not into the bright uplands of clarity, but into the murky, damp, misty, and mysterious forest of uncertainty." Perhaps the reason is that RDoC are based on the brain-disease model of mental health just at a time when the "bio-bio-bio" model (Read 2005; Read, Bentall, and Fosse 2009), which combines neurobiology, genetics, and pharmacology, has come under attack at the epistemic, ontological, sociomoral, and cultural levels.

Crisis of the "Bio-Bio-Bio" Model?

What are the grounds for criticism of the "bio-bio-bio" model? First, recent and in principle better-targeted medication has not worked as anticipated. The new generations of antipsychotics are not more effective than older and (by now) much cheaper drugs like Thorazine, a trademark of chlorpromazine. The new drugs, moreover, have been related to sudden cardiac death, cardiovascular risk, weight gain, and the development of diabetes (Álvarez-Jiménez et al. 2008; Foley and Morley 2011; Luhrmann 2012; Ray et al. 2009; Weinmann, Read, and Aderhold 2009). The pharmacological disenchantment matches the failure to identify genetic and neurobiological biomarkers and is reinforced by evidence about the role of culture in the prevalence and prognosis of disorders such as schizophrenia (Luhrmann 2007, 2012; but as Cohen and Gureje 2007 document, producing and assimilating evidence about these matters in ways that are not heavily determined by interests and preconceived views is as difficult in relation to culture as in relation to biology).

Second, psychological therapies have made a comeback. The cerebralization of psychiatry has conflicted with psychodynamic, mainly psychoanalytic approaches, around issues of efficacy, diagnostic validity, and

prevalence. Current debates around GMH and the articulation of universality and particularity in mental disorders constitute the most recent chapter of this ongoing tension, which seems to be entering a new phase. Indeed, as we shall see below, the recent retreat from prevalent contemporary classifications is at bottom a move toward "denosologizing" mental illness altogether, that is, giving up diagnostic categories as we know them, to focus instead on dimensions that can be variously combined and treated in context-sensitive ways at the behavioral and psychological level.

Given the evidence that the efficacy of antipsychotics has been overestimated—and their toxicity underestimated—as well as emerging data regarding alternative treatment options, it has been argued that patients should be given more choice concerning medication and therapy. A 2012 editorial in the *British Journal of Psychiatry* argued that nonadherence and discontinuation of medication by some psychotic patients may "represent a rational informed choice rather than an irrational decision due to lack of insight or symptoms such as suspiciousness" (Morrison et al. 2012, 83). The authors emphasized the significance of evidence-based alternatives to antipsychotic medication, mainly psychosocial interventions. Studies show the efficacy of cognitive behavioral therapy (CBT) in reducing psychotic symptoms compared to other psychological methods (Turner et al. 2014) and conclude that it "seems to be a safe and acceptable alternative for people with schizophrenia spectrum disorders who have chosen not to take antipsychotic drugs" (Morrison et al. 2014, 1395).

While CBT has been recommended in the United Kingdom for new cases of schizophrenia, long-term psychotherapy has become standard in some parts of Scandinavia (Balter 2014). Contrary to widespread skeptical or negative perceptions about psychodynamic therapies, assessments using randomized control trials support their effectiveness (Bhar and Beck 2009; Fonagy et al. 2015; Leichsenring and Klein 2014; Leichsenring and Rabung 2008, 2011; Rosenbaum et al. 2012; Shedler 2010; Thoma et al. 2012). Carried out in the context of a generalized turn to evidence-based practices in insurance and healthcare policy making, these studies are encouraging for both CBT and psychodynamic therapies and contribute to the credibility of psychological approaches to severe mental disorders at a time when the search for biomarkers and the use of antipsychotics as a first option seem to have stalled. As far as depression is concerned, CBT has become less and

less effective, its effect size falling by half since 1977 (Johnsen and Friborg 2015).

Yet the evidence-based conditions under which psychotherapy is being validated have themselves raised objections. Thus, the emphasis on probabilistic outcomes has been criticized as a threat to the psychotherapeutic focus on the specificity of each patient's experience (McKinley 2011); the generalized use of treatment-as-usual (TAU) as a control condition is problematic, given that in each case TAU encompasses diverse treatments and that its composition varies in ways that affect assessment outcomes in uncontrolled manners (Löfholm et al. 2013); and the real-world *effectiveness* of depression treatments that demonstrated *efficacy* in strict randomized control trials criteria has for the most part not been assessed (Balt 2014, Blais et al. 2013).

In short, the therapy wars are not about to end (Burkeman 2015), and the fact that they likely never will points to a further consideration relative to criticism of the bio-bio-bio model since the late twentieth century. Advocates of psychotherapy do not defend a purely psychological understanding of mental disorders, and the backlash against the model does not deny the etiological role of genetics or neurobiology. Rather, it reflects the emergence of a more systematic focus on the interactions among biological, social, and cultural factors. Epigenetics has come to provide not only empirical data but also a model, insofar as it concerns the study of changes in the regulation of gene activity and expression that do not depend on gene sequence but are heavily influenced by the environment (Carey 2012; Meloni 2013, 2014a, 2014b; Rose 2013b). The epigenetic approach has profound implications for research, mental health services, and prevention, as it replaces the earlier focus on genetic predispositions and inborn susceptibility or vulnerability and opens ways for showing how an adverse social environment "gets into the mind" and "under the skin" (Hyman 2009) and affects mental health (Toyokawa et al. 2012).

For example, while there is evidence of a relationship between childhood trauma and subsequent psychosis, understanding it requires integrating biological and psychosocial paradigms, and that is likely to be done largely via the identification of epigenetic processes (Larkin and Read 2008; Read, Bentall, and Fosse 2009). In the case of schizophrenia, nutritional intake (an environmental factor) can affect epigenetic processes associated with the

disorder. The study of survivors of the Dutch "Hunger Winter" of 1944 and of the Great Chinese Famine of 1959–1961, both of which implied prenatal food deprivation, revealed a twofold increase in the cumulative risk of schizophrenia in the birth cohort. The effect of such deprivation on the *IGF2* gene, which provides instructions for making a protein that plays an essential role in prenatal development, offers a plausible epigenetic mechanism for the environmental roots of schizophrenia (Toyokawa et al. 2012). Epigenetic differences linked to susceptibility to psychiatric disorders might arise through exposure to stress-related factors during critical developmental periods, and various models propose to explain how epigenetic regulatory mechanisms contribute to behavioral phenotypes in schizophrenia and depression, drug addiction, and fear-related anxiety disorders (Dudley et al. 2011). Models are also being developed for gene-environment interactions accounting for the well-documented effects of early life stress (childhood abuse, neglect, and loss) as a risk factor for the later development of depressive disorders (Heim and Binder 2012).

In short, by aiming to give equal weight to genetics and the environment, the epigenetic trend instantiates a sort of social turn in the biological sciences (Meloni 2014a) and, in any case, represents the breakdown of the bio-bio-bio model as far as the understanding of psychic distress is concerned.

Finally, there have been new departures with regard to the moral and political effects of the pure biomedical approach. As long as it was believed that reframing mental illness as brain disease reduced stigma, the bio-bio-bio model could be perceived as nourishing acceptance, diversity, and human rights. Biological explanations seemed to exempt individuals from responsibility for their disease (Corrigan et al. 2002, Lopez-Ibor 2002). Claiming that mental disorders are diseases "like any other," antistigma campaigns espoused cerebralization in the belief that public acceptance of biological causality would inspire more tolerant attitudes (Cheek 2012). Even scholars who debunked the chemical imbalance theory of mental disorders saw in it a "convenient" way of helping to destigmatize psychiatric illness (Angell 2011).

It turns out that this destigmatizing effect has been exaggerated and that the biological conception of mental illness has sometimes even provided new grounds for intolerance (Angermeyer and Matschinger 2005; Bennett, Thirlaway, and Murray 2008; Phelan 2005; Read and Harré 2001; Schnittker 2008). The idea that individuals are not responsible for their disorders and

that, therefore, their brain is to be "blamed" can promote further stigma. Neurobiologization can heighten the perception that mentally ill individuals are dangerous, precisely because they lack control and appear unpredictable. In addition, it contributes to erect boundaries between "healthy" individuals and mental health sufferers, now seen as biologically different. Thus, 42 percent of people interviewed in a Canadian survey would no longer socialize with a friend with mental illness, and 55 percent would not marry someone suffering from a mental disorder (Cheek 2012). Other research suggests that attributing mental illness to genetic or biological underpinnings increases public stigma and social distance and that "people who are the intended beneficiaries of stigma reduction campaigns . . . may internalise a stigma-reduction message while the society around them fails to do so" (Buchman et al. 2013, 71). If the biomedical model of illness ever had a moral justification, it is also now largely gone.

The situation we just sketched is complicated. Different factors drive both the cerebralization of distress and its critique, and they may interlock in various ways. It is therefore more productive to map that complexity than to reproduce dichotomies that are not confirmed in the field. For example, it may seem that the cerebralization of mental distress goes hand in hand with the reification of nosological categories—that, to put it bluntly, neuroimaging and the *DSM* express the same epistemic outlook on mental illness. However, as we saw, while constituting a move away from *DSM* categories, the Research Domain Criteria program pursues the search for neurobiological markers and strengthens the brain model of mental distress. Such ambivalence, we propose, is an essential feature of the cerebralization of psychic distress and therefore also of the processes whereby human experience and views about personhood sometimes come to embody ways of "being brains."

Depression

Perhaps more than any other psychiatric condition, depression remains torn between biomedical and psychological accounts, between neurobiological causes and contextualized explanations. Though generally understood as involving an array of factors, from genetic predispositions to environmental

circumstances, it has turned out to be impossibly challenging to bring these factors together. The stakes of the challenge are considerable, since (in 2010) major depressive disorder (MDD) ranked as the second leading cause of disability worldwide and as the eleventh leading cause of global burden of disease (Ferrari et al. 2013). Depressive disorders have therefore become a global health priority, and the WHO recurrently calls for coordinated global action.[2]

At the same time, there have been intense debates over whether depression is overdiagnosed and antidepressants overprescribed (Reid 2013, Spence 2013) as well as over the efficacy of antidepressants (better than placebo? only in severe cases? Fournier et al. 2010, Gibbons et al. 2012, Kirsch et al. 2008, Turner et al. 2008). The difficulties of seeing clearly in this domain are compounded by the fact, mentioned above, that drug manufacturers withhold negative data and that only positive data tends to be published. A significant turn occurred around 2011, when major companies including Novartis, GlaxoSmith-Kline, AstraZeneca, Pfizer, Merck, and Sanofi decided to stop investing in research on drugs for brain disorders and redirected their efforts toward genetics (see Tracy 2016 on the "neuro funding rollercoaster"). The decision was motivated by commercial considerations: Because many generic psychiatric drugs are available, because new medications do not work better than the older ones, and because most candidates aimed at new brain targets fail after years of clinical trials, companies concluded that there are better chances of identifying genetic biomarkers than neurobiological ones (Abbott 2011). This crisis joins the weakening of trust sketched above, rooted in the practices of industry and the failure to globalize psychiatric diagnoses and classifications effectively.

Again, however, the situation is complex. In the overall picture, the calls for abandoning the bio-bio-bio model and for elaborating local idioms of psychic distress coexist with programs that variously pursue the model as a means to revamp classification so as to make it truly universal and fully transcultural. Our focus here will be on neuroimaging as a major player in this context. As elsewhere, the uses of neuroimaging in the field of depression and the claims that are made for its significance encapsulate epistemic and moral as well as social and psychological mechanisms involved in bringing about cerebral subjects.

Just Like Diabetes?

Even a superficial look at the successive editions of the *Handbook of Depression* (Beckham and Leber 1985, 1995) demonstrates that, like most psychological and psychiatric entities, depression is not a single thing and that no single approach may be considered simultaneously necessary and sufficient for understanding and treating the condition.[3] In the *Handbook*'s most recent edition, thirty chapters address four main areas (Gotlib and Hammen 2014). Part 1 reviews "descriptive aspects" such as the epidemiology, course, outcome, and assessment of depression as well as issues in methodology, classification, and diagnosis (for example, the relations between personality and mood disorders or the comparison of unipolar and bipolar depression). Part 2 moves from the genetics of major depression to the interpersonal and social environment of the condition, dealing along the way with the contributions of neurobiology and affective neuroscience as well as with depression and early adverse experience, children of depressed parents, and the cognitive aspects of depression. Part 3 examines depression in specific populations (with a chapter on understanding the condition across cultures), and Part 4 considers prevention and treatment, not only pharmacological but also cognitive, behavioral, and psychosocial.

Obviously, neuroimaging is used in only a few of these areas. Given the vastness of the field of depression, the condition's high degree of comorbidity with other psychiatric disorders, the heterogeneity of the category, and the diversity of possible approaches, it is one element in a wider framework of investigative, therapeutic, and economic practices and interests. Neuroimaging, however, is not just one more approach in research and assessment. As we shall see, it is endowed with a certain methodological and epistemic primacy and thereby with the authority to prove that, by virtue of actually being brain disorders, mental disorders, depression included, are "just like diabetes."

Depression is of course an organic illness. It is in general reasonable to admit that mental disease is in important ways "like any other medical disease." To begin with, since "all diseases involve the self," the self-affecting aspects sometimes said to be unique to mental disorders are in fact not

exclusive to those conditions (Hofmann 2015). But is depression *just like* an organic illness? Maybe yes, in the trivial sense that it is a biochemical state with potentially discoverable causes. Nevertheless, in addition to being neurobiological and having a cause, depression is a state with contents and "reasons," and it can be judged to be warranted or unwarranted, desirable or undesirable, meaningful or meaningless. You may accept that your depressive symptoms are neurochemical, but if you are told that they are "just like diabetes," you might feel that they are not acknowledged as being also warranted and meaningful (Arpaly 2005).

Less phenomenologically, imagining that depression *is just like diabetes* involves a fundamental confidence in the possibility of discovering biomarkers that will enable diagnoses of the condition according to purely biological criteria. Such redefined criteria might ultimately contribute to "denosologize" psychiatry in the following sense: Presently, categories such as "major depression disorder" are defined on the basis of syndromes, or collections of behavioral *signs* (what is observed) and *symptoms* (the patient's complaints). A denosologized psychiatry would focus on symptoms that, instead of being linked to particular conditions envisaged as discrete entities, would be shared by several conditions (as currently defined) and correlate with *dimensions*, such as aggression, anxiety, or mood. An instance, provided by Herman van Praag, a Maastricht University psychiatrist who has long criticized his field's "nosologomania," is the stress-inducible, "anxiety/aggression-driven depression" (van Praag 2005; on denosologizing, van Praag et al. 1987; van Praag 2000, 2010). For van Praag (2008, 31), the reason why half a century of intensive research has failed to elucidate the biology of depression is that "insufficiently specified diagnostic constructs" will not turn out "to be caused by specific, well-definable pathological processes."

Though not always so strongly formulated, the trend in biological psychiatry moves toward dissolving current nosographic categories and toward identifying the neurogenetic factors involved in depressive symptoms (e.g., Scharinger et al. 2011), diagnostic biomarkers, and biomarkers that will make it possible to prognosticate the illness's evolution and predict treatment efficacy and clinical response. Genetic findings and neural circuit maps link different syndromes or distinct subgroups within syndromes. This is the Research Domain Criteria perspective sketched above. In contrast, diagnosing mental disorders on the basis of clinical observation and patients' reports is

seen as implying that the syndromes embody "unique and unitary disorders" and thus as undercutting the possibility of identifying illnesses linked to pathophysiology.[4] The assumption here is that clinical heterogeneity maps onto biological heterogeneity and that the only way out of the nosographic mess is to replace the examination of clinical symptoms by the identification of biomarkers.

Biomarkers are to be understood in terms of vulnerability and susceptibility, risk and probability; moreover, since they are based on groups, their predictive power as risk factors for individuals is low (Singh and Rose 2009, Walsh et al. 2011). Neuroimaging depression research is essentially about the identification of such biomarkers, which in its case take the form of patterns of neural activation that systematically correlate with a diagnosis (major depression disorder, bipolar disorder), with particular symptoms, or with treatment outcome. The neuroimaging of depression thus looks like the neuroimaging of any other "brain disorder." But there are some significant differences.

In schizophrenia, as we mentioned, social and experiential indicators, such as adversity, stressful life events, or childhood abuse and trauma, have been correlated with chances of developing the disorder; conversely, psychological and social interventions play a role in its management. Nevertheless, more than biopsychosocial models, which emphasize factor interdependence, it is the diathesis-stress model, according to which a stressor may trigger an initial illness episode in persons with a genetic predisposition (*diathesis*), which seems to have become the predominant framework for thinking about the condition (see Jones and Fernyhough 2007 for a discussion of the neural version of this model). In spite of the epigenetic turn and the awareness that culture matters, schizophrenia remains depicted primarily as a brain disease.

The diathesis-stress model is also central in depression research. The etiology of depression, both unipolar ("major" depression) and bipolar (the former "manic depression"), is generally thought to include a significant genetic component in the determination of risk, and the condition correlates with changes in neurotransmitter systems involving serotonin, norepinephrine, and dopamine. Nevertheless, while giving considerable weight to biological factors, depression studies tend to underline the interdependence of a multiplicity of risk and etiological mechanisms. It seems more difficult to turn depression into a purely organic illness than it has been to isolate the

purported neural correlates or "signatures" of schizophrenia or autism spectrum disorder (on the former, see Cabral et al. 2013 and Hart et al. 2013; on the latter, Ecker et al. 2010 and Deshpande et al. 2013, both accompanied by considerable media coverage misleadingly suggesting that henceforth diagnosis can be made on the basis of brain scanning).

Cultural and historical factors hint at the sources of the difficulty. While there is debate on whether depression overlaps with melancholy and on how much continuity there might be between psychiatric categories and the *melancholia* that the Western tradition links to genius and to a superior manner of being in the world, depression sometimes retains the dark luster of the ancient black bile and is often accompanied by an exceptionally penetrating reflexivity.[5] The comparative literature scholar Matthew Bell (2014, xi) insightfully notes:

> One distinctive feature of Western culture is the high status that it has accorded to self-consciousness. Melancholia, or at least the psychological symptoms of melancholia as reported from Hippocrates right down through Western history, depends upon the West's peculiarly introspective culture. The psychological symptoms of melancholia are, to put it crudely, a disorder of malignant self-consciousness.

Certainly some depressed people associate their distress to a large spectrum of causes and reasons, from the random to the meaningful, from the reductively genetic to the deeply psychoanalytic. Nevertheless, in diverse, often contradictory ways, personal accounts by hitherto unknown patients, movie stars, famous writers, diagnosed academics, or mental health professionals have contributed to the modern persona of the depressive and the public image of the condition.

Such autobiographical narratives neither counterbalance nor contradict neurobiological explanations (Dumit 2003). Nevertheless, the evocation of contexts, moments, relationships, and inner lives gives depression cultural resonance as well as meanings that function as a kind of causal interpretation. For the authors of depression memoirs (admittedly a minority of the diagnosed population) such elucidations make more existential sense than the demonstrations of biological psychiatry. Self-reflexive depressed persons may be fascinated by brain scans and acknowledge that depression is biological (Buchman et al. 2013, Cohn 2010, Martin 2010). However, as autobio-

graphical writings show, they wish primarily to understand contextual and relational factors that neuroimages and correlations can hardly reveal and illuminate. While organic explanations of autism or schizophrenia may satisfy the persons concerned (patients or caregivers), they seem intrinsically insufficient to those directly or indirectly touched by depression. For them, depression is not just like diabetes.

Neuroimaging Depression

In 2005, an article in the *New York Times* noted that brain scans, long celebrated as "snapshots of the living human brain," had been counted upon to illuminate the mystery of mental illness but that the promise had not been fulfilled (Carey 2005). The neuroscientists' response, expressed in that article by Steven Hyman, was that those who oversold the technology forgot that "the brain is the most complex object in the history of human inquiry." For him, the key consisted of pursuing the same line of research. Since that is indeed what happened, it is appropriate to ask what kind of progress has been made.

Meta-analytic analyses of neuroimaging publications, which seek to identify consistent patterns and results across a large number of studies, appeared both before and after the *New York Times* asked, "Can Brain Scans See Depression?" In 1998, Wayne C. Drevets, who later became senior investigator at the Neuroimaging Section of the NIMH Mood and Anxiety Disorders Program in Washington, D.C., reviewed the contributions of functional neuroimaging to knowledge of the pathophysiology and "anatomical correlates" of major depression (Drevets 1998, 341). He hoped that such neurocorrelational studies would "ultimately localize specific brain regions for histopathological assessment, elucidate anti-depressant treatment mechanisms, and guide pathophysiology-based classification of depression" (342). At the time, Drevets noted that the capabilities of neuroimaging to determine diagnosis or guide treatment had not yet been established. Functional imaging seemed nonetheless a promising approach: The fact that some depressive symptoms could be experimentally induced in nondepressed subjects opened the way for depressed-control comparisons of the changes in cerebral blood oxygenation and glucose metabolism "associated with" depression.

However, the exact nature of the association remained nebulous. For example, nondepressive conditions sometimes present in depressed patients can affect functional brain imaging measures; regional blood oxygenation or metabolic differences between depressives and control subjects "may thus reflect either the physiological correlates" of depression "or pathophysiological changes that predispose subjects to or result from affective disease" (Drevets 1998, 342). In short, as a 2008 review of biological vulnerability factors in early-onset depression put it, the quest for the "neurobiological roots" of the condition is obscured by the fact that, when assessing differences in brain function or activity between patients and controls, "it is unclear whether we are measuring causal factors making an etiological contribution to the illness, or, conversely, consequences or associated factors of the illness" (Nantel-Vivier and Pihl 2008, 105).

What are these authors saying? On the one hand, their language remains ambiguous: Is *may* freely conjectural or more or less rigorously hypothetical? On the other hand, it conveys ambiguity concerning the nature of the results. The language avoids causal connectives, employing *predispose* and *result* in the context of a speculative remark, yet at the same time, it suggests a capacity to detect and measure causal factors.

On its first page, the review we just quoted explains that the "putative biological, psychological, and environmental etiological mechanisms" of pediatric depression are "intrinsically linked, interactive, and complementary." Starting with the second page, however, it becomes clear that the analyzed research concerns "biological correlates" supposedly pointing the way to a better understanding of "etiological roots" (Nantel-Vivier and Pihl 2008, 103–104). The authors claim that, by studying pediatric populations, they "significantly decrease the likelihood of the occurrence of confounding factors and can therefore more clearly investigate causative neurobiological forces by getting closer to their etiological roots" (105). One of the main goals of "disentangling the neurobiological factors" is to develop a "biological etiology" and, on that basis, a taxonomy of illness that will yield "more homogenous diagnostic categories" (106). But if some factors are "confounding," then they are not "intrinsically" linked to the others. In fact, the purpose of the study is to isolate the "forces" to which causal efficacy can be attributed, that is, the neurobiological ones. As far as we can tell, such

ambiguities in language, as well as the slippage from correlation to causation, are commonplace in neuroimaging depression research and characterize the field of psychiatric neuroimaging as a whole (Boyce 2009).

The same can be noted about the prevalent attitude vis-à-vis the variability of research results. The clinical heterogeneity of depression and the anatomical differences across individuals are major sources of variability; such heterogeneity, as Drevets (1998, 343) explained, also implies that "diverse signs and symptoms may exhibit distinct neurophysiological correlates." "Localization," he wrote, "is now limited as much by the anatomical variability across individuals as by the spatial resolution of imaging technologies" (345). At the time, a related source of confusion came from the fact that imaging results did not differ significantly between subjects with primary depressive syndromes and those whose similar syndromes derived from neurological conditions such as Parkinson's or Huntington's disease (353).

The two chief explanations for the inconsistency of the data (low spatial resolution and the secondary nature of the symptoms) were placed on the same level. Yet, while imaging resolution can improve, as it indeed has since the 1990s, variations in anatomy and brain circuitry are not limitations to be overcome. It is nevertheless hoped that they will cease being an obstacle when the clinically based nosography that still frames neuroimaging studies is replaced by a "pathophysiology-based classification." The stated hope is to refine "our understanding of the anatomical correlates" of depression (358), with the ultimate goal of integrating imaging, neurochemical, and anatomical data so as to move from physiological correlates to anatomopathological localizations. At the same time, the data Drevets reported seemed to support a "circuitry model in which mood disorders are associated with dysfunctional interactions between multiple structures, rather than increased or decreased activity within a single structure" (355). A vocabulary of localization thus coexisted, and still does, with an emphasis on brain circuitry.

In 2002, a shorter overview of depression neuroimaging noted the lack of a "general theory" to integrate the findings about functional abnormalities in the amygdala and hippocampus and reached circular conclusions of confounding generality: Since the medial prefrontal cortex is connected to areas where neuroimaging uncovers structural and functional abnormalities,

> dysfunction in this region may be fundamental to depression. . . . These results thus support a neural model of depression in which dysfunction in regions that modulate emotional behavior may result in the emotional, motivational, cognitive and behavioral manifestations of depressive disorders. (Erk, Walter, and Spitzer 2002, 67)

The recurrent *may* is the hopeful expression that the cause-and-effect connections here envisaged as *possible* will turn out to be true. The ambiguous, evocatively rather than assertively causal language is the same as in Drevets, but Erk and colleagues add an element of self-evidence, since dysfunction in regions that modulate emotion necessarily affect emotion. Insofar as the nosography of depression includes emotional signs, depression necessarily involves brain areas implicated in emotion.

A Quest for "Objectivity"

Also in 2002, an extensive review was coauthored by Richard J. Davidson, the high-profile director of the Laboratory for Affective Neuroscience at the University of Wisconsin–Madison. As a scientist with noticeable media presence and a well-publicized connection to the Dalai Lama, Davidson has been described as "a veritable rock star in the world of neuroscience" (Smith 2009) and was one of the world's hundred most influential people in *Time*'s 2006 ranking.

One of Davidson's best-known messages is that meditation alters the brain. The observation is trivial, since any human activity whatsoever involves and affects the brain. It could be scientifically interesting to know what exactly appears to be altered. In 2003, Davidson and colleagues reported increases in left-sided anterior activation, a pattern associated with positive affect, as well as increases in antibody titers following influenza vaccination in meditators compared with a nonmeditators control group (Davidson et al. 2003). A decade later, Esch (2014) reviewed the effects of meditation and mindfulness that can be detected in the brain as functional and structural alterations, especially in areas related to attention and memory, interoception and sensory processing, and self-regulation, including control of stress and emotions.

While the results are far from surprising and don't really require neuroscience, Davidson's ultimate purpose is to demonstrate that meditation can be put to useful social and psychological uses, such as reducing stress for all or making life easier in maximum-security prisons. Similarly, Tania Singer, the director of the Max Planck Institute for Human Cognitive and Brain Sciences in Leipzig, wishes her neuroimaging research into compassion and empathy to inspire a more peaceful world (Kupferschimdt 2013). In a review of "social influences on neuroplasticity," Davidson and McEwen (2012, 693) write:

> It has also been claimed for thousands of years that specific forms of mental training can produce robust beneficial and enduring effects on behavior. The rigorous investigation of such effects and the neural mechanisms responsible for producing them has only recently become a serious focus of neuroscientific study. The findings that we discuss underscore the structural plasticity of emotional circuitry in response to both acute and chronic stress, particularly alterations of spine density and dendritic length and branching in hippocampus, amygdala and prefrontal cortex.

The modern confirmation of ancient wisdom, lyrically celebrated as "a confluence of streams and a flowering of possibilities" or more soberly as "the convergence of science and the contemplative traditions" (Kabat-Zinn and Davidson 2011, 3) is surely worthwhile for those engaged in the growing enterprise of mindfulness neuroscience (Tang, Hölzel, and Posner 2015) but does not call for spending hundreds of thousands of dollars on brain scans. The empirical results add pieces to our knowledge of the brain, and it is probably relevant to investigate kindness, compassion, and well-being with the same tools that have been used to study hostility, aggression, and suffering. However, the effects of meditation, empathy training, or cognitive therapy do not become more real because they are shown to have neural correlates, nor does knowing that experiential factors shape neural circuits help promote positive social behavior.

Davidson declares that the best way to study the mind is to study the brain (Redwood 2007). Yet neither the neurosciences in general nor neuroimaging in particular can tell us anything about the psychological or social effects of meditation. That is why, when asked about "the link between compassion for others and a sense of personal happiness," Davidson referred to

psychological, not neuroscientific data, citing the well-known experiment "in which participants were given $50 to spend. Half were instructed to spend it on themselves, half to spend it on others. Those who bought gifts for others reported feeling happier after the exercise" (Smith 2009). Illustrating claims for neuroscience by discussing psychological rather than neuroscientific results is a widely shared strategy among neurocultural actors—and one through which they involuntarily reveal the limitations of their own cause (see for example Frith 2007 and the critique by Tallis 2007).

Davidson's 2002 review of affective neuroscience perspectives on depression focused on research about the representation and regulation of emotion in the brain (Davidson et al. 2002a, almost identical to Davidson et al. 2002b). It illustrated a growing emphasis on the brain circuitry "underlying" mood, emotion, and affective disorders and how it coexisted (as it still does) with a focus on brain structures (prefrontal cortex, anterior cingulate cortex, hippocampus, and amygdala). It also illustrated the ultimate goal of the majority of such studies, namely to redefine depression subtypes without relying "on the descriptive nosography of psychiatric diagnosis" but "on a more objective characterization of the specific affective deficits in patients with mood disorders" (Davidson et al. 2002a, 546). In other words, the goal is to deconstruct complex processes into "elementary constituents that can be studied in neural terms" and "examined with objective laboratory measures" instead of self-reports (546).

The heterogeneity of mood disorders is one of the "crucial issues" that the neurologizing of clinical concepts aims to resolve. Symptoms are broadly similar, but the proximal causes can be extremely varied, and even "the underlying mechanisms may differ" (547). Indeed, symptoms come in clusters whose specific features "are likely mediated by different neural circuits despite the fact that they culminate in a set of symptoms that are partially shared" (547). Since descriptive phenomenology does not yield a "clean separation of underlying neural circuitry," one should move beyond it, "toward a more objective, laboratory-based parsing of affective processing abnormalities" (547).

The claim to "objectivity," here identified with what happens in a laboratory, bolsters the ultimate goal of reevaluating the relationships between etiology and nosography by defining symptom clusters "that may arise as a consequence of dysfunctions in specific regions" and thus of offering "sug-

gestions for different ways of parsing the heterogeneity of depression in ways that more directly honor the circuitry of emotion and emotion regulation in the brain" (547). Depression types and symptom profiles "should vary systematically with the location and nature of the abnormality" (565). Thus, the "delineation of brain-based illness models . . . is seen as a promising strategy for redefining our depression nosology" (Mayberg 2007, 729), and neural markers of "at-risk individuals may prove to be more sensitive predictors of subsequent depression and sensitivity to treatment than the clinical predictors we have at present" (Keedwell 2009, 97). From a developmental viewpoint, "identifying depression subtypes based on age of onset and neurobiological characteristics may provide us with more etiologically consistent and uniform diagnostic categories" (Nantel-Vivier and Pihl 2008, 111). We have provided many quotations to show how the usual language, floating between the normative and the expectant (*should*), the permissible, the possible, and the hoped-for (*may*), contrasts with the methodological and empirical technicalities of the research, implicitly favors biological causality over integrative models, and conflates "objectivity" with laboratory research and anatomical description.

A Desire for Causality

In the early 2000s, Davidson and his colleagues' expression "may arise as a consequence of" was as far as they advanced toward understanding the causal mechanisms of depression. In connection with the prefrontal cortex, for instance, they observed that some types of depression "may be caused" by abnormalities in the circuitry that implements positive affect–guided anticipation; similarly, anatomical differences in the brains of patients with mood disorders "might account" for some of the detected functional differences (Davidson et al. 2002a, 548, 550). The existence of hippocampal-dependent Pavlovian conditioning (in the form of an association between places and fear responses) "has important implications for our understanding of the abnormalities that may arise as a consequence of hippocampal dysfunction" (556).

Davidson, however, noted: "Whether hippocampal dysfunction precedes or follows onset of depressive symptomatology is still unknown" (557). "We

do not know," he added, if any of the discussed functional and structural abnormalities "precede the onset of the disorder, co-occur with the onset of the disorder, or follow the expression of the disorder" (565). Such remarks highlight the limits of neurocorrelational research, which is by definition unable to fulfill its own stated goal of differentiating between causes and consequences. By the end of the decade, neither the updated version of the same review (Davidson, Pizzagalli, and Nitschke 2009) nor any of the brain-related articles in the new *International Encyclopedia of Depression* (Ingram 2009) offered a different view or evidence of progress toward the longed-for knowledge of causes and causal mechanisms.

While the scientific literature invariably underlines progress in knowledge of the brain structures said to "subserve" or be involved in depression, it also acknowledges persistent ignorance about causality and localization. In 2008, for example, an article in *Current Directions in Psychological Science* reviewed the status and unresolved issues in neuroimaging and depression. It summarized neurocorrelational research, assessing the role of several brain structures in major depression, and concluded that heightened activity in the limbic structures engaged in emotional experience and expression dampens activation in the dorsal cortical structures involved in affect regulation. The article devoted different sections to distinct structures or systems (the amygdala, the subgenual anterior cingulate cortex, and the dorsolateral prefrontal cortex) and pointed out that identifying "the patterns of functional connectivity that characterize the depressive neural network" was still a challenge for future work (Gotlib and Hamilton 2008, 161).

As the authors made clear, the fact that "neural abnormalities" accompany depression was known before the advent of neuroimaging. But they also recognized that determining the timing of those abnormalities, as can be done by means of activation patterns (for instance, greater-than-normal amygdala reactivity to affective stimuli during a depressive episode), has so far not illuminated their actual connection to the disorder. The results concerning the temporal relation between neural activation and depression as well as the etiological role of neural dysfunction "are complex and do not cohere to tell as clear a story as we would like" (162). Indeed, anomalies can be present in a diagnosed person's brain or precede the onset of the disease "without being involved in its development" (162).

As in earlier literature, the findings discussed in the 2008 *Current Directions* article "underscore the fact that 'depression' refers to a heterogeneous group of disorders that are not carved at their neurobiological joints in *DSM-IV*"; hence the desire to define depression subtypes and symptom profiles "that are related systematically to neural functional and structural abnormalities" (162). In other words, one should go beyond correlations, establish causal links, and amend the nosology of depression on the basis of the disorder's neural substrates. The goal of deconstructing present diagnostic entities in that way is widely shared among researchers in psychiatric neuroimaging (Abou-Saleh 2006). A more recent overview notes again that "the current classification of depression is essentially clinical and aetiological and pathophysiological factors do not play a significant role"; it also comments that thanks to the development of operational criteria, diagnosis has become "reasonably reliable" but that "doubts about validity can be resolved only by a better understanding of pathophysiology" (Cowen 2013, 11).

It is revealing that the metaphor of "parsing" is applied to the *heterogeneity* of depression. It implies that depression *should* not be heterogeneous—or not in the present manner—but, rather, that it should be reconceptualized so as to facilitate its breakdown into clear-cut brain-based nosographic types and components (for example, patterns of brain activation that correspond to individual differences in severity, accompanying symptoms, or treatment response, though some studies also seek biomarkers to differentiate established categories, such as major depression and bipolar disorder; see Kempton et al. 2011).

The main research operation always consists in *correlating*, but the ultimate aim is to *relate causally*. Hence the problem of what to do with the observation (one among dozens of similar ones) that positive correlations between increased functional connectivity in the amygdala network and Geriatric Depression Scale scores in elderly patients with amnestic mild cognitive impairment "suggest" that connectivity in those areas "is related to the degree of depression." It seems impossible to go beyond hazy general conclusions—in this case, that there is an "interactive neural mechanism" between the dysfunction of emotional processing (supported by the amygdala) and cognitive and memory functions (Xie et al. 2008, T259). Although the predominant "functional connectivity" strategy aims at extracting

patterns of covariance, it is assumed that the "activity changes in different locations influence one another" (Mayberg 2007, 729).

The same language that is used when neurobiological interactions and associations are inferred from statistical covariance characterizes a more recent application in psychiatric imaging research, namely diffusion tensor imaging (DTI) studies of white matter hyperintensities. White matter hyperintensities appear on magnetic resonance images as ultrawhite patches that indicate injury to axons. DTI produces neural tract images on the basis of the diffusion of water in tissue (such as the axons in white matter). The variation of diffusion along different spatial directions provides information about diffusion anisotropy (the direction preference of the diffusion process); the results are couched in terms of "fractional anisotropy" (FA), that is to say in degrees of anisotropy (from 0 for isotropic, or homogenous in all directions, to 1 for fully anisotropic). The technique is used to investigate tissue structure and connectivity between regions or points in the brain. While DTI is different from fMRI and other imaging technologies, its basic goal—to correlate pathologies with cerebral locations and circuits—continues to illustrate the assumptions, promises, and limitations of the neurocorrelational logic.

In the field of depression, white matter hyperintensities have been found consistently in elderly unipolar patients. A DTI study of 2009 established that, in comparison with controls, patients with major depressive disorder tend to show lower FA values in the left sagittal stratum; the implied structural changes "may contribute" to the previously detected dysfunction in the limbic-cortical network in depressive patients (Kieseppä et al. 2009, 5). Another meta-analysis of MRI studies of brain volume in MDD observed that some of the areas "involved in" emotion regulation and stress responsiveness exhibit volume reduction. The authors concluded that the integration of MRI and DTI measurements "may improve our understanding of the neural circuitry involved in MDD" and that their own meta-analytic results "strongly suggest that studying brain structure in MDD will contribute to understanding the pathogenesis of this disease" (Koolschijn et al. 2009, 11, 13). They do not explain, however, how pathogenesis can be inferred or demonstrated without some sense of causality or at least temporal direction (see Smith 2015 for numerous other references on white matter hyperintensities).

A 2008 meta-analysis of structural imaging studies remarked that after twenty-five years of scanning bipolar patients and generating over seven thousand MRIs, brain regions "affected in" the disorder remained ill-defined. Given the number of studies considered, significant findings were surprisingly few. There are in fact only three, all "regionally nonspecific." First, bipolar disorder is "associated" with lateral ventricle enlargement and (second) with increased deep white matter hyperintensities; third, lithium use is "associated" with increased total gray matter volume. Conclusion: "There may be genuinely limited structural change in bipolar disorder, or between-study heterogeneity may have obscured other differences" (Kempton et al. 2008, 1026). Regarding major depressive disorder ("unipolar" depression), meta-analytic studies are just as inconclusive: "we still lack information concerning the extent to which structural and functional changes co-occur in a depressed brain," and the "essential neural correlate characteristics for the phenotype of a depressive episode" are still to be discovered (Sacher et al. 2012, 142, 146–147). The high inter- and intrastudy heterogeneity, and the fact that individual investigations are chronically underpowered (that is, they have a small probability of detecting a statistically significant effect), mainly because of excessively small samples, are crucial for understanding such limited achievements. Nevertheless, however much they may be explained by deficient sampling, the occurrence of false positives and false negatives, insufficient control of intervening variables (such as medication), or discrepant nosologies, it is likely that the results also express a variability that is a characteristic feature of the objects and phenomena studied rather than a methodological artifact. (See also Fitzgerald et al. 2008, as well as Hasler 2010, who highlights "the most limited overlap of findings" from functional imaging.)

Neuroimaging depression research has kept looking for treatment-specific biomarkers capable of predicting an individual's improvement in response to a particular treatment and nonresponse to an alternative treatment. For example, it has been suggested that neural response to emotional stimuli in visual cortical areas might be a useful biomarker for identifying patients who will respond favorably to scopolamine (Furey et al. 2013). Similarly, in 2012 another study confirmed earlier suggestions that decreased reactivity to negative words in the subgenual anterior cingulate cortex (sgACC) predicts outcome in cognitive therapy for depression (Siegle et al. 2012; cf. Greicius

et al. 2007). And research published in 2013 found that insula hypometabolism is associated with good results for cognitive behavioral therapy and poor response to escitalopram (a selective serotonin reuptake inhibitor), and insula hypermetabolism, with remission to escitalopram and poor response to the same kind of therapy (McGrath et al. 2013).

Functional, structural, and postmortem studies suggest that sgACC abnormalities are the most solid finding in connection with MDD. This was probably to be expected, given the role of sgACC as a crossroads in a network of structures involving the control of mood, memory, appetite, and sleep. These findings led Helen S. Mayberg, already introduced as a leading figure in depression neuroimaging, to try deep-brain stimulation (DBS) of sgACC as a treatment (the pioneer study was Mayberg et al. 2005; for recent reviews, see Anderson et al. 2012 and Schlaepfer et al. 2014). The apparent discovery of a "depression switch" (Dobbs 2006) received glowing media coverage, with most journalists preferring to ignore Mayberg's failure to disclose her financial ties to medical technology manufacturers (Bass 2010). The hype was dampened in 2013, when the U.S. Food and Drug Administration suspended a trial because it failed the "futility analysis," which monitors whether an experimental treatment has reasonable chances of being shown to be significantly better than the control treatments (Horgan 2014).

While treatments for such devastating conditions as depression are to be welcomed, the cautionary value of the DBS story can be extrapolated to the entire field of predictive neuroimaging and the goal of revamping nosology on purely neurobiological bases. The identification of diagnostic biomarkers is supposed to help redefine bipolarity "in terms of different underlying pathophysiological processes that are likely to include abnormalities in neural circuitry" (de Almeida and Phillips 2013, 115). It is hoped that, in combination with genetics (and taking environmental factors into account), neuroimaging will reveal "neural predispositions" that increase the probability of developing some form of depression (Northoff 2013a). Patients should in the future be "managed" according to "algorithms" based on brain states rather than on clinical examination and patient or professional preference (McGrath et al. 2013). As we saw above, this goal has been a response to the limited efficacy of antidepressants, which is in turn widely attributed to the heterogeneity of the condition and rests on the conviction that "depression" likely refers to multiple diseases, "each with a distinct neurobiology"

(Holtzheimer and Mayberg 2011, 4). However, as meta-analytic studies demonstrate, not only have even the most apparently foundational neuroimaging results been challenged, but the dominant region-of-interest approaches ignore activity (and hence potential anomalies) in regions currently not considered "of interest" for the study of depression, thus sorely weakening the significance of the obtained results (Hamilton et al. 2012).

Again "Just Like Diabetes"

Awareness of these limitations has reinforced the view that abnormalities in neural networks rather than in discrete brain structures underlie psychiatric disorders. It has also contributed to move psychiatric neuroimaging research toward resting-state models (Broyd et al. 2009) and to come in line with emerging approaches to brain connectivity (Price and Drevets 2010) and the concurrent transformation of fMRI research (and brain science in general) into a big-data worldwide endeavor (Lohmann et al. 2013, Thompson et al. 2014). Launched by Marcus Raichle in 2001 (Raichle et al. 2001, Raichle and Snyder 2007), the notion of a "default mode" of brain function has come to describe a "resting state" characterized by very slow neural oscillation (see Callard and Margulies 2011 for a history and larger significance of these notions). The resting state is the "state" of large-scale networks that are active when the subject is awake but not focused on the external environment; their activity is therefore driven neither by tasks nor by external stimuli. Neuroimaging studies of the relationship between the default mode network and mental disorder began in the early 2000s and have shown, for example, that the network is functionally overactive in schizophrenia and hypoactive in Alzheimer's disease (Buckner et al. 2008).

Resting-state research has also gained momentum in the field of depression neuroimaging. A 2012 review of sixteen resting-state fMRI studies published between 2005 and 2011 described various default mode network "abnormalities" in major depression (Wang et al. 2012; see also Veer et al. 2010, not included in the review, as well as the meta-analysis by Alcaro et al. 2010). What is supposed to be their role? The most ambitious resting-state model of major depressive disorder (Northoff et al. 2011) does not aim at "denosologizing" the category. Rather, it preserves *major depressive disorder*

(MDD) in all its heterogeneity—at the level of its symptoms, the affects it encompasses (anxiety, sadness, grief, panic, pain), the bodily systems it involves (from the vegetative and endocrine to the cognitive), the neuroanatomical regions observed to be "abnormal" in the condition, and the biochemistry pertaining to each of those systems and regions. It then seeks to correlate those different levels, mustering a vast amount of neuroanatomical, psychopathological, and biochemical information to turn major depression into a specific brain system–network disorder.

MDD turns out in this model to be characterized by a subcortical-cortical imbalance, with resting-state hyperactivity in some regions and hypoactivity in others. Certain subcortical and cortical regions are hyperactive in the resting state, while others (especially cortical) show hypoactivity. Such abnormal resting-state patterns "may strongly impact the neural processing of external stimuli" in the regions concerned, and that "may enable and predispose the occurrence" of major depression symptoms (7). Higher affective and cognitive functions are "highjacked [*sic*]" by subcortical primary-process emotional systems (1, 11). For example, depressive hopelessness arises by way of a "psychopathologically specific" relationship with resting-state activity in the ventromedial prefrontal cortex (VMPFC). On the one hand, in depressed individuals, elevated resting-state activity in the perigenual anterior cingulate cortex (PACC) and the VMPFC has been found to correlate with high scores on a self-report inventory known as the Beck Hopelessness Scale. On the other hand, in "healthy" subjects, PACC and VMPFC are associated with the slowing of time in subjective perception. The "abnormally elevated" VMPFC resting-state activity therefore "seems to impair anticipation and hence one's experience of extending hopes into the future" and "to block the ability of MDD patients to project hope into the future, thereby promoting hopelessness and ultimately helplessness" (10).

The authors of the model acknowledge that such causal pathways are speculative. For us, the most revealing feature of their work is that they offer as primarily predictive and etiological a neuroanatomical model very largely based on correlational neuroimaging data. For example: "One could expect that elevated resting-state activity in these regions *would lead* to an increased self-related processing and hence to abnormally increased personal concerns in MDD patients" (11). A study actually shows that, in MDD patients as compared to "healthy" subjects, increased self-focus in connection

with negative emotional stimuli correlates with significantly lower signal intensities in various subcortical and cortical regions (Grimm et al. 2009). But here we remain in the domain of correlations, not of factors "leading" anywhere.

The authors of the model we just summarized believe that if social factors known to be associated with the onset of depression were shown "to impact either the resting state-level itself or the degree of rest-stimulus interaction," then the model could become neurosocial (Northoff et al. 2011, 14). They also realize that abnormal resting-state activity is likely to be a necessary rather than a sufficient condition of depression, acting as a "neural predisposition," a "susceptibility marker," a "risk factor" (14). However, in spite of its integrative purpose, the model ultimately accounts for depression in neuroanatomical terms and depicts it as "just like diabetes." And it does so explicitly: Low insulin, the authors explain, "metaphorically corresponds" to the abnormally elevated resting state; like abnormally high blood sugar, which interacts with biochemical mechanisms in diverse bodily systems, such a state has "comparable effects on diverse brain-mind subsystems"—psychopathological effects comparable to those of diabetes, like becoming blind or suffering gangrene (15).

The neurobiologists who, working with laboratory mice, induced a network and behavioral chronic stress phenotype and then reversed it by stimulating the prefrontal cortex-to-amygdala circuit did not hesitate adventurously to attribute a causal nature to the "central brain mechanism underlying MDD" they apparently identified and to announce treatments directly targeting the relevant network interactions (Hultman et al. 2016, 449). In contrast, and quite understandably, authors engaged in neuroimaging depression research abstain from speaking directly in terms of causes. Abnormalities "play a role," are "involved in," "impact on," or "may contribute" to mental disorder; functional and anatomical differences or the activation of brain structures do not reveal the cause of depressive symptoms but only have "temporal relations" with their expression or are "significantly positively associated" with them. In the end, research cannot decide "whether particular brain changes in depression are a consequence of symptoms or due to underlying neural vulnerabilities," which are themselves at the beginning of an etiological chain (Graham et al. 2013, 424). There is much to commend

in a cautious attitude toward causal connections. Yet the intentionally imprecise language not only reveals ambivalence regarding causality but is also symptomatic of a historical situation. Although the existence of a link between brain chemicals and mood disorders has been known since the 1950s, when drugs that alter those substances were found to relieve these disorders, in 2017 it is still unknown if changes in neurotransmitter levels cause depression or the other way around; the same is acknowledged in connection with volumetric, anatomical, and neuroimaging data.

As we have noted, explicit discourses about physiopathology and diagnosis are less about causes than about biomarkers. These are being sought for at different levels: some concern "predisposition" and hence the probability of developing a disorder under particular conditions; others, valuable for diagnosis, are expected to indicate, with a high level of probability, that the patient suffers from such and such pathological condition; yet others bear on treatment and thus on the likelihood that an individual will respond or not to a given pharmacological or psychological therapy. The overall goal of the shift toward biomarkers is to underpin diagnoses with pathophysiological evidence and to allow disorders to be reconceptualized (eventually in a denosologizing perspective) according to biological criteria. As already mentioned, biomarkers reflect correlational results at the population level and lack predictive power for the individual. But the explicit acknowledgment that this is so constantly clashes with the way in which goals and results are presented and discussed.

As far as causality is concerned, the situation is essentially the same with regard to neuroimaging, including resting-state results relevant to ADHD (e.g., Posner et al. 2013), schizophrenia (e.g., Arbabshirani et al. 2013), and autism (e.g., Deshpande et al. 2013). The slippage from neuroimaging correlations to etiological causation observed in all these areas has an equivalent in the diagnostic realm. In the field of depression, as in ADHD, schizophrenia, and autism, neuroimaging has been advertised as capable of becoming a diagnostic tool, and *Time* has even announced, "Brain Scans Could Become EKGs [electrocardiograms] for Mental Disorders" (Khamsi 2013). Everything leads the public not to notice that scans are said to correlate with those disorders only *after* a clinical diagnosis has been made. The hope of bypassing the challenges and apparent messiness of the clinic and of auto-

mating diagnosis by way of brain scans drives the entire field of psychiatric neuroimaging; the probabilistic rationale of the biomarker is inconsistent with the expectations raised by those who, in principle, adhere to that rationale.

Moreover, as often explained, psychopathology "is increasingly viewed from a circuit perspective in which a disorder stems not from circumscribed anomalies in discrete brain regions, but rather from impairments in distributed neural networks" (Posner, Park, and Wang 2014, 3). Yet, in the same way that the shift to biomarkers coexists with a desire for causality, the emphasis on neurocircuitry has in most cases barely altered the localizationist logic that drives the research. Connectome-based imaging studies, also correlational, have identified "disrupted topological organization of large-scale functional and structural brain networks in depression," but these "pathologic networks *associated* with depression" cannot be said to be anything beyond "*potentially* valuable biomarkers" (Gong and He 2015, 223, our emphasis). The establishment of meaningful links is left for an unspecified but intensely publicized future.

Finally, the denosologizing effect of the quest for biomarkers, which is expected to break down categories such as depression, is in tension with keeping depression as a category in Global Mental Health and in "burden-of-disease" calculations; turning away from *ICD*- and *DSM*-based diagnoses clashes with the claim that depression, as defined by these classifications, is a major cause of disability worldwide. GMH advocates propose shifting tasks to communities and local primary healthcare settings with appropriately trained lay workers. There are indications that this mode of intervention is effective for depression (e.g., Patel et al. 2011), and it is praised for bringing together bottom-up consultation processes with a top-down evidence-based approach.

The question whether and how mental illness is universal, as well as the globalization of mental illness and mental health, have given rise to acrimonious controversy (see, for a recent exchange, Miller 2014; Summerfield, 2012, 2014; White 2013).[6] Independently of local knowledge systems, the misery of people who would be diagnosed as suffering from brain disorders according to *ICD* or *DSM* is undeniable, and the way they are treated in many cultures around the world fully warrants the indignation of the medical

anthropologist and cross-cultural psychiatrist Arthur Kleinman (2009), who depicted the current situation as a "failure of humanity." At the practical field level, making up for such a failure must be a top priority. That, however, does not eliminate the difficulty, which global mental health actors recognize, of somehow integrating into GMH the science we have sketched here. Indications of useful synergies between GMH and clinical neuroscience have so far remained at a general level—for example, reinforcing what is known about the impact of social and financial deprivation on mental well-being (Stein et al. 2015).

The challenges are considerable, since the neurobiological outlook on psychic distress, which we have explored via neuroimaging approaches, embodies notions of objectivity and a desire for causality that are difficult to reconcile with phenomenological and first-person understandings. We have considered such a situation by focusing on attempts at revamping nosography and diagnostics on the basis of experimental work and have thus remained in the framework of specialized knowledge production and assessment. We deal next with the other end of the circle, with contexts in which *neuro* idioms and bits of neurobiological information are incorporated into the lives of individuals and groups. Especially in the case of the autism spectrum, such incorporation serves mainly but not exclusively to depathologize and redescribe the diagnosed conditions. But seeing autism as a way of being rather than an illness is not to everyone's liking, and the neurobiologization of the spectrum gives rise to camps that defend conflicting forms of subjectivity and sociality, thus highlighting again the constitutive ambivalence of cerebralizing processes.

Neurodiversity

In an often-quoted article of 1998, significantly entitled "Thoughts on Finding Myself Differently Brained," the autistic self-advocate Jane Meyerding wrote that she "was surprised to find [herself] moving into the realm of neurology." Since the 1990s, indeed, autism advocacy has organized itself largely around "neurology" or, more accurately, as a *neurodiversity* movement. So far the movement has been dominated by people diagnosed with Asperger syndrome and other forms of high-functioning autism (although some

prominent self-advocates, such as Amanda Baggs, do not speak and define themselves as "low functioning").[7] Asperger as a formal diagnosis has disappeared from the *DSM-5* and is subsumed as the high-functioning end of a new "Autism Spectrum Disorder." Individuals at that end of the spectrum believe that their condition is not a disease to be treated and, if possible, cured, but rather a human specificity that must be respected as such.[8] Their being unlike "neurotypicals" derives in their view from a brain "wiring" that is different but not abnormal. Such identity claims manifest what the activist Judy Singer (1999) has called "neurological self-awareness." Indeed, autistics' identitarian claims have gone hand in hand with the cerebralization of their condition. As we shall see, the "person-first language" generally supported by the disability rights movement is not always well received within autism self-advocacy groups, for whom the expression "person with autism" suggests that the condition is not constitutive of the individual.[9] The *neuro-* prefix and a usually imprecise *neuro* vocabulary serve to construe autism as a positive attribute and to demonstrate the legitimacy of the autistic experience. Cerebralization, which as we saw is driven by a quest for causality and "objectivity," thus sustains subjectivation.

Autism as a Biosocial Phenomenon

The emergence of the term "neurodiversity" and the corresponding movement in the late 1990s should be analyzed within a broad perspective. On the one hand, it belongs in the history of disability movements (Charlton 2000; Corker and French 1999; Corker and Shakespeare 2004; Davis 1995, 2002; Shapiro 1993). On the other hand, it instantiates the extensive and diversified societal impact of neuroscientific knowledge and practices. The neurodiversity movement is historically connected to a turn away from psychoanalysis and toward a neurobiological and genetic understanding of autism. Especially in the United States, from the 1940s to the mid-1970s, psychoanalytic explanations were paramount both in psychiatric theory and clinical practice (Nadesan 2005). The later shift was embodied in procure and anti-cure discourses, both expressed in neurodiversity advocacy groups and in parent and practitioner groups favorable to behavioral and

psychopharmacological therapies (Chamak 2008; Silberman 2015; Silverman 2008a, 2008b, 2012).

Other roots of the neurodiversity outlook are to be found in the antipsychiatry movement as well as in the emergence of psychiatric consumer/survivor/ex-patients groups (Graby 2015). From a nosological perspective, the categories comprised under the umbrella of "neurodiversity" (autism is the main one, but see Armstrong 2010 and Hendrickx 2010) are included in the *DSM*, overlap with learning disabilities, and historically fall somewhere between psychiatric diagnosis and disability, between mental illness and mental retardation (Eyal et al. 2010, Graby 2015). Autistic self-advocates frequently associate themselves explicitly with the Deaf rights movements, and some of them have been inspired by the American "independent living" movement (Silberman 2015).

Disability studies and the disability rights movement share a commitment to the social model of disability and the rejection of the medical model (Oliver 1990, Shakespeare 2006, Wendell 1996). The social model, which distinguishes between *impairment* and *disability*, has been criticized for downplaying the importance of impairment and consequently mixing up disabled and nondisabled people. However, underlying the pursuit of a barrier-free world is the assumption that discriminations are not attributable to individuals' impairments but result from society's failure to accommodate them. Similarly, autistic self-advocates do not reject impairment labels (for they consider their autism to be neurologically real) but reclaim them from medicine and turn them into the basis of positive identities and into the justification for claiming rights and compensations. Impairment has thus become a "difference to be expected and respected on its own terms in a diverse society" (Cameron 2008, 24), and its biological nature has allowed activists to redescribe mental disorder as a sui generis form of human diversity or even of human consciousness (Boundy 2008).

As mentioned, the neurodiversity movement also draws on antipsychiatry and the "survivor's" groups, including radical strands such as Mad Pride and its appropriation of traditionally derogatory terms such as "psycho," "crazy," and "nut." These movements wish to deconstruct stereotyped and stigmatizing representations in science, medicine, and public culture at large (Rowland 2015). While they developed as "revolts from below" (Crossley 1998), they historically followed the "revolt from above" that

began inside professional psychiatry with Robert Laing and David Cooper in the United Kingdom, Thomas Szasz in the United States, and Franco Basaglia in Italy. Laing's *The Divided Self* was published in 1960, Cooper's *Psychiatry and Anti-Psychiatry* in 1967, and the Mental Patients' Union appeared in Britain in 1973. Antipsychiatry politicized the psychiatric field, opened up a space for the expression of diagnosed persons, and offered them alternative discourses about madness and normality. Although users were involved in the antipsychiatry movement from the beginning, "survivor's" groups emerged a decade later. While the former belongs in terms of style and tactics to the counterculture of the early 1960s, the latter were more politicized and, influenced by Marxism, located patient politics in the context of revolutionary action and a general class struggle (Crossley 1998, 2006).

In spite of many overlaps in inspiration and rhetoric, the neurodiversity movement differs from antipsychiatry and patients' crusades in its interpretation of the ontological status of mind, brain, and body. While neurodiversity advocates admit that their conditions are neurologically real and therefore represent physically based differences, the earlier movements tended to reject the idea of fundamental and materially real divergences between them and "normal" individuals (Graby 2015, Jones and Kelly 2015). Thus, in each camp the brain ended up fulfilling opposite ideological functions. The neurodiverse see the brain as the somatic seat of legitimate identities, different from the "neurotypical"; antipsychiatry identifies it with the reductionism and pathologizing impulse of biological psychiatry. In short, neurodiversity movements share the depathologizing rhetoric of antipsychiatry and user/survivors movements but at an ontological level have more affinities with the disability rights movements, which instead of downplaying impairment redescribe it in positive terms.

The first associations for the parents of autistic children appeared in the mid-1960s. The National Autistic Society was founded in London in 1962. In 1965, Bernard Rimland, author of *Infantile Autism: The Syndrome and Its Implications for a Neural Theory of Behavior*, together with the pioneer activist mother Ruth Sullivan and other parents, established the Autism Society of America.[10] Similar groups soon mushroomed in other countries (Chamak 2008, Chamak and Bonniau 2013, Dekker 2006, Shapiro 2006, Wing 1997). The rise of the Internet in the early 1990s was a major turning point for both

parent and self-advocacy groups. One of the earliest online parents lists, the Autism and Developmental Disabilities List (AUTISM List), promoted applied behavioral analysis (ABA), a form of cognitive behavioral therapy, as a treatment for autistic children. Diagnosed adults who felt that both experts and families misunderstood or ignored them resisted the emphasis on curing. Australian and American activists then formed Autism Network International (ANI) in 1992, supplemented since 1994 by Autism Network International Listserv (ANI-L). The first issue of their newsletter *Our Voice* came out in 1992, and the first autistic retreat (called Autreat) took place in 1996 (Bagatell 2010, Chamak 2008, Orsini 2012, Silverman 2008a, Silberman 2015).

Although nonautistics may adhere to ANI, all decision making is done by autistics alone. The motto "By autistics for autistics" captures the network's core values and expresses the principle of the disability movement at large, "Nothing about us without us" (Charlton 2000, Shapiro 1993). Their goal is to fight the vision of autism conveyed by professionals and families who share an "obsession" with a cure, which ANI considers not merely disrespectful of the autistic way of life but an attempt to erase legitimate difference. Hence the strength of the anti-cure attitude in this camp (Sinclair 2005).

In the opposite camp stand organizations such as the National Alliance for Autism Research (NAAR), founded in 1994, and the Cure Autism Now Foundation (CAN).[11] The latter was created in 1995 by the parents of an autistic child and brings together families, medical doctors, and scientists to support biomedical research and education. NAAR and CAN have now merged with Autism Speaks.[12] CAN is a major target for autism movement activists, who accuse it of demonizing autistics and frightening their families, promoting narrow viewpoints on the disorder, and neglecting autistic adults' life experiences.

The growth of self-advocacy movements and their enhanced exposure in the media has intensified the political clash between the anti-cure and the pro-cure.[13] One of the most controversial issues concerns applied behavior analysis, the above-mentioned therapy that employs learning theory to improve "socially significant behaviors."[14] For many parents, ABA is the only way of helping their autistic children make some progress toward establishing visual contact and performing limited cognitive tasks. In contrast, for autism militants, ABA represses autistics' natural modes of expression (Dawson 2004). In the United States and Canada, the debate has reached

the courts: While parents fight to obtain governmental support or make health insurance companies pay for the therapy, which is extremely expensive, neurodiversity advocates maintain that autism is not an illness and that attempts to cure it violate autistic rights (Baker 2011; Dawson 2004; Orsini 2009, 2012). The latter position may provide reasons for refusing to subsidize treatments, but the most adamant partisans of neurodiversity are willing to take the risk. For them, the search for therapies manifests denial and intolerance toward differences and enacts eugenic and genocidal policies; in 2004, some went as far as petitioning the United Nations for recognition as a "minority social group" that deserves protection against "discrimination" and "inhuman treatment" (Nelson 2004). Their position is one of the logical consequences of cerebralization, which in this case acts as a normalizing rather than as a pathologizing mechanism. Thus the only "distress" they claim to suffer is not caused by a pathology but by society's lack of acceptance.

The biosocial field of autism is not entirely structured by such radical polarizations, but it must nonetheless face the question whether mental disorders are necessarily harmful and whether people who have "symptoms" that don't make them suffer or put them at an increased risk of experiencing future distress or impairment should be considered mentally ill (Cooper 2015). Under *DSM-IV*, such people were not diagnosed; in *DSM-5* they are. The high-functioning end of the autism spectrum is a paradigmatic case, since some people (who would have formerly been diagnosed as having Asperger syndrome) meet the diagnostic criteria but are not unhappy and function well in society. Treatments are acceptable to alleviate discomforts they share with millions of undiagnosed individuals. Thus, the prominent neurodiversity advocate Temple Grandin, a high-functioning autistic who provided the title for Oliver Sacks's *An Anthropologist on Mars* (that is how she says she feels around neurotypicals), is not against medication. However, as is clear from her autobiography *Thinking in Pictures* (1995), she wishes to limit it to secondary symptoms such as anxiety and not to autism itself. Judy Singer, another activist, thinks that drugs are acceptable as long as they aim to relieve suffering, not change individuals' personality. Fernando Cotta, the president of the Brazilian Autistic Pride movement, concurs that respecting autistics is not incompatible with medication; for example, if an autistic "has attention problems, he can take something that can help him, just as somebody who has the flu takes an anti-flu medicament" (Lage 2006).

In short, some self-advocates insist that autism itself should not be treated but have a pragmatic attitude toward medical interventions.

Neither do all parents oppose self-advocacy movements, nor do all autistic adults favor neurodiversity.[15] The latter sometimes find it difficult to harmonize their identities in the autistic communities and in the neurotypical world, and this tension can become an important source of anxiety and suffering (Bagatell 2007). Moreover, some autistic adults do want to be cured, but they seem to represent a largely silent population. "Most persons with an autism-spectrum disorder have never expressed their opinions on someone's blog and never will," affirms Jonathan Mitchell, who suffers from a mild autism spectrum disorder, blogs against neurodiversity, and notes that "the neurodiverse often reach a vulnerable audience, as many persons on the spectrum have low self-esteem. Neurodiversity provides a tempting escape valve" (quoted in Solomon 2008).[16] Sue Rubin, a low-functioning autistic who is the subject of the documentary *Autism Is a World*, emphasizes that whereas high-functioning autistics tend to be against a cure, low-functioning autistics generally hold the opposite position. For her, "the thought of a gold pot of a potion with a cure really would be wonderful." She writes:

> As a person who lives with autism daily and will not live a normal life, I find people who are high functioning and saying society should not look for a cure offensive. They have no idea what our lives are like. Killing autism lets me enjoy a life with great friends and allows me to go to college, but I must never let down my guard or autism will take over. I don't want any more children to live, as I must, in this constant state of war. (Rubin 2005)

For persons such as Rubin, it is the advocates of neurodiversity who are insensitive and lack respect.

Finally, the relationship between parent and self-advocacy groups differs considerably depending on national context. Whereas it can be highly conflictual in the United States, the United Kingdom, and Australia, in France autistic self-advocacy remains under the influence of parent associations (Chamak 2008, 2014; Chamak and Bonniau 2013). Thus, while the biosocial field of autism seems to be extremely polarized at first glance, under closer inspection it appears to be more complex and to provide room for a variety of nuanced positions. It is therefore more appropriate to characterize it as including discourses, individuals, and groups that, while antagonistic

in some respects, in others overlap or support one another, rather than to characterize it as a clash of homogeneous groups holding sharply antithetical positions.

Autistic Cultures and Neurodiversity

The term "neurodiversity" is generally credited to Judy Singer, a sociologist diagnosed with Asperger syndrome, who used it in a 1999 article titled "Why Can't You Be Normal for Once in Your Life? From a 'Problem with No Name' to the Emergence of a New Category of Difference" (the title echoes Betty Friedan's 1963 *The Feminine Mystique*, whose first chapter identified American women's dissatisfaction and yearning as "The Problem That Has No Name"). The term also appeared in Jane Meyerding's 1998 "Thoughts on Finding Myself Differently Brained," and Singer herself wrote, "I am not sure if I coined this word, or whether it's just 'in the air,' part of the zeitgeist." As explained, "neurodiversity" proclaims that some features usually associated with illness are in fact only atypical or "neurodivergent" (Harmon 2004a, 2004b, 2004c). Disability studies scholars see the rise of neurodiversity as a critique of the dominant discourse of dependency and abnormality, a celebration of difference, and an assertion of pride that, beyond the circle of the disabled, their families, physicians, and caretakers, reaches into the domain of public health and educational policies (Corker 1999, Swain and Cameron 1999). "If you do not believe there is a disability, if you do not believe there is anything that needs to be 'cured' or genetically prevented—that disability is indeed little more than a social construction—then you will likewise be freed from the need for a cure" (Cheu 2004, 209).

These ideas and the social forms they animate exist chiefly by way of the Internet. "Deaf culture" (Padden and Humphries 2006) has in this connection inspired the development of "autistic culture."[17] An autistic self-advocate states it explicitly: "Much like the deaf community, we autistics are building an emergent culture. We individuals, with our cultures of one, are building a culture of many" (Prince-Hughes 2004, 7; see also Davidson 2008). The web has become the privileged vehicle for advocacy and networks, enabling "what was thought impossible, to bind autistics together into groups" (Singer 1999, 67). That is why it seems to be for them what sign language is

for the deaf or Braille for the blind (Blume 1997a). Self-advocates thus craft themselves as a "new immigrant group on line, sailing to strange neurological shores on the Internet" (Blume 1997b).

Cyberspace has turned into a vehicle and territory for new forms of "biosociality." Prominent among the phenomena sustained by websites and blogs is the emergence of a specific self-advocate vocabulary for categorizing persons (Bagatell 2007): *Aspie*, *Cousin* (someone who is not clinically autistic but still similar enough to autistic people to be part of their culture), *Neurotypical*, *Autistic* or *Autie* (preferred to the politically correct "person with autism"), or *Curebie* (derogatory term for those who wish to cure autism). Websites also recommend fictional and science literature; various online support organizations, blogs, and chat rooms facilitate interaction among autistic individuals, provide clarifications on symptoms, enable the sharing of experiences, and help their users make friends or find partners (Chamak 2008; Jurecic 2007; Silverman 2008a, 2008b). All of this combines to promote awareness and empower a community that (at the initiative of Aspies for Freedom) has since 2005 celebrated on July 18 its own Pride Day.[18]

Websites like Proudly Autistic include a marketplace where one can purchase T-shirts, tote bags, mouse pads, stickers, postcards, and greeting cards proclaiming "No more 'Trained Seal' Treatments!" (against ABA), "Not Being Able to Speak Is Not the Same as Not Having Anything to Say," or "I Am Autistic. What's Your Excuse?"[19] As Nancy Bagatell (2007) shows in the case of Ben's "coming out" as an autistic, these objects may function as powerful "tools of identity." Ben's trajectory recalls the gay and lesbian coming out, which can be understood as a political act with significant liberating or destructive consequences (Davidson 2008; Valentine, Skelton, and Butler 2003).

Identitarian Issues: Being *Autistic or* Having *Autism?*

This brings us back to the fundamental question of identity. Parent and professional associations that support the search for a cure usually refuse to acknowledge the very existence of an identity issue. For them, autism is simply a disease. Children *are not* autistic, they *have* autism. As Kit Weintraub (2005), the mother of two autistic children and a board member of Families

for Early Autism Treatment, wrote in response to the autistic self-advocate Michelle Dawson's (2004) critique of the "autism-ABA industry,"

> I love my children, but *I do not love autism*. My children are not part of a select group of superior beings named "autistics." They have *autism*, a neurological impairment devastating in its implications for their lives, if left untreated. . . . it is no more normal to be autistic than it is to have spina bifida. (Weintraub 2005)

Although online discussion groups demonstrate that some autistics do not see their condition as a positive part of their selves (Brownlow 2007), others do consider it as essentially constitutive of who they are. Autism, they argue, is "pervasive, it colors every experience, every sensation, perception, thought, emotion, and encounter, every aspect of existence" (Sinclair 1993). This is also the reason why many activists adopt self-descriptions such as *autistic* or *aspie*, which present autism as an integral part of their identity (Silverman 2008b). For the autism rights activist Jim Sinclair (1999), "person with autism" suggests that autism "is something *bad*—so bad that it isn't even consistent with being a person." Dawson thinks that using that expression would be as bizarre as using "person with femaleness" to designate a woman (quoted in Harmon 2004c). Attitudes toward cure and therapies are consistent with these various positions.

As mentioned, autistic identity is sometimes experienced as a source of pride, even as a "gift" (Antonetta 2005). The emergence of this feeling may begin with a sense of reassurance. High-functioning autistics have reported the "comfort" they felt upon being diagnosed. "Finally an explanation, finally a sense of why and how," wrote a man diagnosed with Asperger syndrome at age thirty-six, shortly after his four-year-old son was diagnosed with the same disorder (Shapiro 2006). Ian Hacking (2006) has noted that "many misfit adults now recognize themselves as autistics, or so they say. It really helps to be able to put a label to your oddities. It brings a kind of peace: so that is what I am." Judy Singer (1999, 62) expounds on the "benefits of a clear identity," and Jane Meyerding (2003) speaks of the "aha! moment" when she discovered autism as an explanation. That led to her finding a community whose thought patterns and modes of expression she identified with: "All my life, I have been forced to translate, translate, translate. Now, suddenly, I have people who speak my own language." Autistics may use the

diagnostic label positively; the autism idiom generates "signposts" and "shorthands," as Meyerding says, that help them position themselves with respect to the surrounding culture. Labeling thus metamorphoses from signal of stigma to instrument of liberation.

Asserting identity is often associated with rejecting psychological explanations and psychotherapies. The latter can be seen not only as a waste of time but also as downright dangerous. For example, a woman diagnosed with autism said that after spending her teens "in a state of suicidal clinical depression as a result of bullying and feeling that I must be a failure or insane for being different," she found this opinion "only reinforced by the psychotherapist I got sent to, who decided that all my problems must be the result of 'sexual repression.'" Proud to have "walked out after six sessions," she welcomed the autism diagnosis as "the best thing" that ever happened to her (quoted in Blume 1997a).

As highlighted by the very notion of neurodiversity, autistics' claim to a specific identity is linked to the cerebralization of their condition. As the anthropologist Tanya Luhrmann shows in her ethnographic account of American psychiatry, the biologization and neurologization of mental illnesses tend to bracket off subjective and experiential dimensions and to convey the positive message that "the body is always morally innocent" (Luhrmann 2000, 8). Talking about her own experience of manic depression, the anthropologist Emily Martin (2007, 13) recounts, "I often heard from my psychiatrist that my problem was related to my neurotransmitters, and I always found this comforting." In contrast, "if something is in the mind, it can be controlled and mastered, and a person who fails to do so is morally at fault" (8). When a biologically oriented psychiatrist speaks of depression as a cardiologist speaks about cardiopathies, a distance is introduced between the patient and the disease.

We have seen that the destigmatizing effects of the biological interpretation of mental illness have been overestimated. Yet, for all the criticism that "blaming the brain" (Valenstein 1998) may deserve, it sometimes has freed both patients and families from blame for manic depression, eating disorders, anorexia, autism, or schizophrenia. Thus, in the case of families who financially support neuroimaging research, their adherence to a neurobiological approach is consistent with the widespread rejection of the notorious late-1940s theory of the schizophrenogenic mother. At the same time,

it reflects the conviction that psychological ailments can be cured, should be covered by health insurance, and should benefit from other forms of compensation (Martin 2007). As it turns out, it is easier for patients and their relatives to accept a diagnosis of *bipolar disorder*, which has become associated with brain states, than one of *manic depression* or *manic-depressive disorder* (other labels for the same condition), which tend to be perceived as psychological (Montanini and Banzato 2012). In the latter case, "mental illness is in your mind and in your emotional reactions to people. It is your 'you'" (Luhrmann 2000, 6). In contrast, a cerebral disorder is only connected to the body, in the same sense that a heart attack may affect your mind but "is" in your body.

Biological explanations contribute to bring together patients, families, and scientists to spread information about a condition, combat stigma, support patients, and drive the search for treatments (Gibbon and Novas 2008a, Rose 2007). The claim to neurodiversity is connected to a "naturalized" identity, according to which I am who and what I am because my brain is "wired" in a certain way. In his discussion of the "looping effects" of diagnostic labeling, Ian Hacking (1995, 2002) has distinguished labeling from above and from below. Originating as it does with the patients rather than the doctors, neurodiversity illustrates labeling from below, even if it necessarily feeds on information "from above." For autistic self-advocates, neurologizing their condition helps redefine it in terms of an organically localized *difference*. There is, however, no consensus on the neurobiological etiology of autism.

Contemporary research uses several approaches to define biological markers: one may search for the characteristics of the "autistic brain," look for autism genotype(s), or investigate comorbidity and environmental influences (Nadesan 2005). Viewing autism as a brain dysfunction (Fombonne 2003, Freeman and Cronin 2002, Wing 1997), psychiatrists and neuroscientists have tried to discover the disorder's "brain address" (Wickelgren 2005, 1856) and have even suggested that the autistic brain is an extreme form of the "male brain" (Baron-Cohen 2002). In view of such heterogeneity, it has been proposed that autism is best understood as a "multi-system disorder" (Charman 2006). As far as neuroimaging approaches are concerned, they have generated great expectations; actually, however, they "seldom provide data on an individual level, do not yet have well-accepted standards or replicability

across time or site . . . and have rarely addressed questions of specificity of findings" (Lord and Jones 2012, 491). In other words, and despite considerable amounts of research, there is still no convincing, well-replicated brain-based autism biomarker (Walsh et al. 2011). In spite of that, "autism has retained its identity as a genetic disorder of the brain" (Silverman 2012, 155).

Autistic advocates place less emphasis on particular biomarkers than on the more general fact that, as Temple Grandin put it, autism "is a neurological disorder. A child is born with it. It's caused by immature development of the brain . . . and not by bad parenting or the environment" (quoted in Blume 1997a). Similarly, for the Dutch self-advocate Martijn Dekker (2006), autism "is neither a physical (bodily) disability, nor a mental illness: it is a neurological disability." His making the brain something different from the body illustrates that organ's special ontological and functional status. All sides of the autism community share his position. Thus, self-advocates' bête noire, Cure Autism Now, has sponsored the creation of an Autism Genetic Resource Exchange (Silverman 2008a), the "world's first collaborative gene bank for autism."[20] (Gene expression patterns in the brain, rather than genomes per se, are increasingly understood as crucial; hence the rising importance of *neurogenetics*. See for example Jones 2012).

Even though some groups have supported alternative causes for autism (for example, mercury poisoning; Bumiller 2008), there is a generalized preference for brain-based explanations, which have emerged as part of the spread of neuroscientific claims beyond the laboratory. It is therefore not a concern for mental disorders that contributes to the "elevation of neurology" but the other way around: this "elevation" provides a major reason for the increasing attention paid to those disorders, including autism (Blume 1997a).

Loving and Hating One's Brain

Whether superficial or well informed, wacky or serious, neurodiversity advocates' engagement with the neurosciences has become a major vehicle for fashioning personal identities. The process began in the late 1990s. We have already mentioned Jane Meyerding's remembering how "surprised" she was to find herself "moving into the realm of neurology." What does her recol-

lection tell us about subjectivation processes within autistic culture? Can we say that some self-advocates become cerebral subjects by way of their engagement with the neurosciences and their claims to *neuro*diversity? Does defining oneself as *neurodiverse* illustrate what Joseph Dumit (2004) calls "objective self-fashioning," that is, the incorporation into one's self-definition of scientific or expert ideas, terms and metaphors? Do all self-advocates mobilize brain vocabularies in the same way? Are there distinctive versions of the "brain story" (Martin 2009)? And how are they shaped differently in blogs, discussion groups, autobiographies, conferences? What sort of information is being used? Is it drawn from scientific articles, popular accounts in magazines, movies, or novels? Who is addressing whom, and in which arenas? How are discourses adapted to different contexts and audiences? Answering these questions is not easy.

There is a substantial amount of social science research on these matters, conducted especially on online materials or by way of ethnographic approaches, focusing on the social construction of disability in the new media (Coleman 2010; Goggin and Newell 2003; Hallett and Barber 2014; Jaeger 2012; Keim-Malpass, Steeves, and Kennedy 2014; Kozinets 2010; Snodgrass 2014). The blogosphere being a major setting for the development and consolidation of disabled identities, the use of the Internet, especially by blind, deaf, and autistic people, has gained considerable attention (Goggin and Noonan 2006). The web has become an essential space of debate and identity development for autistic persons (Biever 2007, Blume 1997a, Dekker 2006, Kenway 2009). Many empirical studies deal with autism in cyberspace.[21] Qualitative research is also being conducted on autistic people's writings, particularly autobiographies and memoirs (Chamak et al. 2008; Davidson 2007, 2008; Hacking 2009; Osteen 2008). Books such as *Voices from the Spectrum* (Ariel and Naseef 2006) collect first-hand stories by parents, siblings, people diagnosed with autism, and mental health professionals, and ethnographic accounts examine identity construction by autistic individuals (Bagatell 2007, 2010; Bertilsdotter Rosqvistab, Brownlow, and O'Dell 2013; Jurecic 2007; Ochs and Solomon 2010; Prince 2010).

The range of subjects is very broad, encompassing differences in parents' and patients' understanding of autism, social interaction and alienation, perceptual differences and sensory distortion, the expression and management of emotion, comprehension and communication difficulties, desire and

relationships, the role of the Internet and support communities, diagnosis, self-diagnosis, and the role of "expert" knowledge. But it is self-advocates' already mentioned "neurological self-awareness" and "preference for neurology" (Singer 1999) that most immediately captures the dynamics of the cerebral subject.

Muskie, the creator of a satirical online Institute for the Study of the Neurologically Typical, declares: "My brain is a jewel." "I am," he writes, "in awe of the mind that I have. I and my experience of life is not inferior, and may be *superior*, to the NT experience of life." Though also branded a "Curebie," aspie Michael John Carley (whose son has likewise been diagnosed) rejoices:

> *I love the way my brain works*, I always have and it's one of the things I can now admit to myself. I like the way I think in terms of numbers. I like the way I visualize things. I like the way most especially that I can bury myself in work that I love to a degree that makes everybody else in the world look at me and go, "God! I wish I could do that." No, I am not changing anything. (Quoted in Shapiro 2006, our emphasis)

Meyerding (1998) illustrates a similar reification of the brain when she notes that her employer and friends "think they have conveyed what it is they expect [her] to do, but they have been speaking in a language [her] brain doesn't understand."

Note how these testimonies slide without warning from "my brain" to "I": I love the way my brain works / I like the way I think; those people speak *to me* in ways *my brain* doesn't understand. Beyond metaphorically personifying the brain, such language conflates persons and brains.[22] The creator of the audio post "Asperger's Conversations"[23] says that "we are a world of *funny brains*" and claims that "neuroscience will help us to understand and appreciate the new mix" (our emphasis). Instead of curing autism, some activists propose curing "Neuro-bigotry,"[24] while others dream of Aspergia, a utopian, autism-friendly "neuro city."[25] "Danni's Blog," by an English self-advocate who defines herself as a "Christian Socialist Computer Addict," is filled with references to the way her brain works:

> *I am hating my brain*. . . . I can't deal with the scary thoughts and brain misfiring that makes me too scared to sleep. . . . I need a *brain transplant*, or for River Tam to kill me with her *brain* I don't want to let people down, and I'm even less reliable now than I was before my *brain* went all bad-funky. . . . I had

> an appointment with the learning support officer. . . . Was weird, as my *brain* wasn't working right. . . . By this point my *brain* was making weird associations. . . . My *brain* feels all sluggish and blocked. . . . My anxiety is pretty bad and I have other *brain weirdness* things that mean that normal coping methods and stuff don't help. . . . I can't do the homework, partially because . . . my *brain weirdness* is getting worse. If I fail it, I can retake it (most likely when my *brain* is working better). It can be hard when my *brain is hating me* and I'm struggling to keep calm.[26]

Such language is common. In a post on "Identity Politics and the Language Controversy," Dora Raymaker, co-director of the Academic Autistic Spectrum Partnership in Research and Education and a member of the Autistic Self Advocacy Network's Board of Directors, confides: "My *brain* has been terribly 'sticky' on a proposal I'm writing for a conference presentation, and tearing my *brain* away to even read a news story let alone write about it has failed some uncountable number of times. And not only has my *brain* been sticky on the topic of the proposal but my *brain*'s been sticky on identity politics and language."[27]

We could give many other examples. The question is: What do these individuals mean when they say their brains are jewels or that they hate their brains, or when they refer to their brains as being in a particular state or doing this or that? Are they using merely figurative language, or do they mean to say that they are essentially their brains, that their identity and subjectivity can be somehow reduced to brain neurochemistry and processes? Actually, like the many other protagonists of the *neuro*, from reputed neuroscientists to phony brain trainers, who also speak of brains that think, feel, decide, believe, know, desire, and do various other things only persons can do, they do not seem to believe that they are their brains alone. Yet they rely on brain language to talk about themselves. Why? One clear reason is that they live in an environment where *neuro* talk (if not always neuroscience itself) has become a major source and sign of legitimacy. My brain is more authorized "to do it" than myself.

Sometimes "brain" and "mind" are interchangeable: Muskie simply juxtaposes "My *brain* is a jewel" with "I am in awe of the *mind* that I have." But he is obviously talking about the same thing, and the same applies to Carley's enthusiasm about how his brain works. The brain sometimes stands metonymically for the person or "I," as when self-advocates write "my brain

doesn't understand," "whatever phrase that non-voluntary portion of my brain happens to be using," or "we are a world of funny brains." On other occasions, the state of the brain—by which a state of being is designated—inspires self-reproach: "sometimes I hate my brain or my brain hates me." That, however, seems to mean that the brain hates itself, since the brain, identified to "I," is also said to feel "all sluggish and blocked," or depicted as not "working right," or making "weird associations."

Neuroscientific metaphors and vocabularies contribute to give the differences between neurotypicals and people on the autistic spectrum a "real" and "natural" character (Brownlow 2007, Brownlow and O'Dell 2006). Neuroscience helps justify those differences, as when a self-advocate declares, "I know they are all individuals, and that we shouldn't blame every NT [neurotypical] for the action of every other NT . . . but there is a common thread that ties them together, and it is at the core of their being. It is more than cultural; it is *how they are hardwired from the factory*" (quoted in Brownlow and O'Dell 2006, 319, our emphasis). Meyerding too neurologizes difference:

> Here came neurology and the possibility that *my brain really was different*. . . . If I could understand my life for the first time only by understanding *how my brain was different* from the majority of brains, how much did I really have in common with all those neuro-typicals (NTs) out there, compared to whom I'd been judged inadequate so many times? . . . Imagine my surprise, then, when I realized I was able to feel "aligned" with this disparate group of individuals joined together by *neurological differences*. . . . *My brain works somewhat differently* from most brains (from "*normal*" *brains*). . . . Most of the ways *I'm different from the neural norm* can be disguised as eccentricities. (Meyerding 1998, our emphasis)

Thus, a neuroscientific idiom is exploited to discard mere eccentricity and place autism in a positive light. Yet both autistics and neurotypicals believe in the "neurological origins of [their] exclusiveness" (Brownlow 2007, 138; Brownlow and O'Dell 2006, 319). Neurobiology thus functions as an instrument to erect identity frontiers, yet it does so on the basis of an underlying commonality: we all are our brains.

By a largely rhetorical reversal of the normalcy discourse, autistics may stress neurotypicals' strange behavior and satirically pathologize neu-

rotypicality. Once he realized "how bizarre and illogical the NTs really are," the self-advocate Archie found "that their comments and insults" had a greatly reduced effect; he could not "blame the people that are afflicted with neurotypicality," but he added: "that does not mean that I am obligated to change my views to see values in traits I dislike" (quoted in Brownlow 2007, 140–141). Neuroscientific claims are mobilized in the construction of NT and autistic experiences so as to highlight their natural difference, yet at the same time even extreme self-advocates know how inextricably they are linked to the neurotypical world. It would be, for example, unfeasible to keep the utopian island Aspergia free from NTs. Indeed, "if an aspergian man and woman get married and have an NT child would we have to kick it out of the country?"[28]

The counterpart of the construction of differences as ontologically real because neurobiologically based is the belief in a certain ontological homogeneity across the autism spectrum. Some activists consider that "low-" and "high-functioning autism" are variations of degree without fundamental "underlying neurological differences" (Nadesan 2005, 208–209). In 2002 Jane Meyerding explained that, since publishing her 1988 essay "Thoughts on Finding Myself Differently Brained," she had realized that classifying people under different categories within the autism spectrum was "seriously misleading" and declared her preference for seeing herself "as autistic, period."[29] Identity politics here implies both essentializing neurological uniqueness and typologizing brain difference. Of course, criticism has also been addressed to the goal of homogenizing the autism spectrum. We have already mentioned that the *DSM-5* eliminated several forms of autism (the most familiar one being Asperger syndrome), integrating them within "Autism Spectrum Disorder." While this shift corresponds to the aspiration of autistic self-advocates such as Meyerding, an online petition by the Global and Regional Asperger's Syndrome Partnership, carrying over eight thousand signatures, regards it as return to a past "when so many of us grew up thinking of ourselves as bad, broken and damaged, not unique and differently-wired."[30] The same petition points out that many children and adults, particularly those with Asperger syndrome, will lose the diagnosis and, with it, "crucial supports, services and legal protections" (see also Lutz 2013). The situation doesn't lack irony since, as the psychotherapist and cultural commentator Gary Greenberg (2013, 182) remarked: "Four decades

after homosexuals demanded to be released from their diagnostic chains, groups of patients were pleading with the APA [American Psychiatric Association, which publishes the *DSM*] *not* to set them free."

Ontological homogeneity is to a large extent a linguistic effect. The world of autistic self-advocacy offers the same phenomenon that Emily Martin observed during her fieldwork on bipolar disorder: Remarks about the brain seemed to be "like clones: endlessly replicating but not generating new connections" (Martin 2009, 7). The brain works like a "confining metaphor" that cuts off links among domains and groups of people. The brain-centered lingo is "folk neurology" (Vrecko 2006) or "folk neurosychology" (Rodriguez 2006), that is, the kind of parlance with which eliminative materialists such as Patricia Churchland (1981) would like to replace folk mentalistic idioms. It has not, however, superseded psychological descriptions of subjective experiences. No amount of neuroscientific progress can suffice to make the mind go away. Indeed:

> If a more reductionistic and brain-based picture of human action displaced our current everyday mental concepts, it would not be because (or solely because) the neural net theory had won in the court of scientific opinion. It would be because the environment we live in (and that scientific theories are produced in) had shifted so that a brain-centered view of a person began to make cultural sense. (Martin 2000, 575)

The neurodiversity universe thus exhibits a cohabitation of everyday ontologies (a phenomenon we discussed in Chapter 1). When acting, thinking, or speaking about themselves and their relations to others, individuals shift ontological registers, and *my brain* may designate *my mind* or, perhaps more precisely, just *I* or *me*. Presumably this does not mean that people are unaware of what they are talking about and say "brain" when they mean something else. Rather, metaphors and metonymies express a more or less harmonious cohabitation of everyday conceptions of the self while at the same time contributing to give a bodily organ—the brain—the kind of psychological depth usually, or formerly, attributed to the mind. The pervasive presence of the *neuro* idiom is thereby legitimized and gives expression to the supposed "neuroscience revolution" in the making of identities.

Identity Politics and the "Neuroscience Revolution"

The combination of "neuro" and "diversity" is by no means self-evident. The term locates difference and singularity so as to naturalize or, rather, physicalize human identity thoroughly. Of course, as we saw in previous chapters, research on neuroplasticity demonstrates that experience shapes the brain in hitherto unimagined ways and to an unexpected extent. Networks of neurons are formed and changed by habits, conscious decisions, acts of the will or attention, physical exercises, food intake, or meditation practices. This supports the shift of diversity, singularity, and creativity to the brain. Neuroscientific research, however, is also characterized by the search for regularities and neuroanatomical and neurophysiological constants that would make it possible to distinguish (ideally on the basis of neuroimaging) between autistic, depressed, schizoid, and normal brains. As we documented in some detail for depression, much neuroscience aims at identifying the brain circuits responsible for normal and pathological mental states. This brings about a paradoxical situation: While neuroplasticity helps account for neurodiversity, neurodiversity advocates tend to minimize the differences among brains within the autism spectrum so as to support their claims for the existence of a brain-based autistic identity. Thus, the "autistic brain" is displayed as ontologically homogeneous and radically different from the comparably homogeneous "neurotypical brain." (The move is analogous to that performed by cultural neuroscience when it implicitly turns cultural difference into an instance of neurodiversity and assumes a fundamental neurobiological homogeneity within each of the groups it studies, "East Asian" and "Western.")

Neuroimaging here plays an extremely powerful role by visually confirming the diagnosis and deepening (whether to celebrate it or pathologize it) a person's sense of autistic identity:

> Joe Powell was diagnosed with Asperger's syndrome, a form of autism, 14 years ago. Before his diagnosis, he didn't speak at all.
>
> Since then, he says he's made big progress in managing his condition.
>
> His brain scan confirms his ASD [Autism Spectrum Disorder]. He says seeing his diagnosis charted in black and white made a big difference to him.

> "You need to physically see it," he says. "I know the autism is still there. The progress I've made in managing my condition is real, but it's still there." (Hughes 2010)

Powell had participated in a study identifying morphometric features and structural patterns of gray matter anatomy in adults with ASD (Ecker et al. 2010; see Deshpande et al. 2013 for a different fMRI approach to identifying "neural connectivity signatures of autism").

The cerebralization of autism may contribute to reify and naturalize differences between autistic and so-called neurotypical brains; the celebration of disability may open the way to an emphasis on difference via comparison and may even sustain hostility toward nondisabled people (Swain and Cameron 1999). Self-criticism, however, has gained ground within the neurodiversity movement. Sinclair (2005) has condemned antineurotypical prejudice, and some Aspergers consider Aspergia as an "Aspie 'Warsaw ghetto.'"[31] Judy Singer (2007) herself warned that the movement is walking on the "dark side" of identity politics, through "its eternal victimhood, its infantilism, its demand for unconditional love and acceptance without concomitant adult self-reflection, self-criticism, a measure of stoicism, and a willingness to see light and dark in oneself as well as in 'the Other.'"

Singer's criticism implies that self-advocates' use of brain-related terms has contributed to the concealment of individual and institutional dimensions that deserve to be openly discussed. In her description of how Ben, a college student she met at a group called Autistic Adults Coming Together, constructed a positive autistic identity, Nancy Bagatell (2007, 423) observes that having to orchestrate the different discourses around him produced "a lot of discomfort—depression, anxiety and sensory overload—and he desperately wanted relief." One of the bipolars interviewed by Emily Martin (2009, 16) remarked that his "brain contains both health and illness, strength and weakness, darkness and light." Self-advocates tend to neglect such tensions and the fact that, as Singer (2007) points out, "not all is for the best in this brave new world that the 'neuroscience revolution' delineates." Some antipsychiatry advocates "fear that the neurodiversity movement too readily embraces a neurological and medical model for all human behavior."[32] On the one hand, seeing oneself as a cerebral subject bolsters one's sense

of identity and may help erase the social stigma often associated with mental pathology. On the other hand, however, it can solipsistically narrow the notion of what it is to be a person. Such, then, are the dilemmas and controversies of the neurodiversity movement. Its members' search for community and relation is in tension with its own reductionistic identity politics, in which selves and difference result from the mechanics of the brain.

Finally, the case of the neurodiversity movement contradicts those who believe that the neurosciences fail to provide "a common ground for shared moral intuitions and values" and therefore lead to an "anthropological and ethical vacuum" (Metzinger 2009, 213). In fact, persons' considering themselves essentially as cerebral subjects has sustained identity formation processes at the individual and the social and community levels. The process is far from straightforward since, as we have seen, autistic self-advocates must negotiate their neurocentric identity politics, a desire for significant forms of sociality, the brain as "confining metaphor" and as liberating condition, and various ways of relating to medicine and to the "neurotypical" world.[33] Nevertheless, across the spectrum of positions, neurodiversity has operated both as an empirical fact and as a shared fundamental value—and one whose status and legitimacy rest largely on its being taken as a validated scientific fact.

Depression and autism, two nosological entities, highlight the ambiguities of cerebralizing processes and the versatility of the *neuro*. But don't those features result from insufficient scientific knowledge? Some day in the future, different biomarkers will delineate with certainty various forms of depression, autism, and other forms of mental distress. Nomenclatures, classifications, and etiologies will finally "carve Nature at its joints"; they will follow from and point to causal mechanisms and neurobiological foundations. Such are the hopes. The cases just sketched, however, suggest that the uncomfortably protean nature of the *neuro* does not represent a problem to be solved or temporary flaws to be superseded but instead highlights its fundamental attribute. There are different ways of being a cerebral subject, ways that do not depend directly on scientific results and idioms but on choices of a different nature (psychological, moral, political, social, even rhetorical) that use those idioms and results as resources.

The last contexts we shall deal with here, literature and film, display such distinctive features and functions as through a magnifying glass. Precisely because they are intrinsically independent from issues of factuality and validity, they can do so in radical form, putting at center stage the dilemmas of the cerebral subject, using them as their most substantive raw material, and performing them without trying to settle them.