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CHAPTER FIVE:
Profit, Risk, and Bare Life

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Introduction

In 2007, the U.S. Centers for Disease Control reported that autism spectrum disorders affect 1 in 150 children (CDC 2007). Parents, educators, medical professionals, and social service providers demand that autism causes be identified and its social and economic risks be addressed and managed, despite considerable controversy over what autism actually is (see Nadesan 2005). Some autism advocates see autism as an inborn or environmentally caused medical condition that requires treatment and cure while others see autism as a difference that requires accommodation and cure.

This chapter considers competing causal explanations and discusses the politics inherent within and informing these competing frameworks for interpreting and treating autism. The chapter critiques the politics of the inborn and genetic-based dominant frame governing autism research and public funding for autism in the U.S., by focusing on the allocation of funding for autism, the prioritization of pharmaceuticals, and expanding efforts to create autism susceptibility tests. The chapter addresses the marginalization of environmental explanations for genetic changes that arguably derives from the

research emphasis on finding susceptibility genes. The emphasis on inborn susceptibility has implications for people with autism and their families as the search for genes may be prioritized over research and spending related to support, therapy, and accommodation in an economic context of public spending cuts.[1] People with autism and their families should consider carefully this formulation of autism as a heritable, genetic deficiency because they will shoulder a growing share of the economic costs of accommodation and treatment as the austerity state cuts services.

Some autism advocates reject altogether the idea of "deficiency"; thus, the construct of autism as a heritable, genetic based disorder may be troubling as autism susceptibility testing expands. The heritable model of genetic deficiency could legitimize a new pre-natal eugenics as families seek to limit the economic and social impacts of having a child with autism. Autism becomes in this dystopic vision a marker of a devalued life form, or bare life.

Autism Research Priorities: Genes, Dollars and Susceptibility Tests

Expert discourses have carved out a definition of autism operationalized through behavioral criteria. The DSM-IV stipulates that a diagnosis of autism (299.00 Autistic Disorder) requires that the patient exhibit symptoms (a total of six or more items) from a triad of behavioral/communication impairments including: (1) impairments in social interaction, including impairments in non-verbal

behaviors ("eye contact", "facial expressions", "body postures", "gestures to regulate social interaction"), impairments in ability to develop appropriate peer relationships, and impairments in emotional reciprocity (e.g., pleasure in other people's happiness); (2) impairments in communication including delays in expressive language, impairments in conversational competence, use of stereotypic or repetitive language, and lack of spontaneous make-believe play; and (3) "restricted repetitive and stereotyped patterns of behavior, interests, and activities". Onset of delays and/or impairments must occur before the age of three for a diagnosis of autistic disorder (pp. 70-71). With few substantive differences, the diagnostic criteria stipulated in the ICD-10 by the World Health Organization basically mirror these criteria. Neither system specifies that mental retardation be an essential diagnostic feature of autism. Because of the heterogeneity of autistic expressions, both the ICD-10 and the DSM-IV include a diagnostic category for atypical expression: "Atypical Autism" in the ICD-10 requires the patient meet fewer of the diagnostic criteria stipulated for autism as does Pervasive Developmental Disorder: Not Otherwise Specified (299.80 PDD-NOS) in the DSM-IV. In DSM-IV, late age of onset requires a diagnosis of PDD-NOS in the absence of other diagnostic possibilities (e.g., schizophrenia).

Most medical and popular accounts of autism represent the disorder as involving brain damage, usually perceived as caused by genetic mutations or alleles (see Herbert 2005). The brain damage is believed to manifest in the triad of impairments that constitute the disorder's diagnostic criteria. Today functional

magnetic resonance imaging (fMRI) and other brain scanning technologies can identify precise areas of the brain believed to be affected. Brain imaging technologies can be employed to create "neural phenotypes" (Ramus 2006, 3), which are seen as necessary for establishing clear relationships between (1) brain and mind and (2) brain and gene, as researchers seek to map cognitive symptoms of autism (mind) onto specific brain areas. After this first step, researchers strive to link targeted brain areas with gene alleles that may contribute to the disorder by affecting the brain's development in that area.

Finding autism genes might be regarded as the Holy Grail of autism research. Autism is typically understood in the medical and scientific literature as primarily a genetic disorder, although the literature occasionally acknowledges the mediating role of exogenous factors (e.g., see Lauritsen and Ewald 2001; Ozand, Al-Odaib, Merza and Harbi 2003). Findings of areas linked to autism include regions on chromosomes 7q21-35, 17q and 11. However, this research has revealed no consistently replicable genetic markers despite over two decades of research (Geschwind 2008; Losh, Sullivan, Trembath and Piven 2008). The phenotypic and etiological complexity of the disorder may complicate discovery of replicable genetic or other bio-based markers. Autism may be caused by multiple risk alleles of small effects, making it difficult to identify using genetic linkage analysis. New DNA imaging technologies have revealed de novo (not seen in parents) structural variations, such as microdeletions and duplications in DNA segments linked to autism, particularly on chromosome 16.

However, most of the copy number variations detected in microarray research appear to be "rare, essentially private, mutations in simplex (one affected child) autism families" (Geschwind 2008, 394). Viruses, environmental contaminants and ionizing radiation all emerge as potential exogenous factors figuring into susceptibilities for mutations. However, very little research actually addresses the role of environmental factors in producing the de novo genetic mutations that have been recently linked to autism. Hence, genetic explanations remain severed conceptually from environmental contaminants.

Despite the ambiguity about the role of genes in causing autism, the public has been educated to regard autism as a hereditary disorder. Popular websites such as WebMd typically prioritize genetics in their introductory accounts of autism, as illustrated here at WebMd's "Autism: Topic Overview": "Autism tends to run in families, so experts think it may be something that you inherit. Scientists are trying to find out exactly which genes may be responsible for passing down autism in families. Other studies are looking at whether autism can be caused by other medical problems or by something in your child's surroundings" (http://www.webmd.com/brain/autism/autism-topic-overview). Likewise, Parenting Magazine notes in their section on "New Autism Facts and Figures" that "We're getting closer to understanding the possible causes. Groundbreaking research last year pinpointed what scientists are calling autism 'susceptibility' genes, which regulate how the brain develops and how connections between cells are made . . . " (http://www.parenting.com/article/

new-autism-facts-and-figures). The article does acknowledge, however, that individuals who have the susceptibility genes do not necessarily have autism.

This framing of autism as a heritable genetic disorder has dominated public research priorities, expert discourse about autism in the media and commercial product developments pertaining to autism. For instance, the same WebMd article explains under the heading of "What Autism Research Is Being Done?" that "The National Institute of Neurological Disorders and Stroke (NINDS), part of the National Institutes of Health, is studying brain abnormalities that may cause autism and is looking for genes that may increase the risk of autism" ("What", 2010). Funding agencies such as the NIH have prioritized the research goal of finding autism susceptibility genes. For instance, in 2004 the NIH issued a Request for Proposal for Identifying Autism Susceptibility Genes" (RFA-MH-05-007) that would make $4,295,000 available in FY 2005.[2] The focus on autism susceptibility genes in the absence of discussion of environmental mediations implies that autism is simply inherited through the convergence of susceptibility genes.

Furthermore, in 2007, three of the six Autism Centers of Excellence[3] research awards focused on brain imaging studies, two focused on genetic based causes (one of which also used neuroscience) and one focused on behavioral interventions.[4] In 2008, the ACE program Center award was for a study seeking to identify rare genetic variations believed to play a role in autism.[5] In 2008, ACE program Network award recipients included three grants studying: genetics;

pharmaceutical treatment with Buspar; and, atypically, an environmental risk study. A study of NIH funding priorities for autism from 1980 to 2007 by Blaxill, Bernard and Wrangham (2008) found that genetic and imaging grants were strongly favored. Treatment using pharmaceuticals and screening were also prioritized, while environment-only studies were strongly de-emphasized when funding was allocated. In 2009, Johns Hopkins University announced receipt of an ACE award to study the intersection of genetics and autism.[6] This list of funded research priorities illustrates the strong emphasis placed on genetic research and neuroscience and, to a lesser extent, research on biomarkers, screening and drug treatment. It also illustrates the de-emphasis on environmental factors, which, when investigated at all, are cast in relation to genetic susceptibilities. Funding prioritizes research that simply aims to identify genes linked to the disorder and/or brain anomalies that deviate from the neural profiles of 'normal' populations. Research assessing the appropriateness and effectiveness of actual therapeutic practices is almost non-existent. Federal and private funding for autism privileges research that studies autism as a genetic and brain based disorder. In contrast to research funding, public funding for services for people with autism does not exist to any significant degree at the national level in the US. Furthermore, national funding for research developing and assessing non-pharmaceutical treatment protocols is extremely limited, as I discuss below. States within the U.S. are therefore responsible for providing the limited funding that does exist for actually treating people diagnosed with

autism. These treatment protocols are primarily non-medicinal and emphasize instead sensory-integration therapy, speech therapy and cognitive and behavioral skills training. Some autism advocates argue that the transmission of the majority of private and public autism dollars to large, institutional and often commercially-driven research programs may result in relatively fewer social or medical benefits for people diagnosed with the disorder. Increasingly, the prioritization of genetic and, increasingly, neuro-scientific research frameworks that presume biological deficiency and damage, raises concern among some autism advocates (e.g., Blaxill, Bernard and Wrangham 2008; Rimland 1997; UK Autism 2004).[7] Advocates worry that public funding for practical therapies for people with autism may be rejected in favor of investing those same public dollars in commercial-viable or commercially 'sexy' research projects conducted at high-profile public and private research labs. Genetics and neuroscience are 'in', while cognitive therapy is not.

This attitude toward prioritizing high-profile, commercially-viable research in genetics and neuroscience was illustrated by the appointment of Frances S. Collins to head the National Institutes of Health, after he stepped down from his position as director of National Human Genome Research Institute at the National Institutes of Health, from 1993-2008. Although Collins publicly eschews genetic determinism, his research has prioritized it and after his appointment to the NIH Collins's first research priority for mental health was "Applying Genomics and Other High Throughput Technologies" (Collins 2010;

see also Blumberg). Collins' 2010 "Director's Report to the 224th National Advisory Mental Health Council Meeting" emphasized genetic approaches to autism and schizophrenia (Collins 2010). In his role as Director at the NIH, Collins encouraged the National Institutes of Mental Health, which is subordinate to the NIH, to embrace genetic explanations and pharmacological treatment of mental illness. Collins' stance reinforced the trend to approach autism as a brain-based disorder caused by genetic mutations or alleles.

Despite its popularity and institutional support, the genetic-brain based approach has failed to deliver on its promises to explicate the 'causes' of autism and has failed to lead to new autism treatments. Genes believed to increase susceptibility in one population are difficult to find in other populations. Penetration is typically uneven as identical twins with autism do not uniformly share the disorder (Bruder et al. 2008). Unknown environmental factors and other independent variables such as father's age may contribute to genetic mutations linked to the disorder. The relationship between phenotypic autistic traits (such as behavioral and cognitive traits) and specific genes remains very unclear although researchers suspect that genotype-phenotype relationships may exist (Thompson, Cannon and Toga 2002).

It is difficult to deny the monetary significance of genetic research. Many gene alleles and processes believed to play a role in the disorder have been patented. Genes can be patented after their sequence is decoded. The World Intellectual Property Organization recognizes patents on at least two autism

susceptibility genes (Application No. PCT/1B2005/002630 and Application No PCT/1B2005/002319). Patents also exist for autism susceptibility genes (as well as ADHD susceptibility genes) (Nadesan 2010). The U.S. Patent office has also patented a method for screening genetic markers associated with autism (Application No. 95117 filed in 1998). Genes have been transformed into commodities, shaping research trajectories and problem-solution frames (Nelkin 2001, 558; Rose 2008).

Patenting of autism genes and other genes linked to illness or undesirable states raises a variety of ethical issues and concerns. Patents can dictate and restrict research trajectories and be used to generate revenue streams. Thus, disorders such as autism can become business opportunities for bioengineering and pharmaceutical companies. Autism circulates in a growing bio-economy that is viewed as vital to re-establishing national economic competitiveness (Gottweis 1998; Rajan 2006). Although geneticists studying autism formally acknowledge the role of environmental mediations, it is their ability to target susceptibility genes (over environmental contributions) that produces commercial revenue streams. By shifting our focus from environment to genes, the marketization of autism genes absolves the state of regulatory responsibility for monitoring and governing those diverse contaminants known also to confer risk and susceptibility to developmental disorders, such as lead, mercury, ionizing radiation, and industrial chemicals (e.g., phthalates) and pesticides and (e.g., organophosphate pesticides). Genes are viewed as risk-laden, as opposed to

environments. The shift in risk from environments to persons implied in the formulation of gene-based autism susceptibility can usher in a dangerous politics of life and death.

Not all genetic research is driven by the profit motive, however. Moreover, not all biological research is genetic. Nonetheless, the seductive combination of genes and dollars has the potential to crowd out other research trajectories and autism funding priorities. The concern here is that knowledge and products developed by the pharmaceutical-research complex will likely promote autism detection (tests) and pharmaceutical 'management' over the more costly (in terms of initial investments) therapeutic approaches that are labor- and resource- intensive (e.g., sensory integration therapy and special education services) and over alternative research approaches that might curtail industry profits by forwarding environmental explanations for autism and other syndromes and diseases.

Environmental research on autism receives limited federal funding and non-pharmaceutical research on autism prevention emphasizing environmental remediation is almost non-existent.[8] Environmental health research addresses environmentally mediated causes of autism and other syndromes, including cancer. This research seeks to identify toxic or carcinogenic environmental contaminants or processes that give rise to diseases. Sometimes this research examines genetic susceptibility in conjunction with environmental harms. At other times, this research emphasizes the role of chemicals and ionizing radiation

in destroying sub-cellular processes, for instance, in breaking the chemical bonds of DNA. This biological research assumes there are causes to disorders such as autism and that these causes can be identified and acted upon (e.g., see Landrigan 2010). However, this biological research does not typically lead to profitable pharmaceuticals and may be unpopular with industries that are responsible for producing environmental hazards. As I shall examine in greater detail, this research is underfunded and underreported in the popular media because financial incentives and domestic austerity are together crowding out alternative approaches and basic treatments for people with autism.

Risk Management and Autism Susceptibility Testing

Autism susceptibility tests are seen as a strategy for detecting childhood autism early in development, before behavioral based testing outlined by the DSM-IV can be employed. As Walsh, Elsabbagh, Bolton and Singh remark (2011, 603): " . . . there is an intensive search for biological markers for autism. Such biomarkers could not only reveal causes of the condition but could also be clinically useful in complementing or improving the behavioral diagnosis of autism and in enabling earlier detection of the condition.

Autism susceptibility tests presume that people with autism are born biologically different and that their differences from 'normal' populations are significant enough to enable detection by focusing on biological markers.

Susceptibility testing could involve pre-natal testing for the presence of autism susceptibility genes. This approach would be problematic for the reasons discussed in the previous section but may in fact be developed if costs for sequencing are reduced. Commercial efforts to develop susceptibility tests are also pursuing other bio-markers that might be linked to the disorder. Bio-markers are the 'surface' manifestations of the underlying biological differences that constitute the disorder.

The possibility that gene-based susceptibility tests might be developed has raised considerable concern within particular sub-sets of the autism advocacy movement. For example, there exists online an Autism Genocide Clock that purports to countdown years, days and minutes to the seeming inevitable development of an autism prenatal test that will result in an autism holocaust (see http://ventura33.com/clock/). This clock was uploaded in 2001 in response to concerns that genetic knowledge about autism would lead to the patenting of susceptibility genes, which could be used to develop commercial pre-natal tests. Private fertility clinics have been scanning embryos for gene-linked diseases for years to these concerns were not unwarranted (Naik 2009). A recent survey of 999 U.S. people seeking genetic counseling found that 75 percent supported genetic tests for the elimination of mental retardation (Naik 2009).

The online research site "Autism Research and Prenatal Testing" observed in July 2011 that today concerns about a gene-based autism susceptibility test have been largely put to rest because the genetic research has failed to identify

consistent markets and because of opposition from autism advocates who fear an impending autism holocaust. Still, each new announcement of the identification of autism susceptibility alleles affords clinical testing services a new opportunity to capitalize on autism. For instance, in 2010 identification of yet another autism susceptibility allele was broadcast through the media (see Sousa et al. 2010). Furthermore, Walsh et al. (2011) report that chromosomal microarray testing can be used to identify submicroscopic deletions and duplications that have been linked with autism. These findings could stimulate further efforts to develop commercial autism-susceptibility tests.

Indeed, patents have been granted for screening techniques aimed at detecting genetic errors linked to birth defects. A new method called comparative genomic hybridization was patented in the 1990s to detect sequence deletions, duplications, and translocations in targeted areas of an individual's genome (U.S. Patents 5,665,549 in September of 1997 and 5,965,362 in October of 1992). The abstract for Patent 5,965,362 explains:

> Disclosed are new methods comprising the use of in situ hybridization to detect abnormal nucleic acid sequence copy numbers in one or more genomes wherein repetitive sequences that bind to multiple loci in a reference chromosome spread are either substantially removed and/or their hybridization signals suppressed. The invention termed Comparative Genomic Hybridization (CGH) provides for methods of determining the relative number of copies of nucleic acid sequences in one

> or more subject genomes or portions thereof (for example, a tumor cell) as a function of the location of those sequences in a reference genome (for example, a normal human genome). . . . Amplifications, duplications and/or deletions in the subject genome(s) can be detected (http://patft.uspto.gov/netacgi/nph-).

Comparative genomic hybridization is currently being used for pre-natal diagnostics to identify fetuses potentially susceptible to particular syndromes caused by, or linked to, deletions or additions of genetic material (Stein 2008). Concern already exists that this type of genetic screening could be used to identify susceptibilities for a wide range of disorders linked, but not caused by, genetic factors. Comparative genomic hybridization transforms a child's risk into suspect absences or duplications of alleles prior to his or her birth. Risk becomes the punctuation of one's genome.

Researchers recently announced they were able to sequence the entire genome of a fetus using a sample of the mother's blood (Marcus 2012). The news article heralding this accomplishment stated that the development would make it easier to predict fetal genetic defects with little risk. The researchers demonstrated their technique on a mother with DiGeorge syndrome, a genetic condition that can cause cardiac problems. They successfully determined that the fetus inherited the condition. The article notes that making sense of the information poses the most significant challenge because of limited information available about the relationships between genes and diseases. This innovation

reinforces the emphasis on the punctuation of one's genome as the precursor for adult health and cognition, thereby marginalizing important environmental factors, including the conditions of the prenatal environment.

Autism susceptibility tests need not rely on genes. In 2010, researchers declared that they could use MRI scans to measure and detect deviations in brain circuitry representative of autism (Lang et al. 2010). The research was promoted as offering an alternative route for detecting autism in very young children, although the researchers acknowledged their system was not ready for clinical practice (McClean 2010). Other strategies suggested for early detection of autism include using serum samples for proteomic profiling and urine samples for metabolic profiling (see Walsh et al. 2001). Saliva-based susceptibility tests have also been proposed (see Castagnola et al. 2008).

The article by Walsh et al. (2011) on the ethical implications of autism susceptibility testing raises ethical quandaries stemming from the contingency and uncertainty of autism diagnoses and outcomes. The authors recognize the potential for eugenic applications of autism testing, a concern that I raised in previous work (Nadesan 2005). Given the risk of eugenic applications, Walsh et al. recommend that autism advocates be involved with the development and assessment of autism susceptibility tests. Their recommendations are worthy, but the commercial enterprises that develop autism susceptibility tests--to be marketed with other tests for home and clinical use--are not likely to be swayed by these recommendations, which could impinge against profitable products.

Indeed, in a 2010 article published by the Canadian Globe and Mail, the Director of Canada's Centre for Applied Genomics, Dr. Stephen Scherer, observed that he believes people will be able to purchase mail-order genetic tests that scan for autism related genes in the "near future", even if the information is "dubious at best" (Abraham 2010).

Moreover, it is possible that even the most sensitively developed and marketed autism susceptibility tests could have adverse effects for public support and medical insurance for people diagnosed with the disorder. Parents who 'choose' to carry a susceptible embryo to term could be held responsible for their child's care by insurance companies and society alike if the child develops autistic symptoms. Likewise, it is possible that susceptibility tests marketed directly to prospective or new parents (see Castagnola et al. 2008) might be implemented in the absence of social and therapeutic supports, leaving parents terrified, but ill-equipped, to evaluate the long-term prospects for their 'risky' fetus or infant.

In 2006, The New York Times published a troubling essay by Elizabeth Weil titled, "A Wrongful Birth." In her essay Weil describes a legal suit of wrongful birth by a woman whose baby was born with a gene duplication and a gene deletion on his fourth chromosome. The baby was diagnosed with Wolf-Hirschhorn syndrome, which often involves physical and mental disabilities. The mother sued her medical providers for failing to detect problems with the fetus, whose weight and size were abnormally low. The multimillion-dollar lawsuit

charged that the obstetrician's neglect disallowed her from the right to abort the fetus. Weil observes that this case and others like it reveal new beliefs about reproduction:

> And what those cases are exposing is the relatively new belief that we should have a right to choose which babies come into the world. This belief is built upon two assumptions, both of which have emerged in the past 40 years. The first is the assumption that if we choose to take advantage of contemporary technology, major flaws in our fetus's health will be detected before birth. The second assumption, more controversial, is that we will be able to do something--namely, end the pregnancy--if those flaws suggest a parenting project we would rather not undertake.

A third assumption not identified by Weil is that the measure of a child's perfection exists in a form of genetic normalization that be read off genetic sequences. Development of ever more sophisticated and less invasive pre-natal testing devices encourages the idea that the seeds of perfect normalization are located in our genes, that pre-natal testing can measure a child's propensity for this form of perfection, and that we have an implicit responsibility to know and decide upon a child before s/he is born.

Pre-natal testing is an ethically complex field. It is therefore not surprising that autism advocacy surrounding issues of autism detection and treatment is a heterogeneous terrain notable for its fragmentation. Some autism advocates push for finding autism causes and cures and might welcome autism susceptibility

tests. The organization Defeat Autism Now (DAN) illustrates this approach to advocacy. This approach toward cause and cure is itself fragmented between those who see autism as a gene based order and those who stress environmental causes for the disorder, ranging from vaccinations to inorganic chemicals such as pesticides and endocrine disrupting phthalates.

In contrast to the approach emphasizing cause and cure, other autism advocates argue that people with the disorder are different, not deficient.[9] Advocates within this group argue that the overarching emphasis on cause and cure can distract attention from the more important goal of helping people diagnosed with the disorder live meaningful and comfortable lives. Some advocates within this second group reject the disease model altogether and all approaches that aim to cure the disorder. Advocacy by this second group, often by people diagnosed with autism, has grown in visibility as the Internet enables autism spectrum individuals to communicate comfortably their concerns about how 'neuro-typical' persons view and attempt to normalize autistic differences (Nadesan 2005). Advocates in this group often share a sense of a unifying common biology that presents challenges, but also offers special contributions to society. These advocates promote assistance that does not seek to eradicate difference, but rather seeks to promote individual growth and development. For advocates who reject medicalization, autistic individuals are simply 'wired differently' (Nadesan 2005). The idea that people with autism are 'wired differently' has been promoted by the field of cognitive neuroscience. Autism has

been a cash cow for neuroscience research and has garnered publicity for the discipline's efforts to map cognitive traits, aptitudes and behaviors directly onto the brain (see Ortega and Vidal 2011; Nadesan 2005, 2008). Neuroscience aims to create neural phenotypes of autistic brains that may be linked to physiological causes, such as gene alleles. Using technologies such as fMRI and PET (positron emission tomography), researchers claim to identify patterns of neural activity representing 'normal' and 'deviant' (e.g., autistic) cognition and affect. Autistic phenotypes can be generalized from patterns of 'autistic' cognition and affect created through the neural scans. Ultimately, such research hopes to identify gene alleles that correlate with specific neural phenotypes, bridging brain and gene (and behavior or trait). This research has a variety of ontological and methodological problems that will not be rehearsed here (see Ortega and Vidal 2011; Ortega this volume; Uttal 2001). What is of interest for this chapter is that neuroscientific knowledge claims have been used by some autism advocates to affirm autistic differences.

Yet this strategy of affirming difference by seizing upon neuroscience's ontology of autistic brains can have dangerous implications, particularly for those who fear susceptibility testing will produce a veiled eugenics. The growing awareness of autism may actually increase parental fears about having and raising a child with autism, particularly in a contracting economy characterized by government austerity. Biological susceptibility tests may effectively reduce unborn autistic persons into a form of biological, but nonhuman 'bare life' that

enables annihilation by reducing the entire person to a small set of biological markers regarded as deviant or abnormal. Tests that evaluate genetic and/or biomedical susceptibility to autism risk producing a 'thanatopolitics'--a politics of death--for autism. Pre-natal autism tests, for instance, have the potential to reduce life from bios--the life proper to an individual within a polity--to zöe, or bare life that can be killed with impunity (see Agamben 1998).

The decision to abort an autistic fetus stems from a perception of irreparable damage done to the fetal brain by deficient or damaged genes, or by other physiological events/processes. The potential becoming of the fetus is calibrated only in terms of autistic characteristics or deficiencies. The fetus becomes bare life, denied becoming and personhood (see, for example Tremain 2005, 2006). To raise concern about this type of reduction of human life to disposability does not necessarily imply a strident 'pro-life' bias, nor does it necessitate sentimentalizing autistic persons. Rather, the concerns being raised are twofold. First, the cultural adoption of pre-natal testing signals ascendancy of a technocratic determinism that rejects uncertainty, the unpredictable processes of becoming, and the work of parenting. Second, the technologies of pre-natal testing may responsibilize parents for choosing children in the context of increasingly 'austere' social supports. Finally, technologies of detection may usurp environmental policies and technologies of prevention, because the former provide opportunities for capitalization, while the latter imply greater

monitoring and regulation of industry, processes which are costly to government and industry.

Austerity and Personal Responsibility in the Great Recession

Having now explored the limitations and financial and ethical implications of genetic based and deterministic models of autistic brains, this chapter turns to examine autism within the contemporary socio-economic context. In Chapter Six, "Caring for Autism: Toward a More Responsive State," Kristin Bumiller demonstrates that the life chances of people with autism is significantly impacted by the state policies that structure and enable opportunities and financial support. She argues that recent trends toward privatization of care shifts responsibility onto families. Bumiller's concerns about the responsibilization of families for the care of those with disabilities takes on heightened significance in the context of the massive cuts in social spending that have occurred in the US and Europe over the last five years. The shift in governmental priorities away from the social welfare of dependent and vulnerable populations may impact their valuation in public attitudes and expert opinions. Formulations of disability that emphasize growth and development through intensive supports and accommodation may fade from public consciousness as more deterministic and less resource-intensive formulations emerge. Indeed, the formulation of 'irrevocably brain-damaged' autistic persons might gain credence within this

social context because of its economic expediency. Irrevocably damaged persons demand less therapeutic remediation than persons who have the capacity to grow and develop within intensive (and costly) therapeutic supports and accommodations. Early detection of people who get classified as autistic could be used for nefarious purposes as well as for optimizing ones. This section examines these arguments, focusing on the challenges of providing educational and therapeutic services for autistic people in the context of economic recession.

The global recession that began in 2007 has hit the U.S. particularly hard, exacerbating three decades of slow decline as U.S. jobs were automated and outsourced abroad. The American middle and working classes have been severely impacted. The Center for American Progress issued a report titled, "Recession, Poverty, and the Recovery Act: Millions at Risk of Falling Out of the Middle Class" in February 2009 (Kvaal and Furnas 2009). The report cites rising unemployment, loss of health insurance, and growing poverty before concluding, "Our economy is in a perilous state. Millions of middle-class families are likely to fall into poverty if Congress does not take swift action". In September 2011, 49.9 million Americans had no health insurance and employer-sponsored private insurance covered only 55% of the population ("Bleak News" 2011). The U.S. stimulus, the National Recovery Act, fell short of delivering adequate relief and job growth to an increasingly fragile middle-class and an increasingly desperate working class (Hilsenrath and Leo 2009; Kroll 2011).

Calls for fiscal austerity in the U.S. by 'deficit hawks' and the decimated budgets of public entities such as states, school districts and cities are eroding health, social-welfare and educational support and spending in the U.S. The reign of "disaster capitalism" (Klein 2007, 6) terrorizing all those citizens dependent upon public spending promises to significantly curtail supports for children and adults whose social vulnerability and/or bio-medical differences require greater health, welfare, or educational spending than allocated to wealthier, healthier, or more 'optimized' populations.

Public services are stretched thin as demand is rising while public budgets are contracting. Public education has been particularly affected by the recession. Public schools in the US derive most of their funds from local property taxes. Today's property taxes reflect the inflated housing values of the real-estate bubble, the collapse of which precipitated the recession. As property valuations are adjusted downward over the next several years, schools will face significant declines in revenue. The National Conference of State Legislatures predicts that schools funding will, metaphorically, drop 'off a cliff' when the American Recovery and Reinvestment Act (i.e., the stimulus) funds expire (see Shreve 2009). Even the socially and economically conservative Forbes magazine observes: "Unless major changes are made, or taxes raised sky-high, spending on baby-boomer retirements (Social Security, health care, public employee pensions, etc.) will eat up almost every morsel of discretionary funding that could otherwise go to services like schools" (Finn and Petrilli 2009). Out of necessity,

schools will raise class sizes and cut 'elective' programs such as art, music, and physical education. For instance, the Wall Street Journal reported that 75 percent of California's elementary schools will increase class size in 2010 (Tuna 2010). Likewise, Georgia school districts are facing $700 million in cuts, leading state lawmakers to consider temporarily waiving state-mandated class-size limits.

Strapped schools will have difficulty providing aids and needed resources such as speech therapy, occupational therapy and physical therapy to children with special needs. Federal funding for special education is limited. The 1975 Individuals with Disabilities Education Act (IDEA) specified that the federal government would compensate schools for 40 percent of excess special education costs by 1982. However, funding has never reached the 40 percent level and is believed to account for far less. The Department of Education's own data for 2009 only attributes $1,700 in federal dollars towards states grants funding students qualifying under IDEA in 2009.[10]

The crisis in funding is also stressing state, county and city budgets. These entities are cutting funding for programs for the poor, the elderly and the disabled. The Center on Budget and Policy Priorities reported in May of 2010 that at least 30 states have cut funding for low-income families eligible for health insurance or care, at least 25 states are cutting programs for the elderly and disabled (Johnson, Oliff and Williams 2011). Efforts to halt budget cuts to people with disabilities using injunctions have not been successful in states such as Arizona.[11] State budget shortfalls anticipated for 2011 are expected to total

approximately $121 billion and will be greater than the 2010 and 2009 shortfalls (Center for Budget and Policy Priorities 2010). Further cuts for services and programs for disabled and vulnerable populations seem inevitable. Home health care services for the elderly and disabled have been targeted for cuts, even though these programs are cost efficient and are a vital lifeline for those that receive their services (Leland 2010).

U.S. states such as Arizona and California must run balanced budgets but the federal government operates using a fiat currency model and therefore can run extensive deficits. The financial bailout of the insolvent US banking system and unprecedented military spending have resulted in massive federal deficits. Given the ease with which the federal government runs up deficits, one must question why austerity is being enforced in social welfare spending. The federal government could replace lost state funding necessary for operating social services. Instead, the federal government is allowing extreme state-level austerity measures that cripple services to the old, disabled and ill.

Disasters allow neoliberal authorities and various other 'reformers' to downsize social-welfare programs and services (Klein 2007). Austerity measures typically target vulnerable populations lacking the resources to lobby against them, as evidenced in the disaster capitalism that occurred in the wake of Hurricane Katrina (Nadesan 2008b). Austerity measures are often accompanied by the circulation of neoliberal cultural discourses that responsibilize individuals, while delegitimizing the logic of social-welfare (Nadesan 2008a,

2010). Poor populations, those with disabilities or chronic illnesses, the long-term unemployed, the elderly, children and every other group conceivable that fails to achieve the neoliberal idealization of entrepreneurial autonomy become second-class citizens.

Neoliberalism dovetails smoothly with the amorality of Randian narcissism. Indeed, Ayn Rand's philosophy, marked by intolerance for anything less than perfect human autonomy, captured the imagination and loyalty of the long-term Federal Reserve Chairman Alan Greenspan (Taibbi 2010). The fusion of neoliberal market valorization and Randian intolerance and arrogance enabled and legitimized the plundering of the wider population and the systematic dismantling of Keynesian social-welfare equalization and re-distribution systems. The ruthless, predatory capitalist ethos held in check by Keynesian era reforms and values has now been fully unleashed. The vast majority of the American population is vulnerable to wage repression and austerity measures and no population is more vulnerable than those with autism.

Cuts in state and community level public funding for autism research are likely to exacerbate the already existing public policy bias that frames autism as a biological, gene-based disorder in search of a pharmaceutical cure. Future autism research priorities and commercial product developments are likely to reflect the interests of the pharmaceutical and bio-tech companies still able and willing to 'invest' in autism.

Family members of people with autism will be responsibilized for their care without the benefits of supports as states, counties, cities and school districts shed services. The potential expansion of autism susceptibility test may have the potential of refiguring risk so that parents of autistic children are responsibilized with "the choice" to have kept (i.e., not aborted) their autistic children. The brain-based model of irreparable autistic harm will circulate more widely as medical and public authorities alike struggle to rationalize elimination of funding for therapeutic programming for people with autism. This dystopic scenario is not the fruit of wild extrapolation, but rather is the logical consequence of current trajectories.

Another important effect of neoliberal approaches toward health research and management is the dis-investment in and symbolic marginalization of, environmental research on autism and related disorders. A recent research study publicized by Reuters found that children exposed to organophosphate pesticides have higher risks of ADHD (Reuters 2010). If these pesticides can be directly and positively correlated with ADHD, it seems probable they could just as likely be correlated with autism given many researchers see ADHD as part of the autism spectrum because of commonalities in executive function deficits (e.g., Corbett, Constantine, Hendren, Rocke and Ozonoff 2009). Indeed, Landrigan's (2010) research suggests that a wide array of common environmental toxins could play a role in producing autism. Martha Herbert, a prominent researcher in the area of autism and environmental health, agrees; although, she is more

optimistic about physiological interventions for children who have suffered environmentally induced injuries (personal correspondence). Yet, the U.S. approach to regulating chemicals remains lax despite growing evidence of chemical harms to human and animal life.

The Children's Environmental Health Center at Mount Sinai reports that only 20 percent of the more than 80,000 new chemicals produced since World War II have been tested for toxicity in children (Kristof 2009). In August of 2007 the U.S. Government Accounting Office (GAO) published a report comparing the lax U.S. regulatory framework for chemicals with a recently enacted European framework, REACH. The GAO report (GAO-07-825) explains that under the current regulatory system in the U.S., companies do not have to develop information on the health or environmental impact of chemicals unless specifically required by EPA ruling. Consequently, the EPA relies on voluntary programs for gathering information from chemical companies in order to evaluate and regulate new chemicals under the provisions of 1976 Toxic Substances Control Act (TSCA) legislation.[12] The GAO report's recommendation that the burden of risk be shifted to the chemical companies was not adopted by the George W. Bush Administration, even after former President Bush's cancer panel found a strong link between environmental toxins and cancer (see Layton 2010b, The President's Cancer Panel 2010). In 2010 a new regulation to overhaul the now outdated 1976 TSCA were introduced, but so far nothing has been passed (see Layton, 2010a; "Momentum" 2010).[13]

Chemicals are not the only environmental factor that may be implicated in autism. Ionizing radiation has received almost no attention in the environmental research on autism, yet in my opinion there exists considerable likelihood that it may play a role in the disorder. Research has demonstrated that even low-doses of ionizing radiation can cause childhood leukemia (Little, Wakeford and Kendall 2009). Environmental researchers have pointed out that radiation risk-exposure standards are based on adult standards and children are a sensitive subpopulation whose biological vulnerabilities have not been properly assessed for risk-management standards (see Fucic, Brunborg, Lassan, Jezek et al. 2008; Preston 2004). Furthermore, natural killer (NK) cells that have been implicated as being deficient in children with autism (Enstrom 2008) are particularly susceptible to damage from ionizing radiation (Vokurková et al. 2010). The nuclear power/weapons industry has been very successful in deflecting attention away from radiation as an environmental risk.

Two research centers study environmental factors and autism, the University of Medicine and Dentistry of New Jersey/Robert Wood Johnson Medical School in Piscataway and the University of California at Davis ("New Centers" 2002). However, efforts to regulate environmental factors implicated in human disease and disorders face formidable challenges from industry, ranging from industry science to corporate lobbyists (see McGarity and Wagner 2008). Epidemiological evidence that chemicals cause harm is often riddled with contingencies that offer fuel to the corporate science aimed at debunking

environmental health research. Most importantly, contingency complicates efforts to calculate definite harms using financial logics and methodologies. Contingency calculates poorly within neoliberal economic calculi used for assessing risk when costs are involved. Neoliberalism is inherently biased toward protecting industry when regulation is at issue. Even when harms are established, opposition to environmental legislation typically overwhelms support. Business claims that environmental regulation will raise costs, or force industry to export production abroad, are powerful persuaders.

The compelling body of research documenting environmental factors in producing autism is likely to be marginalized for the foreseeable future. The fiscal significance of autism may very well wane as public expenditures on health care and services for people with autism are cut as states slash funding. As risk shifts to individuals, government has fewer financial incentives for battling industry and enforcing commercially costly enhanced environmental regulations. In contrast, public dollars for university and commercial research on the genetic causes of autism will continue since this type of funding is represented as promoting bio-tech innovation and professional job creation. The same can be said for pharmaceutical development. Pharmaceuticals that manage autistic symptoms simultaneously provide cost-effective strategies for managing people with autism while also fulfilling the mantra of fostering innovation while promoting economic growth. Risperdal, that corpulent-zombie producing anti-

psychotic, exemplifies the dangers of drugs that promise to 'manage' troubling autistic symptoms.

Conclusion

This chapter considered some of the competing causal explanations for autism and discussed the politics inherent within and informing these competing frameworks for interpreting and treating the disorder. The chapter critiqued the politics of the inborn and genetic-based dominant frame governing autism research and public funding for autism in the U.S., by focusing on the allocation of funding for autism, the prioritization of pharmaceuticals, and expanding efforts to create autism susceptibility tests. The chapter emphasized the latent dangers lurking in a geneticization of autism devoid of environmental mediation. Concerns were raised also about the potential for the dominant framework to produce pre-natal testing, potentially ushering in a new eugenics cloaked in the guise of personalized 'technologies of the self': that is, autism advocates see pre-natal testing as shifting eugenic decision-making to prospective parents who then become responsibilized for their children's conditions. Finally, the chapter raised the possibility that the economic and social stressors of the ongoing financial-economic crisis could amplify the prioritization of inborn-genetic frameworks and that this prioritization could undermine support for costly educational and therapeutic supports, thereby having the

potential to reduce autistic persons to a form of bare life denied social equality and political representations.

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[1] In 2007, three of the Autism Centers of Excellence research awards focused on brain imaging studies, two on genetic based causes (one of which also used neuroscience), and one on behavioral interventions. (See http://www.nimh.nih.gov/science-news/2007/nih-funds-new-program-to-investigate-causes-and-treatment-of-autism.shtml). The Autism Centers of Excellence (ACE) program is a consolidation of two previous programs, the Studies to Advance Autism Research and Treatment (STAART) and Collaborative Programs of Excellence in Autism (CPEA) programs into a single research effort. The 2008-2009 Progress Report for the Autisms Centers of

Excellence lists one funded environmental study on autism in its description of thirteen funded research studies (http://report.nih.gov/biennialreport/ViewSection.aspx?sid=28&cid=4). The study explicitly addressing environmental impacts on autism was for research at Drexel University, which is part of the project of Early Autism Risk Longitudinal Investigation (EARLI) (http://www.earlistudy.org/).

[2] See http://grants.nih.gov/grants/guide/rfa-files/RFA-MH-05-007.html.

[3] The passage of The Children's Health Act of 2000 (P.L. 106-310) m andated that the NIH assist a new platform of at least five centers of excellence focusing on autism and related disorders. The Autism Centers of Excellence (ACE) program consolidates two previously existing programs, the Studies to Advance Autism Research and Treatment (STAART) and Collaborative Programs of Excellence in Autism (CPEA) programs into a single research effort (http://grants.nih.gov/grants/guide/rfa-files/RFA-HD-06-016.html). The CPEA program began in 1997 and emphasized genetic, immunological, and environmental factors. The STAART program emphasized causes, diagnosis, early detection, prevention and treatment (http://grants.nih.gov/grants/guide/rfa-files/RFA-HD-06-016.html). It appears the two programs were combined in 2007.

[4] See http://www.nimh.nih.gov/science-news/2007/nih-funds-new-program-to-investigate-causes-and-treatment-of-autism.shtml.

[5] See http://www.nih.gov/news/health/apr2008/nimh-01.htm.

[6] See http://www.jhsph.edu/publichealthnews/press_releases/2009/earli.html.

[7] See http://www.nichd.nih.gov/autism/research/cpea.cfm for list of past research funded.

[8] See footnote 1.

[9] Autism advocates in this second group are a diverse group. Some notable advocates include Stephen Shore (website is http://www.autismasperger.net/writings_self_advocacy.htm), Phil Schwartz (interview available here: http://aspitude.blogspot.com/2009/03/world-autism-

interviews-phil-schwarznew.html), Frank Klein (website http://home.att.net/~ascaris1), and Jim Sinclair.

[10] See http://www2.ed.gov/about/overview/budget/budget11/summary/edlite-section3b.html#spedstate.

[11] See http://www.acdl.com/legalpolicynews.html.

[12] Passed in 1969, the National Environmental Policy Act (NEPA) initiated policy actions addressing biological and ecological impacts of synthetic environmental chemicals (Frickel 2006). NEPA mandated the creation of the Environmental Protection Agency (EPA) and the Council for Environmental Quality. NEPA requires an annual report on the state of the environment and environmental impact assessment using data collected from the EPA. The EPA was afforded additional regulatory authority with the passage of the 1976 Toxic Substances Control Act (TSCA), which enabled the EPA to control chemicals known to pose unreasonable risks to human or environmental health.

[13] Documents pertaining to the bill can be found here: http://energycommerce.house.gov/index.php?option=com_content&view=article&id=2086:toxic-chemicals-safety-act&catid=169:legislation&Itemid=55 and also here http://www.saferchemicals.org/safe-chemicals-act/index.html.