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Autistic Consumer Audit of UC Davis MIND Institute's Mutant Angelman Mice and Their Translational Value Toward the Human Autistic Experience

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Abstract

On March 10, 2022, six researchers in affiliation with the UC Davis MIND Institute published a mouse gait study in the Autism Research journal, the official journal of the International Society for Autism Research (INSAR). Our team of Autistic scientists set about conducting this consumer audit to evaluate any conflict(s) of interest between the study topic, choice of journal publication, and grant award agenda. To evaluate the impact factor of translational research, we investigated the researchers' strident claims that genetically modified mutant Angelman syndrome mice ("AS mice models") may contribute to the advancement of discoveries of behavioral and cognitive abnormalities in intellectually disabled and developmentally disabled (ID/DD) people, such as Autistics. As a result of our exhaustive review of both broad and narrow claims, a full redaction request is made in our recommendation to the publishers of the Autism Research journal, John Wiley & Sons. In conclusion, we strongly recommend that speculative claims about rodent models and translational research for Autism should not be

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considered meritorious of funding from the National Institutes of Health. Our grassroots initiative is aimed at ameliorating the damaging stereotypes within the medical pathology paradigm under which Autistic people are studied as abnormal human variants under the disease model. Finally, we ask that UC Davis' MIND Institute cease and desist all rodent model research with the aims of investigating the pathogenic mechanism that is presumed to be underlying Autistic behavioral expression.

Keywords
rodent models, research ethics, translational research, autism claims, autistic culture

Autism spectrum disorder (ASD) is classified as a developmental disability (DD) but not intrinsically as an intellectual disability (ID). Autism is behaviorally diagnosed based on clinical observations and external interpretations. Autism is an umbrella term for a variety of neurodevelopmental differences which are diagnosed according to a set of stereotypical presentations. Autism as a neurodevelopmental phenomenon requires a clinical diagnosis, even when the Autistic person is an adult who can reflect on their own inner experience. Distinct behavioral and psychiatric correlates, developmental trajectories, and patterns of social impairment are distinct from those with nonsyndromic ASD.

Angelman syndrome is a DD characterized by severe developmental delay and learning disabilities and often exhibits overlapping symptomatic behaviors with Autistic presentation. However, Angelman syndrome and Autism are not diagnostically equivalent as the former requires a blood test rather than a psychological assessment. It is imperative to preserve clinically important differences that are masked by reliance on the categorical diagnosis of ASD that underlie inadvertent or intentional convergence between syndromic and nonsyndromic ASD (Sztainberg & Zoghbi, 2016). For instance, Abbeduto et al. (2014) observe that individuals with comorbid fragile X syndrome and Translational studies fall under a paradigm that conflates variation along dimensional axes of normal function with quantitative measurements of disease phenotypes and with the occurrence of diseases in overlapping clusters or spectra (Ross & Margolis, 2019).

A total of six researchers, Stela P. Petkova, Anna Adhikari, Elizabeth L. Berg, Timothy A. Fenton, Jessica Duis,[1] and with principal investigator (PI) Jill L. Silverman, investigated motor dysfunction using gait in mice that have been genetically modified for the study of Angelman syndrome, which the

authors term "AS mice models" (Petkova et al., 2022). In ibidem, "the translational power of the DigiGait system lies in its ability to precisely and automatically collect the same gait outcome metrics in rodent models of Angelman syndrome (AS) that are collected clinically in human AS patients." Their clinical observations used automated and digitized metrics for motor movement that were predetermined as deficient, which is an unfounded attempt to quantitatively measure the presence of functional activity that is qualitatively presented in a clinical setting as abnormal.

In addition to standard motor behavioral assays, Petkova and others (2022) used these deficit measures to analyze four phases of temporal gait pattern: swing (during which a paw has no contact with the ground), braking (during which a paw transitions from the swing phase and steps onto the ground into the stance phase), propulsion (during which a paw lifts off the ground and into the swing phase), and stance (when the paw makes full contact with the ground)." The researchers' use of "AS" to group the mice models should not be confused with any pseudonym for Autism spectrum nor Asperger's syndrome, which the researchers have not explicated as a distinct abbreviation.

The mouse gait study was published in the Autism Research journal, which is a U.S.-based journal owned by John Wiley & Sons Inc. The journal is the official journal of the International Society for Autism Research (INSAR). The publisher's policy contains a Wiley Research Diversity, Equity & Inclusion Statement,[2] which states that Wiley is committed to implementing sustainable and positive change to advance diversity, equity, and inclusion through the editorial processes and policies of its publications. We flagged this study as Autistic people, wondering why this gait study on mice was published in an Autism journal and became interested in the conflict(s) of interest with the study topic, choice of journal publication, and grant award agenda.

Especially problematic is the presumption that rodents can be used for modeling human emotional and behavioral differences. The right to a multidisciplinary assessment of research aims is enshrined in the United Nations Convention of the Rights for Persons with Disabilities (CRPD; United Nations, 2007). The National Institute for Clinical Excellence, situated in the United Kingdom, recommends multidisciplinary practice in many education and social and health care fields to avert a "one size fits all" response to heterogeneous diagnoses, such as Autism (Dillenburger et al., 2014).

Given that the Autism Research journal published this gait study for INSAR, we are critical of the peer reviewers who determined that a study that does not relate to Autism and does not study Autistic people, belongs in an Autism journal. To strengthen the relatability to Autistic readership ("consumers" of the research), we recommend that "research teams pay

particularly close attention to language complexity, precision, and concreteness" (Nicolaidis et al., 2020). We believe that consumers and community members add meaningful value to all phases of health and medical research, especially in the translation of such research to clinical results. We strongly encourage all researchers to include Autistic adults in research development and to assist in laying out clear steps for research teams to take to make the process useful and rigorous.

The gait study authors declared their funding sources as: Eunice Kennedy Shriver National Institute of Child Health and Human Development, Grant/Award Number: P50HD103526; National Institute of Neurological Disorders and Stroke, Grant/Award Number: R01NS097808; Intellectual and Developmental Disabilities Research Center; Foundation for Angelman Syndrome Therapeutics. We conducted an audit to determine the hidden costs paid by American taxpayers, based on the above grants, evaluating their impact factor and relevance of the study as it pertains to the body of knowledge relating to Autism in 2022. Our critique of this impact is supported by Shapiro (1998), who states that "Comparable conditions do not occur naturally in nonhuman animals and investigators must attempt to devise situations which induce what are at least analogous versions of the human condition under study." The gait study authors failed to demonstrate how mouse models are comparable to the human model of Autism. As Autistic people, we resist the implied value and merit of such research designs.

Doogri Method of Consumer Audit

This consumer audit research project was designed under the guidelines of Able Grounded Phenomenology (AGP), directing us toward an ethical and humane model for nonautistic researchers conducting Autism research. The checklist within the AGP Model (Kupferstein, 2020) specifies the steps that a nonautistic researcher must implement when conducting meaningful Autism research. In addition, as co-authors, our strengths as Autistic scientists ground our abilities in the phenomenon of conducting Autism research, using the strength-based Doogri Method.[3] This review is a case study that serves as a template for a general audit, and especially primed for investigating translational value research.

To date, publicly funded Autism research appears to privilege nonautistic scientists who do not adhere to current guidance to "facilitate inclusive work" with Autistic scientists (Grant & Kara, 2021). A mere 2.5% of federal funders of Autism research prioritize projects pertaining to "lifespan issues" (Harris et al., 2021). Autistic scientists are systematically denied funding for social support models and quality of life (QoL) research, in favor of biomedical (to

Table 1. Funding Sources for UC Davis MIND Institute's Grants for the Years 2013 to Present.

Funding source	Grant/award	Funding amount
Intellectual and Developmental Disabilities Research Center	U54HD079125	US$8,369,200
Eunice Kennedy Shriver National Institute of Child Health and Human Development	P50HD103526	US$2,589,126
National Institute of Neurological Disorders and Stroke	R01NS097808	US$1,717,190
Foundation for Angelman Syndrome Therapeutics	Not Publicly Available	US$3,000,000

prevent diseases), further perpetuating the medical pathology paradigm. Even in cases where Autistic people feel that they desire a paradigmatic review of their comorbid and degenerative conditions, the progressive blindness of the pathology lens is grossly inadequate for the review of the human experience.

We reviewed the publicly posted disclosures on the Department of Health and Human Services (HHS) Operating Divisions (OPDIVs) Tracking Accountability in Government Grants System (TAGGS) website, which displays detailed descriptions of grants and their intended purpose. We then searched the Guidestar nonprofit database, itemizing the total grant money received, using IRS Form-990 (Internal Revenue Services, 2021). Table 1 lists the funding sources for UC Davis MIND Institute's grants for the years 2013 to the present. We will detail this audit using declared funding grants overseen by the federal Department of HHS for those years.

NIH: The Intellectual and Developmental Disabilities Research Center

In 1963 Congress established "Centers of Excellence" for research of intellectual and developmental disabilities, through the Intellectual and Developmental Disabilities Research Centers (IDDRCs). The IDDRC[4] represents the nation's first and foremost sustained effort to prevent and treat disabilities through biomedical and behavioral research. The 2013 initial application by the MIND Institute resulted in US$1,300,000 for funding to establish an Intellectual and Developmental Disabilities Research Center (IDDRC) at the University of California, which would be fully integrated into, and benefit from, the existing resources of the UC Davis MIND Institute

and its campus partners under award # U54HD079125 (2013). The IDDRC has a cooperative agreement with the MIND Institute for a total of US$8,369,200[5] from 2013 through 2021, with an average annual distribution of a little more than US$1,000,000 per project fiscal year.

In the TAGGS data award application abstract, the MIND Institute's IDDRC states that it will address four specific aims:

1. Aim 1 is to conduct interdisciplinary translational research that yields insights into the nature, causes, and consequences of IDD and leads to innovative evidence-based approaches to prevention and treatment. To this end, we propose 81 externally funded projects that reflect the themes of Integrated Biobehavioral Characterization of IDD, Genetic and Environmental Contributions to IDD, and Treatment of IDD. These projects are led by 43 investigators from 16 academic departments and 5 schools/colleges. 68 projects are funded by the NIH, including 16 by NICHD.
2. Aim 2 is to accelerate the pace of interdisciplinary translational research via the operation of cost-effective, innovative, and widely used scientific cores. In addition to an Administrative Core, we propose four scientific cores. The Clinical Translational Core will facilitate the recruitment of diverse samples of human participants, provide specialized clinical expertise to diagnose and characterize participants, support complex phenotyping, collect and store biospecimens, and integrate digital technologies into treatment studies. The Biological and Molecular Analysis Core will provide access to expertise and technologies in the areas of immune function, cellular and molecular imaging, epigenetics, and environmental exposures as they relate to neural development. The Rodent Behavior Core will provide assays of mouse and rat behavior, guidance in the development of mutant rodent models, and support for preclinical evaluations of drug safety and efficacy. The Biostatistics, Bioinformatics, and Research Design Core will provide support for study design, creation of electronic data capture and management systems, and statistical analysis of complex multidimensional datasets.
3. Aim 3 is to improve the lives of people with IDD and reduce health and social disparities in care by developing a robust plan for disseminating and implementing scientific discoveries. The plan will involve multiple paths of communication to target professionals and policy makers reach diverse communities while being informed by the perspectives of people with IDD and their families.

4. Aim 4 is to conduct a signature research project that examines the heterogeneity of outcomes and mechanisms underlying those outcomes in maternal autoantibody-related (MAR) ASD, which may account for as much as 20% of ASD cases. The project will make use of existing human clinical data to inform the creation of rat model systems to test hypotheses about causal mechanisms. The signature project addresses three RFA themes: Preventing and mitigating the impact of exposures that can cause IDD, Outcome measures or biomarkers for interventions or treatments, and Development of biomarkers or assessment measures in more than one IDD condition.

The last and 4th declared agenda for this grant application is to investigate "maternal autoantibody related (MAR) ASD, which may account for as much as 20% of ASD cases." The only declared Autism agenda of the IDDRC "about" mission[6] is the following: "Biological or biochemical mechanisms that cause behavioral characteristics, such as those found in ASD, self-injurious behavior, and impairments in language development." Mouse studies do not appear on their face to contribute to the Autism related agenda for IDDRCs—especially inducing mice with an altered gait. In addition, it appears that the UC Davis MIND Institute continues a sustained effort to develop and prioritize the medical pathology paradigm for Autism, which sharply diverges from the current Neurodiversity movement (Silberman, 2015).

Most recently, beyond the 2013 application agenda, the IDDRC award # U54HD079125 was also cited as a funding source in the CDKL5 gene deficiency cognitive deficits study (Adhikari et al., 2022) using "Mutant mouse models of neurodevelopmental disorders (NDDs) with intellectual disabilities [as] useful translational research tools," and Silverman is a co-author. This project application, identical to the gait study, does not prioritize Autism in its research agenda but rather Fragile X Syndrome. Again, such speculation of overlaps with Autism is a category mistake (Hall et al., 2010) and not an empirical way of looking at the general benefits of research to Autistic people.

NIH: Eunice Kennedy Shriver National Institute of Child Health and Human Development

The Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD),[7] funded the gait study during 2020-2021 for a total of US$2,589,126[8] (Grant/Award Number P50HD103526). President Kennedy

gave us a great vision of what Autistic people want today when he proposed and signed the Community Mental Health Act of 1963. This legislation was passed as part of his envisioned New Frontier, which led to considerable deinstitutionalization of persons deemed mentally unfit. Kennedy's sister Rose Marie "Rosemary" Kennedy (September 13, 1918—January 7, 2005) spent most of her life being cared for at St. Coletta, an institution in Jefferson, Wisconsin.

This NICHD discretionary award is for scientific/health research and is to include surveys. In the TAGGS data award application abstract, the MIND Institute's proposal states that it will address the same four specific aims as stated in their IDDRC grant as described above. Of significance is the signature project in Aim 4, which dedicates an agenda to "Preventing and mitigating the impact of exposures that can cause IDD" as well as the use of any research outcomes measures, that may indicate "biomarkers for interventions or treatments" as well as "assessment measures in more than one IDD condition." Most of those listed aims seem to require interactions and evidence from actual human beings, not pure-bred mice models.

NIH: National Institute of Neurological Disorders and Stroke

The next grant totaling US$1,717,190[9] for the years 2017 to 2021, is from the National Institute of Neurological Disorders and Stroke (Award Number # R01NS097808). The National Institute of Neurological Disorders and Stroke (NINDS) supports and conducts research on the normal and diseased nervous system. The NINDS mission[10] is to seek fundamental knowledge about the brain and nervous system and to use that knowledge to reduce the burden of neurological disease for all people. This grant application directly pertains to the actual experiments done in the mouse gait study; unlike the other grant applications which are only peripherally related to mouse studies. The project summary per the abstract is provided below:

1. Maternally derived duplicates or triplicates of 15q11.2-q13 (Dup15q) are one of the most common genetic variations associated with ASD detected in 1% to 3% of cases. Prominent features commonly found in Dup15q include ID, epilepsy, developmental delay, hypotonia, speech impairments, and minor dysmorphic features. The ubiquitin E3A ligase gene (UBE3A), which maps to the 15q11.2-q13 region, has been implicated in multiple NDDs, including ASD, Angelman Syndrome (AS), Prader–Willi Syndrome (PWS), and ID. Based on this information, we postulate that dysregulated UBE3A has deleterious outcomes.

2. Because UBE3A is imprinted specifically in neurons, we will use novel mouse models to test the hypothesis that elevated UBE3A in neurons is the major contributor to phenotypes. It is well known that three differentially spliced isoforms of UBE3A exist, propelling us to pursue the secondary scientific question of which isoform plays the most critical role in Dup15q. No in vivo studies, to date, have evaluated the phenotypic contributions associated with each of the three Ube3a isoforms.
3. Preliminary data illustrate our discovery that forebrain, neuronal selective overexpression of Ube3a isoform 2 is sufficient to cause behavioral and anatomical phenotypes. Here, we propose a multifaceted, collaborative project to identify behavior, neuroanatomical and epigenetic mechanisms of isoform-specific Ube3a overexpression. This proposal will directly address the most important questions regarding our main scientific premise that overexpression of UBE3A is the principal pathogenic mechanism causing Dup15q impairments. We will also address our secondary premise that different Ube3a isoforms in neurons cause differential behavioral, pathological, and epigenetic anomalies. We will delineate phenotypes and identify pathologies in each line of isoform-specific Ube3a-overexpressing mice. Significant correlations and corroborations between molecular, cellular, histopathological, and behavioral phenotypes will reveal key information on neural substrates of Dup15q phenotypes. These studies will answer the most important questions regarding the pathogenic nature of mechanisms underlying UBE3A overexpression.

The stated project is to investigate the "molecular, cellular, histopathological and behavioral phenotypes" of Dup15q phenotype, and to answer questions about the pathogenic nature of mechanisms underlying UBE3A overexpression. In the first sentence for this grant, these genetic anomalies are stated to be "the most common genetic variations associated with ASD, detected in 1% to 3% of cases," whereas their other two grants postulate that "testing the causal mechanisms of maternal autoantibodies" is "speculated to account for 20% of ASD cases." Furthermore, for this grant, we detect a direct intention to conflate Autism with Angelman Syndrome as allegedly both are implicated in multiple NDDs. The human clinical data that was used to inform the rat model creations cannot be implicated in the genetic manipulation of nonhuman animals.

Foundation for Angelman Syndrome Therapeutics

The Foundation for Angelman Syndrome Therapeutics ("Foundation") website specifies that their "FAST Targeted Research to Advance a Cure" (FAST TRAC) Program provides targeted funding to enable translational research projects in Angelman Syndrome ("AS"). Angelman syndrome is not clinically related to Autism, despite some diagnostic overlap in the functional presentations of motor movement, communication, and development.

The Foundation specifies that their organization's mission is: "FAST is dedicated to finding a cure for Angelman Syndrome (AS) and related disorders through the funding of an aggressive research agenda, education, awareness, and advocacy." Given that both Autism and Angelman are largely considered developmental disabilities with pervasive deficits in cognition, behavior, and neurological motor movement, the link between the aggressive research agenda and the author's publication in the "Autism Research" journal is in line with their project aim to the NIH. A query on the *FAST-Funded research* database confirmed the posted infrastructure grant announced by the Foundation.

The Foundation's grant webpage *declares an exclusive partnership* with a full-page honorable mention of the UC Davis MIND Institute (the "Institute"). *Jill Silverman's* team at the University of California, received the grant from FAST with the express purpose of building a lab devoted to creating a new generation of scientists focused on researching Angelman syndrome with their combined expertise in molecular and behavioral components, establishing a stable infrastructure in which this team can evaluate multiple therapeutics simultaneously through, at least 2025, confirmed by the *MIND INSTITUTE May 14, 2020* press release to be the amount of US$3 million. The use of this grant has fallen far short of its goals in the hands of Silverman and her team, instead used for an initiative that is definitely not a priority of the Foundation *or* Autistic people; a feat not uncommon for Silverman, as seen in her 2017 grant (Grant #FT2017-002: 2017-2018) from the Angelman Foundation, where similar unfounded conflations were made between AS and Autism.

Jill Silverman from the MIND Institute co-authored and published another study of mutations on the X chromosome CDKL5 deficiency disorder (CDD) in the *Journal for Human Molecular Genetics* (Adhikari et al., 2022). The study looked at cognitive deficits, hyperexcitability, and hyperactivity in males and females using two models of CDKL5 Deficiency. The authors claim that numerous genetic factors have been shown to confer risk for NDDs, including ASD and Intellectual Disabilities (IDs). Not surprisingly, this cognitive deficit study declares an identical federal funding source as the

gait study, with the intention to link rodent models to Autistic people. However, the only single mention of Autism is in the context of one conjecture about severe NDDs, and that they are "resulting from mutations on the X chromosome: CDKL5 deficiency disorder (CDD)."

We do not know yet if the seizures in Autistic people may be linked to CDKL5 mutations in the future. CDKL5 mutations have been identified in many ethnic groups, with more females than males being reported with an approximate ratio of 4:1 (NORD, 2020). The prevalence of mutations in CDKL5 is estimated at approximately 1 in 40,000 to 60,000 live births (Jakimiec et al., 2020). Although rare, CDD is one of the most common forms of genetic epilepsy. In 2017, the total diagnosed prevalence of CDD in the United States was 208 in males and 1,315 in females (Wood, 2020). This prevalence is so rare that it is precariously situated within the grant aims for investigating treatment options for intellectual and developmental disabilities in Autism, which is indicative of a major conflict of interest with funding fidelity.

Only after the completion of the 5-year funded project, did Silverman publicly critique the marginal translational value of rat models, which she originally touted in her grant applications as a primary objective to use rat models for translational research:

> Given behaviors in a model system with apparent face validity to symptoms of ASD (specifically social communication deficits together with the presence of restrictive and repetitive behaviors) are never going to look the same as those displayed in a human, it is imperative that researchers avoid exaggerating the model system's relevance by recognizing and stating that not every behavioral feature in an animal model should be expected to completely phenocopy the complex and heterogeneous features of NDDs in humans (Silverman et al., 2022).

After Silverman's 5-year funded studies of behavioral and developmental deficits of Angelman syndrome, the MIND Institute has completed its grant project intended to find a translational model for developmentally disabled humans. Most notably, Professor David Segal who is a member of the UC Davis Genome Center and a faculty member at the UC Davis MIND Institute was awarded US$1.36 million[11] to develop bioengineered pacemakers, and to find a cure for Angelman syndrome "caused by loss of ubiquitin ligase E3A (UBE3A) gene expression in the brain." This departure from Silverman's rodent model research claims of relatability to Autism directly correlates to the cessation of NIH funding, which we sincerely hope is due to her conclusion about the zero-impact translational value to enhancing the QoL for

Autistic individuals originally proposed to be within the scope of her research aims.

Analysis: Whether a Human Brain Is Comparable to Mouse Brain

There is no proven evidence showing down-regulation of an Angelman gene variant linking biomarkers of gait abnormality to any other ID/DD condition. Researchers who investigate the deficit models of human function are speculating that "Analyzing animal self-grooming also has a broader value in the study of neurobiology underlying complex repetitive behaviors," which may be disrupted in certain neurological diseases (Kalueff et al., 2016). The connection between Autistic people a neurologically diseased human subvariant still cannot be scrutinized under the lens of animal behaviors. This is what makes the use of animal models dehumanizing, because it degrades an incredibly complex human experience down to a set of observable behavioral traits, as though this is all we are.

We reject the UC Davis MIND Institute's further study of immune function, cellular and molecular biology, and environmental contaminants to measure complex human behavior. As a matter of human rights, genetic research cannot be leveraged to impose biological and behavioral determinism (CRPD, 2022). While genetic research can offer life-saving and tangible benefits for people with genetic conditions, the conflation of Autism with co-occurring medical diagnoses disambiguates Autism and becomes an umbrella term for a variety of genetic conditions that may have behavioral implications, with dangerous real-world consequences. It is critically important to maintain the specificity of how each individual genetic condition impacts a whole person and to use the genetic diagnostic language to characterize corresponding conditions without using Autism or ID/DD as an umbrella term.

Autism is not synonymous with genetic expression. For example, left-hand dominance is highly correlated with Autism; however, not all Autistic people are left-handed, and not all left-handed people are Autistic. Disability and educational rights proponents have established a cultural definition of what it means to be Autistic which introduces an "objective self-awareness extracted from the real-world situation" (Vallacher, 1978, p. 66). The use of abnormal gait measures to compare with the restricted and repetitive behaviors that fulfill the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; American Psychiatric Association, 2013) criterion for Autism overlooks that these observable behaviors that are mostly voluntary

and often reportedly used for information processing and emotional regulatory purposes. It also implies each behavior has only one cause and all behaviors deemed abnormal are connected to a syndrome. The notion that an animal model has been manipulated to act in a way that is, to the researcher's view, in an Autistic manner is in and of itself, a fallacy.

Not all Autistics have complex medical or genetic conditions that impact their health, and where they do, the priority for what to study and to what purpose should be guided by the affected people. Autistic people, with the support of professionals and family members adjacent to Autistic people, have built a culture and community around understanding the scientific research in a framework that incorporates our lived experience and voice to create more effective supports and approaches for accommodating Autistic needs (Nicolaidis et al., 2019). The majority of Autistic people do not want a cure for Autism; however, many of us would love to see better medical treatment for co-occurring conditions. Both these preferences are fine choices and should be tolerated and widely accepted without prejudice.

When researchers conflate Autism with a variety of diagnoses associated with only the biased and superficial understanding of what it means to be Autistic, it impedes the progress of human rights and causes undue division in the burgeoning community creating actionable and tangible support for Autistic people. Autism shall not be defined in those frameworks, whereby the pipeline of diagnosis to treatment is based on behavioral and psychiatric observations. The key consideration of valuable research contains measures of well-being options such that it recommends self-sufficiency, financial stability, informed decision-making, and autonomy. Finally, Autism researchers must be in agreement for the participatory coding of themes as it pertains to the QoL of Autistic consumers (McConachie et al., 2018).

Behavioral markers are still widely used as measures of efficacy for applied behavior analysis (ABA) as an end-all recommendation for all Autism presentations but in fact significantly correlates with greater traumatic stress symptoms when measured across the lifespan (Kupferstein, 2018). Classic chronic medical gaslighting results from problematic conflated recommendations. Autoimmune encephalitis/PANS/PANDAS are common conditions often misdiagnosed initially as Autism (or as a worsening of Autism, when they occur later in children already diagnosed with Autism), while those with those inflammatory, immune-modulated conditions are enlisted in intensive behavioral interventions and the fact that they are experiencing active inflammation around their brains causes traumatic brain injury presenting as regressive traits (Rossignol & Frye, 2014; Theoharides et al., 2016). Consequently, any mouse model that mimics these behavioral presentations cannot inform treatments for abnormal gait without these confounding markers.

Disabled people must be intentionally represented such that citizen control, delegation, and partnership are prioritized. Placation, consultation, and informing are mere tokenism, while therapy in the absence of representation goes along with manipulation as nonparticipation (Arnstein, 1969). American taxpayers must encourage a more enlightened typology of citizen participation in federal social programming and funding of representative organizations. Rodent models and pathological review of Autistic typology does not prioritize the well-being of the daily living needs of the Autistic.

Relatability of Angelman Mice to Human Autistic Behavior

The human brain is not comparable to a mouse brain, and there is no shortage of Autistic people who are willing to participate in Autism research when conducted humanely for socially beneficial purposes. Since the advent of the human genome project, scientists may use mice to simulate human genetic disorders to study their development and test new therapies. Mice are far better than flies or worms for studying complex biological systems found in humans, such as the immune system, endocrine (delivers hormones into the body), nervous, cardiovascular, and skeletal systems. Like humans, mice naturally develop diseases that affect these systems, including cancer and diabetes. Mouse studies are grossly inapplicable to the Autism condition, as Autism is not classifiable in the disease model of scientific research. It is widely known[12] that a definitive diagnosis for Angelman syndrome is made with a single blood test of the individual's chromosomes (Mayo Clinic).

The use of mice in biomedical research needs to take into account the evolved differences as well as the similarities between mice and humans. In the early days of biomedical research, scientists developed mouse models by selecting and breeding specific mice to produce offspring with certain desired characteristics. Mice are less reliable as models of human disease because the networks linking genes to disease are likely to differ between the two species.

Many Autistic people believe the term "disorder" to be an oversimplification, or in some cases, even just plain categorically incorrect, because the diagnostic criteria do not reflect some of the most salient aspects of the lived internal experience of the heterogenous group of people identified in this manner; nor do the deficit-based descriptions provided in the diagnostic criteria reflect how autistic people experience their disadvantages internally. Using rodent models deliberately bred with a specific genetic defect should not be funded by organizations that purport to study or diagnose Autistic humans.

In accordance with the ethical principles for medical research per the declaration of Helsinki,[13] vulnerable groups of individuals may have an increased likelihood of being wronged or of incurring additional harm when research is conducted unethically; "Because all vulnerable groups and individuals should receive specifically considered protection," we must evaluate the gait study for the "net benefit from the knowledge, practices or interventions" that result from the research. In a capitalist society, the Autism Industrial Complex (AIC) commodifies Autism and Autistic people as well (Broderick, 2022). A consumer of evidence-based practices is an Autistic person who has no power or say in the underwriting of the research that is about them but without them. Therefore, these practices are largely uninformed by the consumers, leaving us, the Autistics, without any other strategy except to single handedly audit the impact of the Autism Industrial Complex (AIC). By design, the AIC keeps us out of their infrastructure.

Typically a research project only has two to three aims and there would rarely be a time when one grant would find more than one project. Funding agencies, particularly the government, like to find people who have been funded and have been successful. The aims of these projects contain drastic outcomes that leap from rat models to behavior in Autism without a clear link to the trajectory of funding toward each benchmark. Salary for grants can be paid in a number of ways. Some might be a buyout (someone else teaches for me), some could be summer salary, and some could be additional for activities such as conference travel. There are often limits to what we can pay ourselves based on time on grant or role (PI or subcontractor).

Some things to consider, though, are the indirect costs (i.e., what the University takes off of the top) and the personnel costs (i.e., salary for faculty, staff, & students). Both of these things take up the bulk of any funding that comes from the Federal grants listed in the sum of awards table of their TAGGS data. Conservatively, indirect costs and personnel could consume anywhere from 50-70% of a given budget. As an example, a 2.5 million grant may only have about 1 million to spend for the actual project itself.

Prevalence Disparities Between Autism and Angelman Syndrome

Of concern to Autistic people is the NIH's overarching mission to eradicate chromosomal anomalies in society. The Foundation for Angelman Syndrome Therapeutics' prevalence of Angelman syndrome is reportedly one in 15,000 people—about 500,000[14] individuals worldwide. By contrast, the U.S. Autism prevalence is approximately 340× higher, at 1 occurrence in 44 live

births, according to the most recent estimates from the Centers for Disease Control and Prevention (CDC, 2022). The Angelman syndrome prevalence is so minuscule when compared with Autism prevalence that is dubiously situated within the grant aims for investigating treatment options for intellectual and developmental disabilities in ASD, which is again indicative of ethical dishonesty with funding underwriter discretion. Here we see how they leverage concern over the existence of Angelman syndrome as a way to divert research funding from Autism, which has a far higher prevalence. The conflation of Angelman Syndrome with Autism tends to skew the understanding of the public as casual consumers of this agenda-driven aim to confound all ID/DD with Autism.

The Institute in co-authorship with Silverman continues to utilize genetically modified pure-bred rat line models in the evaluation of cognitive-communication disorders based on genetic modification in rats and to advance the agenda that "both the Ube3a maternal deletion mouse and rat models of Angelman Syndrome reliably demonstrate behavioral phenotypes of relevance to AS and therefore offer suitable in vivo systems in which to test potential therapeutics" for developmentally disabled humans (Berg et al., 2021). The CDD study declares their funding source from the National Institute of Neurological Disorders and Stroke, Grant/Award number: R01NS085709; Eunice Kennedy Shriver National Institute of Child Health and Human Development, Grant/Award number #U54HD079125. It is this last Grant/Award number #U54HD079125 that is the same as the mouse Gait study investigated in this audit. Overall, the impact of this mouse Gait study is still scored by the single nonreferential self-citation to evaluate the impact on the body knowledge as it pertains to Autism since it was published in the Autism Research Journal.

In lay terms, the UC Davis MIND Institute's research focus is on early detection, prevention, and cure of intellectual and developmental disabilities. This aggressive agenda is deeply offensive and morally unjust with regard to the humanity of Autistic people, who want to live well in a socially restrictive setting. Autistic people should not be constantly told by researchers that they are aberrant and that the generations in the future would be better off without Autism. This reflects the prevailing opinion of Autistic adults who have generously participated in research toward a social model of Autistic culture, acceptance, and amelioration of the supremacy of the medical pathology paradigm (Rosqvist et al., 2020). There are contactable emerging communicators who have been formally classified as severe cases who are concerned with secondhand reporting to researchers about their lived experiences.

IDDRC funding for Autism research related to the well-being of Autistic persons throughout their lifespan is deprioritized versus funding for research

for a cure and prevention of "Chromosomal conditions that cause intellectual disabilities, such as PWS, Angelman Syndrome, Williams Syndrome, and Down Syndrome." Cure and prevention are both prioritized in the study toward uncovering causal mechanisms for the behavioral characteristics of Autistic people. Autistic people are acutely aware that funded Autism researchers are not concerning themselves with applicable dissemination, as they do not see Autistic people as the direct consumers of their research, which further perpetuates the AIC (Broderick & Roscigno, 2021). This commodification of Autism and the commodification of Autistic people spurs the impetus for our consumer audit and upcoming planned audits of all biomedical and pathophysiological research published in Autism journals.

Implications and Recommendations

It is our first professional recommendation that the *Journal for Autism Research* shall redact the mouse Gait study. This redaction is based on a compromised peer review that is misaligned with the journal's mission statement. Furthermore, it is a concern for public safety that a single gene anomaly in mice models bred to simulate Angelman syndrome is used to speculate about developmental and intellectual disabilities, which is evidence of an older paradigm that has largely been superseded by modern genetic and molecular research. The aim of the grant recipients is to extend the biomedical applications of mice models for human Angelman syndrome. The gait study, after redaction, must be rewritten to steer clear of the unfounded translational value to intellectual and developmental disabilities, especially in the aims of relatability to Autism.

The conjectured Autism link mentioned in their aims is outside the scope of the original research. We recommend legislative action to realize that since the NIH is overseen by the U.S. Food and Drug Administration (FDA), under HHS, and not the Mental Health and Family divisions, or the CDC. It is important to recognize that when there is no disease to underwrite the policy and research decision-making, the U.S. government structure is more likely to defer the research and policy to the NIH, based on its administrative structure. Here too we see the diagnosis-to-treatment pipeline for behavioral interventions even before pharmaceuticals.

This gait study also has not fulfilled the project's aim to make use of existing human clinical trials to test any hypotheses about causal mechanisms. The signature project was proposed to address the prevention and mitigation of the impact of exposures that can cause IDs/DDs. This gait study has not yielded any specific measures or biomarkers for interventions or treatments or assessment measures for any ID/DD human condition. We are requesting

that the MIND Institute shall remove all Autism related language in their grant projects so long as they maintain a strictly nonhuman biomedical and pathological research agenda. A footnote should be added to the grant abstract detailing to the reader that the article has been corrected while it is publicly posted.

We also recommend a specific correction in the event that the study may be later published in a more suitable trade journal. Primarily, we demand a full removal of the speculative link to Autism research and the relevancy of this mouse gait study, given that the research does not meet the grant aims of testing the causal mechanisms of maternal autoantibodies that had been speculated to account for 20% of ASD cases. The correction notice should provide clear details of the error and the changes that have been made to the version of the record. A redaction notice shall be issued to make specific invalid conclusions in the article. Wiley & Sons shall add a "redacted" watermark to the published version of the record of the article to prevent accidental referencing and to prevent undermining efforts to protect the integrity of the scientific literature. Silverman has never returned our calls and emails through July and August 2022, and David Amaral, Director of the MIND Institute who is also the Editor of the INSAR Autism Research journal, was asked to clarify the affiliation of his editorial team, with no response. Amaral was also contacted in regard to an article in the *Autism Research* journal by Waizbard-Bartov et al. (2023).

We hope that future Autism research considers the needs of the Autistic consumer as a primary focus in research design, grant applications, and project language as a pattern of practice. To shift the pathology paradigm embedded in the culture of the AIC, contaminated, dissonant, and polyphonous projects of biocapitalism only offer subjunctive survival of the damaging rubric of the AIC; As such, a systemic dismantling will require a multiplicity of assemblages in subversion of the AIC so that our revolution is not a project of biocapitalism, but one of "collective Autistic—and therefore *human*–survival" (Broderick, 2022). If Autistic scientists merit any sums awarded for this audit, we would consider it largely insufficient to mitigate the damages of the gait study and its egregious harm-inducing medical orientation.

An example of translational research that would be relevant to Austitic people is to investigate the detrimental impact of behavioral interventions potentially directed at the hundreds of millions of Autistics worldwide, which is of great concern, as U.S. policy continuously sets a global precedent. In fact, caregivers who discontinued ABA, and the Autistics who were exposed to no treatments, fared best in adulthood (Kupferstein, 2019; Lord et al., 2022). Beyond translational value speculations, novel research must prioritize a refinement of the clinical and biological concepts of ASD by taking an

appropriate holistic approach at the molecular level (Breitenkamp et al., 2015).

Research on the whole Autism experience and related disorders could be translated into treatments. For example: Voltage-gated calcium channels and inwardly rectifying potassium channels have already been shown to cause problems like hypo- and hyperkalemic periodic paralysis or hypovolemic shock (Wen et al., 2016). A precursor for an episode is usually hypo- or hyperkalemic sensory overstimulation, which Autistic people describe as sensory overload and/or cognitive underwhelm. Yet the knowledge gained from these gene studies has never translated into a treatment regimen for sensory signaling in Autistic people or other ID/DD conditions.

It is time for the U.S. Government to represent the millions of Autistic citizens who have been grossly underserved by grants and legislative initiatives that fail to fund Autism research dedicated to enhancing Autistic peoples' QoL. Although the majority of autism's costs in the United States are for adult services—an estimate nearing US$2 billion a year, these funds are grossly inadequate and not disseminated to families whose members include adults with profound communication deficits and behavioral consequences. Many Autistic people have said that if they want anything cured, it is not their autism, but this endemic funding disparity.

The projected taxpayer burdens for Autism interventions and supports is expected to rise to US$461 billion by 2025.[15] We conservatively estimate that 2.8% (>100 million) of the global population is Autistic, as compared with a mere half a million identified Angelman syndrome diagnoses. Still, 99% of these treatment costs are largely informed by research that does not consider the overall lived experience of the whole human condition across the lifespan. Future researchers may find this audit a guide to how genetic research could serve the needs of Autistic people, but does not yet. Autistic people themselves are left to interpret these findings from medical research publications and teach their doctors and other professionals.

We appeal to the HHS and NIH and its overseeing bodies to spur an Autism Research Act that would mandate the reworking of the taxonomy of Autism research that impacts the daily lives of Autistic people. We are demanding a legislative guarantee for a quorum of qualified Autistic scientists to be intentionally included in the decision-making for research agendas that would be funded from public and federal money, effective immediately.

Autistics have a proven level of interactional expertise necessary for sustaining effective communicable value with nonmarginalized professionals (Milton, 2014). Autistics are capable of claiming those rights and making decisions for their lives based on their free and informed consent as well as being active members of society (CRPD, 2022; Raymaker, 2020). Therefore,

we expect ethical and humane research, such that it contributes to the well-being of Autistic people, as opposed to funding a privatized governmental agenda to generalize from rodent models; the rodent models support an anti-quated medicalized view of Autistic people.

It would be in the best interest of Autistic people that the findings of this audit stimulate the U.S. Government and the NIH to evaluate the damage that high-cost institutional grants based on outdated paradigms can inflict on human beings. As Autistic scientists, we have conducted this audit under the guidelines set forth by our Autism research governing ethos toward social justice change. We, the Autistic consumers who conducted this audit, want nothing more than a fighting chance at a better life but are still forced to step into the unpaid role of negating inequities in research funding as a grassroots effort to amplify our voice. *Nothing about us, without us.*

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Notes

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