JOURNAL OF NEUROTYPICAL STUDIES

Pollute the world, deny the damage, cut the supports

Super Autism, environmental load, and the remarkable habit of blaming the exposed

Axel

Abstract

This paper examines a pattern that is obvious to anyone who has not outsourced their conscience to a quarterly earnings report: some of the loudest calls to cut disability supports come from the same general class of interests that profit from pollution, environmental degradation, and climate decay. Current research does **not** establish a simple direct claim that climate change causes autism. It does, however, increasingly support models in which genetic vulnerability interacts with environmental exposures through mechanisms including inflammation, oxidative stress, mitochondrial dysfunction, and epigenetic regulation (Dawson et al., 2024; Naviaux, 2026).

Recent human studies have also reported associations between autism risk and prenatal exposure to extreme nighttime heat and wildfire-related PM2.5, which is awkward for people who prefer their environmental harms to remain theoretical (Yu et al., 2026; Zhang et al., 2026).

I argue that people with **Super Autism**—my term for autistic people with profound sensory vulnerability, intellectual disability and/or complex support needs, and communication that may be embodied rather than conventionally verbal—are often among the first to be affected, among the first to communicate that something is wrong through distress or behaviour, and among the last to be listened to (Narzisi et al., 2025; de Marchena et al., 2025; Aidonopoulou-Read, 2025).

Introduction

I have noticed a pattern. The environment becomes harsher. The bodies most exposed to physiological stress become less stable under that load. The people who react first are treated as the problem. Then the cost of supporting them is presented as the real crisis. This sequence is not well described as “fiscal responsibility.” It is better described as a social habit of protecting causes while complaining about consequences (Dawson et al., 2024; Yu et al., 2026; Zhang et al., 2026).

The current autism literature does not support a simplistic single-cause claim. What it increasingly supports is a gene-environment model. Dawson et al. (2024) argue that the rise in autism prevalence is partly attributable to diagnostic expansion and advocacy, but that the interplay between genetic predisposition and modern environmental exposures is also likely contributing to a true increase in incidence. Their review identifies prenatal infection, gestational diabetes, maternal obesity, medication exposures, and toxicants as relevant lines of evidence, with mechanisms including immune dysregulation, mitochondrial dysfunction, oxidative stress, hormone disruption, microbiome effects, and epigenetic regulation (Dawson et al., 2024).

That matters because climate-linked and pollution-linked exposures are no longer side gossip in the literature. In a large Southern California cohort, exposure to extreme **nighttime heat** during pregnancy was associated with increased autism risk, particularly during gestational weeks 1–10 and 30–37 (Yu et al., 2026). In a separate California study, prenatal exposure to **wildfire-related PM2.5** was associated with autism risk, adding another very inconvenient data point to the growing pile of “perhaps bodies do, in fact, react to hostile environments” (Zhang et al., 2026).

Super Autism as an early-warning position

I use the term **Super Autism** deliberately. It refers to autistic people whose lives make simplistic theories collapse on contact: people with severe sensory vulnerability, intellectual disability and/or rare syndromic forms of autism, limited or unreliable speech, complex co-occurring distress, and bodies that do not quietly absorb environmental insult for the comfort of policy analysts (Narzisi et al., 2025; Aidonopoulou-Read, 2025).

This is not a sentimental framing. Research on sensory processing in autism argues that sensory differences are central to autistic functioning and regulation, not decorative little quirks to be mentioned once before everyone returns to pretending “behaviour” arrived from nowhere (Narzisi et al., 2025). If sensory processing is central, then heat, smoke, toxic load, sleep disruption, pain, and environmental instability are not background scenery. They are physiological events with consequences, especially for people already living close to the edge of regulatory tolerance (Narzisi et al., 2025; Dawson et al., 2024).

In my own case, this question is not abstract. I have an **ASH1L** change. MedlinePlus Genetics notes that ASH1L provides instructions for making a histone methyltransferase involved in regulating gene activity, including genes important for development, and that variants in ASH1L have been identified in people with autism spectrum disorder and

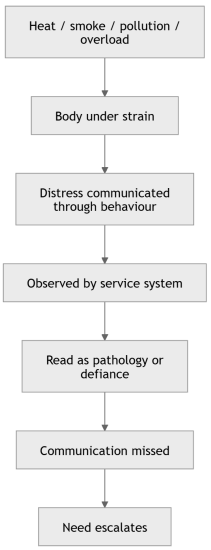
intellectual disability (MedlinePlus Genetics, n.d.). That is gene-regulation biology. Which means the fantasy that genes and environment are separate stories is not science. It is administrative coping (MedlinePlus Genetics, n.d.; Dawson et al., 2024).

Naviaux (2026) takes this further with a three-hit metabolic signaling model in which autism risk reflects: first, inherited susceptibility; second, early environmental triggers; and third, recurrent or persistent exposure during critical developmental windows. One does not need to agree with every sentence of a model to notice when it is pointing in the same direction as lived reality: vulnerability, trigger, persistence, consequence (Naviaux, 2026).

Behaviour as communication

One of the more durable intellectual failures in autism discourse is the belief that if communication is not tidy, verbal, and convenient for professionals, it somehow becomes secondary. The current literature does not support that. A review of communication in autistic adults found that autistic people frequently face barriers affecting relationships, employment, and health, and that communication outcomes depend not only on autistic traits but on interaction partners and environments as well (de Marchena et al., 2025). A framework for ethical listening in research with nonspeaking autistic people likewise argues that non-verbal communication can and should be systematically recorded and interpreted rather than dismissed (Aidonopoulou-Read, 2025).

That matters because people with Super Autism often communicate overload and distress through agitation, shutdown, self-injury, panic, sleep collapse, refusal, loss of regulation, or other embodied forms of communication. The system then performs its favourite trick: it takes communication, relabels it “behaviour,” and uses the new label as permission not to listen. This is not a subtle epistemic issue. It is a repeated institutional failure of interpretation (de Marchena et al., 2025; Aidonopoulou-Read, 2025).

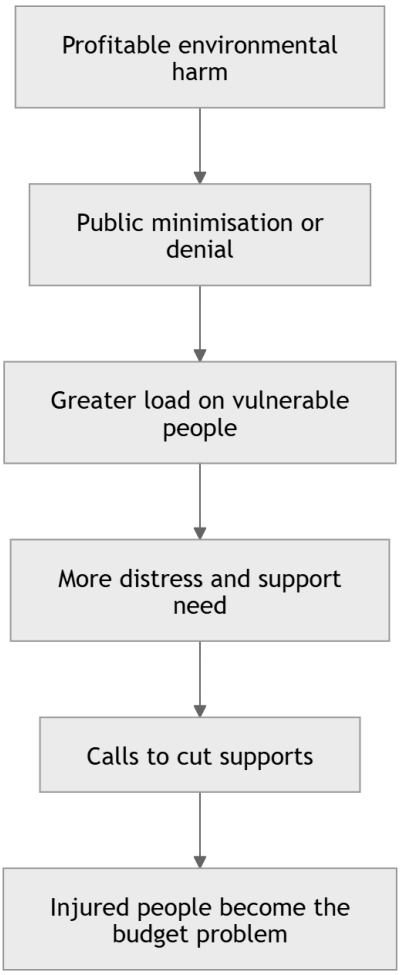


Profit, denial, and the support bill

The social contradiction here is not subtle. If environmental stressors plausibly contribute to neurodevelopmental risk and can intensify instability in susceptible people, then pollution and climate disruption are not external to disability policy. They are part of its upstream architecture (Dawson et al., 2024; Yu et al., 2026; Zhang et al., 2026).

And yet public conversation is often arranged as though environmental harm belongs to one portfolio, disability support belongs to another, and no bridge is permitted between them unless somebody can monetise it. This is especially useful for people profiting from the first while complaining about the cost of the second. The sequence is analytically clear: conditions deteriorate, vulnerable people become less stable, their communication is misread, their support needs intensify, and the resulting support bill is then cited as evidence that supports themselves are the problem (Dawson et al., 2024; de Marchena et al., 2025; Aidonopoulou-Read, 2025).

So when some of the same interests that normalise environmental harm also argue that disabled people are the budget emergency, I do not hear a sophisticated economic thesis. I hear a civilisation trying to invoice the exposed for the cost of exposure (Dawson et al., 2024; Yu et al., 2026; Zhang et al., 2026).



Discussion

The science does **not** yet justify a bumper-sticker claim that climate change directly causes autism. That would be a sloppy overstatement, and I am trying to keep the standards here above the average press conference. But the evidence does justify several narrower and more serious claims.

First, autism risk is increasingly understood through gene-environment interaction rather than genes alone (Dawson et al., 2024; Naviaux, 2026).

Second, mechanisms such as inflammation, oxidative stress, mitochondrial dysfunction, and epigenetic regulation are central to that emerging framework (Dawson et al., 2024; Naviaux, 2026).

Third, recent population studies have identified associations between autism risk and prenatal exposure to extreme nighttime heat and wildfire-related PM2.5 (Yu et al., 2026; Zhang et al., 2026).

Fourth, sensory vulnerability and communication difference are central to how many autistic people experience and signal distress (Narzisi et al., 2025; de Marchena et al., 2025; Aidonopoulou-Read, 2025).

From that perspective, people with Super Autism may not be peripheral to this story at all. They may be among the earliest indicators of it.

They are often among the first to register environmental strain, among the first to communicate that strain through behaviour, and among the last to be believed by systems that still privilege tidy verbal reporting over embodied evidence (Narzisi et al., 2025; de Marchena et al., 2025; Aidonopoulou-Read, 2025).

Conclusion

So no, I am not arguing that science has already drawn a perfect straight line from climate change to autism and hidden it in a drawer labelled “too awkward for donors.” I am arguing something more careful than that. I am arguing that current evidence increasingly supports a model in which vulnerable neurodevelopment is shaped by interaction between biology and environment; that recent studies are identifying associations between autism risk and climate-linked exposures such as extreme nighttime heat and wildfire smoke; that gene-regulatory and metabolic models make these interactions biologically plausible; and that autistic people with high support needs may function as an early-warning population whose distress is routinely downgraded into mere “behaviour” by systems too crude to interpret what they are being shown (Dawson et al., 2024; Naviaux, 2026; Yu et al., 2026; Zhang et al., 2026; Narzisi et al., 2025; de Marchena et al., 2025; Aidonopoulou-Read, 2025).

That leaves one final technical observation. If powerful interests help make the environment harsher, deny the relevance of that harm, ignore the people who react first, and then complain that supporting those people costs too much, the budget problem is not the disabled person.

The budget problem is the civilisation that keeps sending the invoice to the exposed.

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