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SPECTRA

WORLDWIDE EVIDENCE REVIEW

Electromagnetic Fields and Autism Spectrum Disorder

Artificial electromagnetic fields as an environmental stressor in autism

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ABSTRACT

This review examines the peer-reviewed, institutional and public literature on electromagnetic fields (EMF) and autism spectrum disorder (ASD) published or re-verified between 2002 and September 2026. It does not ask whether EMF causes autism. The human evidence for that proposition does not exist, and this review says so plainly. It asks a narrower and more useful question: whether artificial electromagnetic fields, including radiofrequency fields from wireless devices (RF-EMF), extremely low-frequency fields from power systems (ELF-EMF) and the transient noise on electrical wiring known as dirty electricity, behave as an environmental stressor for autistic people in the same way that acoustic noise, artificial light, chemical exposures and sleep loss are already documented to do.

The argument has two halves. First, autistic individuals carry a meta-analytically confirmed physiological vulnerability profile across oxidative stress, mitochondrial function, immune and neuroinflammatory activation, calcium-channel biology, autonomic regulation, hypothalamic-pituitary-adrenal (HPA) reactivity, sensory processing, sleep and melatonin, blood-brain and gut barriers, and seizure susceptibility. Second, EMF exposure perturbs several of these same systems in animal, in vitro and, more weakly, in human experimental work. Where the two halves overlap, EMF is a plausible and modifiable load on an already dysregulated system. The strongest points of convergence are oxidative stress, cortical excitability and sleep. The weakest are autonomic function and HPA reactivity, where the human EMF literature is inconsistent in direction or null.

The review also documents an empty intersection. No peer-reviewed study has exposed an established autism animal model to environmental-level EMF, and no provocation, device-reduction or physiological study of autistic humans under EMF exposure exists. That absence bounds what can be claimed and defines the research agenda. All 22 sources of the original review were link-checked; three of the peer-reviewed anchors were linked to secondary or wrong-publisher pages, three carried factual errors, and one widely repeated dosimetry figure for the 2025 Yale organoid study (“0.025% of the FCC limit”) is a secondary calculation that does not appear in the paper. Roughly 90 additional peer-reviewed and institutional sources were added and every reference was standardized to APA 7th edition.

1. Background and scope

Autism spectrum disorder affects roughly 1% of children worldwide, with higher estimates in recent surveillance from the United States (Zeidan et al., 2022). The prevalence recorded in administrative data has risen steeply since the 1980s. In Denmark, changes in diagnostic criteria and the inclusion of outpatient diagnoses together account for about 60% of the increase observed in children born between 1980 and 1991 (Hansen et al., 2015). Any argument about environmental contributors to autism has to start from that fact: much of the apparent epidemic is a change in counting, and the remainder is shared between genetic architecture, perinatal factors and a still incompletely mapped set of environmental exposures (Bölte et al., 2019; Modabbernia et al., 2017).

Artificial electromagnetic fields sit outside the exposures that umbrella reviews of autism risk have examined. Neither Modabbernia et al. (2017) nor Bölte et al. (2019) lists EMF among the reviewed environmental factors. At the same time, the International Agency for Research on Cancer classifies both radiofrequency fields (IARC, 2013) and extremely low-frequency magnetic fields (IARC, 2002) as possibly carcinogenic to humans (Group 2B), the European Academy for Environmental Medicine has published clinical guidance that names autism explicitly (Belyaev et al., 2016), and a 2025 study in Cell Reports reported that radiofrequency exposure altered radial glia differentiation and induced autism-associated gene expression in human cortical organoids (Cakir et al., 2025). The subject is neither settled nor fringe.

EFEIA approaches this question from the discipline it works in: electromagnetic hygiene, understood as the management of a person's electromagnetic environment in the same way that acoustic, thermal, chemical and light environments are managed. Within that discipline the relevant question is not etiological. It is whether a given exposure raises or lowers the physiological load on a person, and whether reducing it changes anything measurable. This review is organized around that question.

1.1 Scope

The review covers: (a) the physiological vulnerability profile of autism as established in meta-analyses and systematic reviews; (b) the proposed biological mechanisms by which non-thermal EMF exposure interacts with living tissue; (c) in vitro, organoid and animal evidence on EMF and neurodevelopment; (d) human epidemiology on prenatal, parental occupational and residential EMF exposure and child neurodevelopmental outcomes; (e) the convergence between the two halves, system by system; (f) sleep as a candidate mediating pathway; (g) therapeutic applications of applied electromagnetic stimulation in autism; (h) regulatory and institutional positions; (i) public and advocacy discourse; and (j) evidence gaps. Sources span the United States, the United Kingdom, Canada, Spain, Italy, France, Germany, Austria, Switzerland, Sweden, Denmark, the Netherlands, Montenegro, Iran, India, Indonesia, Australia and the international bodies WHO, ICNIRP and IARC.

2. Methods

The review builds on an anchor set of 22 sources compiled through structured literature searches conducted between 2024 and September 2026, weighted toward 2025 and 2026 primary research, institutional statements from WHO, ICNIRP, ARPANSA and equivalent regulators, European medical-society guidance, the mechanistic papers most cited by the precautionary community, and news and advocacy coverage from outside the United States. That set was then extended in two stages. The first traced every anchor citation back to its primary source, resolving each link and DOI and adding the human epidemiology, animal, mechanistic, regulatory and legal literature that the anchor set cited only indirectly. The second searched each half of the stressor argument on its own terms: the physiological vulnerability literature on autism, and the experimental EMF literature on each of the same systems.

Every URL was opened and compared against the content attributed to it, every DOI resolved through doi.org, and author lists, journal names, volumes, issues, page ranges and dates checked against the indexing record. Where a link pointed to a secondary page, a news release, a wrong publisher or an aggregator, the primary DOI landing page was substituted. Each source carries a 0 to 10 quality score that reflects, weighted roughly equally, the peer-review or editorial rigor of the outlet, the methodological strength of the underlying evidence, the independence and transparency of the source, and how well the claims are supported by the data presented. Scores are the reviewers' judgment for this document, not an external metric. All references follow the seventh edition of the Publication Manual of the American Psychological Association, with a DOI expressed as a URL and a retrieval date on pages designed to change.

Table 1 lists the 22 sources that anchored the review, with their type, year, country of origin, stance and quality score. Each score is the reviewers' judgment under the rubric above, not an external metric. The anchor set is not the evidence base of this review. Roughly 90 further peer-reviewed and institutional sources were added in the two stages described above, and the sections that follow rest on those primaries rather than on the anchor items.

Table 1. At-a-glance source table (22 items)

#	TITLE / SOURCE	TYPE	YEAR	COUNTRY	STANCE	SCORE
1	Radiofrequency regulates the BET-mediated pathways in radial glia differentiation in human cortical development Cell Reports 44(10), art. 116238 — DOI 10.1016/j.celrep.2025.116238	Experimental / study	2025	USA (Yale, Colorado State, Minnesota State, U. Bridgeport) + present-address collaborators in Canada and China	Supportive	9.5
2	Yale Study: Wireless radiation increased the expression of autism-related genes in laboratory models Environmental Health News (EHN.org)	News / advocacy	2026	USA	Supportive	6.5

#	TITLE / SOURCE	TYPE	YEAR	COUNTRY	STANCE	SCORE
3	Expert reaction to review linking wireless technology to childhood development, autism and ADHD Science Media Centre (UK)	Other	2026	UK	Skeptical	8.0
4	Electromagnetic Fields, Pulsed Radiofrequency Radiation, and Epigenetics: How Wireless Technologies May Affect Childhood Development Child Development 89(1):129–136 — DOI 10.1111/cdev.12824	Review / policy	2018	USA (Sage) + Italy (Burgio, ISDE)	Supportive	6.0
5	Effects of Extremely Low-Frequency Electromagnetic Field Treatment on ASD Symptoms in Children: A Pilot Study Brain Sciences 14(12):1293 — DOI 10.3390/brainsci14121293 (MDPI, Basel)	Experimental / study	2025	International (MDPI publisher, Switzerland); author affiliations not fully listed on page	Supportive	5.5
6	Central Causation of Autism/ASDs via Excessive [Ca2+]i Impacting Six Mechanisms Controlling Synaptogenesis during the Perinatal Period: The Role of Electromagnetic Fields and Chemicals and the NO/ONOO(-) Cycle, as Well as Specific Mutations Brain Sciences 14(5):454 — DOI 10.3390/brainsci14050454	Review	2024	USA	Strong support	5.0
7	Findings in Autism (ASD) Consistent with Electromagnetic Fields (EMF) and Radiofrequency Radiation (RFR) BioInitiative Report 2012, Section 20 (BioInitiative Working Group)	Review / policy	2013	USA (both authors); influential internationally	Supportive	7.0
8	Autism and EMF? Plausibility of a pathophysiological link — Parts I & II Pathophysiology 20(3):191–209 and 211–234 (Elsevier)	Review	2013	USA	Supportive	6.5
9	Electromagnetic fields and oxidative stress: The link to the development of cancer, neurological diseases, and behavioral disorders Electromagnetic Biology and Medicine 45(1):94–118 — DOI 10.1080/15368378.2025.2567872	Review	2026	Montenegro	Supportive	5.5
10	Determining the Impact of Nonionizing Electromagnetic Waves on Human Pregnancy and Teratogenicity: Protocol for Systematic Review and Meta-Analysis JMIR Research Protocols — DOI 10.2196/86479	Protocol	2026	Indonesia	Neutral	7.5
11	La Complutense acoge un congreso con expertos que relacionan el 5G con el autismo y el cáncer El Confidencial (Salud section)	News / advocacy	2026	Spain (event); USA + Canada (featured researchers)	Supportive	5.5
12	EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses Reviews on Environmental Health — DOI 10.1515/reveh-2016-0011	Review / policy	2016	Europe (Germany, Austria, and other EU authors)	Supportive	6.5
13	FAQ — ICNIRP RF EMF Guidelines 2020 (and related ICNIRP statements) icnirp.org	Review / policy	2026	International (secretariat in Germany; membership WHO-recognized)	Skeptical	9.0

#	TITLE / SOURCE	TYPE	YEAR	COUNTRY	STANCE	SCORE
14	Electromagnetic fields — WHO topic page who.int	Review / policy	2026	International (Geneva)	Neutral	9.0
15	Review on radiofrequency electromagnetic fields and pregnancy outcomes misses the mark arpansa.gov.au	Review / policy	2026	Australia	Skeptical	8.5
16	Wireless Radiation and Fetal Brain Development latitudes.org	News / advocacy	2025	USA	Supportive	5.0
17	Yale Study Finds Low-Level Wireless Radiation Altered Developing Brain Cells and Increased Autism-Related Gene Expression childrenshealthdefense.ca	News / advocacy	2026	Canada	Strong support	4.5
18	Cell phone use in pregnancy may cause behavioral disorders in offspring Scientific Reports 2:312 — DOI 10.1038/srep00312	Experimental / study	2012	USA	Supportive	7.5
19	Wissenschaft (Science) esc-info.eu	News / advocacy	2025	Germany / EU	Supportive	4.0
20	Babies in Wi-Fi-dense homes face triple risk of developmental delays NaturalNews.com	Review	2025	India (study) / USA (outlet)	Strong support	3.5
21	EMF and Autism: Can Electromagnetic Fields Cause Autism? emfradar.com	News / advocacy	2026	Unspecified	Neutral	5.0
22	Electromagnetic Fields and Autism: What the Research Actually Says blossomabatherapy.com	News / advocacy	2026	USA	Neutral	4.5

3. Autism as a physiologically vulnerable state

The first half of the stressor argument rests on a body of literature that has nothing to do with electromagnetic fields. Over the past two decades, meta-analyses and systematic reviews have established that autism is accompanied, in a large fraction of cases, by measurable abnormalities in the systems that regulate cellular energy, oxidative balance, immune signalling, ion transport, autonomic tone, stress hormones, sensory gating, sleep and tissue barriers. Each of these is summarized below, because each is a system that the EMF literature also claims to perturb.

3.1 Oxidative stress

A meta-analysis of 87 biomarker studies found that blood glutathione, glutathione peroxidase, methionine and cysteine were significantly reduced, and oxidized glutathione significantly increased, in autistic individuals compared with controls (Frustaci et al., 2012). Post-mortem brain studies place oxidative stress, mitochondrial dysfunction and neuroinflammation in the same regions, notably the cerebellum and temporal cortex (Rossignol & Frye, 2014). The hypothesis was first articulated in detail by Chauhan and Chauhan (2006) and remains current in the biomarker literature (Liu et al., 2022).

3.2 Mitochondrial dysfunction

The prevalence of clinically diagnosable mitochondrial disease in autism is about 5.0% (95% CI 3.2 to 6.9%), roughly 500 times the general-population estimate, and a further 30% or so of autistic children show abnormal lactate, pyruvate or carnitine without meeting full diagnostic criteria (Rossignol & Frye, 2012). Frye and Rossignol (2011) argue that mitochondrial dysfunction links the gastrointestinal, immune, seizure and metabolic comorbidities of autism; Palmieri and Persico (2010) raise the question of whether it is cause or consequence. For the stressor argument the direction does not matter. A population with reduced mitochondrial reserve has less capacity to buffer any additional oxidative load.

3.3 Immune activation and neuroinflammation

Vargas et al. (2005) documented marked microglial and astroglial activation, with elevated MCP-1 and TGF-beta1, in the cerebellum, midfrontal and cingulate cortex of autistic brains. Meta-analyses of peripheral cytokines confirm a pro-inflammatory profile: higher IL-1beta, IL-6, IL-8, IFN-gamma, eotaxin and MCP-1 and lower TGF-beta1 across 743 autistic participants (Masi et al., 2015), with a later and larger update adding IL-17 and TNF-alpha (Saghazadeh et al., 2019). Estes and McAllister (2015, 2016) review immune dysregulation in brain and periphery and the role of maternal immune activation.

3.4 Calcium signalling and ion channels

Timothy syndrome, caused by a gain-of-function mutation in CACNA1C encoding the CaV1.2 L-type calcium channel, produces autism in a majority of surviving patients (Splawski et al., 2004). It is the clearest genetic demonstration that excess voltage-gated calcium-channel activity can generate an autism phenotype. Broader reviews place calcium signalling at the convergence point of many autism-associated genes (Krey & Dolmetsch, 2007), describe autism as in part a channelopathy (Schmunk & Gargus, 2013), and report association of common calcium-channel variants with ASD (Lu et al., 2012). This row matters because voltage-gated calcium channels are also the mechanism most often proposed for non-thermal EMF effects (Section 4.1).

3.5 Autonomic regulation

A meta-analysis of heart rate variability found significantly lower vagally mediated HRV at rest in autistic individuals (Cheng et al., 2020). Systematic reviews describe a pattern of elevated baseline sympathetic tone and reduced parasympathetic activity (Benevides & Lane, 2015; Arora et al., 2021), and experimental work shows elevated baseline heart rate with blunted reactivity, consistent with chronic autonomic arousal (Kushki et al., 2013).

3.6 HPA axis and stress reactivity

Cortisol rhythm in autism is flatter and more variable, and cortisol responses to social and sensory stressors are heightened or dysregulated (Taylor & Corbett, 2014). Autistic children show higher and more variable evening cortisol, associated with sensory sensitivity (Corbett et al., 2009), and a significantly larger cortisol rise to a mild stressor such as a blood draw (Spratt et al., 2012).

3.7 Sensory over-responsivity and cortical excitability

Sensory modulation differences are among the most consistent findings in autism. A meta-analysis of fourteen studies reported large effect sizes for under-responsivity, over-responsivity and sensation seeking, greatest between ages 6 and 9 (Ben-Sasson et al., 2009), confirmed a decade later with 55 additional studies (Ben-Sasson et al., 2019). Youth with autism and sensory over-responsivity show heightened amygdala and sensory-cortex responses to mildly aversive stimuli and fail to habituate (Green et al., 2015). At the circuit level, atypical sensory perception is linked to an altered balance of cortical excitation and inhibition (Robertson & Baron-Cohen, 2017; Marco et al., 2011).

3.8 Sleep and melatonin

Sleep problems affect 50 to 80% of autistic children, against 9 to 50% of typically developing children (Souders et al., 2017). Meta-analysis confirms longer sleep latency, shorter total sleep time and lower efficiency by both parent report and polysomnography (Díaz-Román et al., 2018). Melatonin and its metabolites are significantly lower in autism, and supplementation improves sleep onset and duration with large effect sizes (Rossignol & Frye, 2011; Tordjman et al., 2005). Sleep loss is not a passive comorbidity. In 81 autistic children, night wakings, short duration and parasomnias predicted aggression, irritability, inattention and hyperactivity (Mazurek & Sohl, 2016), and in 2,714 children of the Simons Simplex Collection shorter sleep was associated with greater social impairment and more comorbidities (Veatch et al., 2017).

3.9 Blood-brain barrier and gut permeability

Post-mortem autistic brains show altered blood-brain-barrier gene expression, including increased MMP-9 and disrupted claudins and tight-junction components, and intestinal biopsies show reduced barrier-forming elements (Fiorentino et al., 2016). Abnormal intestinal permeability was found in 36.7% of autistic patients and 21.2% of first-degree relatives against 4.8% of controls (de Magistris et al., 2010).

3.10 Seizure susceptibility

The pooled prevalence of epilepsy in autism is about 12%, and of autism among people with epilepsy about 9%, both far above population baselines (Lukmanji et al., 2019; Tuchman & Rapin, 2002). Epileptiform EEG abnormalities without clinical seizures are more common still. This is the clinical face of the excitation-inhibition imbalance described in Section 3.7.

3.11 Environmental stressors already documented in autism

The stressor model is not new. Higher measured classroom noise correlates with more repetitive behaviours in autistic children, and acoustic modification is reported by teachers and parents to reduce disruptive behaviour (Kanakri, Shepley, Tassinary, et al., 2017; Kanakri, Shepley, Varni, et al., 2017). A small early crossover study found more repetitive behaviours under fluorescent than incandescent lighting (Colman et al., 1976). The same logic underlies the allostatic-load framework: repeated activation of stress mediators in a system with limited reserve produces cumulative wear (McEwen, 1998). Autism is, on the evidence above, a condition of limited reserve across several systems. The question for the rest of this review is whether EMF is one of the loads.

4. Proposed mechanisms of non-thermal EMF interaction

International exposure guidelines are built on established thermal effects: tissue heating from RF absorption, and nerve stimulation from ELF induced currents (ICNIRP, 2020). The stressor argument depends on effects below those thresholds. Four mechanisms dominate the literature. None is accepted by ICNIRP or WHO as established; each has a body of experimental support and a body of null or inconsistent findings.

4.1 Voltage-gated calcium-channel activation

Pall (2013) proposed that EMF acts primarily through activation of voltage-gated calcium channels, with downstream calcium-dependent signalling, nitric oxide and peroxynitrite production, and that calcium-channel blockers abolish EMF effects in a large number of published studies. He extended the argument to neuropsychiatric effects (Pall, 2016) and, most recently, to autism, proposing that elevated perinatal intracellular calcium disrupts six mechanisms of synaptogenesis and that EMF and calcium-elevating chemicals act as joint drivers (Pall, 2024). The classic calcium-efflux experiments of Blackman et al. (1982) and the immune-cell work reviewed by Walleczek (1992) are the historical basis. Panagopoulos et al. (2021) offer a biophysical model in which polarized man-made fields force ion oscillation and gate voltage sensors. The mechanism is contested at sub-thermal intensities on biophysical grounds, and ICNIRP does not accept it. Its relevance here is the overlap with Section 3.4: the same channel family implicated genetically in autism is the one proposed as the EMF sensor.

4.2 Oxidative stress

This is the best-supported mechanism. Yakymenko et al. (2016) reviewed 100 studies of low-intensity RF and found oxidative effects reported in 93. Schuermann and Mevissen (2021) reviewed the 2010 to 2020 animal and cell literature and found that a majority reported increased oxidative stress after RF or ELF exposure, often within regulatory limits, while noting methodological weaknesses in many studies. The Swiss expert group BERENIS reached the same conclusion and added that vulnerable groups, including the very young, the elderly and people with pre-existing conditions such as immune deficiency or diabetes, may be more susceptible (BERENIS, 2021). The WHO-commissioned systematic review by Meyer et al. (2024) confirmed elevated oxidative-stress biomarkers in exposed animals but rated the certainty of evidence as very low to low because of risk of bias, imprecision and heterogeneity. That certainty rating must travel with every citation of this pathway. The Swedish Radiation Safety Authority's Scientific Council, in its twentieth annual report, notes that oxidative-stress findings in animals continue to appear, some below current reference levels, and that effects are reported more often when RF exposure is combined with another stressor than when it is given alone (Strålsäkerhetsmyndigheten, 2026). That last observation is the multi-stressor model in the words of a skeptical regulator.

4.3 Mitochondrial and DNA effects

Primary rat neurons exposed to 1800 MHz at SAR 2 W/kg showed increased 8-OHdG in mitochondrial DNA and reduced mtDNA copy number (Xu et al., 2010). Quail embryos exposed to GSM 900 MHz showed superoxide and nitric oxide overproduction and DNA strand breaks (Burlaka et al., 2013). Santini et al. (2018) review mitochondrial membrane potential and respiratory-chain changes after EMF exposure. Leković (2026) proposes an "electromagnetic pathogenesis" model built on increased electron tunnelling in the mitochondrial electron transport chain; it is a conceptual paper without original cohort data.

4.4 Blood-brain barrier, glial activation and cortical excitability

Salford et al. (2003) reported albumin leakage and dark neurons in rat brain after two hours of GSM exposure; Nittby et al. (2009) found leakage persisting seven days after a single exposure; Tang et al. (2015) linked 28 days of 900 MHz exposure to tight-junction loss, blood-brain-barrier leakage and spatial-memory deficits through an identified molecular pathway; Sırav and Seyhan (2016) found increased Evans-blue leakage in male but not female rats. Replication across laboratories has been mixed. Acute 900 MHz head exposure increased GFAP in several rat brain regions, indicating astroglial activation (Barthélémy et al., 2016), and twelve weeks of 835 MHz at SAR 4 W/kg produced hyperactivity, demyelination and autophagy in mouse cortical neurons (Kim et al., 2017). In humans, 45 minutes of GSM exposure reduced intracortical inhibition and increased facilitation in the exposed hemisphere as measured by transcranial magnetic stimulation (Ferreri et al., 2006), and in patients with focal epilepsy the excitability response differed from that of healthy controls (Tombini et al., 2013). Immune effects more broadly are reviewed by Johansson (2009) and the international perspective on thermal and non-thermal effects by Belpomme et al. (2018).

4.5 The ASD-EMF overlap argument

The overlap between these mechanisms and the autism physiology of Section 3 was first laid out systematically by Herbert and Sage in the BioInitiative Report (Herbert & Sage, 2012) and in a two-part peer-reviewed paper (Herbert & Sage, 2013a, 2013b). Their position was explicit: the parallels do not prove causality, but they justify precaution and biologically based exposure standards. Sage and Burgio (2018) added epigenetic and microRNA mechanisms. A panel of UK academics convened by the Science Media Centre, including Dorothy Bishop of Oxford and Sue Fletcher-Watson of Edinburgh, criticized that paper as non-systematic, selectively cited and closer to polemic than to evidence synthesis (Science Media Centre, 2017). Both readings are correct. The overlap is real and the reviews that document it are not systematic. This review treats the overlap as a hypothesis to be tested system by system against meta-analytic evidence, which is the approach of Section 8.

5. In vitro and organoid evidence

The most consequential recent result is Cakir et al. (2025). Using human cortical organoids derived from embryonic stem cells, together with non-human primate radial glia, the Yale group reported that radiofrequency exposure in the 800 to 2,400 MHz band regulated the differentiation of radial glia progenitors, maintaining stem-cell identity and delaying differentiation. Single-cell transcriptomic and chromatin-accessibility profiling showed that exposure induced expression of autism-associated genes and human endogenous retroviral elements through BET (bromodomain and extraterminal) protein-mediated pathways, and that BET inhibitors rescued the exposure-induced developmental defects. The authors frame the work as mechanistic evidence relevant to neurodevelopment. They do not claim that wireless devices cause autism in humans.

The paper also reports that RF-treated cortical neurons showed a significant increase in dendritic-spine density and in MAP2/PSD95-positive synapses, together with increased spontaneous calcium activity. That finding is in the primary text and can be cited. Two details in the way the study has circulated require care. First, the exposure system was a DSD TECH HC-05 Bluetooth module at 2.4 GHz with a transmission output of 4 dBm (2.4 mW), placed one inch from the organoids; the paper reports the source power and distance, not a specific absorption rate. The statement that exposure was "0.025% of the FCC limit" or "4,000 times below it" is a calculation made by Environmental Health Sciences and repeated by advocacy outlets, not a figure the authors report, and should be attributed accordingly. Second, the study has no mouse or in vivo arm. Its value is as a human-tissue demonstration that a low-power consumer RF source can shift a developmental program toward an autism-associated transcriptional and morphological state. Its limits are that organoids lack vasculature, immune cells and a body, that the exposure system has not been replicated elsewhere and its dosimetry has not been independently characterized, and that a transcriptional and synaptic signature is not a phenotype.

6. Animal studies of prenatal and early-life exposure

Aldad et al. (2012) exposed pregnant mice to an active 800 to 1900 MHz cellular telephone for the duration of gestation. Adult offspring showed hyperactivity, impaired memory and altered glutamatergic transmission in prefrontal cortex. The phenotype resembles attention-deficit hyperactivity disorder rather than autism, and the authors say so. Othman et al. (2017a) exposed pregnant rats to 2.45 GHz Wi-Fi with or without maternal restraint stress and found anxiety, motor and exploratory deficits and brain oxidative stress in offspring; a companion study without restraint found neurodevelopmental delays over the first 17 postnatal days, brain oxidative stress and altered cholinesterase activity, but no behavioural differences at postnatal days 28 and 43 (Othman et al., 2017b). Zhang et al. (2015) found anxiety-like behaviour and impaired spatial memory after prenatal 9.417 GHz exposure. Stasinopoulou et al. (2016) found delayed developmental milestones and reduced pup weight after pre- and postnatal DECT exposure.

None of these radiofrequency studies used the endpoints that define autism-domain behaviour in rodents: the three-chamber social test, ultrasonic vocalizations, marble burying or stereotyped grooming. A search of the 2015 to 2026 literature for prenatal or early-life RF exposure with social or repetitive-behaviour endpoints returned nothing. One extremely low-frequency study did use them. Alsaeed et al. (2014) exposed BALB/c mice to a 1 mT, 50 Hz magnetic field continuously during the last week of gestation and the first postnatal week and reported reduced sociability and reduced preference for social novelty in the exposed males, with no difference in anxiety on the elevated plus maze. The groups were small (8 exposed, 9 controls), the field intensity is well above residential levels, and the finding has not been replicated. The animal literature therefore supports a general claim that prenatal EMF exposure can alter offspring behaviour and brain redox status, and contains a single small ELF study with an autism-domain social endpoint. It does not support a claim that environmental-level exposure produces autism-like behaviour.

7. Human epidemiology

7.1 Prenatal and early-life wireless exposure and child behaviour

The Danish National Birth Cohort is the largest source. Prenatal and postnatal maternal cell-phone use was associated with behavioural problems on the Strengths and Difficulties Questionnaire at age 7 (Divan et al., 2008), and the finding replicated in a separate group of 28,745 children with an adjusted odds ratio of 1.5 (95% CI 1.4 to 1.7) for combined exposure (Divan et al., 2012). The authors state in both papers that the association may be non-causal because of unmeasured confounding by maternal behaviour. A pooled analysis of five birth cohorts totalling 83,884 mother-child pairs found maternal cell-phone use associated with child hyperactivity and inattention, again not judged causal and with a call for adjustment for parenting style and maternal hyperactivity (Birks et al., 2017). The Dutch ABCD cohort found similar associations at age 5 (Guxens et al., 2013). A Spanish cohort found no association with neurodevelopment at 14 months (Vrijheid et al., 2010). No cohort in this literature used a physician-diagnosed autism endpoint.

7.2 Objective dosimetry in infancy

Setia et al. (2025) followed 105 infants in Navi Mumbai with in-home RF measurements using a Narda SRM-3006 analyzer and screened development with the Ages and Stages Questionnaire. Across 261 observations, infants in the high-exposure group were more likely to be classified as needing monitoring or referral for fine motor skills (adjusted OR 2.74, 95% CI 1.10 to 6.78) and problem solving (adjusted OR 3.67, 95% CI 1.41 to 9.55). Autism was not an outcome, the sample is small and the instrument is a screener. The study's value is that it is one of very few to combine objective in-home dosimetry with a developmental outcome, and it should be cited under its authors rather than through the alternative-media coverage that misattributed it.

7.3 Parental occupational exposure

A case-control study of 206 children aged 16 to 36 months evaluated at the Early Intervention Service of Ciudad Real, Spain (70 with ASD, 136 controls) examined parental night work, solvent handling and occupational EMF exposure. The risk of ASD was multiplied by 2.22 (95% CI 1.42 to 3.48) when either parent worked in an exposed occupation, with solvents (OR 2.81) and night work (OR 2.18) carrying the effect; EMF exposure was not separately significant in the reported results (Pino-López & Romero-Ayuso, 2013). This is the study that practitioner blogs describe as "a Spanish case-control study of paternal EMF exposure." It is small, retrospective and exposure was assigned from job titles.

7.4 Speech and neurodevelopmental outcomes

Two small Iranian studies reported associations between maternal cordless-phone use and power-line proximity before and during pregnancy and offspring speech problems (Zarei et al., 2015, 2019). Maternal magnetic-field exposure measured by personal dosimeter in a Kaiser Permanente cohort of 1,482 mother-child pairs was associated with ADHD, not ASD, in the offspring (Li et al., 2020); the article was subsequently retracted and replaced after errors in the exposure analysis were identified, with the replacement reporting weaker and less consistent associations (Li, 2021). It should be cited only with that history.

7.5 Systematic reviews of RF and child neurodevelopment

The WHO-commissioned systematic review and meta-analysis of human observational studies found only a few studies providing very low to low certainty evidence of little to no association between RF-EMF exposure and learning, memory, executive function and attention, with no exposure-response relationship (Benke et al., 2024). A systematic review of experimental and epidemiological studies in children and adolescents rated the body of evidence as low to inadequate (Bodewein et al., 2022). A registered protocol from Universitas Diponegoro for a systematic review of non-ionizing EMF and pregnancy outcomes is in progress (Armalina et al., 2026). A 2026 narrative review claiming that precaution is warranted for mobile-phone use in pregnancy (Chatha et al., 2026) was criticized by the Australian regulator for not reporting the results of its own quality assessment (ARPANSA, 2026).

7.6 What the human literature does and does not show

Taken together, the human evidence supports a modest, replicated, non-causal association between self-reported maternal wireless use and child behavioural problems; one small objectively measured association between home RF levels and infant developmental screening flags; and no study that combines objective dosimetry with a diagnosed autism endpoint. That last gap is the reason the original review's first conclusion stands unchanged: there is no epidemiological evidence that EMF causes autism.

8. Convergence: the same systems, side by side

Table 2 sets the two halves of the argument against each other for each physiological system. The third column records whether any study has examined the intersection directly, meaning an autism model or autistic people exposed to environmental-level EMF. The fourth grades the strength of convergence. The grade reflects the quality of evidence on both sides and the coherence of the proposed pathway, not the size of any single effect.

Table 2. Convergence between autism physiology and reported EMF effects, by system

SYSTEM	ALTERED IN AUTISM	PERTURBED BY EMF	DIRECT INTERSECTION	CONVERGENCE
Oxidative stress	Frustaci et al. (2012): GSH reduced, GSSG elevated across 87 studies; Rossignol & Frye (2014) brain evidence.	Meyer et al. (2024): biomarkers elevated in vivo, certainty very low to low; Schuermann & Mevissen (2021); BERENIS (2021) names pre-existing conditions as susceptibility factors.	None	Moderate. Both halves meta-analytically supported; no study measures oxidative markers in autistic subjects under EMF.
Mitochondria	Rossignol & Frye (2012): 5% mitochondrial disease, ~30% biomarker abnormalities.	Xu et al. (2010): mtDNA oxidation after 1800 MHz in neurons; Santini et al. (2018) review.	None	Moderate to weak. Autism side strong; EMF side in vitro and rodent at SAR near or above limits.
Immune and neuroinflammation	Vargas et al. (2005) microglial activation; Masi et al. (2015), Saghazadeh et al. (2019) cytokine meta-analyses.	Barthélémy et al. (2016) astroglial activation, rat, 900 MHz; Kim et al. (2017) demyelination, mouse, SAR 4 W/kg.	None	Weak to moderate. EMF glial findings at high SAR and not independently replicated.
Calcium and ion channels	Splawski et al. (2004) Timothy syndrome; Krey & Dolmetsch (2007); Schmunk & Gargus (2013).	Blackman et al. (1982), Walleczek (1992) efflux data; Pall (2013) VGCC hypothesis; Panagopoulos et al. (2021) model; not accepted by ICNIRP at sub-thermal levels.	None	Moderate in plausibility, weak in evidence. Genetic autism side strong; EMF mechanism contested.

SYSTEM	ALTERED IN AUTISM	PERTURBED BY EMF	DIRECT INTERSECTION	CONVERGENCE
Autonomic and HRV	Cheng et al. (2020): reduced vagal HRV; Benevides & Lane (2015); Arora et al. (2021).	Misek et al. (2018): parasympathetic increase, opposite direction; Parazzini et al. (2007) null; Havas et al. (2010) small, non-standard outlet.	None	Weak. Human EMF-HRV findings inconsistent in direction.
HPA and cortisol	Taylor & Corbett (2014); Spratt et al. (2012) enhanced reactivity.	Touitou & Selmaoui (2012): no cortisol effect from 50 Hz fields; no robust RF-cortisol study.	None	Weak or none.
Sensory processing and cortical excitability	Ben-Sasson et al. (2009, 2019); Green et al. (2015); Robertson & Baron-Cohen (2017) excitation-inhibition imbalance.	Ferreri et al. (2006): reduced intracortical inhibition after GSM; Tombini et al. (2013): altered excitability in epilepsy; alpha-band EEG effects (Croft et al., 2010; Ghosn et al., 2015; Zhang et al., 2017).	Tombini et al. (2013), epilepsy, nearest analogue	Moderate. The most mechanistically coherent row: a shift toward excitation in a population defined partly by excitation-inhibition imbalance.
Sleep and melatonin	Rossignol & Frye (2011); Díaz-Román et al. (2018); Souders et al. (2017); sleep loss worsens symptoms (Mazurek & Sohl, 2016; Veatch et al., 2017).	Loughran et al. (2005, 2012), Schmid et al. (2012) spindle EEG changes; Lustenberger et al. (2013) reduced slow-wave activity; Danker-Hopfe et al. (2011, 2016) null macrostructure with individual variation; Touitou & Selmaoui (2012) null melatonin.	None in autism	Moderate. Strongest candidate mediating pathway; see Section 9.
BBB and gut barrier	Fiorentino et al. (2016); de Magistris et al. (2010).	Salford et al. (2003), Nittby et al. (2009), Tang et al. (2015), Sırav & Seyhan (2016), rodent leakage.	None	Weak to moderate. Rodent findings reproducible within groups, contested across laboratories.
Seizure threshold	Lukmanji et al. (2019): ~12% epilepsy in autism.	Ferreri et al. (2006); Tombini et al. (2013).	Tombini et al. (2013), epilepsy	Moderate as analogue. RF alters excitability in already hyperexcitable cortex.
Gut microbiome	Extensive autism literature (not reviewed here).	No verified EMF-microbiome study located.	None	None. Omitted until a verified study exists.

8.1 The empty intersection

A systematic search for studies exposing established autism animal models (BTBR, prenatal valproate, Shank3, Cntnap2, Fmr1) to RF or ELF fields, and for provocation, device-reduction or physiological studies in autistic humans under EMF exposure, returned no peer-reviewed results. The closest animal evidence is the small ELF study of Alsaeed et al. (2014), which used wild-type mice and a social-behaviour endpoint rather than an autism model. Parent-report surveys of screen time and symptom severity exist but confound content, behaviour and exposure and cannot serve as EMF evidence. No electrohypersensitivity study has stratified by neurodevelopmental diagnosis or reported autism comorbidity (Belpomme & Irigaray, 2020). The only direct evidence that autistic nervous systems respond to applied electromagnetic fields comes from therapeutic stimulation (Section 10), which demonstrates responsiveness, not harm.

This is the honest limit of the stressor argument. It is a convergence-of-mechanisms case built on two independently strong literatures that have never been tested together. The nearest analogue is the epilepsy work of Tombini et al. (2013), where a population with a known excitability disorder responded differently to RF than controls did. Whether autistic people do the same is unknown because nobody has looked.

9. Sleep as the candidate mediating pathway

Of the rows in Table 2, sleep is the one where the pathway from exposure to symptom load is fully articulated on both sides. On the autism side, sleep disruption is highly prevalent, melatonin is reduced, and both shorter sleep and poorer sleep quality predict aggression, irritability, inattention, hyperactivity and social impairment (Mazurek & Sohl, 2016; Rossignol & Frye, 2011; Veatch et al., 2017). On the EMF side, pre-sleep GSM exposure reproducibly increases EEG spectral power in the sleep-spindle range during NREM sleep (Loughran et al., 2005), the effect depends on pulse modulation rather than carrier frequency (Schmid et al., 2012), pulsed RF during sleep reduces slow-wave activity and impairs overnight motor-memory consolidation (Lustenberger et al., 2013), and the effect is consistent within individuals but highly variable between them (Loughran et al., 2012).

The null findings belong in the same paragraph. Two large double-blind crossover studies found no group-level effect of GSM or UMTS exposure on sleep macrostructure (Danker-Hopfe et al., 2011, 2016), although the second identified a minority of participants with consistent individual-level changes. Controlled human exposure to 50 Hz magnetic fields produced no effect on melatonin or cortisol rhythm (Touitou & Selmaoui, 2012), and the human melatonin literature is largely negative (Lewczuk et al., 2014), with the observational cell-phone finding of Burch et al. (2002) and the review by Halgamuge (2013) as the main dissents. The picture is of a small, real, modulation-dependent effect on sleep EEG that does not degrade sleep in the average healthy adult but shows marked inter-individual susceptibility.

A population with 50 to 80% baseline sleep disturbance, reduced melatonin and a demonstrated link between sleep loss and symptom severity is precisely the population in which a small, susceptibility-dependent sleep effect would be expected to matter. EFEIA's own data point the same way. In the EHS Global Census 2025, sleep disruption was the strongest single predictor of self-reported electrohypersensitivity symptoms, explaining 40.7% of variance, more than reported exposure levels themselves (EFEIA Foundation, 2025). Sleep is therefore the first place a direct test of the stressor hypothesis should look.

10. Therapeutic electromagnetic stimulation in autism

Electromagnetic exposure is not one thing. Frequency, modulation, intensity, timing and the biological window all change the effect, and the clearest demonstration of that is the therapeutic literature. A meta-analysis of 23 studies found that repetitive transcranial magnetic stimulation produced moderate improvements in repetitive behaviours and social functioning in autism, with a high risk of bias (Barahona-Corrêa et al., 2018). An expert roadmap concludes that rTMS can modulate cortical excitability and behaviour in autism but that trials remain small (Oberman et al., 2016). Sokhadze et al. (2014) reported altered event-related potentials and reduced repetitive behaviour after rTMS combined with neurofeedback in 42 children.

At environmental rather than clinical intensities, Pietramala et al. (2024) reported that whole-body extremely low-frequency exposure delivered through a Helmholtz-coil mat produced statistically significant improvements in receptive vocabulary (PPVT-4, $p = 0.002$), expressive vocabulary (EOWPVT-4, $p = 0.041$) and several Achenbach behavioural subscales in 20 autistic children. The study is open-label, has no sham arm, and one co-author was employed by the device manufacturer. It should not be read as evidence of benefit. It should be read, together with the rTMS literature, as evidence that autistic nervous systems are responsive to applied fields, which is the premise the stressor argument requires and the reason a blinded, sham-controlled replication would be informative in either direction.

11. Regulatory and institutional positions

The World Health Organization's International EMF Project reviews evidence on possible health effects across 0 to 300 GHz and does not identify autism as an EMF outcome (World Health Organization, n.d.). ICNIRP's 2020 guidelines are built on established thermal and nerve-stimulation effects, and the Commission's position is that exposures below its limits do not cause substantiated adverse health effects, including neurodevelopmental effects (ICNIRP, 2020; ICNIRP, n.d.). Health Canada's Safety Code 6 (2015) and the Swedish Radiation Safety Authority's Scientific Council, which reports annually, hold the same line. The Council's twentieth report finds no new established causal relationship between EMF exposure and health, while recording that oxidative-stress effects in animals continue to be reported below reference levels and that most included experimental studies used only male animals (Health Canada, 2015; Strålsäkerhetsmyndigheten, 2026). ARPANSA maintains that exposure below its RPS-S1 standard does not affect health and has publicly criticized precautionary reviews for methodological weakness (ARPANSA, 2026).

Two official positions are more precautionary. The French agency ANSES concluded in its 2016 collective expert report on children and radiofrequency that the data did not allow a conclusion for behaviour, development or teratogenic effects, that a possible effect on cognitive function and well-being could not be excluded, and that children's exposure to wireless devices should be limited and regulatory exposure indicators reconsidered (ANSES, 2016). The Swiss BERENIS expert group, in its 2021 special issue on oxidative stress, identified people with pre-existing conditions as potentially more susceptible (BERENIS, 2021). The Parliamentary Assembly of the Council of Europe called in 2011 for precautionary exposure reduction with particular attention to children (Council of Europe, 2011). IARC classifies RF-EMF and ELF magnetic fields as Group 2B (IARC, 2002, 2013).

Two medical-society and working-group documents take the precautionary position explicitly on autism. The EUROPAEM guideline recommends exposure limits far below ICNIRP for prevention, diagnosis and treatment of EMF-related illness and lists autism among the conditions sharing oxidative, mitochondrial and calcium-related features with EMF effects (Belyaev et al., 2016). The BioInitiative Report's Section 20 is the anchor document for the overlap argument (Herbert & Sage, 2012). Neither is endorsed by WHO or ICNIRP.

One legal ruling is relevant. In Environmental Health Trust v. Federal Communications Commission (2021), the United States Court of Appeals for the D.C. Circuit granted the petitions in part and remanded to the Commission to provide a reasoned explanation for its determination that its 1996 guidelines adequately protect against harmful effects of radiofrequency exposure unrelated to cancer. The court faulted the agency's process, not the science. Neither the FCC limits nor the scientific consensus changed as a result.

Table 3 records the institutions, regulators and researchers active on the question worldwide, with the position each holds.

Table 3. Who is talking about this — organizations, researchers, regulators

ORGANIZATION / RESEARCHER	COUNTRY / REGION	POSITION ON EMF ↔ AUTISM
WHO — International EMF Project	International (Geneva)	Neutral. Does not identify autism as an EMF outcome; assessment ongoing across 0–300 GHz.
ICNIRP	International (Germany)	Skeptical / null. Exposures below current guidelines are not accepted as causing substantiated health effects, including neurodevelopmental effects.
ARPANSA	Australia	Skeptical of precautionary reviews; reaffirms RPS-S1 standard as protective.
Science Media Centre	UK	Skeptical. Expert panels (King's College London, Open University, Royal Berkshire NHS) have publicly critiqued EMF-autism reviews as non-systematic.
EUROPAEM (European Academy for Environmental Medicine)	EU (Germany-based)	Supportive / precautionary. Clinical guideline states fetal and early-childhood wireless exposure "may be a risk factor" for hyperactivity, learning and behavioral problems and cites autism parallels.
BioInitiative Working Group (Herbert, Sage, Carpenter, Hardell, Lai, Carlberg, Oberfeld)	International (US-anchored)	Supportive. Section 20 of the 2012 Report documents biological overlap between ASD and EMF/RFR effects, explicitly not claiming causality.
Yale School of Medicine — In-Hyun Park lab; Hugh Taylor lab	USA	Mechanistic evidence (organoids, mice) consistent with RF-related neurodevelopmental effects; authors are cautious and do not claim wireless causes autism in humans.
Environmental Health Trust (EHT)	USA	Supportive / precautionary. Curates evidence and hosts webinars (e.g. with Cindy Sage and Martha Herbert) framing EMF as a modifiable risk factor.
Environmental Health Sciences / EHN.org	USA	Supportive / precautionary science-news outlet.
Children's Health Defense (US and Canada chapters)	USA / Canada	Strongly supportive; policy-campaign framing.
ACN Latitudes	USA	Supportive / patient-education framing.
ISDE (International Society of Doctors for Environment) Scientific Office	Europe (Italy)	Supportive. Burgio co-authored the 2018 Child Development paper with Sage.
Universidad Complutense de Madrid (2026 conference)	Spain	Hosted precautionary experts (Pall, Havas); the university itself does not take a formal position.
Universitas Diponegoro (Armalina et al.)	Indonesia	Neutral. Running a registered systematic review of EMF and pregnancy outcomes (2026).
Organization "Spektar Podgorica" (Leković)	Montenegro	Supportive. Proposed the "Electromagnetic Pathogenesis" (EMP) model in 2025.
Trent University (Magda Havas, emerita)	Canada	Supportive of an EMF health-effects link generally.
Washington State University (Martin Pall, emeritus)	USA	Strongly supportive of a mechanistic EMF → ASD/ADHD hypothesis via VGCC.

ORGANIZATION / RESEARCHER	COUNTRY / REGION	POSITION ON EMF ↔ AUTISM
Harvard Medical School / MGH TRANSCEND (Martha Herbert)	USA	Supportive of pathophysiological plausibility; conservative on causality.

12. Public and advocacy discourse

The reception of Cakir et al. (2025) illustrates how the same result is read by different constituencies. Environmental Health News (2025), ACN Latitudes (2025), Children's Health Defense Canada (2026) and the European Sensitivity Community (2025) presented it as evidence that current exposure limits are outdated, frequently attaching the unverified "0.025% of the FCC limit" figure. The UK Science Media Centre's earlier expert reaction to the Sage and Burgio review (Science Media Centre, 2017) represents the mainstream academic response to overlap-based arguments: non-systematic, indirect, polemical. Consumer explainers such as EMF Radar (2026) and Blossom ABA Therapy (2026) occupy a middle position, acknowledging mechanistic overlap while stating that causation is not established. Spanish coverage of the X Congreso Internacional de Medicina Ambiental at the Universidad Complutense de Madrid, where Martin Pall and Magda Havas presented, shows the topic is being discussed, and contested, inside European academic institutions: El Confidencial (2026) reported the claims descriptively, while the science-communication blog Radiandando (2026) argued that the Faculty of Medicine should not have hosted the event and cited the WHO-commissioned reviews against it. One alternative-media item (NaturalNews, 2025) misattributed the authorship of the Mumbai infant study and extrapolated from developmental delay to autism editorially; it is retained here only to document the error.

13. Evidence gaps and research agenda

The review identifies five gaps in order of the leverage a single well-designed study would have.

1. A blinded, sham-controlled provocation study of sleep EEG, melatonin and heart rate variability in autistic adolescents under pulse-modulated RF exposure, with a typically developing comparison group. The sleep-EEG effect is reproducible in healthy adults and shows inter-individual susceptibility; whether autistic adolescents sit at the susceptible end is unknown and directly testable.
2. An environmental-level RF or ELF exposure study in a validated autism mouse model (BTBR or prenatal valproate) with autism-domain endpoints: three-chamber social test, ultrasonic vocalizations, marble burying, grooming, plus brain oxidative and cytokine markers. No such study exists; the only social-endpoint study (Alsaeed et al., 2014) used wild-type mice, a 1 mT field and 17 animals, and has not been replicated.

3. Independent replication of Cakir et al. (2025) in at least one other laboratory, with different stem-cell lines, a different exposure system, and published dosimetry, so that the intensity relative to regulatory limits can be stated from primary data.
4. A prospective birth cohort with objective personal RF dosimetry and a pre-registered, physician-diagnosed autism endpoint. Every existing cohort relies on maternal self-report and behavioural questionnaires.
5. A randomized, blinded, sham-controlled replication of the Pietramala et al. (2024) ELF pilot without manufacturer involvement, to establish whether the responsiveness signal is real.

Two further items would clarify the institutional picture: completion of the Universitas Diponegoro systematic review of EMF and pregnancy outcomes (Armalina et al., 2026), and a WHO-commissioned systematic review that explicitly addresses neurodevelopmental outcomes, including autism, at sub-thermal exposure levels. The existing WHO series covers cognition (Benke et al., 2024), oxidative stress (Meyer et al., 2024) and pregnancy outcomes (summarized by ARPANSA, 2026) but not neurodevelopmental diagnosis.

14. Conclusion

As of September 2026, artificial electromagnetic fields are not established as a cause of autism, and the human epidemiology needed to test that proposition has not been done. What the evidence does support is narrower and more actionable. **Autism is a condition of reduced physiological reserve across oxidative, mitochondrial, immune, ionic, autonomic, sensory, sleep and barrier systems. EMF exposure perturbs several of those systems in experimental settings, with the best-supported effects on oxidative balance, cortical excitability and sleep EEG, and with marked inter-individual susceptibility in the human data. The two literatures have never been tested together, and until they are, the correct framing is the one this review adopts: EMF is a candidate environmental stressor for autistic people, comparable in kind to noise, light and chemical load, plausible on mechanism, unproven in the population, and modifiable at low cost.**

For a clinical or environmental-medicine audience the practical implication follows from that framing rather than from any causal claim. **Where a stressor is plausible, modifiable and cheap to reduce, and the population is one of reduced reserve, exposure reduction is a reasonable component of environmental management, in the same way that acoustic treatment of a classroom is reasonable without proof that noise causes autism.** The LEDNA principle of designing low near-field exposure into buildings, wiring, room layout and communication systems, and the EFEIA evaluation protocol that records how people experience their electromagnetic environment, are the operational expression of that position. Neither depends on the causal question being settled. Both would benefit from the studies listed in Section 13, and the first of those, a blinded sleep study in autistic adolescents, is within reach of any environmental-medicine department with a sleep laboratory.

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