

Commentary

Vaccines, Revisited: The Case Against ‘Natural Immunity’

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The idea that childhood diseases strengthen the immune system persists because it feels intuitively true, yet biology reveals the opposite: even mild infections trigger a metabolic storm that depletes the body’s core maintenance systems. Although surviving an infection does create immune memory, the same “reference material” can be gained through vaccines, which deliver the necessary information without causing illness or risking the serious complications natural infections can produce. This commentary reframes the debate over “natural immunity” by showing how the erosion of maternal antibody transfer caused by rising cesarean delivery, shortened gestation, and reduced breastfeeding leaves many infants with less inherited protection than evolution once provided. These changes coincide with a developmentally fragile stage when children begin encountering pathogens in daycare, playgroups, and the broader community, creating a period of heightened immunological vulnerability. The risks of infection extend beyond early childhood: several viruses historically encountered in infancy — such as measles, mumps, and rubella — cause far more severe disease when first experienced in adulthood, a consequence rarely acknowledged in discussions of the timing of the MMR vaccine, which protects against all three diseases. The commentary also examines the most persistent vaccine concerns — autism claims, clustered dosing, manufacturing errors, and additives such as preservatives and adjuvants — and clarifies what the evidence shows.

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Introduction

For generations, a quiet assumption has shaped how many people think about childhood illness: that surviving infections is a kind of biological training, a rite of passage that strengthens the immune system through adversity. It’s a worldview rooted in “survival of the fittest,” where each fever is a test that leaves

a child tougher than before. But biology tells a different story: infection triggers a metabolic storm that initially weakens the body, leaving a small population of immune-memory cells in its wake. This commentary focuses on childhood infections, the vaccines that prevent them, and developments in obstetric and infant care that have produced a historically unprecedented reduction in the maternal immune protection young children once received.

For most of human history, infants were protected by a stable evolutionary sequence—spontaneous labor at full term, vaginal birth, immediate maternal contact, and exclusive breastfeeding. But in the last century, a cascade of obstetric interventions reshaped that landscape: cesareans, once rare, became common only after the 1970s, driven in part by the widespread adoption of continuous electronic fetal monitoring, which increased surgical deliveries without improving maternal or neonatal outcomes ^{[1][2]}. Pharmacologic labor induction accelerated after mid-century ^[3]; and baby formula, introduced in the late 19th century and widely adopted after the 1950s ^[4], displaced breastfeeding for millions of babies. These shifts increased infant exposure to pathogens at the very moment when urbanization, crowding, and global travel were accelerating the spread of infectious disease. Vaccines emerged during the same historical period – late in the 19th and early in the 20th centuries – when the traditional biological protections of birth and early feeding were being eroded. Taken together, these changes mean that many babies today receive far less maternal immune protection than full-term pregnancies and extended breastfeeding once reliably provided.

The appeal of “natural immunity” rests on a partial truth: surviving an infection does leave behind an immunological memory, providing the tools necessary to quickly quell the same pathogen when it is next encountered. But the cost of acquiring this information is high – days or weeks of metabolic diversion, tissue damage that must be repaired and the risk of serious complications. Vaccines bypass this series of events while providing the information the immune system needs to recognize and combat pathogens when encountered. The array of vaccines currently available are not just a public health add-on – they are arriving at an unprecedented time in human evolution when the immune protections provided by nature are not fully available to many children.

Mother Nature’s plan, interrupted

For most of human history, infants entered the world with two layers of maternal immune protection — one delivered through the bloodstream during pregnancy, and one delivered via breast milk after birth.

These systems evolved to bridge the gap between birth and the slow maturation of the infant's own immune defenses. Modern birth interventions and feeding practices have disrupted both.

1. The first layer of protection is maternal immunoglobulin G (IgG), transferred across the placenta. IgG transfer begins modestly in mid-pregnancy, rises sharply in the third trimester, and reaches its highest levels in the final days before birth. Infants born prior to full gestation or by pre-labor cesarean receive substantially less maternal IgG and therefore begin life with reduced passive immunity ^[5]. These IgG antibodies, which the mother developed either during infections or due to vaccinations, protect against the major systemic infections that once posed the greatest threat to infants, such as:

- measles
- mumps
- rubella
- varicella
- tetanus
- diphtheria

Pertussis, commonly called “whooping cough,” is a notable exception: maternal pertussis antibodies are not reliably long-lasting and do not protect infants unless the mother was vaccinated during pregnancy.

To provide an understanding of how many births this shortfall affects, more than 10 percent of U.S. infants are born prematurely, missing much of the late-pregnancy transfer. Labor induction now accounts for more than 34 percent of all births, a substantial share of which occur before spontaneous full-term labor would have begun, shortening gestation by varying amounts. And roughly one-third of all births occur by cesarean section ^[6], most before labor begins, so they miss the highest levels of IgG transfer. These infants start life with only a portion of the intended antibody supply. Maternal IgG then wanes steadily after birth and is largely gone by 6-12 months ^[5], just as infants begin encountering pathogens in daycare, playgroups, and the broader community.

2. The second maternal layer is secretory immunoglobulin A (IgA), delivered through breast milk. Unlike IgG, which circulates in the bloodstream, IgA coats the infant's mucosal surfaces — the respiratory tract, the gastrointestinal tract, and the ear–nose–throat passages. These antibodies do not prevent the major systemic childhood diseases, but they dramatically reduce the everyday infections that destabilize young children:

- respiratory infections
- ear infections
- diarrheal and gastrointestinal infections
- some viral exposures that would otherwise cause prolonged illness

Breast milk also contains smaller amounts of other antibodies and a suite of antimicrobial and immune-modulating factors — including lactoferrin, lysozyme, human milk oligosaccharides, and maternal immune cells — all of which help reduce the everyday infectious burden during early life ^[7]. In natural settings, breastfeeding continues well into the second year of life, providing a long, steady supply of mucosal protection during the period when toddlers are most exposed and their immune systems are still maturing. In modern settings, breastfeeding is often shortened or absent, removing this second protective layer just as the placental IgG disappears. Unlike placental immunities, which provide months of protection, the immune protection provided by breastfeeding ends as soon as it is discontinued ^[4].

The cost of infection

Any infection that triggers an immune response draws on the same three biological pathways the body uses to maintain health: mitochondrial energy production, proteostatic balance, and neuroimmune regulation. When a pathogen forces the body into high-intensity inflammation, a predictable sequence of events engages these pathways in rapid succession, as follows:

- **MITOCHONDRIA ALERT (Minutes)** As soon as a pathogen replicates enough to be detected, mitochondria shift from powering normal physiology to helping coordinate the immune response. The body's early-warning sensors activate within minutes, sending signals that raise body temperature and increase metabolic demand. Mitochondria release distress cues that amplify this response, pulling energy away from physical endurance, cognition, and mood regulation ^[8].
- **PROTEOSTATIC STRESS (Hours)** As viral replication accelerates, the cell's protein-handling systems are overwhelmed by misfolded proteins and debris. The resulting emergency cleanup diverts energy away from normal functions — a maintenance detour that can continue long after the infection has cleared ^[9].
- **NEUROIMMUNE RESPONSE (Hours to days)** As the immune response ramps up, signals from the inflamed tissues reach the brain and trigger the coordinated shift known as sickness behavior:

fatigue, cognitive slowing, reduced motivation, and social withdrawal. These changes aren't psychological or optional — they're a built-in energy-conservation program that helps the body redirect resources toward fighting infection and repairing tissue ^[10].

The only lasting benefit of an infection is the formation of immune memory — a small subset of B and T cells set aside as long-lived memory after the pathogen is cleared ^[11]. However, that benefit arrives only after the body has paid the full biological cost of “natural immunity.”

Vaccine fears

No discussion of vaccine concerns can begin without acknowledging the most persistent one: the belief that vaccination can trigger autism. This fear is rooted in timing, not biology. Large, carefully conducted studies across multiple countries have found no association between vaccination and autism ^{[12][13]}. The underlying causes of autism are still being investigated, but current evidence does not implicate vaccines. A plausible biological explanation for why autism traits often become evident in the second year of life is that this is a uniquely vulnerable developmental window: mitochondria have not yet fully proliferated, synapses remain overabundant, vascular networks are still maturing, and myelination is ongoing — a convergence that makes the brain less resilient to metabolic and inflammatory stress. In earlier human environments, toddlers would still have been nursing during this period, receiving ongoing maternal antibodies that buffered fevers and infections. Modern use of formula or early weaning practices removed that layer of protection at the very moment when children are most sensitive to inflammatory stress. Since this same period is when children receive several routine vaccinations, coincidence has been mistaken for causation. But for every parent who says, “My child was fine until he was vaccinated and became autistic,” countless others could truthfully say, “My children received all their vaccinations and remained healthy for years.” The second story is not told because nothing frightening happened.

Concerns about vaccines often arise because they can trigger fevers and, very rarely, more serious reactions. These responses are real, but they are fundamentally different in scale and consequence from the commonly experienced metabolic storm of a full infection. A vaccine activates the immune system in a controlled, low-load way: enough to build the reference library of immune memory, but not enough to force the body into days or weeks of mitochondrial diversion, proteostatic overload, or inflammatory damage. Natural infection delivers the same information at a vastly higher biological cost. When a fever precedes autistic regression, the timing can reflect an underlying vulnerability that the fever simply

reveals — whether a limited ability to meet sudden metabolic demand (mitochondrial), strain on protein-maintenance systems (proteostatic), or difficulty regulating inflammatory signals (neuroimmune). Any of these vulnerabilities can temporarily power down the complex neural circuits involved in verbal and social engagement, and in some children those circuits do not fully rebound. However, the susceptibility likely existed before the fever; an unvaccinated child could have shown similar regression after a standard illness with high fever.

Serious adverse reactions to vaccination have been documented but are exceedingly rare. Large surveillance systems consistently show that these same reactions occur far less often after vaccination than after the infections vaccines prevent. In other words, the biological cost of vaccination is not zero, but it is dramatically lower than the cost of natural infection [\[14\]\[15\]\[16\]](#).

Another reason parents remain wary is that there were serious mistakes in the early history of vaccine manufacturing — contamination events, live vaccines that were either strong enough to cause disease or too weak to reliably prevent it, and inconsistencies in production that affected safety or effectiveness. These failures reflected early industrial processes due to insufficient purification, limited regulatory oversight, and the primitive technology available at the time [\[15\]](#). Acknowledging these events is important but using them to reject today's vaccines — built with stringent quality control and decades of accumulated safety data — is a costly overreaction.

There are also concerns about the number and clustering of vaccines in early childhood. It is true that the schedule has expanded over the decades, but the biology has not changed: these injections do not infect children — they simply deliver information. Even in the most extreme example, the MMR vaccine — which protects against measles, mumps, and rubella in a two-dose series — does not expose the body to three infections at once; it provides three sets of instructions on how to recognize three pathogens. Mild reactions such as fever, fussiness, or achiness reflect this brief immune activation, not the metabolic storm that real infections would trigger. More noticeable reactions, such as higher fever or swelling of a limb, are uncommon but self-limited. Pediatricians generally recommend treating high fevers for comfort, but even one of the rarest reactions, a febrile seizure, is triggered by the fever itself rather than the vaccine and is not known to cause lasting harm. Calls to “separate” the MMR misunderstand how the vaccine works. The MMR is already a two-dose series and splitting the first dose into three separate visits does not reduce biological burden -- it only increases the risk of incomplete vaccination, leaving children unnecessarily exposed to infection [\[13\]\[14\]\[15\]\[16\]](#).

Some parents also worry about the preservatives or adjuvants contained in some vaccines, but these concerns stem from misunderstandings about what these components do and where they are used. Thimerosal was removed from childhood vaccines more than twenty years ago, despite extensive research that found no evidence of harm beyond minor local reactions. Adjuvants, such as aluminum salts, are added to some vaccines to help the immune system notice the antigen, allowing for lower doses and fewer injections. They have been used safely for decades ^[17]. These components have proved to be reliable and effective, not threats — and they are largely irrelevant to modern platforms like messenger RNA (mRNA) vaccines, which contain no preservatives, no adjuvants, and no viral material at all.

RNA vaccines – an elegant adaptation of nature’s own machinery

Where there is fear regarding mRNA vaccine technology, there should be wonder. mRNA technology is not a manipulation of genes; it is a way to co-opt nature’s own communication system. RNA is the molecule that cells already use to pass along temporary instructions — messages that disappear once they show immune cells how to identify a pathogen. It provides the immune system with important blueprint details. Nothing is altered, nothing is integrated, and nothing persists ^[18] — so there is no need to “shoot the messenger.” But mRNA vaccines are just one example within a broader array of established and emerging vaccines. No single approach is universally optimal, because each pathogen presents its own biological challenges — how much antigen is needed to generate protection, what kind of immune response is required, and how long that protection must last. Some pathogens are best countered at the body’s surfaces, which is why mucosal vaccines — such as those used against cholera and typhoid in low-resource settings — provide short-lived protection at the point of entry, rather than long-term immune memory. Different pathogens require different tools. When more than one vaccination is required, it usually reflects either that the pathogen does not generate durable immune memory, as with tetanus, or that it mutates so rapidly that vaccines must be updated, as with influenza.

When childhood diseases strike adults

Several infections that are usually mild in young children can become far more serious when someone first encounters them as an adult. Hepatitis A, which may cause few or no symptoms in young children, is far more likely to cause prolonged illness in adults. They are much more likely to develop jaundice, weeks of fatigue, and complications that sometimes require hospitalization ^[19]. Varicella, known popularly as “chickenpox,” is usually mild in children, who typically experience an itchy rash. But it becomes far more

dangerous when first encountered in adulthood, when the risks of pneumonia and other severe complications — including the rare possibility of fatal outcomes — are substantially higher ^[20].

When the MMR vaccination is given to children, it protects them against the following potential adult scenarios:

- **Measles**, already a serious illness in childhood, becomes even more dangerous when first encountered as an adult. Adults experience higher rates of pneumonia, hospitalization, and brain inflammation ^[21]. Multiple studies have shown that measles can erase 20-70 percent of a person's existing immune memory cells, leaving them vulnerable to other infections for months or years afterward ^[22].
- **Mumps**, which typically causes swollen cheeks in children, can trigger pancreatitis or meningitis in adults. In men, in about 20-30 percent of cases, mumps causes orchitis -- a swelling of the testicles. Among those who develop orchitis, 30-50 percent are left with permanent testicular atrophy, and a smaller proportion go on to have impaired fertility ^[23].
- **Rubella**, an infection so mild it can be virtually asymptomatic, poses a particular danger to the fetuses of women who may not even realize they are pregnant. Infection during the first 12 weeks of gestation can cause congenital rubella syndrome, which can result in cataracts, congenital heart disease, hearing loss, bone abnormalities, and developmental delay, among other consequences ^[24]. During the 1960s epidemic, 8-13 percent of children with congenital rubella syndrome later developed autism ^[25]. Preventing rubella infection before pregnancy is one of the central public-health achievements of the MMR vaccine.

Taken across the lifespan, the MMR is not just a childhood vaccine — it is an essential tool that prevents severe adult disease, infertility, and devastating pregnancy complications, providing protection that endures long after childhood has passed. But the risks of natural infection are not limited to acute illness or to the age at which it occurs. Some pathogens leave behind long-term liabilities that surface years or decades later.

When 'recovery' is not the end of the story

Even when an infection is over, it can cause serious health issues later in life. For instance, after an initial chicken pox infection is resolved, the virus that causes it remains dormant within sensory nerve cells clustered along the spinal cord. If the immune system starts to wane, as it often does with age, the virus can reactivate, causing shingles or chronic nerve pain that can last months or years ^[26].

Polio leaves a different kind of long-term footprint. During the initial infection, the virus destroys a portion of the motor neurons that control movement. Recovery is possible because the surviving neurons sprout new branches to take over the lost connections, but this compensation comes at a cost: those overextended neurons spend decades working beyond their intended capacity. In mid- to late-life, they can begin to fail, leading to new weakness, fatigue, and muscle loss — a condition known as post-polio syndrome. It is not reinfection or viral reactivation, but the delayed collapse of a system that has been running on borrowed capacity since childhood ^[27].

Measles carries its own long-term danger: a rare but almost always fatal brain disease called subacute sclerosing panencephalitis (SSPE), which can emerge years after the initial infection. Children who contract measles in early childhood are at highest risk, and vaccination eliminates this threat entirely ^[16].

Discussion

The idea that childhood illness strengthens the immune system has endured because it feels intuitively true. Illness is visible; recovery is visible; and the child who emerges afterward appears tougher for having endured it. But childhood diseases do not just train the immune system — they strain resources by way of mitochondrial diversion, proteostatic overload, and neuroimmune inflammation that accompany infection. These metabolic events force the body into defensive overdrive, diverting resources intended to support normal growth and maintenance. What looks like “training” from the outside is, internally, a temporary depletion of resources required for health.

Modern birth practices amplify this vulnerability by disrupting the evolutionary sequence that once buffered infants and toddlers. A substantial share of babies are now born preterm, induced before spontaneous labor, or delivered by cesarean prior to labor. Although these categories overlap, together they show that many infants are born either early or without labor — both of which reduce the amount of maternal antibody transfer that nature intended.

And while baby formulas provide robust nutritional support, they cannot replace the extended period of passive immune protection that once shielded toddlers as they encountered a widening range of pathogens. Together, these departures from ancestral conditions mean that many children now enter early childhood with a smaller amount of inherited protection than has ever been experienced in human history.

These shifts are structural, not personal. No mother chooses a cesarean lightly, and many have no choice at all. While some inductions are elective, many are medically indicated. And most mothers who do not breastfeed, or who do so only briefly, face practical, economic, medical, or logistical barriers that shape those decisions. The point is not to blame parents but to recognize that the biological landscape has changed.

Even children born full term and breastfed for extended periods benefit from vaccination, because neither placental nor breast milk immunities create durable immune memory. Vaccines do. They supply the child with a permanent reference library for recognizing and neutralizing pathogens long after maternal antibodies have waned – a reference library that will serve them throughout their lives. Natural infection also generates durable immune memory, but only after the body has paid the full biological cost of illness. In earlier human environments, when vaccines were not an option, natural labor, extended breastfeeding, and limited pathogen exposure might have carried a child safely through the vulnerable early years. In the modern world — with higher population density, global travel, and rapid pathogen spread — durable immunity is essential. Further, natural exposure leaves the development of immune memory to chance, whereas a vaccine schedule ensures that children acquire protection reliably and before they encounter the pathogen.

People who refuse vaccines because they don't want anything "foreign" in their bodies are overlooking something essential: the real foreign threat is the pathogen. A vaccine is more like emergency preparedness training — a controlled, harmless rehearsal that prepares the immune system for a real event. Rejecting it because it is "foreign" is like refusing to leave the building during a fire drill because there is no actual fire.

Vaccine fears persist not because the biology is unclear, but because the psychology is powerful. Autism traits often emerge in the same developmental window when children receive multiple vaccines, and coincidence can be mistaken for causation. Fevers after vaccination are obvious, while the infections that never occur offer, quite literally, nothing to see. Early manufacturing errors from a different technological era still echo in public memory. And preservatives or adjuvants — tools for safety and effectiveness — have been misunderstood as threats. These fears are understandable, but they are not aligned with the biology of how vaccines work or the decades of data supporting their safety.

Conclusion

Whereas the fitness value of natural immunity is a myth, characterizing vaccines as “heroic” is also wrong, because it suggests great effort when it is simply an elegant way to sidestep the full cost of infection. The different types of vaccines are practical tools for closing the evolutionary gap created by modern birth interventions, shortened breastfeeding, and the increased pathogen exposure of contemporary life. They provide durable immune memory that maternal antibodies cannot supply. Natural exposure leaves immune protection to chance — dependent on when and whether an individual happens to encounter a pathogen — while a vaccine schedule ensures that all major threats are covered deliberately and safely, without the risk of infection. In a world where infants begin life with less inherited protection than evolution once provided, vaccines not only restore but expand the margin of safety that earlier environments made possible.

About the Author

Caroline C. Rodgers is an independent science researcher whose peer-reviewed work examines obstetrical interventions -- including preterm induction, prenatal ultrasound, electronic fetal monitoring, and kangaroo mother care -- and their biological implications. Her broader theoretical work extends to neurodevelopment, including the potential etiology of autism, as well as neurodegeneration.

Statements and Declarations

Funding

The author received no external funding for this work.

Conflicts of Interest

The author declares no conflicts of interest.

Author Contribution

The author is solely responsible for the conception, development, and writing of this manuscript.

Use of Generative AI

Bing Copilot was used as an editorial tool during manuscript preparation to check factual claims, logical sequence, refine phrasing, and locate supporting citations.

Acknowledgements

My thanks to all six reviewers of V1 for their careful and constructive comments, which improved the accuracy and clarity of this revision.

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Declarations

Funding: No specific funding was received for this work.

Potential competing interests: No potential competing interests to declare.