

Interactive Risk: Whole-Cell Pertussis as a Co-Factor in a Toxic Web

Brazil's mass vaccination programs didn't occur in sterile clinical settings. They were deployed in the favelas of the Northeast, regions suffering from chronic malnutrition, infectious disease burden, and environmental contamination. These areas were also subject to emergency countermeasures like pyriproxyfen treatment of drinking water and multiple concurrent vaccinations. Yet, no official risk models ever evaluated the combinatorial effects of these exposures.

wP + Aluminum + Malnutrition: Whole-cell pertussis vaccines in Brazil contained 1.25 mg of aluminum hydroxide. Malnourished mothers—common in slums—have impaired detoxification pathways. Combined, this creates a sustained neuroinflammatory state in the fetus, particularly dangerous during early brain development.

wP + Mercury (Thimerosal): Each dose included 0.2 mg of thimerosal. Ethylmercury is a mitochondrial toxin. Coupled with the cytokine storm induced by wP, fetal mitochondrial injury and apoptosis become likely. This combination was never evaluated in gestational safety studies.

wP + Pyriproxyfen: The larvicide was added to drinking water. Vaccine-induced inflammation may compromise placental integrity, increasing fetal exposure to small molecules like pyriproxyfen and amplifying any teratogenic effects.

wP + Zika Virus: Even if Zika has limited standalone teratogenic potential, it may act as a co-factor. Immune activation from wP may heighten Zika's neuroinflammatory impact on the fetal brain.

wP + Concurrent Vaccines: Brazil's maternal immunization schedule often included multiple simultaneous vaccines. This immune burden could exceed tolerable limits during critical gestational windows.

wP + Background Infection Burden: Superimposing vaccine-induced inflammation on a high-infection background may tip immune balance into crisis, with devastating fetal outcomes.

Not one of these interaction pathways was modeled, measured, or ruled out by the WHO or CDC. Their approach was linear, binary, and dangerously simplistic. The phrase "no evidence" was weaponized against every potential co-factor, even when the interacting variables were known teratogens, inflammagens, or immune disruptors.

In February 2016, I submitted a manuscript to *PLOS ONE* documenting the timeline: the rising microcephaly cases predating Zika, the introduction of wP vaccine campaigns, the environmental co-exposures, and the suspiciously timed collapse in microcephaly cases following the cessation of those campaigns. The manuscript was rejected without scientific rebuttal. The editorial team provided vague procedural objections. Around the same time, Dr. Peter Hotez remarked via peer review of our article, submitted to PLOS One article that such a paper would "not be helpful." Helpful to whom? The women who lost their babies? The families who were told a virus did it, when no such evidence held up under scrutiny? To science?

Zika virus has existed for decades without ever causing birth defects. In Colombia, Zika spread widely in 2016. Microcephaly did not. One of my colleagues who was instrumental in keeping people reasonable about Ebola – Dr. Gavin MacGregor-Skinner- traveled to Colombia during the height of the microcephaly crisis and confirmed – Zika, no microcephaly. I called a doctor in Suriname – Zika, no microcephaly. Decades prior – Nigeria, Java, French Polynesia, similar story: many infections, no microcephaly. Only in Brazil—only in the slums, only in the years wP was administered—did this cluster of tragic birth outcomes occur.

This is not coincidence. It is a signal. And IPAK was on it in real time. Peter Hotez and PLOS One want to sanitize any potential link to vaccination; my manuscript is still unpublished, a testimonial to their destructive effect on science.

Here is our best three-factor causal hypothesis that could explain MC in Brazil from 2012-2015.

Agent	Mechanism	Synergistic Interaction
Whole-cell pertussis vaccine (wP)	Pro-inflammatory cytokine storm (IL-6, TNF-α), aluminum & thimerosal load	Upregulates oxidative stress, impairs placental barrier
Glyphosate-based herbicides (GBH)	Elevates RA signaling, suppresses Shh and Obf2, induces apoptosis	RA pathway is already sensitized by systemic inflammation
Zika virus	Infects neural progenitors (but only produces defects in high-inflammatory conditions)	Amplified teratogenicity in immune-primed gestation

Science, if it is to mean anything, must be honest. And honesty requires that we acknowledge uncertainty, revisit our assumptions, and follow the data wherever it leads—even when the destination is uncomfortable. To this day, no major health authority has investigated whether the use of whole-cell pertussis vaccine in pregnant women caused Brazil's microcephaly crisis. But the burden of proof does not lie on the victims. It lies on the institutions that unleashed untested interventions on vulnerable populations and then buried the consequences beneath a viral scapegoat.

Zika virus may have been present, but it was never proven to be the cause of microcephaly. Whole-cell pertussis vaccine, in contrast, was confirmed in use during pregnancy, was known to be inflammatory, was halted just before the crisis resolved, and yet has never been studied in this context. That is not "no evidence." That is willful ignorance.

It is time to ask the questions that were never allowed. Who authorized this? Who funded it? Who ignored the outcomes? And why have the people of Brazil not been told the truth?

The public deserves answers. And science demands accountability. Even for vaccine iatrogenic disease and death.

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Summary of Key Mechanisms Identified in Glyphosate-Induced Teratogenicity

Image is from GMWatch

A landmark study by Paganelli et al. (2010) offers critical mechanistic insight into how glyphosate-based herbicides (GBHs) can produce microcephaly and other neural defects—findings that mirror the patterns observed during the microcephaly surge in Northeast Brazil.

Microcephaly and Cephalic Defects from GBH Exposure

In both *Xenopus laevis* (frog) and chicken embryo models, sublethal exposure to GBH—at concentrations far lower than those used in agricultural settings—resulted in striking developmental abnormalities. These included microcephaly, cyclopia, and severe craniofacial malformations. Gene expression analyses revealed that key developmental markers such as *slug*, *krox-20*, *N-tubulin*, *shh* (Sonic Hedgehog), and *otx2* were all significantly downregulated in exposed embryos. Additionally, the migration of neural crest cells—essential for craniofacial patterning—was visibly delayed or disrupted. These experimental outcomes directly parallel the clinical phenotypes reported in microcephalic infants born during the Zika-associated outbreak in NE Brazil.



Mechanistic Driver: Disruption of Retinoic Acid Signaling

The study identified a central mechanistic driver: disruption of the retinoic acid (RA) signaling pathway. Using a RA-responsive reporter assay (RAREZ), the researchers demonstrated that GBH exposure caused a significant increase in endogenous RA signaling. This hyperactivation of RA is known to induce developmental defects that resemble those seen in retinoic acid embryopathy. Notably, the teratogenic effects induced by GBH were **rescued** when embryos were co-treated with **Ro 41-5253**, a known RA pathway antagonist. This confirms that the observed neural and craniofacial defects are, at least in part, mediated through retinoid dysregulation. The phenotypic outcome mimicked well-documented human syndromes such as holoprosencephaly and otocephaly—both of which involve impaired midline brain development and neural crest function.

Interaction with Other Stressors

The study further raises concerns about synergistic toxicity. It demonstrated that the teratogenicity of glyphosate is potentiated by adjuvants—especially surfactants—commonly included in commercial formulations to improve absorption. Glyphosate alone was capable of impairing mitochondrial function, triggering apoptosis through the activation of caspases 3 and 7, and further suppressing *Shh* signaling, which is critical for neural tube and facial development. When these effects occur in tandem, they likely compound embryonic damage and increase the probability of severe birth defects. In real-world contexts—especially in vulnerable populations exposed simultaneously to vaccines, malnutrition, infections, and chemical agents—these interactions could explain the geographic and demographic clustering of microcephaly.

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