

The Susceptible Host Hypothesis in Vaccine Adverse Reactions

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Abstract

Population-average safety estimates cannot reveal every individual risk pattern. Certain recognized vaccine reactions vary by age, sex, product or medical history. This paper explores whether more refined immunologic or genetic host phenotypes could sometimes explain concentrated adverse-event susceptibility, while avoiding the claim that such phenotypes have already been validated for most disputed outcomes.

The Clinical Question and Its Context

Host response depends on innate and adaptive immune activity, prior antigen exposure, inflammatory state, age and concurrent medical conditions. Some vaccine contraindications already reflect known susceptibility—for example, prior severe allergic reaction to a relevant component. Product-specific myocarditis patterns after mRNA vaccination demonstrate that aggregate rates can conceal meaningful demographic variation. Such observations justify stratification, but they do not prove a general “toxin-sensitive genotype.”

Proposed Biological or Explanatory Model

An interpretable susceptibility model specifies an outcome before searching for biomarkers. It must distinguish a cause of baseline disease from a cause of the vaccine-attributable increment in disease. A genetic association present in patients with spontaneous myocarditis may not be specific to postvaccination myocarditis. Discovery cohorts need independent replication and clinically meaningful discrimination before biomarkers guide individual care.

Mechanism-Specific Interpretation

A plausible susceptibility model starts with an outcome known to vary across subgroups. For myocarditis, age and sex differences after certain vaccine products suggest that risk is not uniform. The next question is whether pre-existing biology adds reliable information beyond those obvious factors. Biomarkers might involve prior immune exposures, inflammatory history or other characteristics, but a biological story is not enough. Rare adverse events mean that even a test with good relative prediction could have poor positive predictive value. A risk marker must be sufficiently precise to help rather than simply label thousands of healthy people vulnerable.

Competing Explanations and Limits of Inference

There are strong reasons for caution: multiple comparisons can produce spurious genetic signals, rare-event studies may be underpowered, and testing can generate needless anxiety. Population findings for one vaccine cannot be generalized to others with different components. The best current evidence does not support a simple inherited susceptibility profile for vaccine-caused autism, and vaccine-autism causation has not been established.

Discriminating Observations and Evidence Thresholds

The hypothesis should be treated as weaker when researchers search retrospectively through hundreds of variables and report only the most attractive signals. Independent validation is nonnegotiable before using biomarkers to advise vaccine decisions. A known serious allergic reaction or a documented product-specific contraindication is qualitatively different from a speculative inherited “toxin-sensitive” phenotype. Precision means preserving that evidence distinction.

Implications for Interpretation and Future Inquiry

The paper should encourage product- and outcome-specific precision pharmacovigilance rather than blanket assertions about “vaccine-damaged children.” A host-risk model is credible only when it improves prediction beyond available demographic and clinical factors and when its use creates measurable benefit.

Selected Evidence and Further Reading

1. Patone et al. Risks of myocarditis, pericarditis, and arrhythmias associated with COVID-19 vaccination or SARS-CoV-2 infection
2. Andersson et al. Aluminum-Adsorbed Vaccines and Chronic Diseases in Childhood: A Nationwide Cohort Study

Source lineage: Vaccine book; autism and host-susceptibility investigations

Editorial note: Conceptual scientific commentary; not an original clinical trial, systematic review, peer-reviewed publication, or medical advice. Hypotheses are not established treatment effects.