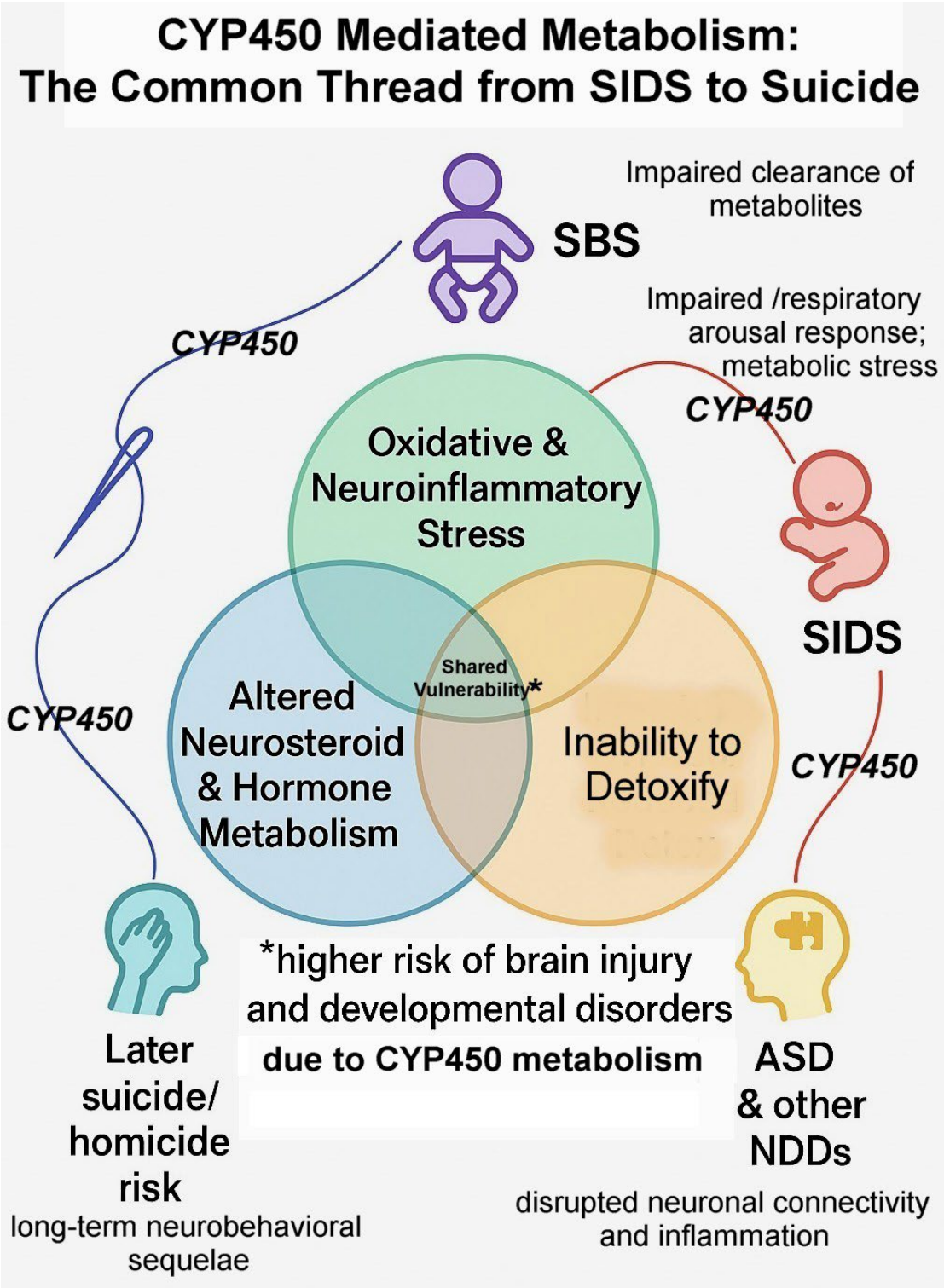
CYP450 Mediated Metabolism: From SIDS to Suicide



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October 24, 2025

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What common thread links Shaken Baby Syndrome, Sudden Infant Death Syndrome, Autism Spectrum Disorders, Neurological Developmental Disorders, and outcomes such as suicide or homicide?

Cytochrome P450 (CYP450) refers to a large family of 57 enzymes found mainly in the liver, but also present in the intestines, lungs, kidneys, and within certain mitochondria. These enzymes help the body process many endogenous substances, such as hormones and fatty acids, as well as exogenous substances such as medicines, environmental toxins, and vaccine ingredients. Their main job is to make these substances easier for the body to neutralize and remove through urine or bile after further processing. Together, CYP450 enzymes handle about 70–80 percent of all prescription drugs and also take part—directly or indirectly—in the metabolism or immune-related effects of vaccine excipients. CYP450 enzymes also participate in the *fetal* metabolism of testosterone and estrogen, which are critical to neurodevelopment.

Due to the immaturity of cytochrome P450 (CYP450) enzymes in infants and young children, capacity to detoxify vaccine excipients and adjuvants such as aluminum, mercury, formaldehyde, polysorbate 80, and ethanol is limited. **Immature CYP450** systems create a shared biological vulnerability—marked by reduced detoxification capacity, altered neuroactive steroid metabolism, and heightened oxidative and inflammatory stress—that exacerbate brain injury and disrupt neurodevelopment, thereby increasing risk for a spectrum of adverse

outcomes, including SBS, SIDS, ASD, other neurodevelopmental disorders, and later violent or self-harm behaviors.

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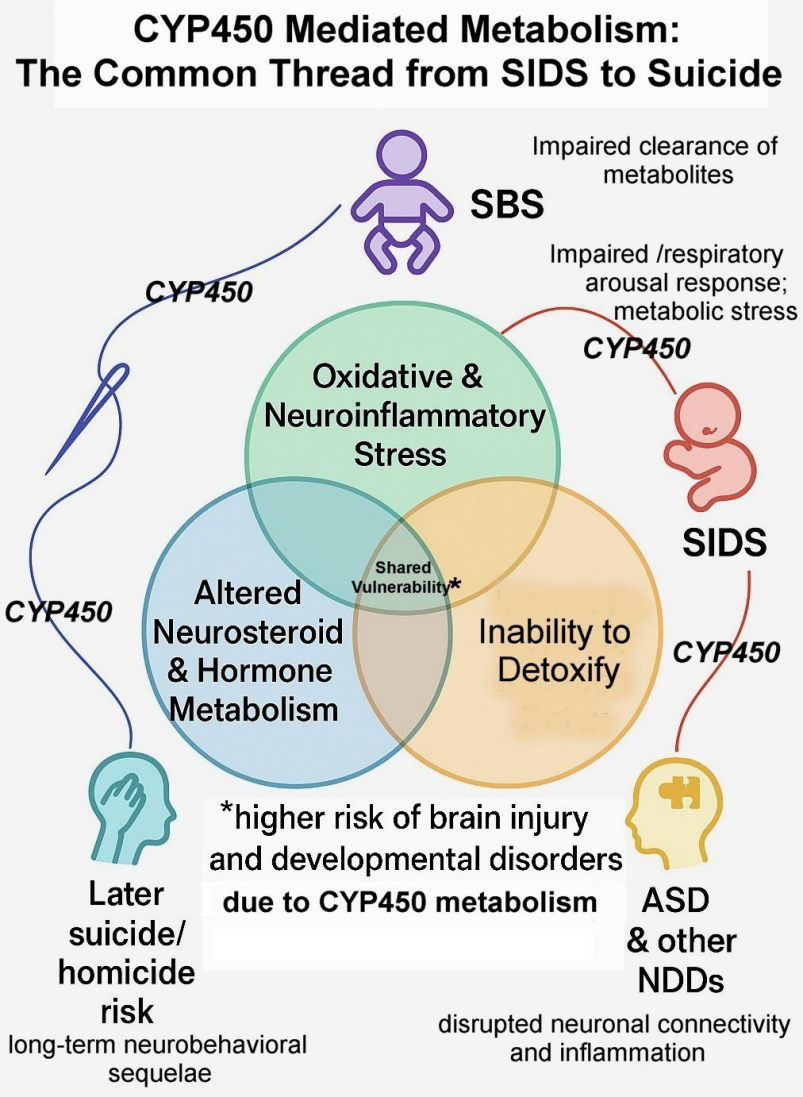
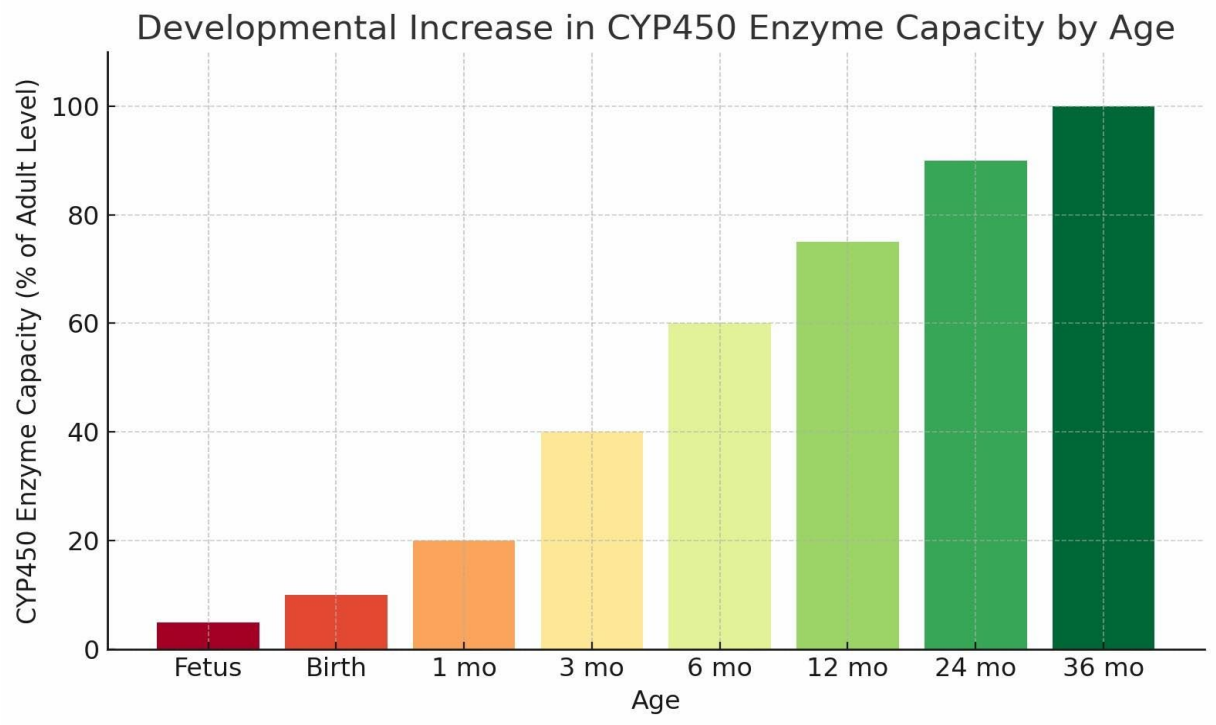


Developmental immaturity of CYP450 enzymes

Before modern medicine, infants were exposed to natural toxins and contaminants—mainly through breast milk and the environment. Without vaccines, infants had no need to metabolize synthetic excipients or aluminum-based adjuvants.

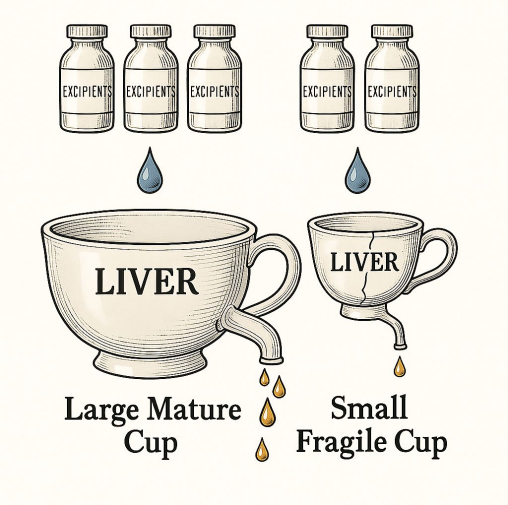


At birth, CYP450 enzyme activity is markedly immature and in newborns it is typically only 30-60% of adult levels. Although enzymes, such as CYP3A4, mature rapidly within the first year, others—including CYP2C9 and CYP2D6—may not reach adult activity until 5 to 10 years of age. The majority of overall hepatic metabolic capacity, however, typically develops by three years. This extended maturation period implies that infants and young children experience reduced clearance of certain vaccine components.

Under such conditions, repeated exposure to and the accumulation of vaccine excipients or adjuvants exceed detoxification capacity, increasing the likelihood of accumulation in sensitive organs such as the brain. These dynamics suggest a biologically plausible framework linking early-life metabolic immaturity to differential neurodevelopmental vulnerability, a relationship that warrants systematic and longitudinal investigation.

The illustration of the adult vs infant cup

The Adult vs. Infant Cup



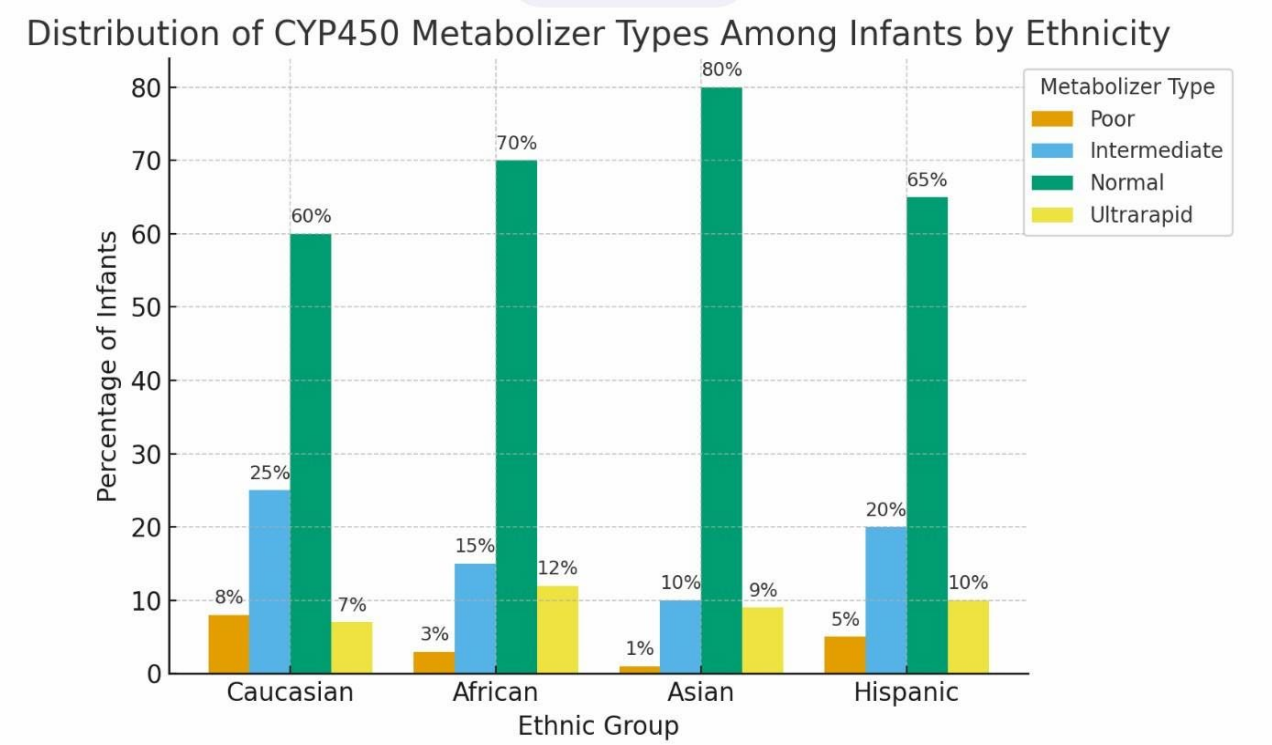
An infant's liver can be compared to a small, fragile cup with a narrow drain—with limited capacity to clear what enters. By 12 months, infants have received 20 vaccine doses—by 36 months, nearly 30. Each dose adds "trace" amounts of toxic excipients such as aluminum salts or polysorbate 80 that accumulate in the infant's cup.

A single dose may not overflow the cup. But multiple inputs, before the drain widens, can lead to a quiet overflow—subtle, unseen, yet capable of disrupting immune signaling, hormonal balance, or neural development.

This is why a dose safe for adults may overwhelm an infant's developing system, and why developmental timing matters profoundly. After decades of vaccinating without fully considering the infant's metabolic maturity, we may have forgotten what true physiological resilience in an unburdened infant once looked like. Recognizing this early window of vulnerability reminds us why understanding infant metabolism matters.

Over twenty (20) vaccines are scheduled in the first year of life—each containing adjuvants, stabilizers, and/or preservatives. These trace amounts of excipients multiply reaching unsafe levels, in all infants and children under three years of age, precisely because their developing systems lack the metabolic and renal capacity to effectively eliminate these compounds.

Genetic Variations in CYP450 Enzymes



In addition to developmental immaturity, **genetic metabolic differences** introduce substantial interindividual variability. Identifying individuals as poor, intermediate, normal, ultra-rapid metabolizers or polymorphisms result in safe and effective drug dosing. This variation, affected by ethnicity, helps explain why some infants and adults experience disproportionate adverse reactions to drugs or vaccine excipients despite standardized dosing and timing.

Pharmacokinetics

Pharmacokinetics is the study of how the body interacts with an administered substance, such as a medication, throughout its entire duration in the body. It is often abbreviated as ADME, which stands for absorption, distribution, metabolism, and excretion.

Pharmacogenetics

Pharmacogenetics is the study of how inherited genetic differences influence an individual's response to medications, particularly through their effects on drug-metabolizing enzyme systems - cytochrome P450 (CYP450) pathways**.**

These CYP450 enzymes are responsible for the biotransformation (activation or breakdown) of most drugs in the body. Variations in the genes that code for these enzymes (e.g., CYP2D6, CYP2C9, CYP2C19, and CYP3A4) can determine whether a person metabolizes a drug rapidly, slowly, or not at all, which in turn affects the drug's safety, efficacy, and risk of adverse reactions. In the case of preterm, neonates, infants, and children under three years old, without exception, their detox activity is immature and severely limited.

Fetal immaturity

Despite a normal maternal detoxification of Tylenol, the **immaturity (or near absence)** of the Cytochrome P450 enzyme system in the embryo and fetus combined with low glutathione reserves may result in an embryo and/or fetus being susceptible to acetaminophen's toxic metabolites. Further research independent of stakeholder influence is warranted. It must be noted, however, that the Standard of Care, recommending post-vaccination Tylenol for fevers and discomfort is an additional burden to the detoxification process.

Shaken Baby Syndrome (SBS)

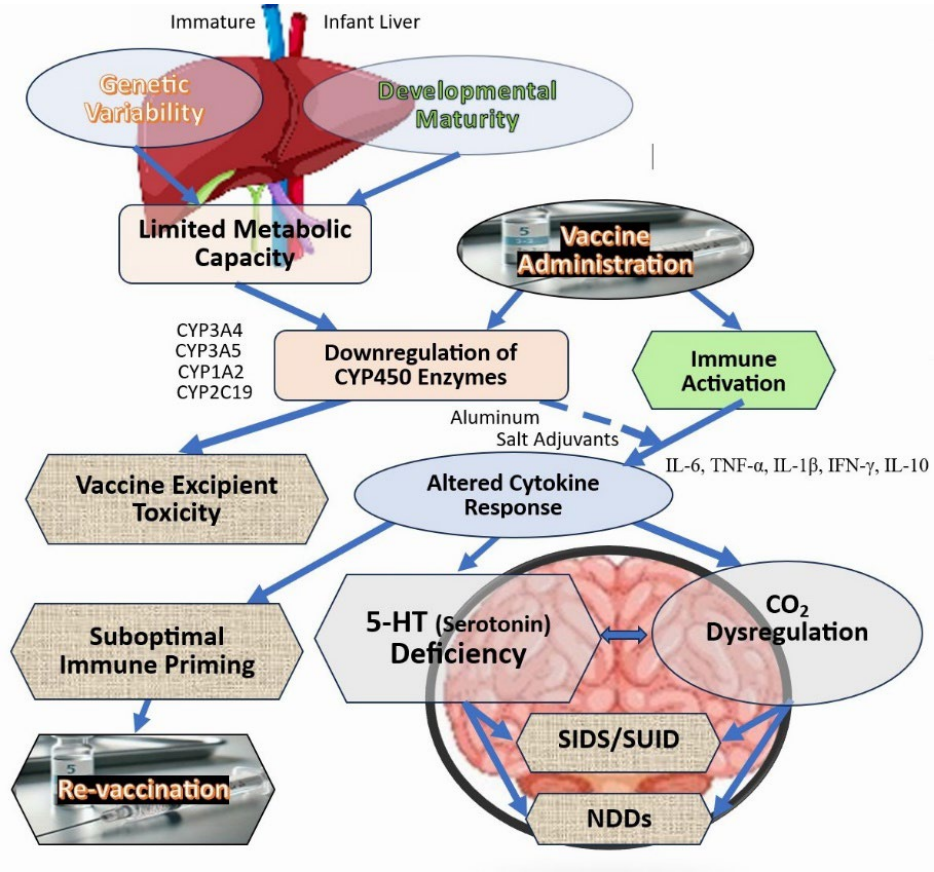
Immature CYP450 enzymes as an underlying potential differential diagnosis for subdural bleeding and fractures in SBS:

Subdural bleeding and unexplained fractures in infants are often attributed to abuse, leading to diagnoses of Shaken Baby Syndrome (SBS). However, biological factors may be the underlying cause for abnormal fragility. One such factor is immaturity of cytochrome P450 (CYP450) enzymes, which help the body process both endogenous compounds (naturally produced substances like hormones and neurotransmitters) and xenobiotics (foreign substances such as medications or environmental chemicals).

Due to immaturity of CYP450 activity, prenatal and postnatal exposure to vaccines and medications—including SSRIs, opioids, antiepileptics, antidepressant, sleeping aids, and allergy drugs—stress immature metabolic systems. In the context of immature CYP450 activity, detoxification is limited, increasing oxidative stress and resulting in fragile capillaries that are susceptible to microbleeds and prone to hemorrhage, as well as impairing bone development (impaired bone mineralization). These vulnerabilities may contribute to subdural bleeding or fractures, even in the absence of trauma.

Assessment of maternal drug exposure, fetal and infant metabolic capacity, and CYP450-related genetic polymorphisms would help distinguish non-traumatic from traumatic etiologies, improving diagnostic accuracy and guiding safer care for vulnerable infants.

Sudden Infant Death Syndrome (SIDS)



Research shows that Immature CYP450 enzymes prolong exposure to toxic vaccine components and, in addition, it alters cytokine response. Cytokine-mediated inflammation impairs serotonin-mediated arousal in the medulla, increasing fatal apnea risk. Multiple independent datasets have confirmed that approximately 70% of SIDS cases involve the medullary serotonin (5-HT) system.

Current medical consensus promotes that SIDS coincidentally occurs during periods where infants are administered vaccines every several months during the first year. However, there are seven independent studies that fail to report a uniform distribution of SIDS cases following vaccine administration, as would be expected if the pattern were coincidental. Approximately 75% of SIDS reports

occur within the first week of vaccination with the remaining 25% occurring between 8 and 60 days.

Neurodevelopmental Disorders (NDDs)

A 2025 retrospective study by Mawson and Jacob published in Science, Public Health Policy and the Law, based on 47,155 Forida Medicaid records from 1999 to 2011, found 9-year-old children had 2.7 times increased odds of ASD and 6.2 times increased odds of tic disorders. Vaccinated preterm children had 3.14 times ASD, 3 times hyperkinetic syndrome, 4.17 times seizures, 9.84 times learning disorders, 7.12 times encephalopathy. While not directly measuring CYP450 activity, these findings highlight concerns about early exposure to vaccination during metabolic vulnerability.

Autism Spectrum Disorder (ASD)

Why 4:1 male predominance in ASD?

In the fetus, CYP3A7 predominates, transitioning postnatally to CYP3A4. Some reports show faster CYP3A4 maturation in females. Male infants may be more vulnerable to ASD because their immature CYP450 enzymes must process higher levels of testosterone, which places greater metabolic demand on an already limited detoxification system. In contrast, estrogen in females can enhance certain CYP450 activities and provide antioxidant protection, helping to reduce oxidative stress in the developing brain. These subtle metabolic and hormonal differences could partly explain the higher prevalence of ASD among boys. No single factor explains the 4:1 male-to-female ratio; it is likely multifactorial (genetics, environment, sex hormones, immune factors).

Mental illness/suicides and homicides

Consider a normally functioning individual who has an acute a mental health event such as depression, anxiety, panic disorder, or post-traumatic stress disorder (PTSD). The pre-prescription pharmacogenetic test would ensure that the individual was treated with a drug that performs efficiently with the metabolic rate of the corresponding CYP450. If the individual is prescribed medication or uses a recreation drug without an individualized phenotype profile and they happen to have a slow rate of metabolism or have no activity at all(polymorphism) of the corresponding CYP450 the drug will not be eliminated but will accumulate in the body to potentially toxic levels. Well established research in the field of pharmacogenetics state that the elevated drug levels cause agitation, confusion, mood instability, suicidal ideations, and violent thoughts leading to homicide and events such as school shootings. Such risks are recognized in black box warnings on many antidepressant and antipsychotic medications. Additionally, corresponding CYP450 pathways are detailed in their inserts. Pharmacogenetic testing for CYP450 variants should guide safe, personalized prescribing, as a standard of care. Today, Pharmacogenetic research has taken virtually every drug and identified the corresponding cyp450 needed to efficiently metabolize it and establish the correct dosage.

In 2011 Lucire et al. [1] published a case series of 85 persons who had developed suicidal and/or homicidal ideation and behaviors only after being prescribed antidepressants augmented by atypical antipsychotics with similar side effects. [2]

14 of the 85 had committed homicide. All had problematic drug metabolizing genes (DMGs). Akathisia (a drug-induced movement disorder marked by severe inner restlessness, agitation and tremors), suicide, and homicide are listed as side effects of drug toxicity and are included in prescriber information. These adverse outcomes occur when the level of the drug or drugs in the blood exceeds the therapeutic window of opportunity and becomes both toxic and ineffective.

Toxicity is dose related but also affected by diminished activity drug metabolizing genes and co-prescribed CYP inhibitors or inducers (i.e., other drugs that a patient is taking at the same time that increase or decrease enzyme activity, increasing the risk of toxicity or causing withdrawal-like effects, respectively).

CASE STUDIES

#1. A young woman had taken Ambien for many months. After making detailed arrangements to see her friends on the next day, she sleepwalked to her death jumping from the Sydney Harbour Bridge. She had four out of six low *metabolizing alleles in three genes.* [1]

#2. An 18-year-old male with no activity of CYP2D6 (polymorphism), was prescribed half the standard dose of fluoxetine, 10 mg/day, for 14 days. **Here is his account in his own words [1]:**

"I felt better, energized, then restless, no appetite, no sexual desire; became aggressive, paranoid, violent, could not sleep, walked around thinking constantly about suicide. Tried to kill myself twice, cutting my wrist, became depressed, and cried.

I ran out of Fluoxetine and felt like a ticking time bomb. I had no thoughts for consequences. Fearless, I challenged strangers to fight, punched out windows, … running over street signs, crashed my truck; stood at the top of a bridge wanting to jump. Wanting to die, I walked a long way to a friend's house where I found a pistol, walked eight miles to my father's house, talked to him for a few minutes {killed him}.

What had I done? I wanted to shoot myself. I confessed immediately."

#3. A young man, after completing Bible school, married his sweetheart and began taking *Coricidin* nightly for allergy relief. *Coricidin* is an over-the-counter medication containing dextromethorphan and chlorpheniramine. Dextromethorphan is primarily metabolized by CYP2D6, and enzyme with marked genetic polymorphism affecting drug metabolism.

After routine use of *Coricidin,* the man experienced a horrible nightmare in which he imagined violently attacking his wife. Upon awakening, he discovered she had been fatally stabbed 125 times. He immediately contacted police. Although *Coricidin* use was discussed during the trial, no pharmacogenetic testing was performed to assess his CYP2D6 phenotype. Consequently, the potential role of aberrant dextromethorphan metabolism in precipitating psychosis or dissociation was not evaluated. The defendant was convicted and remains incarcerated. [3]

[1] Lucire Y, Crotty C. Antidepressant-induced akathisia-related homicides associated with diminishing mutations in metabolizing genes of the CYP450 family. Pharmgenomics Pers Med 2011; 1(4):65-81. https://pmc.ncbi.nlm.nih.gov/articles/PMC3513220/
[2] Clarke C, Evans J, Brogan K. Treatment emergent violence to self and others; a literature review of neuropsychiatric adverse reactions for antidepressant and neuroleptic Psychiatric drugs and general medications, Adv Mind Body Med. 2019; 33(1):4-21. https://pubmed.ncbi.nlm.nih.gov/31370036/
[3] Dannemann E. Documented anecdotal case.

Conclusion

- All preterm infants, neonates, and children possess incompletely developed cytochrome P450 (CYP450) enzyme systems. This developmental immaturity limits their ability to detoxify xenobiotics—including vaccine excipients, environmental chemicals, and prescription drugs. Moreover, CYP450 enzymes influence immune activation indirectly through modulation of steroid hormones and cytokine pathways. Consequently, immature or genetically variable CYP450 expression may alter both the efficacy and safety profile of pediatric vaccines and drugs.
- Before administering vaccines or any pharmacologic agent—prescription, non-prescription, or recreational—each individual should undergo pharmacogenetic testing for key CYP450 variants. Such testing would allow physicians to individualize care based on metabolic capacity rather than rely on a one-size-fits-all approach. The results should become a permanent component of the patient's medical record, ensuring accountability and long-term safety.
- Medical education must evolve accordingly. Training in pharmacogenetics, pharmacokinetics, and orthomolecular nutrition should be standard so that clinicians understand how metabolism, detoxification, and nutrient status interact with immune function. These foundations would help physicians promote natural immunity and reduce the need for aggressive early-life vaccination schedules, postponing such interventions until a child's metabolic and neurological systems reach greater maturity.

- Current pediatric health outcomes demand that we reassess medical consensus. With autism spectrum disorder now affecting roughly one in every thirty-one children, and sudden infant deaths continuing without adequate mechanistic explanation, it is irresponsible to dismiss possible metabolic and iatrogenic contributors. Our conclusion does not seek to appease entrenched financial interests or preserve institutional reputations. Rather, it seeks to restore medical integrity by placing the child—particularly the most vulnerable—at the center of all safety considerations.
- It is essential that medical interventions designed to save lives be fully compatible with the developmental stage of those lives. Just as pharmacogenetics has transformed our understanding of adult drug safety, similar attention to infant metabolic capacity can illuminate and prevent early-life vulnerabilities that have been overlooked for far too long.

Notes:

1. This paper draws substantially on the publication: Goldman GS, Cheng RZ. *The immature infant liver: Cytochrome P450 enzymes and their relevance to vaccine safety and SIDS research*. Int J Med Sci. 2025;22(10):2434–2445. PubMed PMID: 40386062 https://pubmed.ncbi.nlm.nih.gov/40386062/
2. Pharmacogenetic testing can be performed using a simple at-home DNA cheek swab (e.g., GeneSight® or comparable services). The approximate cost is $350, and testing is often covered by Medicaid and other insurance providers.
3. Contacts: Goldman Pearblossominc@aol.com; Dannemann Ncowmail@gmail.com
4. Digital copy: https://vaccineliberationarmy.com/medical-information-and-research-data/goldman-dannemann-cyp450-mediated-metabolism-from-sids-to-suicide/