Sudden infant death:

what if the maternal terrain played a role?

A signal in the data

In the PMCHS survey (N=423, 10+ countries), 9.7% of respondents report a history of sudden infant death syndrome (SIDS) in their family or close circle. For context: the prevalence of SIDS in the general population is approximately 0.05%. This signal, observed in a cohort of people presenting a mast cell hyperreactivity terrain, is not a coincidence — it is consistent with 30 years of published research on the subject.

What science has known since the 1990s

The work of Holgate et al. (1994) and Platt et al. (1994) independently demonstrated significantly elevated post-mortem beta-tryptase levels in SIDS infants versus explained-death controls — with a 20-fold increased risk of elevated tryptase (p=0.0004). This mast cell activation was IgE-independent: it is not a classical allergy, it is MCAS-type activation.

In 2000, Gold et al. took a decisive step: by testing living family members of SIDS-affected families, they showed that 73% of them presented mast cell hyper-releasability — proving that this terrain is heritable and present in the family, not only in the deceased infant.

“ SIDS may not be an unforeseeable tragedy. In a subset of cases, it could be the most dramatic expression of a heritable mast cell terrain — transmitted by the mother, amplifiable by perinatal history. ”

The PMCHS scenario

In the PMCHS framework, the infant is born with a mast cell hyperreactivity terrain inherited predominantly from the maternal line. This terrain, combined with perinatal stressors (difficult delivery, separation, infections), lowers the degranulation threshold further. In some cases, massive systemic mast cell activation — triggered by a minor stimulus (temperature change, mild infection, sleep position) — may provoke acute cardiorespiratory failure.

This mechanism does not explain all SIDS cases. But it potentially explains a familial subgroup — where SIDS recurs in siblings or appears across multiple generations of the same family.

A simple screening proposal

We propose a two-point neonatal urinary histamine screening protocol, non-invasive and low-cost:

- **Day 2–3:** measurement of the post-birth histamine peak (any delivery is a mast cell stress — this first point establishes the level of inherited reactivity)

- **Day 15:** measurement of return to equilibrium — if histamine remains elevated at D15, the system is not self-regulating: this is the signal of a hyperreactive terrain that does not spontaneously return to normal

This protocol measures regulatory dynamics — the capacity to return to equilibrium — not simply baseline levels. Applied as a priority to newborns of mothers with known MCAS/PMCHS terrain, it would identify at-risk dyads before any complication.

What families can do today

- If you have a SIDS history in your family, **mention it systematically** at each pregnancy — it is important clinical information, not a taboo.
- Ask your doctor for a **24-hour urinary histamine test** during pregnancy — the measurement exists, it is simply rarely prescribed.
- **Maternal mast cell stabilisation** during pregnancy and breastfeeding could reduce terrain transmission — a therapeutic avenue to explore formally.
