PMCHS RESEARCH NOTE · DEFINITION & NOMENCLATURE

What is PMCHS, and why does it

need a new name?

The problem with 'idiopathic'

Millions of people suffer from conditions labelled 'without known cause': idiopathic mast cell activation syndrome (MCAS), fibromyalgia, chronic fatigue syndrome, lipedema, PCOS, long COVID. The word 'idiopathic' literally means the cause is unknown. But is that truly the case? Or is siloed medicine looking for the cause in the wrong place — at the level of the affected organ, rather than at the level of the underlying terrain that connects them all?

Mast cells: dysregulated sentinel cells

Mast cells are immune cells present in every tissue of the body — skin, gut, lungs, brain, heart. Their normal role is to trigger a defensive reaction in the face of a threat: inflammation, allergy, stress response. In the PMCHS terrain, their activation threshold is durably lowered — they fire too easily, too often, in response to stimuli that should not activate them.

This dysregulation is not a classical allergy. It does not require IgE antibodies. It does not necessarily produce frank anaphylaxis. It expresses itself silently, chronically, through dozens of symptoms in different organs — which explains why these patients wander from specialist to specialist without ever obtaining an integrated picture.

PMCHS: a terrain, not an isolated disease

Programmed Mast Cell Hyperreactivity Syndrome (PMCHS) — French: SHMP — designates this heritable biological terrain. 'Programmed' refers to two realities: a genetic predisposition (ACKR3 variants, HLA immune axis, mitochondrial genes) and epigenetic amplification — meaning that the environment, notably maternal stress, perinatal history and childhood trauma, can worsen the expression of this terrain from generation to generation.

“ PMCHS is not one more disease. It is the common underlying denominator of many chronic conditions that have been treated separately for decades. ”

Why a new name?

Idiopathic MCAS as currently defined applies to patients without clonal disease (no mastocytosis) and without identifiable IgE-mediated allergy. But this definition by exclusion leaves unanswered an essential question: where does the hyperreactivity come from? The PMCHS framework proposes an answer: it is programmed, heritable, and transmitted predominantly along the maternal line. This is not idiopathic — it is epigenetic.

The 2017 consensus (Akin) explicitly posed as an open research question whether a chronic form of MCAS exists with continuous mediator release, without frank anaphylactic episodes. PMCHS answers this question affirmatively and proposes a mechanism.

What the survey data shows

- 96.0% female — consistent with oestrogenic amplification of the mast cell terrain
- Maternal-to-paternal transmission ratio of 5.4:1 — consistent with epigenetic, predominantly maternal inheritance
- 77.4% adverse childhood events — HPA axis and mast cell reactivity programming via NR3C1
- 94.8% difficult diagnostic trajectory — the human cost of an unrecognised terrain
- 9.7% SIDS history in family or close circle — a signal warranting formal investigation

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