PMCHS PRACTICAL RESOURCE · FOOD & MEDIATORS

Trigger foods by mediator:

leukotrienes, prostaglandins, non-IgE release, serotonin

In the PMCHS terrain, mast cells release several types of mediators depending on the stimulus and the degranulation phenotype. Histamine is the best known — but leukotrienes, prostaglandins, non-IgE release (tryptase) and serotonin produce distinct symptoms that sometimes resist antihistamines alone. Identifying your personal triggers by mediator allows for more targeted intervention.

Leukotrienes (LTC4, LTD4, LTE4)

Involved in: bronchoconstriction (asthma), mucosal inflammation, abdominal pain, persistent skin reactions. Montelukast is the reference treatment when this phenotype dominates.

Category	Foods / mechanism
Fatty meats	Fatty pork, excess red meat, processed meats — rich in arachidonic acid, leukotriene precursor
Omega-6 oils	Sunflower, corn, soy, grapeseed — linoleic acid -> arachidonic acid -> leukotrienes
Ultra-processed foods	Industrial fried foods, crisps, ready meals with hydrogenated omega-6 oils
Alcohol	Promotes leukotriene release in addition to histamine — dual effect
Natural salicylates	Tomatoes, strawberries, raspberries, blueberries, grapes, apricots, cherries, kiwi, pineapple — COX-1 inhibition, diversion to leukotriene pathway
Spices & herbs	Cinnamon, curry, paprika, chilli, oregano — high in salicylates
Dyes E102, E210-213	Tartrazine, benzoates — COX inhibitors with leukotriene diversion

→ To favour: omega-3 oils (rapeseed, linseed, walnut), oily fish (sardines, mackerel, wild salmon). Omega-3 compete with arachidonic acid and reduce leukotriene synthesis.

Prostaglandins (PGD2, PGE2)

Involved in: intense menstrual pain, intestinal cramps and diarrhoea, skin flushing, vascular headaches, asthma aggravation. NSAIDs (ibuprofen, aspirin) block their synthesis but may worsen leukotrienes — use with caution in PMCHS terrain.

Category	Foods / mechanism
Saturated fats	Butter, fatty cheeses, cream, fatty meats — substrates for COX-2 pathway -> PGE2
Arachidonic acid	Liver, kidneys, brain, excess egg yolk — direct PGE2/PGD2 precursor
Alcohol	Stimulates COX-2 and PGE2 synthesis — worsens pain and flushing
Refined sugar	Hyperglycaemia -> NF-kB activation -> COX-2 induction -> PGE2

Category	Foods / mechanism
Omega-6 oils (reminder)	Same mechanism as for leukotrienes — dual impact
Caffeine (high doses)	Can stimulate PGE2 release via intestinal mast cell activation
Glutamate MSG, E621	Non-IgE mast cell activator -> PGD2 release in sensitive individuals

→ To favour: turmeric + black pepper (natural COX-2 inhibitor), ginger, quercetin. Reduce animal fats in favour of omega-3.

Non-IgE release & tryptase

Mast cell activation without allergic antibodies — directly by chemical, osmotic or mechanical stimuli. Released tryptase degrades collagen and nerve fibres. This phenotype does not respond to antihistamines alone — mast cell stabilisers (cromoglycate, quercetin) are necessary.

Category	Foods / mechanism
Direct liberators	Strawberries, tomatoes, spinach, aubergine, chocolate, raw egg white, shellfish — activate mast cells without IgE
Additives	Sulphites E220-228 (wines, dried fruit, tinned food), benzoates, nitrites (processed meats), carrageenan
Alcohol (all forms)	Direct mast cell liberator — independently of tannins or yeasts
Fermented foods	Sauerkraut, kefir, kimchi, kombucha, aged cheeses, vinegar — preformed histamine + non-IgE activation
Tinned fish	Tinned tuna, sardines, mackerel — high bacterial histamine + non-IgE activation
Irritant spices	Black pepper, strong chilli, wasabi, horseradish — TRPV1 activation -> non-IgE degranulation
Alcohol + foods	Alcohol increases intestinal permeability -> increased absorption of liberators -> cumulative effect

→ To favour: sodium cromoglycate (oral mast cell stabiliser), quercetin, luteolin. Long steaming or poaching reduces liberator load compared to high-temperature cooking.

Serotonin — precursors & enterochromaffin cells

95% of the body's serotonin is produced in the gut by enterochromaffin (EC) cells. In the PMCHS terrain, two mechanisms overlap: (1) excess dietary tryptophan may feed the IDO/kynurenine pathway at the expense of serotonin synthesis — contributing to treatment-resistant depression and brain fog; (2) certain foods directly activate EC cells, triggering excessive intestinal serotonin release responsible for diarrhoea, cramps, hypermotility and post-prandial nausea. The serotonin/mast cell relationship is bidirectional: mast cells both release serotonin and are sensitive to it via 5-HT receptors.

Category	Foods / mechanism
High tryptophan	Turkey, chicken, eggs, hard cheeses (parmesan, cheddar), pumpkin seeds, soy, cashews — direct serotonin precursor, possible IDO pathway overload

Category	Foods / mechanism
EC cell activators	Bananas, pineapple, kiwi, tomatoes, walnuts, cashews, dark chocolate — directly stimulate intestinal serotonin release
Fermented high-serotonin	Aged cheeses, sauerkraut, kefir, miso, tempeh — contain preformed serotonin + activate EC cells
Alcohol	Short-term intestinal serotonin release then depletion — activation/deficit cycle
Caffeine	Increases EC serotonin release at high doses — agitation, insomnia, intestinal hypermotility
Refined sugar & fast carbs	Insulin spike -> temporary increase in brain tryptophan -> serotonin surge followed by trough
Glutamate MSG, E621	Directly stimulates intestinal EC cells -> serotonergic release -> digestive symptoms

→ To favour: moderate tryptophan sources paired with complex carbohydrates (brown rice, sweet potato) for steady serotonin synthesis without spikes. Magnesium and zinc — cofactors of the tryptophan -> serotonin pathway — are frequently deficient in the PMCHS terrain.

Note on the IDO/kynurenine axis: in the PMCHS terrain, chronic mast cell inflammation activates the IDO enzyme (indoleamine 2,3-dioxygenase), diverting tryptophan towards kynurenine production (neuroexcitatory) rather than serotonin. This is one of the mechanisms explaining SSRI-resistant depression in this terrain: the problem is not the serotonergic receptor but the availability of the precursor.

■ *This sheet is a guidance tool, not a prescribed diet. Every PMCHS profile is different — a personal food diary remains the best tool for identifying your specific triggers.*

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