The Cumulative Logic of a Sensitised System

How Repeated Exposure Trains the Body – and the Mind – to Detect Threat

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Sensitisation in allergies is often treated in clinical settings as a discrete, static status - a binary marker of presence or absence. In practice, sensitisation functions as a cumulative biological trajectory; it is an expanding catalogue of environmental data that physiologically trains the system to narrow its margin for error. Repeated exposure does not merely increase reactivity; it alters pattern recognition across immune, neurological and cognitive domains. This article examines sensitisation as a progressive adaptation to threat, using allergic disease – particularly histamine intolerance, fungal allergy and cross-reactivity – as the primary lens.

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1. My Experience

I have lived the slow accumulation of sensitisation as well as multiple defining allergic events. What has unfolded over half a century was a progressive specificity of thresholds – foods, environments and exposures that were once tolerable became unreliable, then actively harmful. Reactions were not consistent, which made them easy to dismiss from the outside, but the pattern was consistent from the inside: repeated exposures produced disproportionate consequences, recovery took longer and the cost of lacking fundamental information escalated with each attack. Each episode trained my body further, not only immunologically but systemically, until vigilance became a rational adaptation rather than a choice.

Over time, this training extended into cognition. I became acutely attentive to variables most people never have to consider – batch differences, airborne exposure, cross-reactivity, delayed effects. What might appear as hyper-focus or over-caution is, for me, the residue of consequence-based learning. When combined minor exposures result in days or weeks of impairment, pattern recognition sharpens whether one wants it to or not. This has been isolating, particularly when these observations are treated as speculative rather than evidentiary, but they are grounded in repeated outcomes. My experience is not one of fragility. It is one of adaptation to a system that no longer tolerates error.

My experience is not isolated. Other severely sensitised individuals occupy a similar position. In Australia, allergic disease is among the fastest growing chronic conditions, now affecting roughly 30 percent of the population – around 8.2 million people – up from about 20 per cent in 2007. Food allergies alone impact about one in ten infants and one in twenty children, with hospital admissions and deaths from anaphylaxis rising significantly over recent decades.

This represents a massive cohort whose cumulative trajectories remain largely undocumented - not because the data doesn't exist, but because clinical frameworks are not designed to capture progression patterns, threshold drift, cross-reactivity emergence or the layered environmental and dietary interactions that shape long-term sensitisation.

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2. Sensitisation as a Learning Process

In immunology, sensitisation refers to the process by which the immune system becomes primed to respond to a specific antigen. What is often under-acknowledged is that this priming does not occur on its own. It unfolds over time, across repeated exposures and within a body that is simultaneously managing stress, inflammation, environmental load and prior injury.

Each exposure functions as a data point. The immune system updates its internal model accordingly. With sufficient repetition – particularly when exposures are intense, poorly resolved, or layered – *the system stops treating the antigen as a discrete insult and begins treating the surrounding context as hostile as well*.

This is not dysfunction. It is learning.

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3. Thresholds Drift Before They Collapse

Clinical discourse often relies on thresholds: safe versus unsafe, allergic versus non-allergic, tolerable versus intolerable. In lived experience, thresholds are not static. They drift.

Before overt reactions become unavoidable, there is usually a long intermediate phase characterised by Increasing sensitivity to trace exposures, inconsistent reactions to nominally identical inputs, delayed or prolonged symptom resolution and cognitive and affective changes that precede physical symptoms.

This phase is frequently misinterpreted as anxiety, somatisation, or inconsistency. In reality, it reflects a system whose thresholds are in flux.

When enough variables accumulate – fungal load, histamine burden, enzymatic insufficiency, stress, prior sensitisation – the system reaches a state of allostatic load (the cumulative wear and tear on the body's regulatory systems). At that point, the threshold effectively collapses. "Minimal" exposures are no longer tolerated.

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4. Cross-Reactivity Is Not an Edge Case

Cross-reactivity - the reaction of an antibody (developed against a specific antigen) with a different, but structurally similar, antigen or protein - is often treated as a specialist concern, relevant only in narrow occupational or clinical contexts. This minimisation obscures its practical significance.

In my case, proteins from moulds, insects, storage mites and enzymatic additives are not theoretical contaminants; they are biologically active components of common foods and environments. This is driven by the specificity of Tropomyosin, a "pan-allergen" protein found in the muscle structures of mites, cockroaches, crickets, locusts and shellfish. Because this protein is evolutionarily conserved, its molecular structure remains nearly identical across these

diverse species. My IgE to tropomyosin for some species is high and others low. Clinically, I have only known of my allergy to mites all my life but significantly not to the tropomyosin in mites. Only in the last few months were the others revealed in a comprehensive allergy blood test. I have no certainty as to whether these reactions have been occurring or not, I had been able to eat prawns for my entire life up until 3 years ago. It is likely I was having a minimal reaction that was raising my baseline and leaving me more sensitive to other triggers.

For a sensitised individual, the immune system isn't making a "mistake" or reacting to everything; it is accurately recognising an identical molecular structure in different "packaging". In this state, exposure does not require ingestion or contact. Inhalation alone can be sufficient. The result is a pattern many people report but few frameworks explain: One batch can be tolerated, another is not. One exposure produces mild symptoms, the next produces systemic collapse (eg a high vs low pollen day, a different species of prawn etc), avoidance appears excessive until it proves necessary.

This variability is not psychological noise. It is biological heterogeneity interacting with a sensitised system.

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5. Cognitive Changes Are Downstream Signals

One of the most persistent errors in interpretation is treating cognitive and behavioural changes as primary pathology rather than downstream signals.

Hyper-vigilance, intrusive thoughts, obsessive checking and heightened threat awareness are commonly framed as anxiety disorders. In sensitised individuals, these features often emerge after sustained physiological injury.

When the body has learned – through repeated consequence – that minor exposures can lead to days or weeks of impairment, the brain adapts accordingly. Pattern recognition sharpens. Attention narrows. Risk tolerance drops.

Again, this is not irrationality. It is calibration.

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6. The Cost of Invalidating Pattern Recognition

Individuals managing complex allergic disease often become highly skilled observers. They track inputs, outputs, timing, lag effects and recovery curves with a precision rarely matched in clinical encounters. Misattribution is also possible due to the number of variables and combinations. In my case I have made connections that were not there, suspecting one thing and for another to be revealed - years later - like yeast. A connection I couldn't see until the data existed to reveal it.

When these observations are dismissed – as overthinking, coincidence or bias – the individual loses access to collaborative validation, the system continues to train itself in isolation, and treatment opportunities are missed.

This is how epistemological loneliness develops: not from lack of evidence, but from lack of interlocutors willing to engage with it to assist in creating a comprehensive reactivity profile. This dismissal is particularly unjustified given how allergy diagnosis actually functions.

Clinical diagnosis in allergy routinely relies on patient-reported patterns (anamnesis). Skin prick tests and IgE blood tests show sensitisation, not necessarily clinical symptoms; the definitive diagnosis requires patient history - what was eaten, when symptoms appeared, how long recovery took. This accepted diagnostic method is fundamentally anecdotal: the clinician trusts the patient's observations (including elimination diets, food diaries, and temporal correlations) as diagnostic and valid evidence.

What’s good for the goose is good for the gander. The observations documented here follow identical diagnostic methodology. The distinction lies not in evidential validity but in temporal scope and the complexity of variables tracked.

Conclusion

Framing sensitisation in clinical settings as a discrete, static status remains incomplete.

In practice, sensitisation functions as a cumulative biological trajectory; it is an expanding catalogue of environmental data that physiologically trains the system to narrow its margin for error. It is not confined to the immune system alone or specific body parts. Lifelong sensitisation – occurring through either repeated low-level contact or acute sensitising events – trains the body, and the brain follows.

Integrating these signals requires a shift towards personalised environmental management and biological interventions that at the very least acknowledge the significance of an individual’s cumulative trajectory over the broad categorisation of triggers, rather than just the class of the allergens in isolation.

Understanding this does not require abandoning scientific rigour. It requires expanding the frame to include time, accumulation and lived pattern recognition as fundamental.

A comparable shift is already visible in Australian occupational health regulation (discussed in an earlier post), where flour and grain dust – long minimised as “nuisance dust” – are now formally classified as respiratory sensitisers (RSEN), with dramatically reduced enforceable Workplace Exposure Limits coming into effect nationally. This reclassification did not arise from new biology, but from the slow accumulation of evidence and case numbers that made continued dismissal untenable.

What remains unresolved is that this recognition is currently confined to workplace exposure, while the same biological agents persist largely unacknowledged in consumer food systems and clinical allergy frameworks. Ignoring those signals has consequences – not just for individuals already affected, but for the many who will follow the same trajectory until the numbers can no longer be dismissed as individual aberrations.