A Systems-Biology Model of Diet-Induced, Histamine-Mediated, Site-Specific Acne Recurrence

The "Hamer Loop Hypothesis"
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Category: Hypothesis / Theoretical Note
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Section 1

Preamble

I am not a medical doctor or scientist — I am a 71-year-old retired nurse with 45 years of experience across acute care environments, from bedside nursing to leadership and hospital supervision. My career spanned institutions across Canada and the United States, including Vancouver General Hospital, the Texas Heart Institute, Ben Taub General Hospital, Memorial Hermann, and multiple regional medical centres in British Columbia, Alberta, Oregon, and Washington.

Throughout my career I cultivated a deep commitment to clinical observation and connecting cause and effect. A nurse's superpower is assessment — the ability to notice patterns that others overlook. This paper is the product of that skill, applied over 23 years to a condition that has affected me personally and that I believe carries profound implications for human health far beyond dermatology.

Section 2

Abstract

This paper proposes an integrated model of acne recurrence — the Hamer Loop — that incorporates established pilosebaceous biology with systemic immune activation, histamine signaling, and site-specific immune memory. The model identifies Neu5Gc (N-Glycolylneuraminic acid), a non-human dietary sialic acid, as the primary upstream trigger, and proposes that ammonium compounds in processed foods dramatically amplify Neu5Gc bioavailability and immune reactivity. A complete, observable, timed clinical sequence is documented: from immediate rhinorrhea through abdominal bloating, to skin lesion formation and nocturnal amplification. Multi-system inflammatory expression — including nasal mucosa, gut mucosa, and skin — is documented across a range of dietary triggers including Neu5Gc sources, wheat/gluten, sulphites, and alcohol derivatives. The model further proposes that acne represents a visible warning signal for a systemic inflammatory process affecting all 8.3 billion humans — the majority silently and without warning.

Section 3

Background & Personal Origin

As a lifelong acne sufferer, I had exhausted every known medical treatment — including three rounds of Accutane, tetracycline, nitrogen washes, and numerous trial therapies. Acne was simply my normal.

That changed in my late 40s.

While following a modified Atkins diet — raw spinach, strawberries, tuna in water, hard-boiled eggs, no dressings — I lost approximately 35 pounds over three months. One morning I noticed something I had never experienced: a completely clear, non-oily complexion. Pristine skin, for the first time in my life.

Celebrating this, I ate a Dr. Oetker strawberry mousse pudding. Within two hours, small whiteheads appeared, along with early deep lesions I recognized as the beginning of larger aggressive breakouts. I examined the ingredients: milk, gelatin, and several chemicals I could not immediately identify. In that moment a question crystallized: *Was this an immune response?*

From that day forward, through 23 years of disciplined trial, error, and observation, I developed a thorough understanding of what triggers acne — and began to understand the broader systemic implications of chronic exposure to those triggers. The pattern was consistent, reproducible, and — once understood — completely predictable.

Section 4

Scientific Foundation: Neu5Gc and the Varki Research

The scientific basis for this hypothesis is not mine alone. The work of Dr. Ajit Varki and Dr. Nissi Varki at the University of California, San Diego — among the world's foremost researchers in glycobiology — provides a compelling mechanistic framework that directly supports these observations.

Their published research identifies N-Glycolylneuraminic acid (Neu5Gc), a non-human sialic acid found predominantly in red meat and dairy products, as a dietary molecule that triggers a chronic immune-inflammatory response in all humans. Because humans lost the ability to produce Neu5Gc approximately three million years ago, the human immune system recognizes it as a foreign xeno-antigen and mounts an antibody response. This process — termed **Xenosialitis** by the Varki group — occurs in every person on the planet, without exception.

Dietary Neu5Gc is incorporated into human epithelial and sebaceous cell surfaces, creating xeno-antigen targets. The resulting antibody-antigen reaction generates chronic low-grade inflammation — the same mechanism the Varki team has linked to cardiovascular disease, cancer progression, and type 2 diabetes. I have been in active correspondence with Drs. Ajit and Nissi Varki and have shared my full findings with their team for review.

The Critical Public Health Implication: Approximately 1.6 billion people manifest the Neu5Gc immune response visibly — as acne. The remaining 6.7 billion show no external sign. They are not immune. The inflammation is happening silently — and may be quietly driving cardiovascular disease, cancer, and type 2 diabetes. Acne sufferers may be the fortunate ones. Their bodies are sounding an alarm. Everyone else has no warning whatsoever.

Section 5

The Complete Observable Clinical Timeline

One of the most significant contributions of this research is the documentation of a complete, timed, multi-stage warning sequence following trigger ingestion. This sequence is reproducible, consistent, and observable without any diagnostic equipment — making it immediately verifiable in a clinical setting:

STAGE 1 Minutes	STAGE 2 ~20 Minutes	STAGE 3 2-4 Hours	STAGE 4 Overnight
Watery rhinorrhea — nose runs like a faucet. Uncontrollable nasal drip. First visible alarm. Identifies: Neu5Gc OR Wheat/Gluten trigger.	Abdominal bloating. Occurs with BOTH dairy/beef AND wheat/gluten. Commonly dismissed as "holiday meal bloating."	Skin lesions begin forming at primed sites. Same locations. Same morphology. Every time.	Nocturnal amplification. Fluid shifts to head/neck during sleep. Morning lesions more pronounced. Conges-tion on waking.

Clinical Significance: This four-stage timeline — rhinorrhea within minutes, bloating within 20, lesions within 2-4 hours, nocturnal amplification overnight — represents a complete, observable, verifiable sequence that no existing acne model describes or explains. Each stage is a direct expression of the same underlying Neu5Gc immune mechanism at different anatomical sites and timescales.

Section 6

Two Distinct Trigger Pathways: Neu5Gc and Wheat/Gluten

A critical refinement of this model is the identification of two distinct but overlapping dietary trigger pathways, both of which produce the same early warning sequence (rhinorrhea, bloating, lesions)

through related but potentially distinct mechanisms:

Trigger Pathway	Primary Sources	Early Signal	Bloating	Skin Response
Neu5Gc Pathway	Red meat, dairy, gelatin, cassel/natural flavour, cassel colour, sulphites	Immediate watery rhinorrhea	Yes — within 20 minutes	Acne lesions 2-4 hrs. Same-site recurrence. Dose-dependent morphology
Wheat/Gluten Pathway	Wheat, barley, rye, grain-based products, grain alcohols	Immediate watery rhinorrhea	Yes — within 20 minutes	Acne lesions 2-4 hrs. Often more inflammatory with ammonium present

The Acnerase app incorporates a **toggle feature** allowing users to identify and distinguish between Neu5Gc-pathway triggers and wheat/gluten-pathway triggers — making it a genuinely sophisticated two-pathway diagnostic and avoidance tool, not merely an ingredient scanner.

Section 7

The Ammonium Catalyst: Why Processed Foods Are the Worst Offenders

One of the most clinically significant findings of this research is the role of ammonium compounds as amplifiers of the Neu5Gc immune response. This explains a pattern that conventional acne models cannot account for: why highly processed foods — which may contain only trace or derivative Neu5Gc — frequently produce the most severe and aggressive outbreaks.

The hypothesis is that systemic ammonium levels (NH_4^+) influence the **bioavailability and tagging efficiency** of Neu5Gc as a xeno-antigen. Ammonium compounds — widely used in food processing — may enhance the uptake and incorporation of Neu5Gc into human tissues, making it more accessible to circulating anti-Neu5Gc antibodies and thus dramatically amplifying the inflammatory response.

This also explains why stress and metabolic shifts exacerbate flares — both conditions alter ammonia processing pathways, potentially increasing ammonium availability and therefore Neu5Gc reactivity independent of diet.

Hidden Danger: Caramel colouring, caramel flavouring, "spice", "spices", "flavour", "natural flavour" and other processed food derivatives frequently contain Neu5Gc compounds processed with ammonium. These ingredients are ubiquitous in packaged foods and beverages — virtually invisible to consumers. The combination of Neu5Gc + ammonium consistently produces the most severe lesion responses observed — in some cases exceeding even bovine-derived gelatin in triggering deep, aggressive, painful lesions.

Section 8

Dose-Response Model: Trigger Severity and Combination Effects

This model proposes that acne response is graded — determined by Neu5Gc concentration, bioavailability, and the presence of ammonium as an amplifying agent:

Severity	Trigger Type / Source	Ammonium	Typical Lesion Response
HIGHEST	Caramel colour/flavour, natural flavour, "spice", "spices", "flavour" — Neu5Gc derivatives + ammonium processing	YES — amplifies bioavailability	Most severe, deepest, most aggressive nodular lesions. Worst outcomes. Consistent.
VERY HIGH	Bovine-derived gelatin (packaged desserts, jellies, gummies)	Often present	Deep nodular / inflammatory lesions, painful, slow to resolve
HIGH	Dairy concentrates — cream, cheese, condensed milk, high-fat dairy	Variable	Papules / pustules, moderately inflammatory
MODERATE	Standard dairy — milk, yogurt, soft cheese	Low / absent	Papular lesions, less severe
LOWER	Indirect / trace exposure — processed derivatives without ammonium	Absent	Superficial or mild lesions, comedonal activity

Section 9

The Hamer Loop: Proposed Mechanistic Pathway

The following five-stage sequence describes the complete inflammatory cycle from dietary trigger to site-specific lesion formation:

Stage 1: Antigen Ingestion
High-bioavailability Neu5Gc and/or wheat/gluten introduced via food or beverage. Ammonium compounds amplify Neu5Gc uptake and xeno-antigen tagging efficiency.

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Stage 2: Systemic Immune Activation
Rapid immune recognition of Neu5Gc as foreign xeno-antigen. Anti-Neu5Gc antibody-antigen reaction initiates Xenosialitis. Transient systemic inflammatory state established within minutes.

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Stage 3: The Histamine Bridge
Histamine release triggers immediate rhinorrhea (nasal mast cell response). Capillary permeability increases — pro-inflammatory cytokines infiltrate dermis. Skin oiliness increases. Abdominal bloating within 20 minutes.

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Stage 4: Localized Recruitment via Immune Memory
Inflammatory markers recruited preferentially to Tissue-Resident Memory (TRM) sites. "Immunological blueprints" from prior lesions activate. Same sites. Same patterns.

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Stage 5: Glandular Activation, Pressure, Lesion Formation & Nocturnal Amplification
Sebum production increases. Follicular pressure rises. Lymphatic stasis modulates severity. Recumbent sleep amplifies fluid shift to head/neck — accelerating lesion formation overnight.

Section 10

Multi-System Inflammatory Expression

The Hamer Loop does not express itself exclusively through the skin. The same Neu5Gc-driven systemic inflammatory mechanism produces responses across multiple body systems — each representing the same immune event at a different anatomical interface. This is perhaps the most clinically significant aspect of the complete model:

Body System	Observable Response	Timing	Trigger

Nasal Mucosa	Immediate watery rhinorrhea — uncontrollable, no congestion, pure watery drip	Within minutes	Neu5Gc OR Wheat/Gluten
Gastrointestinal	Abdominal bloating	~20 minutes	Both pathways
Gastrointestinal (severe)	Severe watery diarrhea lasting 24-48 hours — gut mucosal Xenosialitis	Hours	Beer / grain alcohol primarily
Skin — Face/Body	Acne lesions at primed sites — same location, same morphology, every exposure	2-4 hours	Both pathways
Skin — Nasal (Rhinophyma)	Progressive nasal enlargement, waxy comedone plugs from nasal pores — fully reversible upon trigger cessation	Chronic exposure	Alcohol (all types)
Oral/Mucosal	Lesions on lips, inside cheeks, gums	2-4 hours	Both pathways
Eyelids / Ears	Stye-type and deep tender lesions	2-4 hours	Both pathways
Neurological	Disproportionate dizziness/stupor — amplified with sulphite + Neu5Gc combination (e.g., 1 glass grape wine)	Rapid	Sulphites + Neu5Gc combined

The Rhinophyma Observation: The progressive nasal enlargement and waxy comedone-like plugs from nasal pores — classically attributed to rosacea — observed during a period of heavy alcohol consumption, and which completely resolved upon cessation of alcohol, suggest that rhinophyma may represent chronic Neu5Gc-driven sebaceous inflammation of the nasal skin rather than a separate rosacea subtype. This is a testable hypothesis with significant dermatological implications.

Section 11

Alcohol as a Multi-Pathway Trigger

Alcohol represents a uniquely complex trigger because virtually all commercially produced alcoholic beverages contain Neu5Gc derivatives, wheat/gluten, sulphites, or ammonium processing compounds — often in combination. The following response profiles have been consistently observed:

Alcohol Type	Primary Triggers	Observed Response
Grain spirits (whisky, wheat vodka, grain gin)	Wheat/gluten + Neu5Gc flavourings/colourings + ammonium compounds	Most severe acne lesions. Pronounced rhinorrhea. Ammonium amplification likely responsible for severity exceeding standard dairy exposure.
Beer	Wheat/gluten + Neu5Gc dairy derivatives + yeast byproducts	Severe watery diarrhea 24-48 hours (gut mucosal Xenosialitis). Acne outbreaks. Congestion. Most severe GI response observed.
Grape wine	Sulphites (dairy derivatives used as preservatives) + Neu5Gc stabilizers	Acne outbreaks. Disproportionate dizziness and stupor from even 1 glass — consistent with dual-pathway histamine amplification: sulphite-triggered + Neu5Gc-triggered simultaneously.
All alcohol	Varying Neu5Gc derivatives, dairy/wheat additives, colourings, preservatives	Rhinorrhea, bloating, acne outbreaks. No alcohol type found to be consistently trigger-free.

The dual-pathway amplification observed with grape wine — where sulphite-triggered histamine release combines simultaneously with Neu5Gc-driven Xenosialitis — produces a disproportionately intense neurological and inflammatory response from even minimal intake. This may explain why sulphite-sensitive individuals consistently report reactions far exceeding what the alcohol content alone would predict.

Section 12

Nocturnal Amplification and Fluid Dynamics

A clinically significant and previously undescribed pattern has been consistently observed following trigger ingestion: a Nocturnal Inflammatory Pattern characterized by upper-respiratory congestion and morning peripheral edema.

The hypothesis is that the recumbent position during sleep acts as a physiological amplifier of the Hamer Loop. Fluid shifts toward the head and neck during recumbency increase localized edema in the facial dermis, compounding the histamine-mediated vascular permeability already initiated by the trigger. This creates a convergence of inflammatory forces — systemic immune activation combined with positional fluid redistribution — that accelerates the loop toward follicular rupture.

This nocturnal amplification explains the pattern familiar to many acne sufferers of going to bed with a mildly reactive complexion and waking to pronounced new lesions — particularly following an evening meal containing trigger ingredients. The morning congestion and facial puffiness that so frequently accompanies acne flares, but has never been mechanistically connected to them, is part of the same event.

Section 13

Extension Beyond Classical Acne

This model extends beyond sebaceous-rich skin. All affected sites share glandular or secretory structures, high vascularity, and strong local immune responsiveness — consistent with systemic inflammatory activation rather than local follicular pathology:

Location	Structure	Lesion Expression
Scalp / Neck / Back	Classic pilosebaceous — high sebum	Classic acne, repeat patterns
Lips / Oral Mucosa / Gums	Mucosal + ectopic sebaceous glands	Inflammatory bumps, same-site recurrence
Eyelids	Meibomian glands (modified sebaceous)	Stye / chalazion-type lesions
Ear Canals	Ceruminous glands	Deep, tender lesions
Nasal Skin	Dense sebaceous follicles	Waxy plugs, progressive enlargement (rhinophyma) with chronic exposure
Nasal Passages	Mucosal secretory glands	Internal inflammatory lesions, congestion

Section 14

Mechanism of Acne-Related Scarring

Within the Hamer Loop model, scarring is the predictable end result of repeated site-specific inflammatory cycles at the same immunologically primed locations — not random damage.

1. **Trigger-induced inflammation:** Systemic Neu5Gc immune activation, amplified where ammonium is present, initiates histamine release and vascular permeability

2. Sebaceous activation: Increased oil production raises internal follicular pressure, weakening the pilosebaceous unit wall

3. Follicular rupture: Contents spill into surrounding dermis, triggering a pronounced secondary immune response and enzymatic tissue breakdown

4. Collagen degradation: Inflammatory mediators destroy structural dermal tissue — depth of damage determined by trigger severity and lymphatic clearance efficiency

5. Imperfect repair: Fibroblasts deposit disorganized collagen — atrophic (sunken) scars from structural loss, hypertrophic (raised) scars from excess deposition

Repeated activation of the same site — driven by cutaneous immune memory — causes cumulative structural damage with each cycle. Scars map precisely to individual recurrence patterns. Mechanical manipulation dramatically worsens outcomes. Lymphatic stasis prolongs the inflammatory window, increasing both severity and scarring risk.

Section 15

Testable Hypotheses for Formal Research

• **Antihistamine Intervention:** Assess H1/H2 receptor antagonist efficacy in mitigating post-prandial flares within the 2-4 hour trigger window

• **Rhinorrhea as Diagnostic Signal:** Validate immediate watery rhinorrhea as a reproducible early biomarker of Neu5Gc or wheat/gluten immune activation

• **Antigen Mapping:** Controlled re-challenge studies using Neu5Gc-free vs. Neu5Gc-rich diets tracking lesion coordinates, morphology, and timing

• **Ammonium Amplification:** Comparative studies of Neu5Gc response with and without co-ingested ammonium compounds to quantify the bioavailability effect

• **Gut Mucosal Xenosialitis:** Investigate beer/grain alcohol-induced 24-48 hour watery diarrhea as a manifestation of acute gut mucosal Xenosialitis

• **Rhinophyma Mechanism:** Assess rhinophyma as chronic Neu5Gc-driven sebaceous nasal inflammation rather than rosacea subtype — via controlled alcohol cessation studies

• **Dual-Pathway Amplification:** Investigate the disproportionate neurological response to sulphite + Neu5Gc co-exposure as evidence of dual simultaneous histamine pathway activation

• **Nocturnal Pattern Study:** Correlate morning lesion formation with evening trigger ingestion and recumbent fluid redistribution

• **Real-Time Demonstration:** The primary author is able to demonstrate trigger-to-lesion onset in real time, in a person presenting with a completely clear complexion, consistently within 2-4 hours of ingestion — available for controlled clinical observation

Section 16

Implications

- Acne is a **visible manifestation of Xenosialitis** — a systemic-cutaneous interface condition, not a local follicular disorder
- Dietary Neu5Gc avoidance, particularly hidden derivatives in processed foods, represents a primary prevention strategy for susceptible individuals
- Ammonium compounds in food processing represent an unrecognized amplifier of dietary inflammatory response — requiring urgent attention to food labelling
- Immediate watery rhinorrhea following food ingestion is a previously undescribed but reproducible early diagnostic signal for Neu5Gc or wheat/gluten immune activation
- Chronic gut mucosal inflammation from alcohol may be Xenosialitis — not alcohol toxicity — with profoundly different prevention and treatment implications
- Rhinophyma may represent chronic dietary Neu5Gc-driven sebaceous inflammation rather than a rosacea subtype — fully reversible with trigger avoidance
- The 6.7 billion people with no visible acne are experiencing identical Neu5Gc-driven inflammation silently — potentially driving the chronic disease burden defining modern global health

Section 17

Conclusion

The Hamer Loop shifts the focus of acne understanding from topical suppression to systemic avoidance and vascular modulation. By viewing the skin as a check-engine light for systemic immune sensitivity — specifically Neu5Gc-driven Xenosialitis — we open new therapeutic doors for dermatological care that prioritizes arresting the systemic response at its source.

> **Acne recurrence is a trigger-driven, site-specific inflammatory loop: dietary Neu5Gc (amplified by ammonium) initiates systemic immune activation, histamine-mediated vascular changes deliver inflammatory mediators to immunologically primed skin sites, nocturnal fluid dynamics amplify the response, and localized immune memory ensures the same sites activate in the same patterns — every time. This is not a skin condition. This is the skin reporting a systemic event. And for 6.7 billion people, that report is being filed in silence.**

Section 18

About Acnerase

Acnerase (Acnerase.ca) is a free educational app developed over 23 years of independent research to help individuals — particularly young people — identify Neu5Gc-containing and wheat/gluten dietary triggers associated with acne. It identifies varying concentrations of Neu5Gc hidden within ingredients in foods, beverages, and some medication delivery systems, including the dangerous combinations of Neu5Gc derivatives with ammonium compounds that produce the most severe responses.

- Free and publicly accessible — no cost, ever
- No advertising or data collection of any kind
- **Toggle feature** distinguishes between Neu5Gc pathway triggers and wheat/gluten pathway triggers — enabling users to identify which mechanism is driving their individual response
- Identifies Neu5Gc + ammonium compound combinations — the highest severity trigger category
- Designed to support — not replace — medical care
- Focused on education, self-awareness, and the psychological wellbeing of sufferers — particularly youth

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Author Note: This model is based on over 23 years of personal observation, clinical reflection, and pattern recognition by a retired registered nurse with 45 years of acute care experience across Canada and the United States. It is presented as a testable clinical hypothesis intended to complement — not replace — existing dermatological understanding. The author welcomes formal research collaboration, peer review, and correspondence.

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