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What is Cystic Fibrosis?

An Essay on the Genetic Claim That Cannot Explain Its Own Disease

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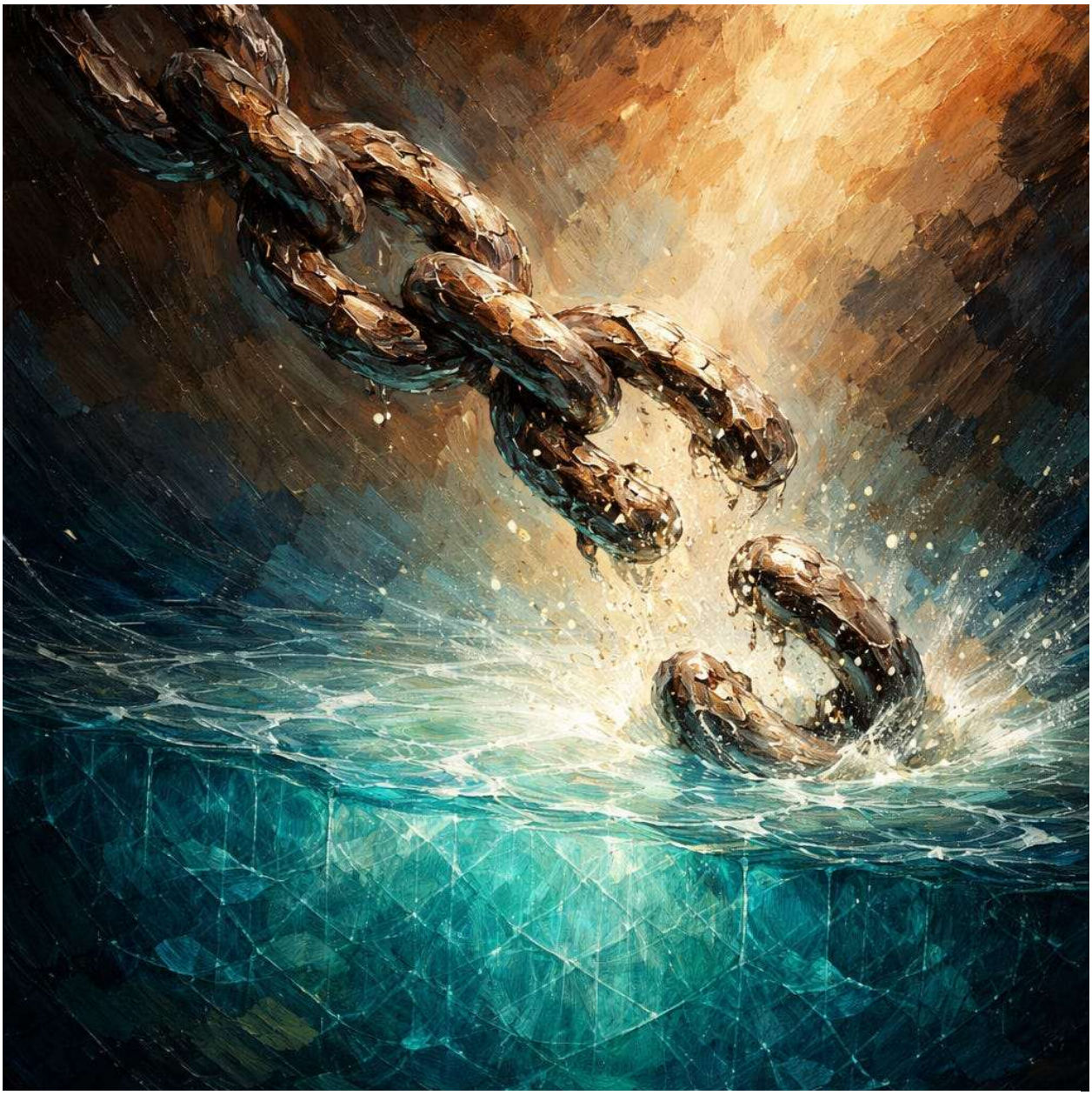

The story of cystic fibrosis is one of medicine’s tidiest genetic narratives. A

mutation in a single gene — CFTR, located on chromosome 7 — produces a defective protein. That protein fails to regulate chloride transport across cell membranes. Thick, sticky mucus accumulates in the lungs and digestive tract. The patient suffers. The gene is to blame.

It is a clean, satisfying, textbook-ready explanation — and it falls apart under examination.

Dr. Tom Cowan has spent years investigating the foundational claims of genetic medicine, and his examination of cystic fibrosis exposes a pattern that runs far deeper than one disease. The CF story is built on a chain of assumptions, each link of which, upon inspection, turns out to be weaker than the one before it. The protein the gene supposedly codes for has never been demonstrated to exist as a genuine cellular structure. The foundational paper that supposedly proved genes code for proteins explicitly states it has no direct evidence and simply assumes it. The clinical presentation of CF is so wildly heterogeneous that the single-gene explanation cannot account for its own patients. And the mainstream medical literature — published in the *New England Journal of Medicine*, no less — documents cases of cystic fibrosis phenotypes in the complete absence of any CFTR mutation.

This essay traces that chain of assumptions to its origin, link by link.

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The Claim

The official account runs as follows. Every person has two copies of the

CFTR gene. A mutation in both copies disrupts production of the cystic fibrosis transmembrane conductance regulator protein — a chloride channel that sits on the surface of epithelial cells and regulates the flow of salt and water. When the protein malfunctions, cells produce abnormally thick mucus, leading to chronic lung infections, pancreatic insufficiency, and elevated salt in sweat. The sweat chloride test detects this abnormality and confirms the diagnosis.[1]

Since the CFTR gene was identified in 1989, more than 2,500 different mutations have been catalogued.[2] The most common, F508del — a deletion of a single amino acid at position 508 — accounts for roughly 70 percent of CF alleles in Caucasian populations.[3] The remaining 2,499-plus mutations range from common to vanishingly rare, found in only a handful of people worldwide.

The narrative has the appeal of a solved problem. Gene identified. Protein characterised. Mechanism understood. Treatments — including the blockbuster CFTR modulators like ivacaftor and lumacaftor — designed to address the molecular defect directly.

But every link in this chain deserves scrutiny.

“We Shall Assume This to Be So”

The entire edifice of genetic medicine — not just CF, but all claims that genes code for specific proteins that produce specific diseases — traces

back to a single foundational paper. When Cowan investigated which paper established that segments of DNA code for specific proteins, the same reference came up repeatedly across multiple search engines and academic sources: Crick, Barnett, Brenner, and Watts-Tobin, “General Nature of the Genetic Code for Proteins,” published in *Nature* on December 30, 1961.[4]

This is the paper that introduced the triplet codon model — the idea that sequences of three nucleotide bases code for specific amino acids, which assemble into proteins. It is the paper to which all subsequent genetic medicine ultimately refers. After 1961, the gene-codes-for-protein assumption became axiomatic — an established fact requiring no further proof.

The paper itself tells a different story. Under the section titled “The Explanation in Outline,” Crick and colleagues write:

> “Our explanation of all these facts is based on the theory set out at the beginning of this article. Although we have no direct evidence that the B cistron produces a polypeptide chain (probably through an RNA intermediate), in what follows we shall assume this to be so.”[4]

The foundational paper of molecular genetics — the paper that every subsequent claim about genes coding for proteins ultimately rests upon — contains an explicit admission that its authors possessed no direct evidence for their central claim. They assumed it. And everything that followed — the triplet code, the reading frame, the entire apparatus of genetic medicine — was built downstream of that assumption.

The rest of the 1961 paper consists of elegant experimental work on

bacteriophage T4 mutations and mathematical analysis of reading frames. The science within the paper's own scope is rigorous. But Crick's experimental results demonstrate properties of genetic sequences in phage; they do not demonstrate that those sequences produce proteins. The leap from "we can manipulate reading frames in phage DNA" to "genes code for proteins" is precisely the leap the authors themselves acknowledge they are assuming rather than proving.

This is not a minor caveat buried in supplementary material. It is the stated foundation of the paper's entire explanatory framework.

General Nature Of The Genetic Code For Proteins 1961
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The Protein That Has Never Been Seen

The CFTR model depends on the existence of a specific transmembrane protein — a chloride channel embedded in cell membranes. This protein is the entire mechanism by which the genetic mutation supposedly produces disease.

Dr. Harold Hillman (1930–2016) spent his career investigating a problem that most biologists prefer to ignore: the preparation procedures used in electron microscopy and other laboratory techniques generate artefacts — artificial structures not present in living tissue — which are then mistakenly treated as genuine features of cells.[5]

Hillman's critique is devastating and specific. When tissue is prepared for electron microscopy, it undergoes a series of violent interventions: the animal is killed, the tissue is excised, fixed or frozen, embedded, sectioned, dehydrated, stained with heavy metals, and bombarded with electron beams in a vacuum chamber. To claim that none of these procedures alters the appearance of the tissue is, as Cowan puts it in *Breaking the Spell*, "beyond ridiculous."[6]

The structures Hillman identified as artefacts include some of the most fundamental components of the standard cell biology model. Ribosomes — the supposed sites where mRNA is translated into protein — appear as perfect circles on every electron micrograph image. A perfectly circular two-dimensional image implies a perfectly spherical three-dimensional structure. But ribosomes are found only after the cell has been homogenised — essentially put through a blender. As Cowan explains the geometry: when any perfectly spherical structure is put through a blender, it is impossible that every resulting slice would be a perfect circle. The laws of spherical geometry rule it out. The perfect circles are stained gas bubbles, an inevitable product of tissue preparation.[6]

The endoplasmic reticulum — the tube-like structure supposedly connecting the nucleus to the rest of the cell — is likewise visible only on electron micrographs, never in living cells under a light microscope. It was invented to solve a specific problem: if DNA lives in the nucleus, bound by a membrane, how does mRNA get from the nucleus to the cytoplasm where it is supposed to be translated into protein? The endoplasmic reticulum was the answer — a tube connecting nucleus to cytoplasm, with ribosomes attached, providing a pathway for mRNA to travel and be translated.

But the endoplasmic reticulum creates a problem of its own. Direct measurement shows that the pH inside the nucleus differs from the pH in the cytoplasm. This means hydrogen ions — among the smallest ions in existence — cannot freely pass between nucleus and cytoplasm. If the endoplasmic reticulum provides a tube connecting the two, it must have an exit large enough for mRNA molecules, which are thousands of times larger than a hydrogen ion. Any exit that permits mRNA to pass would easily permit hydrogen ions to equilibrate between the two compartments. The pH difference would disappear. It does not.[6]

There is a second problem. When living cells are observed under a dark-field microscope, the nucleus rotates continuously, sometimes through full 360-degree turns. If tube-like structures were tethering the nucleus to the outer cell wall, such rotation would be mechanically impossible. The cords would wrap and break. The endoplasmic reticulum is likely a precipitation artefact created by the destructive preparation techniques used in electron microscopy.[6]

Hillman identified membrane receptors as preparation artefacts.[7] This is the critical point for cystic fibrosis: if membrane receptors and transmembrane channels are artefacts of the preparation process — structures that do not exist in intact, living cells — then the CFTR transmembrane conductance regulator protein does not exist as described. As Cowan has observed in multiple webinars and writings, when you take an electron microscope image of a cell membrane, you never see pumps, gates, or channels. All you can say is that certain ions have certain effects. The mechanism is an invention grafted onto an observation.[8]

The gene supposedly codes for a protein. The protein supposedly sits in a

membrane structure. Neither the protein's existence in living tissue nor the membrane structure it supposedly inhabits has been demonstrated by methods that don't destroy the very thing being studied.

One Gene, 200,000 Proteins

The CF genetic claim rests on a broader assumption: that each gene codes for a specific protein. The Human Genome Project was expected to confirm this. Since the human body contains at least 200,000 proteins, the genome was expected to contain a comparable number of protein-coding genes.

It did the opposite. The human genome contains roughly 20,000 to 30,000 protein-coding genes.[9] That leaves well over 170,000 proteins with no corresponding genetic blueprint. Dr. Barry Commoner, senior scientist at the Centre for the Biology of Natural Systems at Queens College, observed in his 2002 article "Unraveling the DNA Myth" that one gene giving rise to multiple proteins destroys the theoretical foundation of genetic medicine.[10]

Scientists attempted to rescue the theory by postulating that enzymes cut and splice the 20,000-odd genes, rearranging them to produce the missing proteins. This is possible. But it means the one-gene-one-protein model — the very model on which the CF genetic explanation depends — has already been falsified by the medical establishment's own data. If 20,000 genes must somehow account for 200,000 proteins, then the relationship between gene and protein is not the simple, deterministic, one-to-one correspondence that the CF narrative requires.

Genes Don’t Issue Orders

Even setting aside the protein-count problem, the assumption that a defective gene *causes* a disease requires that genes function as controlling agents — upstream commanders issuing instructions to passive cellular machinery.

This assumption has been undermined by epigenetics. Dr. Bruce Lipton’s research demonstrated that genes respond to environmental signals; they do not issue commands. In his laboratory experiments, changing the culture medium altered the behaviour and health of genetically identical cells. The same genome, the same DNA sequence, produced different outcomes depending on environmental conditions.[11]

Dr. Robert Hedaya described the mechanism succinctly: genes are turned on or off by environment, diet, stress, and experience.[12] If genes are downstream of environmental signals — responding to conditions rather than dictating them — then the entire framework of “a defective gene causes CF” requires re-examination. The gene is not issuing the orders. Something in the terrain is.

2,500 Mutations, One Diagnosis

The clinical reality of cystic fibrosis presents the single-gene theory with a problem it cannot solve.

A single mutation in a single gene producing a single defective protein should produce a reasonably consistent clinical picture. Cystic fibrosis does not. The 1989 foundational paper on CFTR genetics — published in the *American Journal of Human Genetics* — states plainly that the clinical expression of CF is "heterogeneous."[13] Some patients have chronic obstructive lung disease. Some have pancreatic insufficiency. Some have problems digesting fat. Some have none of these.

Fifteen percent of CF patients have sufficient pancreatic exocrine function for normal digestion and require no enzyme supplementation with meals.[13] This figure is confirmed across the mainstream literature: approximately 85 percent are classified as pancreatic insufficient, while the remaining 10–15 percent retain normal digestive function despite carrying the same diagnosis.[14] [15]

The mutation landscape is even more troubling. Over 2,500 different mutations on the same gene have been identified, all supposedly producing some variant of the same disease.[2] These mutations have been sorted into six classes based on how they affect the CFTR protein — from mutations that prevent any protein production to those that merely reduce its stability. The mainstream literature acknowledges the resulting complexity: different mutations, different protein defects, different clinical presentations, different prognoses.

CF twin and sibling studies reveal that the proportion of CF variance attributable to genetic factors — the heritability — ranges from 0.6 to 0.8.[16] In plain language, 20 to 40 percent of the variation in CF outcomes has nothing to do with the gene. The diagnostic literature itself concludes that genotype is "not a good predictor of clinical outcome and should not be

used as an indicator of prognosis."[16]

Forty years of attempting to match specific mutations to specific prognoses has produced, as Cowan observes, almost nothing. The claim was: one gene, one protein, one disease, one mechanism. The reality is: 2,500 mutations, six classes of protein defect, wildly variable clinical presentation, 15 percent with normal digestion, and a heritability that leaves a full quarter to two-fifths of outcomes unexplained by genetics.

Cystic Fibrosis Without the Gene

The most damaging evidence comes from a 2002 study published in the *New England Journal of Medicine*.

Groman et al. performed extensive genetic analysis on 74 patients with nonclassic cystic fibrosis referred from 34 medical centres. Of the 74 patients, 29 had two CFTR mutations. Fifteen had one mutation. And 30 — more than 40 percent — had no CFTR mutations whatsoever.[17]

The study's most striking finding: patients with CFTR mutations could not be clinically distinguished from those without mutations. Sweat chloride concentrations, degree of organ involvement, and clinical presentation were statistically indistinguishable between patients carrying two mutations, one mutation, or none. The degree of multiorgan-system involvement — in the sweat gland, gastrointestinal tract, and respiratory system — was similar across all three groups.[17]

The study also examined two families in which siblings both had nonclassic CF but carried no identified mutations. Haplotype analysis revealed no linkage to the CFTR locus. Despite elevated sweat chloride concentrations, measurements of CFTR-mediated ion transport in the sweat gland and nasal epithelium demonstrated the presence of functional CFTR protein. The protein was working. The patients were still sick.[17]

The study's conclusion, stated directly: "Factors other than mutations in the CFTR gene can produce phenotypes clinically indistinguishable from nonclassic cystic fibrosis caused by CFTR dysfunction."[17]

This is published in the *NEJM*, not a fringe journal. It means that whatever is producing the constellation of symptoms we call cystic fibrosis, a CFTR gene mutation is neither necessary nor sufficient to explain it. People with the mutations can have normal digestion. People without the mutations can present with the full clinical picture. The gene, at best, is one factor among many. At worst, it is a correlate being mistaken for a cause.

What the Sweat Test Actually Measures

The sweat chloride test — the standard diagnostic tool for CF — measures the concentration of chloride in a patient's sweat. Elevated sweat chloride is taken as evidence of defective CFTR-mediated chloride transport.

There is an additional wrinkle worth noting. The CFTR gene is supposed to code for a *chloride* channel. The entire CF model centres on chloride

transport. But research documented by Daniel Roytas reveals something curious: the hypertensive effect attributed to table salt (sodium chloride) is absent when people consume other sodium-containing compounds like sodium bicarbonate, sodium ascorbate, or sodium citrate. Meanwhile, other chloride-containing salts — potassium chloride, calcium chloride — *do* increase blood pressure. The physiological effects attributed to "salt" appear to be driven by chloride, not sodium.[21]

This doesn't directly refute the CF model. But it does suggest that chloride biology is more complex than the simple channel model implies, and that chloride imbalance may have causes far upstream of any single gene.

From a terrain perspective, the sweat test is measuring something real — but not what the genetic model claims. It is detecting a disturbance in the sodium-potassium-chloride balance of the tissues. The question is not "which gene controls the chloride channel?" but "what environmental conditions are disrupting electrolyte balance in this person?"

The work of Dr. Gilbert Ling provides the framework. Ling demonstrated that the distribution of sodium and potassium across the cell is not maintained by membrane pumps — another structure never visualised in living tissue — but by the configuration of structured water in the intracellular space. When the intracellular gel is healthy and properly structured, its molecular mesh inherently binds potassium and excludes sodium. No pump is needed. The gel itself, through its physical structure, creates and maintains the electrolyte gradient that keeps the cell charged and functional.[18]

Dr. Gerald Pollack's research on the fourth phase of water — exclusion zone (EZ) water — extended this understanding. EZ water forms a

crystalline gel structure against hydrophilic surfaces, carrying a negative charge and excluding minerals and dissolved compounds from its structure. This fourth-phase water is the basis of the gel state in living cells.[19]

Cowan applies this framework directly to CF. The thick, sticky mucus characteristic of CF is not the product of a defective gene coding for a defective protein in an imaginary membrane channel. It is the result of disrupted water structure in the tissues — a disturbance in the sodium-potassium-chloride balance that alters how water organises itself in the cells of the lungs and digestive tract. The sweat test detects this mineral imbalance. The genetic model interprets it as evidence for a defective gene. The terrain model interprets it as evidence that something has disrupted the fundamental structuring of tissue water.[20]

Cowan has described this gel-disruption model across multiple disease contexts. The lens of the eye is a crystalline water structure; when toxins or electromagnetic exposure disturb its crystalline nature, the gel becomes distorted and opaque — what we call a cataract. Joints are surrounded by negatively charged gels; when these gels are disrupted, the protective repulsion is lost and movement becomes painful — what we call osteoarthritis. When toxins dissolve into the gels, the body elevates temperature to partially liquify them and flush them out in mucus — what we call fever.[19]

CF fits the same pattern. The mucus in the lungs and digestive tract is thick and sticky not because a gene has produced a defective protein, but because the water structure in those tissues has been disrupted. The gel has lost its proper crystalline organisation. The electrolyte balance — sodium, potassium, chloride — is a downstream indicator of this disruption,

not a consequence of a faulty membrane channel.

As Cowan puts it: anytime you're seeing an abnormality in the sodium, potassium, or salt balance, you're looking at a situation where the water structure inside the tissues is out of balance. The question to ask is not "which gene is broken?" but "what has disturbed the fundamental structuring of the water in this person?"[20]

The Chain of Assumptions

The CF genetic narrative is a chain, and each link depends on the one before it:

The gene codes for a protein — but the foundational paper explicitly states there is no direct evidence for this and assumes it.[4]

The protein is a transmembrane chloride channel — but transmembrane receptors and channels have never been demonstrated in living tissue and fall under Hillman's artefact critique.[5] [6]

Each gene codes for one protein — but the Human Genome Project found roughly ten times more proteins than genes, demolishing the one-to-one model.[9]

The gene mutation causes the disease — but epigenetic research shows genes respond to environment, not the reverse.[11]

One specific mutation produces one specific disease — but 2,500 different mutations produce wildly variable clinical pictures, 15 percent of patients

have normal digestion, and genotype cannot predict clinical outcome.[13] [14] [16]

The gene is necessary for the disease to occur — but
the *NEJM* documents CF phenotypes in patients with no CFTR mutations at all.[17]

Every one of these links is compromised. The chain does not hold.

So What Is Cystic Fibrosis?

If not a genetic disease, then what? The honest answer is that we don't know with certainty — but not because the question is unanswerable. We don't know because the research to answer it has never been funded. Virtually every dollar spent on CF research for the past 35 years has followed the genetic model. This is the streetlight effect: scientists look where the funding is, not where the answer is likely to be. The absence of a specific identified terrain cause for CF is not evidence against the terrain framework. It is evidence of where the money went.

The Streetlight Effect
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JAN 31

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What we can do is reason from the evidence that exists and identify the

most probable mechanism and the most likely category of causes.

The mechanism is the clearest part. The symptoms of CF — thick, sticky mucus in the lungs and digestive tract, elevated chloride in sweat, disrupted pancreatic function — are all consistent with a single underlying disturbance: the degradation of structured water in the affected tissues. When the intracellular gel loses its proper crystalline organisation, it can no longer maintain the correct sodium-potassium-chloride distribution. The electrolyte imbalance follows. The mucus thickens. The organs that depend most heavily on properly structured water — lungs, pancreas, sweat glands — are the ones that fail.

This is not speculation layered on speculation. It follows directly from Ling's demonstrated mechanism for electrolyte distribution (the gel structure itself maintains the ion gradient, not membrane pumps) and Pollack's laboratory work on the fourth phase of water. The sweat chloride test — CF's own diagnostic gold standard — is measuring exactly this: a disruption in the electrolyte balance of the tissues. The test is real. What it measures is real. The disagreement is about what is causing the disruption.

The causes, in the terrain framework, fall into a recognisable category: anything that degrades the structure of intracellular water. Environmental toxins that dissolve into the gels and disrupt their crystalline organisation. Electromagnetic field exposure — Pollack's own research shows that a nearby Wi-Fi router reduces exclusion zone water by approximately 15 percent.[19] Nutritional deficiencies, particularly in potassium and the minerals required to maintain proper electrolyte balance. Endocrine-disrupting chemicals — relevant here because the NIDDK classifies CF as an endocrine disease, placing it alongside conditions driven by hormonal

disruption rather than genetic fate.[7]

The body's response to toxins dissolved in its gels is to raise temperature — fever — which partially liquifies the gels so the toxins can be flushed out as mucus. CF, in this framework, is what happens when this detoxification process becomes chronic: tissues perpetually trying to clear insults they cannot fully expel, producing the thick mucus that defines the diagnosis.

No specific toxin or exposure has been identified as *the* cause of the CF symptom cluster. That research hasn't been done. But the mechanism is coherent, the observable evidence supports it, and it explains what the genetic model cannot: why genotype doesn't predict outcome, why people without the mutation get the disease, and why 35 years of genetic research has failed to identify the "primary defect" that the 1989 foundational paper admitted was still unknown.

A Different Question

The people diagnosed with cystic fibrosis are genuinely ill. The thick mucus, the lung infections, the digestive problems — none of this is in dispute. The question is what is making them sick.

The genetic model has had over 35 years since the CFTR gene was identified in 1989. Billions of dollars in research funding. Thousands of papers. The clinical result: a set of modulator drugs that partially improve chloride transport in some patients with some mutations, while the fundamental disease process remains poorly understood, genotype cannot predict prognosis, and the "primary defect in cystic fibrosis" was described

as “still unknown” in the very paper that claimed to have found the gene responsible.[13]

Meanwhile, six percent of people with CF have mutation combinations that produce no CFTR protein at all, rendering them ineligible for modulator therapies.[1] For these patients, the genetic approach offers nothing. They are told the cause of their disease is known — it’s genetic — but the treatment pipeline built on that knowledge cannot reach them.

The terrain model asks a different and more productive question: what has disrupted the water structure — the gel phase, the electrolyte balance, the fundamental organisation of the living tissue — in this person? What toxins, what deficiencies, what electromagnetic or environmental insults have degraded the crystalline structure of the intracellular water to the point where sodium-potassium-chloride balance can no longer be maintained?

That question leads somewhere. It points to actionable environmental factors rather than an inherited fate written in code. It explains why genotype cannot predict prognosis — because the gene is not the driver. It explains why people without the mutation can present with the disease — because the mutation was never the cause. And it explains why 35 years of genetic research has produced so little clinical progress — because the research has been asking the wrong question from the start.

The genetic story of cystic fibrosis is a story told from within a framework that has already been undermined by its own data. The Human Genome Project demolished the one-gene-one-protein model. Hillman’s work challenges the existence of the very membrane structures the model depends on. Crick’s own paper admits the central assumption was never proved. The clinical heterogeneity makes a mockery of single-gene

determinism. And the *NEJM* documents the disease occurring in the absence of the gene.

All of these findings point in the same direction — away from genetic determinism and toward the terrain.

Four Causes, Seventy Thousand Diseases
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Explain It to a Six-Year-Old

Imagine you have a recipe book (your genes) and a kitchen (your body). The doctors say one page of the recipe book has a spelling mistake, and that spelling mistake is why the kitchen keeps making thick, sticky soup instead of nice thin soup.

But scientists found out the recipe book has way fewer pages than they thought. And the pages don't actually tell the kitchen what to do — the kitchen decides what to cook based on what ingredients are available and what temperature it's at. Change the ingredients, change the temperature, and the kitchen makes something completely different — even with the

exact same recipe book.

And some kitchens with the spelling mistake make perfect soup. And some kitchens with no spelling mistake at all make the thick, sticky kind.

So maybe the problem isn't a spelling mistake in the recipe book. Maybe the problem is what's in the kitchen.

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New Biology Clinic

For those of you looking for practitioners who actually understand terrain medicine and the principles we explore here, I want to share something valuable. Dr. Tom Cowan—whose books and podcasts have shaped much of my own thinking about health—has created the **New Biology Clinic**, a virtual practice staffed by wellness specialists who operate from the same foundational understanding. This isn’t about symptom suppression or the conventional model. It’s about personalized guidance rooted in how living systems actually work. The clinic offers individual and family memberships that include not just private consults, but group sessions covering movement, nutrition, breathwork, biofield tuning, and more. Everything is

virtual, making it accessible wherever you are. If you've been searching for practitioners who won't look at you blankly when you mention structured water or the importance of the extracellular matrix, this is worth exploring. Use discount code **"Unbekoming"** to get $100 off the member activation fee. You can learn more and sign up at newbiologyclinic.com

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