Episode Book

EPISODE #5668

Why Depression Drains Your Energy

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SYNOPSIS

Daniel wrote in about a friend with seasonal affective disorder, and asked a question that turns out to be two questions: how do light and mood actually connect, and why does non-seasonal depression drain energy at all? This episode traces the light pathway — from the intrinsically photosensitive retinal ganglion cells that don't help you see, to the contested question of whether light moves your circadian clock or acts on mood circuitry directly. Then it turns to the harder half: a condition where fatigue shows up in over ninety percent of patients, where reward processing and inflammation each play distinct roles, and where the "hibernating" symptom profile looks suspiciously like an old mammalian program still running.

TRANSCRIPT



Corn

It's the first of October.



Herman

Which means somewhere out there, somebody is already feeling the floor drop out.



Corn

Right. The days are getting shorter, the light is getting thinner, and for people with seasonal affective disorder, this is the week it starts.



Herman

Not the calendar week. The light week. It just happens to land around now.



Corn

Which is where Daniel's question comes in. He wrote in about a friend of his who struggles with this, and he says it sounds debilitating. And what he wants to unpack isn't really SAD itself. He says SAD he can sort of understand. We associate light with vitality. That part tracks. What he can't get his head around is why depression has such a hold on energy at all. He put it as: for him, that connection has never been obvious.



Herman

Because it isn't obvious.



Corn

Right. And he's careful about it. He says most people with depression probably don't have SAD, and the connection there is a lot more complicated than "I don't get enough natural light so I feel sad and tired." So the ask is really two things. First, the relationship between mood, light, and energy. Second, and this is the harder one, why non-seasonal depression



Corn (continued)

drains you the way it does.



Herman

Two different mechanisms wearing the same symptom.



Corn

That's the episode.



Herman

Start with what SAD actually is, because people use the term loosely. It's a type of depression with a recurrent seasonal pattern. Symptoms typically run four to five months, starting in late fall or early winter and lifting in spring and summer. And the diagnosis is stricter than people assume. You need depressive episodes in a specific season for at least two consecutive years, occurring more frequently at that time of year than at any other.



Corn

So it's not "I feel a bit flat in January."



Herman

One in five people in the UK report winter blues. About two percent have actual SAD. That's a big gap.



Corn

And the timing. Why October, if the diagnostic window is late fall?



Herman

Because it tracks daylight, not the calendar. The depression is related to changes in daylight hours. Norman Rosenthal, who named the condition, makes the point that it isn't strictly a winter thing. Symptoms can show up during cloudy stretches, in dark offices, in dark apartments. Whenever there's insufficient sunlight.



Corn

So a person in a windowless office in September could be in it already.



Herman

If they're vulnerable, yes.



Corn

What does it actually feel like? Because the symptom profile is strange.



Herman

It's atypical. That's the clinical term, and it's the opposite of what you'd expect. Classic non-seasonal depression tends toward insomnia and appetite loss. Winter-pattern SAD goes the other way. People oversleep. They overeat, specifically carbohydrates, and they gain weight. And they withdraw socially. The clinical word for it is hibernating.



Corn

Hibernating is the word they chose. That's not a metaphor anyone had to reach for.



Herman

It's almost embarrassingly on the nose.



Corn

So you've got a condition that looks like a mammal winding down for winter. Which raises the question of whether that's literally what it is.



Herman

Hold that. That's actually one of the live hypotheses and we'll get to it.



Corn

Lay out the light pathway first. Because Daniel's intuition, that light equals vitality, turns out to be more mechanically real than most intuitions people have about mood.



Herman

It's real, and the reason it's real is that the eye does more than see. Everyone learns about rods and cones. Those are the image-forming photoreceptors. But there's a third class, the intrinsically photosensitive retinal ganglion cells. They express a pigment called melanopsin.



Corn

And they don't build pictures.



Herman

They don't build pictures at all. They're not there for vision. They innervate dozens of brain areas to influence physiology, behavior, perception, and mood. That's the phrase from a 2019 review by Do in Neuron. Dozens of brain areas.



Corn

For cells that don't help you see anything.



Herman

They're doing something else entirely. The term is non-image-forming functions. Circadian photoentrainment, which is how your internal clock gets set to the actual day. The pupillary light reflex. And modulation of sleep, alertness, and mood.



Corn

So there's a dedicated channel from the retina to the mood and alertness systems that bypasses vision entirely.



Herman

A separate wire. And here's where it gets contested. The standard story is that light affects mood by shifting your circadian clock. You get out of sync, you feel bad. That's the phase-shift hypothesis for SAD.



Corn

Which is what?



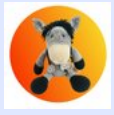
Herman

Later winter sunrises delay your internal rhythms relative to your sleep and wake schedule. So your mood lows land during the day instead of at night, and you wake up in what amounts to night mode. Tired, sluggish, flat.



Corn

That's a satisfying story. Light sets the clock, clock drifts, mood follows.



Herman

It is satisfying. And Lazzerini Ospri, Prusky and Hattar, in the Annual Review of Neuroscience in 2017, put forward an alternative. They argue light's effect on mood may run through an ipRGC-dependent mechanism independent of circadian disruption. Meaning light might affect mood directly, not only by moving the clock.



Corn

So two models. Light moves the clock, mood follows. Or light hits mood circuitry in parallel and the clock is a separate thing.



Herman

And the review explicitly presents a model that dispenses with assumptions of causality between circadian disruption and mood. That's a real fork in the field, not a settled question.



Corn

Which matters for treatment, presumably.



Herman

It matters for what you'd even try to fix. If it's the clock, you fix the clock. If light is acting on mood directly, then timing might matter less than we think, and it's more about total light exposure.



Corn

Rosenthal's own suggestion was genetic, right?



Herman

He suggested some people with SAD may have abnormalities in genes for serotonin transmission or in retinal light sensitivity. And he specifically flagged the retinal pigment melanopsin as a possible site.



Corn

So the receptor itself might be tuned differently in different people.



Herman

Which would explain why one person in a dim office is fine and the person at the next desk is not.



Corn

Give me the history. Because this condition has an origin date.



Herman

1984. Rosenthal and colleagues described it in Archives of General Psychiatry, based on twenty-nine patients in Maryland. And here's the detail that surprises people. Twenty-seven of those twenty-nine had bipolar disorder.



Corn

So the first cohort was almost entirely bipolar.



Herman

Almost entirely. Which is not how SAD is popularly imagined at all.



Corn

Anna Wirz-Justice, at Basel, said of that paper that it was as though this something had been there all the time, but it didn't have a diagnosis or label until then.



Herman

Which is the story of a lot of diagnoses. The thing existed. The name didn't.



Corn

And Rosenthal wasn't theorizing from a distance. He came to the US from South Africa in 1976 and described his own experience of it. A draining of energy, difficulty getting through his tasks once the days drew short and dark.



Herman

He lived the thing he named.



Corn

Numbers. How common is this?



Herman

By the standard questionnaire criteria, up to three percent of Europeans, ten percent of North Americans, one percent of Asians. And there's a latitude gradient in the US data. New Hampshire around nine point four percent. New York four point seven. Maryland six point three. Florida four.



Corn

That's not a clean line.



Herman

It isn't, and I'd flag that. Florida at four percent is not trivial, and Maryland comes in above New York. Latitude is doing something, but it isn't doing everything. Cloud cover, indoor life, who responds to the survey, all of it muddies the picture.



Corn

Women?



Herman

About four times as likely as men.



Corn

And the evolutionary hypothesis. The one I told you to hold.



Herman

This is the part that makes the hibernating language look less like a metaphor. The idea is that SAD may be a retained mammalian mechanism. Sheep track seasons by the duration of melatonin release. Something analogous may be sitting in us. And becoming more lethargic and depressed during colder months made sense when food was scarce. You conserve energy. You wait it out.



Corn

So the low-energy state isn't a malfunction. It's an old program running in an environment that no longer needs it.



Herman

That's the argument. And there's a counterexample that complicates the whole framing. The Sami, in northern Scandinavia, embrace the seasonal slowdown rather than fighting it. Which challenges the assumption that year-round high energy is the healthy norm and everything else is pathology.



Corn

That's the part I want to sit with, because it reframes the question. Daniel asked why depression drains energy. But if there's a version of the low-energy state that's culturally normal and not distressing, then the drain isn't automatically the disease.



Herman

The distress is the disease. The slowdown might just be October.



Corn

Alright. That's the easy half. Light, melanopsin, circadian phase, an old mammalian program. All of it intuitive, because light and vitality feel connected.



Herman

And now the harder half.



Corn

Most depression isn't seasonal. What's the actual split?



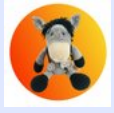
Herman

In both SAD and general depression, somewhere between ten and twenty percent of patients report a seasonal variation in their symptoms. So there's overlap in both directions. But the trigger differs. SAD onset is usually triggered by shortened daylight exposure, as opposed to negative life events.



Corn

Which is a clean distinction on paper and probably messier in a person.



Herman

Almost certainly messier. But it's the right frame. One is triggered by the sky. The other is triggered by life, or by biology, or by both.



Corn

So take the seasonal piece out. A person with major depression, in July, with plenty of light. Why are they exhausted?



Herman

Start with how common it is, because this is the part that gets underestimated. Fatigue occurs in over ninety percent of patients with major depressive disorder.



Corn

Ninety.



Herman

Over ninety. This isn't a side symptom. For most people with the condition, it's one of the defining features.



Corn

And the folk explanation is laziness. Or lack of willpower.



Herman

Which is wrong, and it's wrong in a specific, mechanistic way. There are at least two distinct systems involved. The first is reward processing.



Corn

Meaning what, exactly?



Herman

Depression involves impaired reward processing and a reduced willingness to exert effort for reward. And that's linked to dopamine function. There's a 2024 framework paper in Translational Psychiatry on exactly this. And separately, anhedonia severity predicts lower motivation for both physical and cognitive effort. Both kinds. Not just getting off the couch. Thinking hard, too.



Corn

So it's not that the reward isn't there. It's that the system that converts reward into effort has stopped paying out.



Herman

The person can still want things. The machinery that turns wanting into doing is what's degraded.



Corn

And the second system?



Herman

Inflammation. This is the one I find fascinating, and it's the one that connects back to the hibernation thread. Proinflammatory cytokines, interleukin-one, interleukin-six, TNF-alpha, produce a specific syndrome. The list is lethargy, depression, anorexia, energy conservation, fever, anhedonia, cognitive impairment.



Corn

Read that list again and tell me it isn't a description of being ill.



Herman

It's a description of being ill. That's the point. It's called sickness behavior, and it's not a malfunction. It's what the body does when it's fighting something. You lose your appetite, you lose interest, you want to lie down in the dark. It's an energy-conserving response.



Corn

So the body has a program for "shut down and recover," and it runs it whether or not there's an infection.



Herman

And Maes and colleagues, in BMC Medicine in 2012, described depression and sickness behavior as Janus-faced responses to shared inflammatory pathways. Same pathways, two faces. And they framed it as having a good, acute side and a bad, chronic side.



Corn

The acute side being: you get sick, you shut down, you recover, you get up.



Herman

And the chronic side being: the shutdown never lifts. The program that was supposed to last a week is still running in month four.



Corn

That reframes the whole thing. Low energy in depression isn't a bug. It's a conserved adaptive response that's gone chronic.



Herman

That's the thesis. And it's testable, which is what makes it more than a nice story. If you activate the innate immune system in healthy people, you induce fatigue, worsened mood, and pain sensitivity. That's from Lasselin and colleagues in Frontiers in Neuroendocrinology in 2018. You give someone an inflammatory challenge, and they start reporting exactly the symptoms we're describing.



Corn

So the causation runs in the direction the model predicts.



Herman

In healthy volunteers, yes. Which is about as close to a controlled experiment as you can ethically get.



Corn

And the two systems, reward and inflammation, they're not separate, are they?



Herman

They appear to converge. There's a scoping review in Translational Psychiatry from 2020 arguing that fatigue and anhedonia may share a common mechanism, running through dopamine and inflammatory cytokines. So the motivational account and the immune account may be two descriptions of one underlying process.



Corn

Which would explain why fatigue and anhedonia so often arrive together.



Herman

They cluster. And if they share a mechanism, that's not a coincidence.



Corn

So now pull the two halves together. SAD and non-seasonal depression.



Herman

They may share a final common pathway, which is energy conservation, but they arrive there differently. SAD gets there through seasonal light and circadian signals. Non-seasonal depression gets there through inflammation and reward-circuitry failure. Same destination. Different roads.



Corn

The hibernation program and the sickness program, both ending in the same place.



Herman

And both of them, in their original context, being sensible. Conserve energy when food is scarce. Conserve energy when you're infected. The problem is a system designed to run for a season, or a week, running indefinitely.



Corn

Does the treatment split follow the mechanism split?



Herman

Partly, and this is where it gets interesting. Light therapy works for SAD. The standard dose is ten thousand lux for thirty to forty-five minutes daily, usually in the morning, from fall through spring. The box is about twenty times brighter than ordinary indoor light.



Corn

Twenty times.



Herman

That's why a bright kitchen doesn't do it. You need the intensity, and you need it at the right time of day.



Corn

And for non-seasonal depression?



Herman

The evidence for bright light in non-seasonal major depression is newer and more mixed. It's not the same slam dunk. Which is what you'd expect if the mechanisms are different. If the problem isn't light, adding light is a weaker bet.



Corn

There's a trial from last year worth mentioning.



Herman

Volf and colleagues, in Chronobiology International. They used dynamic LED lighting that mimics a daylight cycle, rather than static lighting, in female inpatients with major depression. Daytime melanopic illuminance peaked at five hundred and seventy-six lux, versus sixty-six in the static condition, and dimmed to zero point three lux in the evening.



Corn

And the result?



Herman

A significantly greater antidepressant effect at week three in the female inpatients. Seventy-one percent response. But not in the whole group. And I'd flag that honestly, because that's a partial result, not a clean one.



Corn

Seventy-one percent in a subgroup is real, but it's a subgroup.



Herman

It's a signal, not a verdict. And there's a bigger point sitting under it. The reason this matters is that it suggests the shape of the light, the dynamic rise and fall, might matter more than the raw dose. Which loops back to the mechanistic fork. If timing and rhythm matter, the clock is involved. If raw intensity matters, it's more direct.



Corn

And then there's the finding that cuts against the pure-photon story entirely.



Herman

Cognitive behavioral therapy for SAD outperformed light therapy at long-term follow-up. Kelly Rohan, at the University of Vermont, who does that work, put it this way. She doesn't argue there's no strong physiological component, and she agrees it's tied to the light-dark cycle. But she argues the person has some control over how they respond to and cope with that.



Corn

So the meaning we assign to winter matters, not just the photons.



Herman

Which is a awkward finding if you're a strict mechanism person. The light is doing something. But so is what the person believes about the light, and what they do when it goes.



Corn

And the timeline on treatment is worth saying out loud, because people quit early. Antidepressants take four to eight weeks to work.



Herman

Four to eight weeks. And SAD symptoms last an average of forty percent of the year. So you're talking about a condition that eats two fifths of the calendar, and a treatment that takes two months to show up.



Corn

Which is a brutal mismatch.



Herman

It's a real one.



Corn

Before we go further, I want to flag something about Daniel's framing. He said SAD is easier to understand because we associate light with vitality. And that intuition is correct, but it's correct for a reason most people don't know. It's not a metaphor. There's a dedicated photoreceptor system for it.



Herman

The intuition is right and the mechanism is stranger than the intuition.



Corn

People think "light makes me feel good" the way they think "sunshine is nice." It's actually a separate neural channel that doesn't involve seeing at all.



Hilbert

It's the flicker.



Corn

Sorry?



Hilbert

The flicker matters more than the brightness. I maintained the lighting in a greenhouse for two years, late seventies. Twelve hundred tubes, and the ones that were failing didn't go dark. They flickered at a rate you couldn't consciously see. The plants didn't care. The people did.



Herman

Because flicker at certain frequencies is known to affect...



Hilbert

The women on the potting line would move. Not complain, just move. They'd drift down the bench to a section where the tubes were newer. Same lux, same color, but they'd end up in the same six feet of bench by the end of a shift. Took me a while to work out why.



Corn

And the plants didn't care.



Hilbert

Plants don't have melanopsin.



Herman

You had a natural experiment. Identical measured light, different subjective effect, and the people self-corrected without being able to explain it.



Hilbert

I had four of those ballasts in my garage for years. Never got around to them.



Corn

Four.



Hilbert

Four. Anyway, the level on your second mic is running about three decibels hot. I'll fix it in the edit.



Herman

The point he's making is real, though. It connects to the individual-sensitivity question. If the response depends on the quality of the light and not just the quantity, then two people under identical measured conditions can have completely different outcomes. Which is exactly what the melanopsin-variant hypothesis would predict.



Corn

It also explains why the latitude data is messy. Florida at four percent. Maryland above New York. If it were purely about photon count, those numbers would line up in a straight line.



Herman

They don't. Because the photon count isn't the whole variable.



Corn

Where does that leave Daniel's question? Why does depression have such a strong impact on energy?



Herman

The honest answer is that there isn't one mechanism. There are at least three, and they overlap. Light and circadian timing. Reward and effort circuitry. Inflammation and sickness behavior. And the reason the connection isn't obvious is that the folk model only has one of them, which is "you feel sad, so you don't do things."



Corn

The folk model has the causality backwards.



Herman

It does. The energy drain isn't a consequence of the sadness. In a lot of cases it's upstream of it. The sickness behavior program produces the lethargy and the anhedonia together, as one package. The low mood is part of the shutdown, not the cause of it.



Corn

Which is why telling a depressed person to push through it doesn't work. You're asking them to override a program that their immune system is actively running.



Herman

It's why the "just get some sunlight" advice fails for non-seasonal depression. You're applying the seasonal fix to a non-seasonal problem.



Corn

What's still unresolved?



Herman

Most of it. There's no single definitive cause of SAD established. The serotonin, melatonin, vitamin D, retinal-sensitivity, and phase-shift hypotheses are all live, and they may all be partly true at once. The vitamin D evidence specifically is mixed. Some studies find it as effective as light therapy, others find no effect at all.



Corn

And summer-pattern SAD?



Herman

Under-researched. Less is known about it, and more research is needed. Which is odd, because it's the natural control group. If the mechanism is "not enough light," summer-pattern SAD shouldn't exist.



Corn

The mechanistic fork, clock versus direct?



Herman

Unresolved. The Hattar review explicitly presents the alternative model that dispenses with the causality assumption. Nobody has closed that question.



Corn

Here's the thing I keep circling, and it's what Hilbert's flicker story actually points at. If individual sensitivity varies that much, and the quality of light matters as much as the quantity, then the line between "this person has a disorder" and "this person is responding normally to a bad environment" gets very thin.



Herman

The Sami point again.



Corn

If a culture can embrace the seasonal slowdown and not call it illness, then some fraction of what we're measuring as pathology might be a mismatch between an old program and a modern expectation.



Herman

Year-round high energy as the healthy norm. Which is a recent assumption, historically.



Corn

Very recent. And possibly wrong.



Herman

The forward-looking version of that is interesting, though. If the shared pathway really is energy conservation, and inflammation is one of the roads into it, then the next generation of treatments might not be about light at all. They might target inflammation directly, or target the reward circuitry, or both.



Corn

Treating the shutdown program instead of the mood on top of it.



Herman

Treating the mechanism rather than the symptom. And the reason that's plausible is that we now have a candidate mechanism. Twenty years ago, "depression drains energy" was just an observation. Now it's a set of pathways you can point at.



Corn

Which is progress, even if none of the questions are closed.



Herman

Especially because none of the questions are closed.



Corn

That's the episode, then. No single cause, several live theories, and a mechanism that looks less like a malfunction and more like an old program that won't switch off.



Herman

Thanks to Hilbert Flumingtop for producing.



Corn

This has been My Weird Prompts. If you enjoyed it, leave us a review on whatever platform you're listening on. It helps.



Herman

We'll be back soon.



Corn

See you tomorrow.