Episode Book

EPISODE #6646

How Magnetic Pulses Target Depression Circuits

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SYNOPSIS

Transcranial magnetic stimulation uses Faraday's law to induce electric currents in the cortex, aiming at a prefrontal spot anticorrelated with the subgenual cingulate. Rather than simply exciting neurons, TMS reshapes directional network signaling and synaptic plasticity—converging on the same molecular door as ketamine and ECT. Yet the clinical evidence lags behind the mechanism: randomized trials show 24% response versus 12% for sham, even as three-minute theta-burst sessions and accelerated protocols like Stanford's SAINT reshape treatment.

TRANSCRIPT



Corn

Five days. A coil held against your scalp, a clicking sound for a few minutes at a time, and by the end of the week your depression scores cut in half. That's the brochure version of where transcranial magnetic stimulation has gotten to, and Daniel wants the version behind the brochure. What is a magnetic pulse physically doing to brain circuits, why would that lift mood, and how strong is the evidence really for the new protocols — theta-burst, and the accelerated courses like Stanford's SAINT.



Herman

And this is the right week to ask, honestly, because the gap between what regulators are approving and what anyone can prove is wider right now than it's ever been in this field.



Corn

Then let's start with the physics, because the physics is the one part that's actually settled.



Herman

It is, and it's beautiful. The stimulator drives a fast-changing current through a coil sitting against the scalp. That current makes a magnetic field, the field passes straight through the skull — bone is transparent to it — and inside the cortex it induces a secondary electric current. That current depolarizes neurons. It's Faraday's law applied to the brain. The magnet isn't doing anything mystical; it's just the delivery vehicle for an electric current you can't otherwise aim through the skull.



Corn

So it's a transcranial battery.



Herman

It's a transcranial battery with astonishingly good aim in one dimension, actually — the induced current is strongest in the cortex right under the coil, a couple of centimeters down. Deeper than that and it falls off fast. Which is why where you put the coil is nearly the whole question, and that's where the story stops being physics and starts being psychiatry.



Corn

Walk me through it, because the standard target has a history, doesn't it.



Herman

It does, and the history is the interesting part. The target is the left dorsolateral prefrontal cortex — DLPFC if you want the acronym. And the reason it's the target is not that somebody worked out causally that perturbing that spot lifts mood. It's that in the early neuroimaging studies of depression, in the nineties, that region looked hypometabolic — quiet, underactive on the scans. Downar's group in Toronto put this plainly in their twenty twenty-four mechanism review: the DLPFC target "emerged from early neuroimaging studies in depression." A correlation, not a circuit map.



Corn

They aimed at the dim light on the scan.



Herman

That's exactly what they did. And the thing is, it kind of worked anyway. Which is either luck or a hint that the dim spot was a decent doorway into something bigger.



Corn

And the something bigger is what changed.



Herman

The whole framing changed, yes. The newer work — lesion mapping, focal stimulation mapping, where you look at what happens to depression symptoms when specific brain areas are damaged or stimulated — reveals not a spot but whole-brain networks. The target isn't a point, it's a web, and the best point in the prefrontal cortex to put your coil is defined by one specific property: it's the spot most functionally anticorrelated with the subgenual anterior cingulate cortex.



Corn

Say the anticorrelation part in plainer words, because that's carrying a lot.



Herman

The subgenual cingulate is a region deep in the midline that runs hot in depression — overactive, metabolically loud. Two regions are functionally anticorrelated when activity in one goes up while the other goes down, like a seesaw. So you want to stimulate the prefrontal spot that pushes down hardest on the subgenual cingulate. Stimulate the top of the seesaw, quiet the bottom. That's how Stanford Neuromodulation Therapy picks its target for each patient individually, using resting-state functional MRI — they find your personal best anticorrelated spot, because it's in a slightly different place in everyone.



Corn

Which explains the old clinic method I've read about — measuring five and a half centimeters forward from a scalp landmark and putting the coil there.



Herman

The five point five centimeter rule. And the MRI-guided version, and the scalp-measured version, are both sold as TMS — same word, same device category, and the results are not the same. Hold onto that, it's the thread through the whole evidence story later.



Corn

So the pulse goes in at the top of the seesaw. What's the actual therapeutic effect — is the coil exciting that patch of cortex like a muscle?



Herman

That's the misconception, and it's the one I most want to kill. The classical framing is that high-frequency stimulation is "excitatory" and low-frequency is "inhibitory," as though you're turning a dial up or down on a spot. Downar's review takes that apart directly. The actual effects in individuals are quite variable, and the better way to think about it is at the level of populations of synapses rather than individual synapses. What improvement looks like is not a juiced-up patch of cortex — it's a change in directional signaling between the DLPFC, the subgenual and dorsal anterior cingulate, and salience-network regions. You're changing the conversation between regions, not the volume of one of them.



Corn

The dial metaphor is a seesaw metaphor.



Herman

Worse than that, it's a misleading one, because it predicts things that don't happen. If it were simple excitation, more should always be better — turn it up, excite harder. And there's a built-in reason it isn't. The same review proposes that synaptic meta-plasticity — the synapses adjusting their own thresholds in response to what's been happening to them — acts as, quote, "a built-in protective mechanism to avoid runaway facilitation or inhibition during treatment." The brain pushes back. It has a governor on the plasticity you're trying to induce.



Corn

Which might explain the patients who get worse instead of better.



Herman

Downar's group suggests exactly that — the same protective governor that stops runaway facilitation in most people may be what tips a small number the other way. It's a hypothesis, not settled, but it's the kind of explanation that changes how you'd think about dosing. If the brain is defending a set point, then timing and rhythm might matter more than raw total.



Corn

Now — the durability question. The benefit persists for some people for months. What's persisting? You can't hold a magnetic field in place after the machine stops.



Herman

The synaptic story. The persistence of benefit seems to depend on actual structural change at synapses, and Downar's review names two molecular dependencies: dopamine acting at the D1 receptor family, and NMDA-receptor-dependent synaptic plasticity. Those are the same molecular players you'd recognize from an entirely different conversation.



Corn

Ketamine.



Herman

Ketamine, and ECT too. There was a Nature Reviews Neuroscience paper in twenty twenty-five — Liao and colleagues — framing impaired prefrontal excitatory synapses as a core feature of depression, and arguing that ketamine, ECT and non-invasive stimulation all "seem to converge on enhancement of neural plasticity." Fifteen years of preclinical work showing ketamine triggers dendritic spine growth on cortical pyramidal neurons, and rTMS looks like a candidate for the same synaptic remodeling, just slower to read out.



Corn

So the coil, the infusion, and the seizure are all knocking on the same door.



Herman

Different fists, same door. And to me that's the most underreported thing in this whole field — it reframes the question. It stops being "device versus drug" and becomes "why does the same plasticity machinery answer such different knocks, and why does it refuse to answer for some people?"



Corn

You did get unreasonably excited about the door metaphor, Herman.



Herman

I stand by the door. When three interventions with nothing physically in common converge on one mechanism, that mechanism is probably the real thing. It's when they diverge that I'd start doubting.



Corn

Fair. Where I get stuck is the causal chain from "synapses got more plastic" to "mood lifted for four months." Plasticity is capacity, not content. What did the brain relearn?



Herman

Nobody can fully answer that, and I'll say so plainly. The honest framing is that the stimulation perturbs a circuit whose healthy configuration has been lost, the perturbation reopens a window for it to reconfigure, and what gets reconfigured depends on what the patient does, thinks, and experiences during and after the window. Which is why the same pulse can lift one person and do nothing for another.



Hilbert

Medieval Pottage Facts. Most peasants ate meat only as a little bacon in the pottage. That was the meat. It was a small piece of bacon and a long year.



Corn

Put that on a motivational poster.



Herman

The thing is, the mechanism story is the elegant half, and I think we should say that out loud before we move to the messier half. Physics settled. Target reframed from a spot to a network, with a principled way to find the entry point. Effect understood as directional signaling change with molecular fingerprints we can name. Converging with two other treatments on a shared mechanism. It's good science.



Corn

It's the brochure's favorite half too, which is my problem with it. The mechanism can be elegant and the evidence can still be thin, and the field has a habit of letting the first vouch for the second.



Herman

Then let's go look at the evidence, because it's more interesting than either the enthusiasts or the debunkers say. Start with conventional TMS — the old six-week, once-daily course. The tri-society consensus, three professional societies reviewing something like two thousand four hundred abstracts, concluded it continues to demonstrate broad evidence for safety and efficacy, and that the newer forms are faster and potentially more effective. That's the calibrated positive. The counterweight is the encyclopedia summary, which is blunt: evidence quality is variable, effect sizes are modest, placebo responses are large.



Corn

Give me the placebo problem in a number.



Herman

Across randomized trials, response rates run about twenty-four percent for real TMS versus twelve percent for placebo. Double — which is a real effect, and also means three quarters of the people who improved on the real thing might have structure in that number they can't see. Meanwhile real-world cohorts, no sham control, report response up to fifty-eight percent. The gap between those two figures is the placebo problem running in both directions — sham trials deflate, open treatment inflates, and the truth sits somewhere in the middle of a fog.



Corn

The rare patient who gets better on the clicking box with nothing in it.



Herman

The clicking box is not nothing, that's the cruel part. It's a convincingly dramatic ritual — a machine, a technician, a chair, a course of treatment. Perfect placebo delivery hardware.



Corn

A placebo with a faraday cage of intent.



Herman

Now, theta-burst — this is where the field changed speed. Theta-burst isn't a device, it's a firing pattern. Intermittent theta-burst fires little bursts of three pulses at fifty hertz, five bursts per second for two seconds, then rests for eight. And a standard session of that is six hundred pulses in about three minutes. A conventional ten-hertz session takes thirty-seven and a half minutes for its dose.



Corn

Three minutes versus thirty-seven.



Herman

And that ratio is the entire unlock, because the old barrier to accelerating TMS was arithmetic. Ten sessions of thirty-seven minutes in one day is over six hours of actual stimulation — nobody was doing that. Ten sessions of ten minutes, with breaks, fits inside a clinic day. Theta-burst made acceleration physically possible before anyone proved acceleration worked.



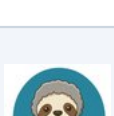
Corn

Did three minutes work as well as thirty-seven?



Herman

THREE-D, published in the Lancet in twenty eighteen — four hundred and fourteen adults with treatment-resistant depression across three Canadian university hospitals, ten-hertz versus theta-burst, both once daily. Hamilton depression scores fell from about twenty-three and a half to thirteen and a half in both groups, and theta-burst was judged non-inferior. Headache was the most common side effect in both arms at about sixty-four, sixty-five percent, and patients rated theta-burst slightly more uncomfortable — three point eight versus three point four out of ten.



Corn

Same result, a tenth of the chair time, marginally more annoying. I'd take that trade.



Herman

Most people would. So then the obvious next question — if the session is three minutes, what happens if you do ten a day for five days? That's Stanford Neuromodulation Therapy. The randomized trial, Cole and colleagues, American Journal of Psychiatry, twenty twenty-two. Thirty-two people enrolled, twenty-nine randomized — fourteen active, fifteen sham. Ten theta-burst sessions a day, five consecutive days, target individually located by fMRI as the spot most anticorrelated with the subgenual cingulate. Stopped early at interim analysis because the effect size was so large they considered it unethical to keep randomizing people to sham.



Corn

Stopped for working too well is not a sentence you hear in this literature often. What were the numbers?



Herman

Mean reduction on the MADRS depression scale at four weeks: fifty-two and a half percent active versus eleven percent sham. Over the four-week follow-up, seventy-eight point six percent of the active group hit remission criteria at least once, versus thirteen percent of sham. Response was eighty-six versus twenty-seven. Those are numbers psychiatry basically doesn't see in depression trials.



Corn

Fourteen active patients.



Herman

That's the footnote that matters, and it gets flattened in every retelling. And before that randomized trial there was an open-label version — no sham, everyone knows they're getting treated — with twenty-two patients, and nineteen of twenty-one completers remitted. Ninety percent. That ninety percent is the number that traveled in press coverage, the one in the clinic marketing, the one people ask their psychiatrist for by name.



Corn

Ninety percent is a billboard. Seventy-eight point six is a study.



Herman

And the newest, best summary is the July twenty twenty-six -analysis in Brain Stimulation — fifteen randomized sham-controlled trials, seven hundred and four adults, accelerated theta-burst versus sham. Risk ratio of about two for response, about one point eight for remission. In plain terms, roughly one extra responder for every five people treated, one extra remission for every ten. Dropout the same as sham, which tells you people can actually tolerate these marathon days. And two protocol details tracked with better outcomes: more total sessions and longer gaps between them, and MRI connectivity targeting rather than scalp landmarks.



Corn

Which is the targeting argument showing up in the statistics. It's the seesaw again — the protocols that actually find the top of the seesaw do better.



Herman

Now the other side of that coin, and it's the study I'd want anyone considering this to know about. St. Michael's Hospital in Toronto, twenty twenty-six: forty-eight adults, seven theta-burst sessions a day for five days — a real accelerated protocol — but targeted with scalp measurements, the Beam F3 method, no MRI. Day-five response rate: sixteen point seven percent. Scores kept falling over the following two weeks, so it's not a clean failure, but still.



Corn

Sixteen point seven in Toronto, ninety on the billboard. Both labeled the same treatment.



Herman

Same label, different medicine. Different targeting, different session counts, different measurement timing — and the label hides all of it. That's my core problem with how this is being sold. "Accelerated TMS" is not one thing; the protocols differ in sessions per day, gap length, days, pulses per session, and targeting method, and a twenty twenty-four review by Emory and Brown psychiatrists said outright that accelerated protocols "have not been standardized." The evidence for one schedule does not transfer to another



Herman (continued)

schedule just because both fit under the same word.



Herman

And the family has kept growing since. UT Southwestern got clearance this year for their Triple TBS protocol — three six-minute sessions per visit with thirty-minute breaks, five days a week for three weeks. Cuts a six-week course to about three. Different again from Stanford's five-day version. Reasonable middle path, honestly, if you live near the clinic.



Corn

Corn, quick check — you've been quiet on the regulators. Where do you land on the clearances?



Corn

That's the part I actually came here to dislike. BrainsWay got an accelerated clearance on a hundred and four patients. Response at six weeks: eighty-seven point eight percent accelerated versus eighty-seven point five daily.



Herman

A dead heat.



Corn

On remission, accelerated was worse — seventy-eight percent against eighty-seven and a half. And headaches ran forty-one percent against fifteen. So the honest summary of the trial behind the clearance is: same response, less remission, nearly triple the headache, done faster.



Herman

Which is a defensible trade for some patients — someone who can't do six weeks of daily visits, someone who lives two hours from a clinic. Faster is worth something real. What it is not, is evidence that accelerated is better, and that's how it keeps getting described.



Corn

Then there's MagVenture, this year. Their expanded clearance covers two to ten sessions a day of either protocol, with or without MRI navigation. Their own announcement gives no trial number, no sample size, and stresses that nothing about the hardware changes.



Herman

The "nothing changes" line is what gets me. The coil is identical. The pulse is identical. The only thing that changed is the paperwork permitting a schedule range wide enough to contain both the Stanford protocol and things nobody has tested. The clearance spans two to ten sessions a day, and a schedule inside that span may have no evidence behind it at all.



Corn

"Two to ten" is not a dose. It's a corridor.



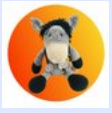
Herman

And the safety numbers are actually reassuring within tested protocols — seizure and fainting run about zero point one percent, usually administration error, headache is the common complaint. The soft spot the field itself names, over and over, is durability. The twenty twenty-four Biological Psychiatry review of accelerated protocols concludes the data support safety and effectiveness "while acknowledging the need for more durability data." Nobody has established how long any of this lasts beyond a few months. Not the five-day miracle, not the six-week classic.



Corn

Which leads us to the part patients actually run into — the insurance letter.



Herman

Aetna's policy bulletin, reviewed this July, covers standard daily TMS and theta-burst on cleared devices when criteria are met, and lists accelerated TMS as experimental and unproven. I don't think that's entirely irrational, by the way.



Corn

You don't?



Herman

No. The insurers are tracking the same thing we just laid out — a clearance that outran its trials, protocols that aren't standardized, durability that nobody's measured. "Experimental" is the wrong word for some of it, but the skepticism lands closer to the evidence than the marketing does. The cruel version is the patient in the middle: a clinic offering a five-day course the FDA has cleared, and an insurer calling it unproven, and no head-to-head trial big enough to settle who's right.



Corn

The head-to-head gap is the scandal nobody's angry about. Fifteen sham-controlled trials, and almost nobody comparing accelerated head to head against the standard course at scale, which is the comparison every patient actually needs.



Herman

Because the sham trials are easier to run and easier to publish. Big comparative trials are expensive and don't clear a device any faster. The incentives point away from the question that matters.



Hilbert

The MagVenture coil. About forty thousand shekels, the one with the cooling. My brother-in-law's clinic has two, second one bought in the spring, and that is what the argument with the supplier has been about since August.



Corn

The supplier argument.



Hilbert

The supplier, Yaakov, at the place off Jaffa Road. I have dealt with him for eleven years, since long before any of this. The clinic ordered the second machine with the German headrests and he delivered the Israeli ones, and he maintains they are the same headrest. They are not the same headrest. I took my brother-in-law to five sessions a week for six weeks, so I have sat in that waiting room longer than anyone should, and I know exactly what the head of the man in the next chair does after minute twenty of the Israeli one. I keep the delivery note in the glovebox. Corn will appreciate this — you spent forty minutes on the word anticorrelated earlier, and the supplier's position is that the two headrests are also anticorrelated. One holds the head, one argues with it.



Corn

The German one wins on comfort, then.



Hilbert

It wins on the neck, which is what you notice when the machine is going. I call the coil the woodpecker. Everyone at the clinic calls it the woodpecker now, including the technician, who got it from me. The sessions are three minutes, and I counted the clicks in the first week because there was nothing else to do — six hundred, and the counter on the machine said six hundred and two, which nobody at the supplier would explain to me either. The earplugs they give out are made for human ears. On me they sit on the shelf of the snout and fall out, so I bring my own, and I have a tin of them in the car, Herman, if your clinic friends ever want to know how it is actually done.



Herman

Six hundred and two — that's probably the coil's self-test pulses at the start, before the session counter properly begins.



Hilbert

That is a better answer than Yaakov gave, which was no answer and a coffee. Fine. Six hundred and two it is. The brother-in-law is still on the daily protocol, six weeks, not the five-day one — the insurer wrote that it was experimental, and I will add that the letter arrived in under a week, which is the fastest anything has moved in that building. I park facing the door of the clinic so he can see the car from the chair. That is all I came to say.



Corn

Under a week for the refusal. The machine that clears in a corridor of two to ten sessions a day, and the rejection letter that beats it out the door.



Herman

The bureaucracy is faster than the physics, which is not a sentence I ever expected to defend. Though — the headrest dispute aside, Hilbert's brother-in-law on standard daily TMS with family driving him there five days a week is actually the best-supported version of this whole story. The boring protocol is the one with the six weeks of trials behind it.



Corn

The cutting-room floor, then, one detail we didn't get to. The earlier open-label Stanford study — the one behind the ninety percent figure — nineteen of twenty-one completers remitted. But the word "completers" is carrying weight: in a five-day intensive protocol, the people who drop out are the ones it isn't working for or can't tolerate, and they vanish from the numerator and the denominator both. The randomized trial kept them and got seventy-eight point six. Same protocol, same building, and the dropout accounting is a third of the difference between a headline and a result.



Herman

And that's before you add the interim stop — thirty-two enrolled, twenty-nine randomized. Every one of those small print choices moves the number in the flattering direction. Not dishonest. Just flattering.



Corn

The open question I'd leave with Daniel is durability, because everything else in this field has an answer or an argument behind it, and that one has a shrug. Nothing establishes how long any of this lasts past a few months — not the five-day protocol, not the six-week one. And if the shared mechanism with ketamine and ECT is real — the same plasticity machinery answering through the same D1 and NMDA receptors — then the interesting question stops being which treatment to pick and becomes why some brains hold the remodeling and others lose it by spring. That's the study the whole field is waiting for someone to fund.



Herman

And a vote for the show on Spotify, or wherever you get your podcasts, helps more people find these rabbit holes. Our producer is Hilbert Flumingtop, and If you enjoyed this one, try episode twenty-five twenty-seven, Do Brain Changes from Therapy or Pills Actually Last; episode twenty-five twenty-nine, Depression Subtypes; and episode four hundred, Rebooting the Brain. this has been My Weird Prompts. We'll be back soon.



Corn

See you tomorrow, everyone.

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