

Hypomania

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THE STORY IN ONE SENTENCE

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“Same circuit as mania, turned up halfway: the person keeps working and stays out of the hospital, but the mood, sleep, and spending have clearly changed.”

Normal anatomy — the body's job

In a healthy brain, the prefrontal cortex (the front part that plans, judges risk, and applies the brakes) balances the reward circuit (the ventral striatum, driven by dopamine — the brain's go-get-it messenger) and the sleep-wake clock in the suprachiasmatic nucleus of the hypothalamus (the tiny timer deep in the brain that sets when you sleep and wake). Mood, energy, and sleep need stay inside a steady range from week to week.

BODY'S JOB

Keep drive, confidence, and sleep matched to real life — enough dopamine to chase goals, enough braking from the prefrontal cortex to stop at the right time, enough sleep to reset the system every night.

1. The prefrontal cortex sets the plan and applies the brakes on impulses and big ideas
2. Dopamine in the ventral striatum (the reward center deep in the brain) supplies drive and confidence when you chase a goal
3. Norepinephrine (the brain's alertness messenger) sets arousal — how awake and fast the system runs
4. The suprachiasmatic nucleus in the hypothalamus enforces the nightly sleep reset, keeping a steady 24-hour rhythm
5. Balanced loop: drive goes up for a goal, then settles back to baseline when the goal is done or sleep arrives

What breaks

The reward and arousal circuits run warm for days at a time. Dopamine (the brain's go-get-it messenger) and norepinephrine (the alertness chemical) tone rise, sleep need drops, and thoughts speed up — but the prefrontal brakes stay partly online. That's the key difference from mania: judgment bends, it doesn't break. No psychosis (losing touch with reality), no hospital-level danger, and daily life keeps going.

CLINICAL TERM

Hypomanic episode — at least 4 consecutive days of abnormally elevated, expansive, or irritable mood with increased energy, plus 3 or more manic-type symptoms (4 if the mood is only irritable), observable by others, WITHOUT marked functional impairment, psychosis, or need for hospitalization (DSM-5-TR).

How the body compensates

The person and the people around them often absorb the episode without naming it. Productivity may even rise at first, which is exactly why hypomania gets missed or is remembered fondly as 'my best week.' The person channels the extra energy into work, projects, exercise, or socializing, so life looks better than fine — until it suddenly isn't.

1. Sleep drops to 3–5 hours a night but the person feels rested — the system runs on raised norepinephrine (alertness messenger) tone, so fatigue doesn't register
2. Extra energy is channeled into projects, work, exercise, cleaning, or socializing, so life looks 'better than fine' — bosses, friends, and family may even praise the burst of productivity
3. Family and close friends notice the change ('you were talking so fast,' 'you started three new things in one day') even when the person doesn't see it as a problem
4. The episode ends on its own within days to weeks — often sliding into a depression that is what finally brings the person to care, and the earlier high gets reported only when the doctor asks the right questions

Domino effect

1. Elevated dopamine and norepinephrine tone raises drive, confidence, and speech speed — The ventral striatum (the reward circuit deep in the brain) fires more easily, so every idea feels excellent and worth starting right now. Norepinephrine cranks up alertness, so thoughts race and words tumble out fast.

2. Sleep need drops for 4 or more days in a row — The suprachiasmatic nucleus (the brain's sleep-wake timer in the hypothalamus) is destabilized by the high norepinephrine and dopamine — and every short night pushes the mood a step higher, because sleep loss itself boosts arousal.
3. Spending, plans, and social contact increase — Warm reward circuit plus softened (not absent) prefrontal braking bends judgment toward optimism and risk. The person sees opportunity everywhere, says yes to invitations, buys things on impulse, and starts projects without weighing the cost.
4. The episode resolves — or escalates — Left alone it usually settles within days to weeks as neurotransmitter tone drifts back to baseline. But stimulants (caffeine, ADHD meds, cocaine), alcohol, shift work, or an antidepressant taken alone (without a mood stabilizer) can push the same circuit into full mania.
5. Depression follows — Bipolar II depression is where most of the illness — and most of the suicide risk — actually lives. After the high burns out, dopamine and norepinephrine tone often crash below baseline, and the person sinks into weeks or months of low mood, no energy, and dark thoughts. The hypomanic episode is the diagnostic key that tells the doctor this isn't simple depression — it's Bipolar II, and antidepressants alone can make it worse.

Symptoms · what the patient feels, and why

- Sleeping much less but not feeling tired: Norepinephrine (a brain chemical that keeps you alert and awake) is running higher than usual, so it blocks the normal signal that tells your body it needs rest. The brain feels charged and ready even after only four or five hours of sleep, like a battery that thinks it is still full when it should be halfway drained.
- Talking much more and much faster than usual: The brain is making thoughts faster than normal, and the prefrontal cortex (the front part of the brain that usually slows you down and helps you take turns in conversation) is not putting the brakes on as strongly. Words spill out quickly because the thinking engine is revved up.
- Feeling unusually confident and optimistic: Dopamine (the brain's reward messenger) is more active, so it stamps more ideas and plans with a 'this is great' feeling than it normally would. The person feels certain that things will work out, even plans that might seem risky or unrealistic. This confidence stays short of the fixed, unshakeable grandiose beliefs you see in full mania.
- Starting many new projects, spending more money, reaching out to more people: The brain's reward circuit—centered in the nucleus accumbens and ventral tegmental area (deep centers that drive motivation and pleasure)—is turned up, so every goal feels exciting and urgent. The person throws energy into new hobbies, shopping, travel plans, or social events because the brain is tagging all of them as high-priority and rewarding.

- Getting irritated or snappy when things do not go as planned: The sped-up system does not tolerate waiting or obstacles well. Some people feel euphoric during hypomania, but others run irritable instead—short-tempered, impatient, easily frustrated. This irritable mood is just as real a sign of hypomania as the happy, energized mood, and it is often mistaken for stress or anxiety instead of recognized as part of the mood episode.
- Racing thoughts or jumping quickly from idea to idea: The flow of thoughts speeds up because dopamine and norepinephrine are both elevated. Ideas arrive faster than the person can finish one, so the mind feels crowded and jumpy. This is different from the distractibility of ADHD because it comes in clear episodes and pairs with the other mood and energy changes of hypomania.
- Seeing or hearing things that are not there, or believing things that are clearly not true: When dopamine activity climbs high enough that the brain starts misinterpreting reality—creating false perceptions (hallucinations) or fixed false beliefs (delusions)—the episode is no longer hypomania. Psychosis means the episode has crossed into full mania, which carries higher risk and requires immediate medication adjustment and close monitoring.
- Mood or behavior so extreme that work, school, or relationships fall apart: Hypomania by definition does not cause marked impairment—the person can still function, even if friends and family notice the change. If the mood becomes so intense that the person cannot do their job, damages important relationships, or needs hospitalization, the episode has escalated to mania. That reclassification changes treatment right away.

Focused exam

- Fast speech that you can interrupt, a bright or snappy mood: When you can interrupt the fast talking and the person still knows what is real, that points to hypomania. If you cannot interrupt them and they believe things that are not true (like thinking they have superpowers), that is mania—a bigger emergency.
- The person knows where they are, what day it is, and is not seeing or hearing things that are not there: If someone sees or hears things that are not real, or believes impossible ideas (psychosis), it is not hypomania anymore—it is mania. That changes the plan to same-day safety help.
- Wide pupils, fast heartbeat, high blood pressure: Drugs like cocaine or methamphetamine can look exactly like hypomania. These body signs plus a drug test can tell the difference—one is a brain condition, the other is the drug.
- Shaky hands, hating heat, fast heartbeat, eyelids that lag when looking down: An overactive thyroid gland (hyperthyroidism) can copy every sign of hypomania. A thyroid blood test (TSH) is required before calling it a mood condition, because treating the thyroid can fix the whole picture.

- No need for sleep but still full of energy after only 3 or 4 hours: Needing less sleep without feeling tired is one of the clearest hypomania signs. Ordinary tiredness with insomnia feels different—people want to sleep but cannot. In hypomania, the brain does not ask for sleep at all.
- New risky choices—spending sprees, sudden road trips, big bets, sexual choices the person would not usually make: The prefrontal cortex (the front of the brain that pumps the brakes on impulses) gets overridden by the revved-up limbic system (the emotional engine). Risky acts that feel exciting in the moment but bring regret later are a red flag that the mood has tipped too high.

Diagnostics · every test answers a question

- Thyroid-stimulating hormone (TSH) → Is an overactive thyroid gland making the mood symptoms, or making them worse?
- Urine drug screen (amphetamines, cocaine, THC) → Are stimulant drugs causing the high energy, fast thoughts, and risky behavior?
- Mood Disorder Questionnaire (MDQ) plus talking to someone who knows the person well → Has this person had mood highs before, even if they were never diagnosed?
- Full medication and supplement list → Did a medicine the person is taking flip the mood upward?
- Pregnancy test (urine hCG) before starting lithium or valproate → Is the person pregnant, which would make certain mood stabilizers unsafe?
- Collateral sleep and activity history over the past week → How many hours is the person actually sleeping, and has anyone else noticed the change?
- Complete blood count (CBC), comprehensive metabolic panel (CMP), and baseline lipid panel if starting quetiapine → Are the kidneys, liver, and blood counts normal before starting medicines that need monitoring?

Treatment logic

- Stop any antidepressant the person is taking without a mood stabilizer → Antidepressants (like sertraline, escitalopram, bupropion) alone can shove the mood upward in someone with bipolar wiring. Stopping it removes the push and prevents hypomania from climbing into full mania.
- Start a mood stabilizer: lithium (if the course is classic highs and lows), or lamotrigine (if depression is the bigger problem) → Lithium calms the manic circuit and is the only mood medicine proven to lower suicide risk. Lamotrigine stops the slide into depression and prevents future lows, though it does little for active highs. Both keep the mood from swinging too far in either direction.

- Use quetiapine when the episode needs faster control, sleep is broken, or depression and hypomania mix → Quetiapine blocks dopamine (a brain chemical that revs up pleasure and energy) and histamine (a wake signal), so it calms hypomanic symptoms, treats bipolar depression, and restores sleep within days. Sleep is the brake pedal—once it is back, the mood usually stops climbing.
- Protect sleep with a fixed sleep and wake time every day (social rhythm therapy) → The brain's circadian clock (the internal 24-hour timer run by the suprachiasmatic nucleus in the hypothalamus) sets mood stability. When sleep shifts—staying up late, sleeping in, or skipping nights—the clock loses its rhythm and the mood circuit destabilizes. A steady schedule is not just advice; it is a real treatment that prevents future episodes.
- Teach the person and their family the early warning signs to catch the next episode early → Most people have a personal pattern—sleeping less (down to 4 or 5 hours without feeling tired), talking faster, spending money in new ways, starting big projects, or feeling 'amazing' after weeks of being low. Writing these signs down while well creates a smoke alarm. When two or three signs show up, the person or family calls early, before the episode is full-blown.
- Avoid caffeine, energy drinks, and skipped meals → Caffeine is a stimulant that can pour gas on a rising mood. Skipping meals causes blood-sugar swings that destabilize the brain's energy supply. Steady fuel (regular meals) and no extra revving (cutting caffeine) help keep the baseline even.

Medications

Lithium · Mood stabilizer

Calms overactive signaling inside brain cells and steadies the mood circuit across both highs and lows. It is the only mood medicine repeatedly shown to lower the risk of suicide.

1. Enters brain cells through tiny gates called sodium channels
2. Turns down two overactive signal-boosting systems inside the cell — the inositol pathway and an enzyme called GSK-3 — like lowering the volume on a speaker
3. Over days to weeks, the mood circuit settles back toward a steady middle zone, preventing both highs and lows

SAFETY WATCH

Very narrow safe window — the blood level must stay between 0.6 and 1.2. Dehydration, ibuprofen or naproxen (NSAIDs), water pills (thiazides), and blood pressure medicines (ACE inhibitors) can push the level too high and cause shaking, confusion, or kidney trouble. · Check kidney function and thyroid at the start and then every 6–12 months — lithium can slow the thyroid over time. · If you might become pregnant, talk to your doctor first — lithium carries a small heart-defect risk in the first trimester, and you need a clear plan.

Lamotrigine · Mood stabilizer (antiepileptic)

Blocks sodium gates on brain cells and lowers the release of glutamate (the brain's main go signal) — best at preventing the depressive lows of bipolar disorder.

1. Slow dose climb over 6–8 weeks lets your immune system adjust safely
2. Blocking sodium gates keeps brain cells from firing too often
3. Less glutamate surge means the circuit stays out of the depressive dive
4. Once at the target dose, ongoing use prevents relapse into depression

SAFETY WATCH

Titrate slowly — doubling the dose too fast raises the risk of Stevens-Johnson syndrome, a life-threatening rash that peels the skin and lines the mouth and eyes. · Any spreading rash with fever, mouth sores, or eye pain = stop immediately and seek emergency care.

Quetiapine · Second-generation antipsychotic

Blocks dopamine and serotonin receptors — cools the overheated reward circuit, restores sleep, and treats bipolar depression too.

1. Blocking D2 receptors lowers the excess dopamine drive — the 'gas pedal' that fuels racing thoughts, big plans, and endless energy
2. Blocking serotonin 5-HT_{2A} receptors steadies mood from the top down
3. Histamine blockade brings sedation — rebuilding the sleep anchor the hypomanic episode destroyed
4. Continued dosing covers both poles: it treats bipolar depression and prevents manic or hypomanic swings

SAFETY WATCH

Metabolic monitoring — track weight, blood sugar, and cholesterol every 3-6 months because quetiapine can shift metabolism over time. · Orthostatic low blood pressure (hypotension) (dizziness when standing) and daytime grogginess are common early on — stand up slowly and avoid driving until you know how you feel.

Valproate (Divalproex) · Mood stabilizer (antiepileptic)

Boosts GABA (the brain's main brake signal) and blocks sodium channels — calms the overactive circuits driving mania and hypomania. Works faster than lithium.

1. Raises GABA levels by blocking the enzyme that breaks it down — turns up the brain's natural calm
2. Blocks sodium channels so brain cells fire less often
3. The overactive mood circuit slows down within days to a week
4. Ongoing use prevents both manic and depressive swings

SAFETY WATCH

Check liver enzymes and platelet count at baseline and during the first 6 months — valproate can stress the liver. · Causes serious birth defects (neural tube defects, lower IQ) — do NOT use if you are pregnant or might become pregnant unless every other option has failed and you have a clear plan with your doctor. · Weight gain and hair thinning are common — taking a zinc supplement may help the hair.

Carbamazepine · Mood stabilizer (antiepileptic)

Blocks sodium channels and calms overactive brain-cell firing — an older mood stabilizer still used when lithium or valproate do not work or cannot be tolerated.

1. Blocks sodium gates so brain cells cannot fire in rapid bursts
2. Mood circuit excitability drops over 1-2 weeks
3. Ongoing use prevents manic and hypomanic episodes

SAFETY WATCH

Check blood counts and liver function at baseline and regularly — carbamazepine can lower white cells and rarely cause a dangerous bone-marrow reaction (agranulocytosis) or liver injury. · Drug interactions are common because carbamazepine revs up liver enzymes — oral contraceptives, warfarin, and many psych meds drop to ineffective levels. Always check with your pharmacist. · Pregnancy planning required — carbamazepine carries neural-tube-defect risk.

Aripiprazole · Second-generation antipsychotic (partial dopamine agonist)

Acts like a dimmer switch on dopamine — turns down the signal when it is too high (mania) and nudges it up when it is too low, stabilizing the reward circuit without full blockade.

1. Partial agonist at D2 — it binds the receptor but only activates it partway, so if dopamine is flooding the synapse aripiprazole blocks the excess, but if dopamine is low aripiprazole gently raises the signal
2. This balancing act cools hypomania without causing the flat, numb feeling full D2 blockade can bring
3. Also modulates serotonin to steady mood and lower agitation

SAFETY WATCH

Metabolic monitoring — weight, glucose, and lipids, though aripiprazole is more weight-neutral than most antipsychotics. · Restlessness (akathisia) — an inner jitteriness that makes you pace — can happen early; lowering the dose or adding a beta-blocker often helps. · Rare but serious: uncontrolled movements (tardive dyskinesia) with long-term use — use the lowest dose that works.

Red flags

- Psychotic symptoms—seeing, hearing, or believing things that are not real—mean this is mania, not hypomania, and the person needs same-day psychiatric evaluation and medication adjustment
- Mood climbing higher despite efforts to protect sleep, or sleep dropping below three hours a night—the episode is escalating toward full mania
- Any suicidal thoughts or plans—people with Bipolar II face their highest suicide risk during depressive episodes and the switches between moods, so any suicidal thinking requires immediate safety assessment
- The person is taking an antidepressant without a mood stabilizer—antidepressants alone can flip a person with bipolar disorder into hypomania or mania, so the medication plan needs urgent review
- Reckless behavior the person cannot undo—spending large amounts of money they do not have, dangerous driving, risky sexual decisions—that causes real harm and cannot be walked back means the episode has crossed into impairing mania

Nursing priorities

1. Screen every 'depression' patient for past hypomanic episodes before antidepressants start — One missed 4-day episode rewrites the diagnosis to Bipolar II and makes antidepressant monotherapy unsafe.
2. Anchor and protect sleep — document actual hours, not just 'slept well' — Sleep is both the earliest warning sign and the most powerful non-drug treatment lever.
3. Bring family or collateral into the history whenever possible — The person often feels great and under-reports; observers supply the diagnostic evidence DSM requires.
4. Assess suicide risk at every mood switch, especially entering depression — The danger in Bipolar II is concentrated in the depressive phases that follow the highs.

Sources

- American Psychiatric Association DSM-5-TR (2022) — diagnostic criteria for hypomanic episode
- APA Practice Guideline for the Treatment of Patients with Bipolar Disorder (2022)
- CANMAT and ISBD 2018 guidelines for the management of patients with bipolar disorder
- National Institute of Mental Health (NIMH) Bipolar Disorder resource

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