
A CLINICAL WHITE PAPER • HORMONE THERAPY INSTITUTE

The 3 a.m. problem

What waking up drenched and wired is telling you about your hormones, your metabolism, and why four doctors could not explain it.

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NORMAL IS NOT THE SAME AS OPTIMAL

Start here

Ever wonder why you are always waking up around 3 a.m.?

Not tossing. Not stirring and drifting back off. Awake. Covers thrown, gown stuck to you, heart going, mind switched on like somebody flipped a breaker. You lie there and do the math on how many hours are left before the alarm, and by the time you stop doing the math, there are two.

I have been practicing obstetrics and gynecology for over twenty-six years and treating hormones specifically since 2008. In that time I have heard the same night described by thousands of women, in almost the same words. One of my patients told me it feels like someone lit a match to her. Another told me she sleeps three hours, wakes up, and is wired for the rest of the night. Another wakes at 3 a.m. every single morning and cannot get back down, so she gets up, and the day starts whether she is ready or not.

Every one of them had already been to a doctor. Most had been told the same three things: your labs are normal, this is a normal part of aging, try to manage your stress.

This paper is about what is actually happening in your body between 2 and 4 in the morning, why it does not stay in the bedroom, and what an honest medical evaluation of it looks like. I am going to show you the mechanism, and I am going to show you the research behind it, including the places where the research is weaker than the internet tells you it is. Normal does not mean you are healthy. It also does not mean I get to tell you things the evidence does not support.

You are not imagining it, and you are not the only one

There is no study that names a clock hour. I want to be precise about that up front, because you will see "the 3 a.m. wake-up" all over the internet attached to numbers that came from marketing surveys, not from research. The 3 a.m. detail is what I hear in the exam room. It is not a data point.

What the data do say is more useful anyway.

The federal government's own numbers, from the National Center for Health Statistics, break sleep down by menopausal status in women aged 40 to 59. Trouble *staying* asleep is more common than trouble falling asleep at every single stage, and it climbs the whole way through: 23.7 percent of premenopausal women report it four or more nights a week, 30.8 percent of perimenopausal women, and 35.9 percent of postmenopausal

women. Fifty-six percent of perimenopausal women are sleeping less than seven hours a night. Fifty-five percent of postmenopausal women do not wake up feeling rested four or more days a week [1].

The Study of Women's Health Across the Nation, the largest and longest-running study of the menopause transition in this country, found the same thing over eight years of follow-up. Frequent awakenings are the most common and the most menopause-specific sleep complaint there is [2].

So the pattern is real, it is measured, and it is specific. It is not that you cannot fall asleep. It is that you cannot stay asleep. Those are two different problems with two different causes, and if the person you are talking to treats them as one problem, you are about to be handed a sleeping pill for a hormone issue.

This is not a phase you wait out

The single most damaging thing a woman gets told in this window is that it will pass in a few months, so just get through it.

In the SWAN cohort, researchers followed 1,449 women with frequent hot flashes and night sweats and tracked how long the symptoms actually lasted. The median total duration was 7.4 years. For women whose symptoms started while they were still cycling or in early perimenopause, the median was more than 11.8 years. Symptoms persisted a median of 4.5 years past the final menstrual period. And the duration was not the same for everyone. Black women in the study had the longest median duration at 10.1 years, Hispanic women 8.9 years, white women 6.5 years, Chinese women 5.4 years, Japanese women 4.8 years [3].

Read those numbers again with your own calendar in mind. Seven years is a job change, a kid's graduation, a parent's illness, and a marriage carried on four hours of broken sleep a night. That is not a phase. That is a decade of your life, and the advice to wait it out is advice to spend it.

What is actually happening at 3 a.m.

Here is the mechanism, and I am going to build it in order, because the order is what makes it make sense.

Your hypothalamus is the temperature control center of your brain. It defends your core temperature inside a narrow band. Go above the top of that band and you sweat and

flush to dump heat. Go below the bottom and you shiver to make heat. Between those two lines is a zone where nothing has to happen, and in a woman with a normal estrogen supply that zone is comfortably wide.

Estrogen helps hold those lines apart. Specifically, estrogen restrains a cluster of neurons in the hypothalamus that signal through a chemical called neurokinin B. When estrogen falls, that restraint comes off. Those neurons enlarge and over-fire. In postmortem studies of human brain tissue, the neurokinin B neurons in postmenopausal women had grown to 194 percent of their premenopausal size [4]. The heat-defense relay downstream gets flooded with signal, and the comfortable zone collapses. In one of the foundational studies on this, the zone measured 0.0 degrees Celsius in symptomatic postmenopausal women, compared with 0.4 degrees in women without symptoms [5].

The analogy I use in the room is a house thermostat. A working thermostat has a dead band built into it. If it is set to 72, it does not fire the air conditioning at 72.1 and the heat at 71.9, because a house that did that would be running the AC and the furnace against each other all day and the system would burn itself out. Estrogen is what keeps the dead band in your thermostat. Take it away and the dead band goes to zero. Now a rise in core temperature so small that you would never have noticed it in your thirties crosses the line, and your brain calls a full emergency heat dump. Blood vessels open, sweat glands fire, adrenaline goes up. At 2 in the afternoon that is a hot flash. At 3 in the morning it is a soaked nightgown and a racing heart in the dark.

We know this pathway is real and not a theory because we can now block it and watch the symptoms fall. Two drugs that block the neurokinin B receptor have been through phase 3 trials and been approved by the FDA. Fezolinetant at the 45 mg dose reduced hot flash frequency by about 2.5 more episodes per day than placebo at twelve weeks, and by about 1.9 at the 30 mg dose [6]. Elinzanetant reduced it by about 3.2 more episodes per day than placebo [7]. When you can interrupt a specific receptor and the symptom drops, you are no longer guessing about the mechanism.

Are you picking up what I am putting down? Good, because here is the part almost nobody tells you.

The flash is not the whole reason you are awake. When researchers measured hot flashes objectively with skin sensors overnight in 168 midlife women, waking coincided with 78 percent of measured flashes, whether or not the woman remembered the flash. But flashes accounted for only about one third of her awakenings [8]. A second study using the same objective approach put the hot-flash-attributable share of nighttime wake time at about 27 percent [9].

So the flash and the awakening are not cause and effect in a straight line. They are two outputs of the same destabilized system. The same hormonal withdrawal that unsteadies your thermostat also unsteadies the machinery that holds sleep together. This matters more than it sounds like it does, because it explains something you may have already lived: women who get their hot flashes handled and still do not sleep. If the plan only chases the flash, the plan is treating a quarter of the problem.

Progesterone was doing more than anyone told you

Estrogen gets all the attention. Progesterone is the one that was putting you to sleep.

Progesterone is converted in the body to a neurosteroid called allopregnanolone, and allopregnanolone acts on the GABA-A receptor, which is the main braking system of the brain. It is the same receptor family that benzodiazepines and alcohol act on. When allopregnanolone binds, the brake gets more responsive. That is a sedative effect, and it is yours, made in your own body, every month, for thirty years [10].

Then perimenopause arrives, ovulation gets erratic, and progesterone production falls off before estrogen does. You did not lose a supplement. You lost your own sedative.

Now, the honest version of the treatment evidence. In a controlled crossover study of eight postmenopausal women, 300 mg of oral micronized progesterone at bedtime cut time spent awake after falling asleep by 53 percent and increased deep slow-wave sleep by about half [11]. Two caveats you deserve. It was eight women, and the effect showed up only on the night when their sleep was deliberately disturbed in the lab. Progesterone did nothing to their undisturbed sleep. It restored sleep that had been broken rather than deepening sleep that was already intact. In a systematic review of ten randomized trials, the four that could be pooled showed progesterone shortened the time it took to fall asleep, while the pooled effect on total sleep time and sleep efficiency was not statistically significant [12].

What I take from that, clinically, is that progesterone is a physiologic regulator, not a sedative you throw at a bad night. It works best in the woman whose sleep is disturbed because her progesterone is gone. That is a specific patient, and finding out whether you are her is the point of an actual evaluation.

Why the weight came on when the sleep went

This is the part women come to me about. Not the heat. The body they no longer recognize.

Almost every woman in my practice has said some version of the same sentence: I am doing exactly what I have always done and my body is not the same. She is usually told she must be eating more than she thinks. She is not.

Two separate things are happening, and both of them are documented.

The first thing is what broken sleep does to your metabolism. In a study at the University of Chicago, researchers took nine healthy young adults and used sound to suppress deep slow-wave sleep for three nights, without shortening total sleep time at all. Insulin sensitivity fell about 25 percent. Glucose tolerance fell about 23 percent. And the effect was dose-dependent: the more deep sleep they took away, the worse the insulin sensitivity got, with a correlation of 0.89 [13]. In a later study, researchers restricted seven young volunteers, six of them men, to four and a half hours in bed for four nights, then biopsied their fat tissue directly. It took nearly three times as much insulin to produce the same signaling response inside the fat cell [14].

I want to be straight with you about what those two studies are. They are small, they are short, and they were done in young adults rather than in women your age. Nobody has run that experiment in perimenopausal women, which is a gap in the field and not a detail I am going to paper over. What they establish is the mechanism: broken sleep degrades insulin signaling in healthy people within days, at the level of the fat cell itself. The mechanism does not switch off at 50.

Insulin is a fat-storage hormone. It is not a fat-releasing hormone. When your cells stop responding to it, your pancreas puts out more, and you spend your days in the physiologic state that stores fuel rather than spends it. That is why the effort stops paying. It is not willpower. It is signal.

Sleep loss also moves your appetite. Pooled across eleven controlled studies, partial sleep deprivation increased next-day energy intake by 385 calories per day, with no matching increase in energy expenditure [15]. Nobody makes their best food decisions on four hours of sleep. Now you know it is not just decisions.

The second thing is what the menopause transition itself does to where your body puts fat. This is the finding I wish every woman in America had seen. In SWAN, researchers followed body composition with imaging, not a bathroom scale, across the transition. Fat mass accrual roughly doubled during the transition window, from about 1 percent a year beforehand to 1.7 percent a year, and lean mass, your muscle, went from slightly gaining to actively declining [16]. Then they looked at where the fat went. Visceral fat, the deep abdominal fat around your organs, was flat or slightly falling before the transition and then climbed 6.24 percent a year during it [17].

And here is the detail that explains the gaslighting. In that same study, waist circumference rose in a straight line the whole time, with no acceleration at the transition at all. The tape measure does not show it. The scale barely shows it. Your clothes show it, your reflection shows it, and your labs eventually show it, and in the meantime you get told your weight is stable so nothing is wrong.

Your body did not fail you and you did not get lazy. Your body changed where it stores fuel, and your nights stopped letting you clear it.

One honest caveat, because you deserve the real version and not the version that sells better. You will read everywhere that 3 a.m. waking spikes your cortisol and that cortisol is what is putting fat on your belly. The first half has experimental support: sleep loss raises next-evening cortisol by 37 to 45 percent in controlled studies [18]. The second half is weaker than it is presented. In the best long-term study of cortisol across the menopause transition in real midlife women, sleep symptoms were not a significant predictor of overnight cortisol at all [19]. And across a population sample of more than 2,500 adults, long-term cortisol correlated with body weight at about $r = 0.10$, which means it explains roughly one percent of the variation [20]. Cortisol is part of the story. It is not the villain the internet has made it, and anyone who builds your whole treatment plan on it is selling you something.

The fog and the mood are on the same clock

Two more things get blamed on you personally, and both track the night.

The fog. SWAN tested 2,362 women repeatedly over four years on processing speed and verbal memory. Normally, when you take the same cognitive test over and over, you get better at it. That is the practice effect, and every group in the study showed it: premenopausal women, early perimenopausal women, postmenopausal women. One group did not. Women in late perimenopause stopped showing the normal improvement [21].

That is the precise, defensible version of brain fog. Your brain is not deteriorating. It stopped picking up the gains it should be picking up. And it comes back. Performance rebounded after the transition. A separate SWAN analysis found that the slow decline you can measure over ten years in midlife tracks chronological age, not the menopause transition [22]. What you are in is a window, not a diagnosis.

The mood. This is the study that ties the whole paper together. Researchers used a medication to induce a rapid, controlled drop in estradiol in 29 healthy young women, average age 27, then monitored their mood and their sleep in the lab. That is an

experimental model of estrogen withdrawal and not a study of natural perimenopause, and you should weigh it accordingly. Inside that model, depression scores rose. And when they looked at what predicted the mood change, each additional *nighttime* hot flash was worth 3.2 points on the depression scale. Daytime hot flashes showed no association at all. Measures of sleep fragmentation predicted mood worsening independently [23].

Same symptom. Different clock. Only the one that happens while you are trying to sleep moves your mood. When your husband asks why you have been so short with everybody, that is the answer, and it is not a character flaw.

Why nobody caught this

I want to be careful here, because pointing at your doctor is the cheapest move in this entire market and I am not interested in it. Your physician is not lazy and is probably not indifferent. The system trained her for this badly and then gave her fifteen minutes.

In a survey of residents across twenty United States training programs in family medicine, internal medicine, and obstetrics and gynecology, 6.8 percent said they felt adequately prepared to manage women going through menopause. One in five reported receiving no menopause lectures at all during residency. Ninety-four percent said the training was important [24]. They knew it mattered. Nobody taught them.

A 2023 survey of obstetrics and gynecology residency program directors found that 31.3 percent of programs had a dedicated menopause curriculum, and among the programs that had one, 71 percent offered two or fewer menopause lectures a year. The directors rated their own programs' effectiveness at preparing residents for menopause care at 3.83 out of 10 [25].

Then add the clock. A national analysis of more than 8 million primary care visits found the average visit runs about 18.9 minutes of face to face time [26]. Eighteen minutes for your blood pressure, your medication refills, your mammogram, your mother's health history, and seven years of not sleeping.

The result of that math shows up in the outcome data. In a Mayo Clinic survey of 4,914 women aged 45 to 60, roughly 87 percent had not sought medical care for their menopause symptoms, and only about one in four of those with symptoms was receiving any treatment [27]. Among women who did raise their most bothersome symptom with a provider, 49 percent described the encounter as negative or dissatisfying [28]. And the gap is not evenly distributed. An analysis of national expenditure data by AARP and NORC found that in 2021, 6.3 percent of non-Hispanic white women received menopause

treatment, compared with 2.5 percent of non-Hispanic Black women and 2.7 percent of Hispanic women, less than half the rate [29].

That is not a character flaw in your doctor. It is a training gap with a measurable body count, and you have been carrying it.

What an honest evaluation actually looks like

Now I am going to tell you something that costs me, because it is true.

There is no blood test that diagnoses perimenopause in a woman over 45. The Endocrine Society states it plainly: in a woman with a uterus, menopause is a clinical diagnosis based on the cessation of menses, and measurements of sex steroids, gonadotropins, inhibin B, or anti-Müllerian hormone do not further inform that diagnosis [30]. The United Kingdom's NICE guideline goes further and instructs clinicians not to use estradiol, anti-Müllerian hormone, inhibin, antral follicle count, or ovarian volume to diagnose perimenopause or menopause in women over 45. FSH has a role in two narrow situations: women aged 40 to 45 with symptoms and a change in cycle, and women under 40 with suspected menopause [31]. The reason is simple. In perimenopause your hormones are not low, they are erratic. A single draw catches one point on a wild line and tells you almost nothing.

So when a clinic promises to find your problem with a hormone panel, or a saliva kit, or a urine metabolite ratio, understand what is being sold. The Menopause Society's position statement says it directly: salivary and urine hormone testing to determine dosing are unreliable and not recommended [32]. The Endocrine Society reaches the same conclusion, noting that salivary hormone assays are not standardized, have no independent quality control program, and lack an accepted reference range [30].

Here is what testing is actually for, and this is what I do in my own practice.

Testing is not there to prove you are in perimenopause. Your history proves that. Testing is there to find what else is in the way, and to establish whether it is safe to treat you. Those are two different jobs and both of them matter.

The things that mimic this picture almost perfectly, and that I want ruled out before anyone writes you a prescription:

- **Thyroid disease.** The European Menopause and Andropause Society named the overlap directly: menstrual irregularity, mood changes, sweating, sleep disturbance, and hair loss appear in both, and they advise a low threshold for testing [33].

- **Iron deficiency.** Heavy or erratic perimenopausal bleeding drains iron, and iron deficiency produces exhaustion, brain fog, and hair loss on its own. It also falsely raises your hemoglobin A1c, and correcting the deficiency brings that number back down, so a metabolic picture read without checking iron can be wrong [34].
- **Metabolic change.** The American Heart Association's scientific statement establishes that the rise in LDL cholesterol and apolipoprotein B in midlife women tracks the menopause transition itself rather than chronological aging [35]. That is precisely why this is the moment to look, not five years from now.
- **Blood sugar.** Screening for prediabetes and type 2 diabetes is recommended for adults aged 35 to 70 who carry extra weight [36], and the menopause transition is when the risk curve bends. In SWAN, the odds of developing metabolic syndrome rose 1.45-fold per year during perimenopause [37].

And one more, where I am going to be transparent about where I stand relative to the guidelines, because you should know the difference between what everyone agrees on and what is my clinical judgment.

I order a fasting insulin. Almost nobody does. It is not part of any standard panel, and I want to be straight with you: the laboratory medicine bodies do not endorse fasting insulin or HOMA-IR as a diagnostic test, and the American Diabetes Association's diagnostic criteria contain no insulin measure at all. That is the guideline position and I am not going to dress it up as something else.

Here is why I run it anyway. Your fasting glucose and your A1c tell me what your blood sugar is doing right now. They do not tell me how hard your pancreas is working to keep it there. A woman can hold a perfectly acceptable glucose for years while her insulin climbs to do it, and every one of those years she is living in a fat-storage state and being told her labs are normal. In the Whitehall II cohort, which followed people for years before they were diagnosed with type 2 diabetes, insulin sensitivity fell steeply in the final five years before diagnosis while the glucose criteria had not yet been met [38]. The disease is late. The physiology is early.

That is the whole difference between normal and optimal. Normal reference ranges were built to diagnose a disease that has already arrived. Optimal ranges are built to catch the dysfunction while it is still a dysfunction. Most medicine is reactive. I would rather be proactive, and I would rather tell you plainly that this particular test is my clinical judgment and not a guideline mandate than let you believe your doctor was withholding something the standard of care requires.

What treatment looks like in 2026, and what it does not

The professional standard is clearer than the conversation around it.

The Menopause Society's 2022 hormone therapy position statement, which is still the current statement as of this writing, concludes that for healthy women younger than 60 or within 10 years of menopause onset, with no contraindications, the benefit-risk ratio is favorable for treating bothersome hot flashes and night sweats and for preventing bone loss. The same statement is equally clear about the other side: women who begin therapy more than 10 years out, or after age 60, have a less favorable benefit-risk ratio [32]. Both halves are the standard. Anyone quoting you only the first half is not giving you medicine, they are giving you a pitch.

The regulatory picture also moved recently, and it moved in a way that is widely misreported. On November 10, 2025, the FDA and HHS announced they were initiating removal of the boxed warning statements regarding cardiovascular disease, breast cancer, and probable dementia from menopausal hormone therapy labels [39]. On February 12, 2026, the FDA approved the actual labeling changes for six products [40]. What that action did *not* do: it did not remove the endometrial cancer boxed warning from estrogen-alone products, it did not change every product on the market, and it was not the publication of new clinical trial data. It was a change in how risk is communicated, and it drew criticism from clinicians who felt the announcement overstated the benefits. You should know the difference between a regulatory decision and a scientific result.

And here is where I part company with a large part of my own industry.

Hormone therapy is not a dementia prevention strategy. I am on record about how much I believe hormones matter, and I still will not tell you that. A 2025 systematic review and meta-analysis covering more than one million participants found no significant association between menopausal hormone therapy and mild cognitive impairment or dementia, in either direction, including when analyzed by timing of initiation [41]. The one long-term randomized follow-up found neither benefit nor harm to cognition roughly a decade out [42]. The cognitive symptoms of the transition are real, measurable, and largely temporary. Treat the symptoms. Do not buy a brain-protection promise the data has not earned.

Testosterone is not an energy prescription. The 2019 Global Consensus Position Statement, endorsed by every major menopause and endocrine society, recognizes exactly one evidence-based indication for testosterone in women: hypoactive sexual desire disorder in postmenopausal women, after a formal assessment. It also states that

a blood total testosterone level should not be used to make that diagnosis, and that compounded testosterone cannot be recommended [43]. I treat low desire, and I use testosterone when it is indicated. I am not going to tell you it is a fatigue drug or a body composition drug, because the evidence does not say that, and a doctor who claims more than the evidence supports has committed the worst sin in this field.

What is actually well supported is simpler and less exciting than what gets sold. Hormone therapy is the most effective available treatment for hot flashes, night sweats, and genitourinary symptoms, and it prevents bone loss. Estrogen has real metabolic effects: in large randomized trials, hormone therapy reduced new diagnoses of type 2 diabetes by roughly 12 to 35 percent depending on the regimen, independent of weight change, though it is not approved or appropriate as a diabetes prevention drug [44]. Progesterone helps the sleep of the woman whose progesterone is gone. Those are true, they are enough, and they are what I will put in writing.

What to do with this

You do not need to become a hormone expert. You need five things, and you can ask for all of them at any office.

1. **Bring the night, not just the day.** Say the words "I cannot stay asleep," give the number of nights per week, and say how long it has been going on. Sleep maintenance is the specific, measurable, menopause-linked complaint, and it is the one most likely to get skipped if you lead with feeling tired.
2. **Ask for the mimics to be ruled out.** Thyroid, iron and ferritin, complete blood count, a metabolic panel, lipids including apolipoprotein B if available, and A1c. If you get pushback, ask for the reason to be documented.
3. **Do not accept a hormone panel as a diagnosis, and do not accept the absence of one as a dismissal.** The right answer is that your history makes the diagnosis and the labs find the obstacles. Both of those sentences should be said to you.
4. **Ask directly whether you are a candidate for hormone therapy under the current position statement, and ask for the risks and the benefits in the same breath.** If you are under 60 or within ten years of your last period, that is a real conversation and you are entitled to have it.
5. **If the answer you get is "your labs are normal, this is just aging," get a second opinion.** Normal does not mean you are healthy. Normal means nobody found a disease yet.

You are not crazy, you are not lazy, and you are not broken. Your hormones changed, your nights broke, and your metabolism followed. That is a medical situation with a mechanism, a literature, and, for most women, a set of options.

I have watched what happens when a woman finally gets tested properly, believed, and treated. My own mother spent years being told her weight and her misery were age. After she started hormone therapy she lost over a hundred pounds, took up Zumba, and rode a bicycle for the first time in her life at 65. Temple, one of my patients, is 54, and her hot flashes are gone and her energy is back. I am on hormone therapy myself. I tend to eat my own cooking.

You have spent long enough deciding whether you feel bad enough to be worth investigating. You do.

The next step

If this is your night, the honest place to start is a baseline. In my practice that starts with the Hormone Blueprint, an at-home lab kit with a recorded explanation of what your report shows. If you want to go over your own numbers with me, one-on-one, that is a separate Lab Review. If you become my patient, we use the 4D Method: Discover, Diagnose, Design, Defend.

You will not be rushed. You will not be told it is in your head. You will not be handed a prescription that nobody explains to you.

Hormone Therapy Institute, Henderson, Nevada

To request a consultation or the Hormone Blueprint, visit HormoneTherapyInstitute.com

This paper is for education. It is not medical advice, and it does not create a physician-patient relationship. Hormone therapy is not appropriate for everyone, and decisions about it should be made individually with a qualified clinician who knows your history.

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