there, the dopamine genes get activated to produce more, and the overall effect is a more stable regulation of these pathways, which are important to controlling addictions.

Serotonin is equally affected by exercise, and it's important for mood, impulse control, and self-esteem. It also helps stave off stress by counteracting cortisol, and it primes the cellular connections in the cortex and hippocampus that are important for learning.

THE TRUE TEST

We've known for a while that exercise influences the same chemicals as antidepressants do, but nobody had done a scientifically sound head-to-head comparison until researchers at Duke University took up the task in 1999. In a landmark study affectionately called SMILE (Standard Medical Intervention and Long-term Exercise), James Blumenthal and his colleagues pitted exercise against the SSRI sertraline (Zoloft) in a sixteen-week trial. They randomly divided 156 patients into three groups: Zoloft, exercise, or a combination of the two. The exercise group was assigned to supervised walking or jogging, at 70 to 85 percent of their aerobic capacity, for thirty minutes (not including a ten-minute warm-up and a five-minute cool-down) three times a week. The results? All three groups showed a significant drop in depression, and about half of each group was completely out of the woods—in remission. Another 13 percent experienced fewer symptoms but didn't fully recover.

Blumenthal concluded that exercise was as effective as medication. This is the study I photocopy for patients who are skeptical of the idea that exercise changes their brain chemistry enough to help their depression, because it puts the issue in terms that are as black-and-white as psychiatry can hope to deliver, at least for now. The results should be taught in medical school and driven home with health insurance companies and posted on the bulletin boards of

every nursing home in the country, where nearly a fifth of the residents have depression. If everyone knew that exercise worked as well as Zoloft, I think we could put a real dent in the disease.

Reading between the lines of the SMILE study gets at the complex issues that have kept exercise from being accepted as a medical treatment. Just as with Andreas Broocks's comparison of exercise to the antianxiety drug clomipramine in 1997, although both groups ended up at the same point of improvement, the patients taking medication experienced more immediate relief. At first glance, this seemed to contradict the cautions from pharmaceutical companies that antidepressants might take three weeks to work. But those time estimates are based on statistics; I have had plenty of patients over the years who have responded within a few days.

Conversely, what about the studies showing that a single bout of exercise can improve mood? For instance, in 2001, Northern Arizona University psychology professor Cheryl Hansen showed that as little as ten minutes of exercise can immediately improve vigor and mood in healthy subjects. But if Hansen had reexamined mood several hours later, she probably would have found that it had returned to baseline. So while it's important for us to realize that we can lift our mood with one session of exercise, it's also important to keep in mind that it takes longer to change our mood day to day.

In Blumenthal's study, he assessed mood once a week, before exercise. He did see immediate improvement in some patients, but not as dramatically as with medicine. A crucial aspect of recovering from depression is being able to predict that you're still going to feel good five minutes from now, and then five hours from now. Eventually you'll be confident that you'll feel better tomorrow morning. That might take a little longer with periodic exercise.

Six months after the study, Blumenthal and his colleagues surveyed the patients to see how they were doing and found that exercise worked even better than medicine over the long term. About

percent of those on medication and 55 percent for those in the combined treatment group. (Blumenthal has some interesting theories about what happened in the combo group, which I'll explain in a bit.) And of the patients who were in remission after the initial study, just 8 percent of the exercise group had a relapse versus 38 percent in the medication group—quite a significant difference.

After the initial four-month trial, the subjects were free to seek treatment (or not) however they chose, which complicated the results. Some signed up for psychotherapy, some in the medication group began exercising, and some in the exercise group started taking medicine, which brings a lot of variables into play. But Blumenthal's group found that the most significant predictor of whether someone felt better was how much they exercised. Specifically, every fifty minutes of weekly exercise correlated to a 50 percent drop in the odds of being depressed. Blumenthal stopped short of concluding that exercise *caused* remission. Maybe the reverse was true: patients who continued to exercise did so *because* they were less depressed. It's the classic chicken-egg problem scientists face when correlating physical activity and mood. Does it really matter whether you're less depressed because you exercise or whether you exercise because you're less depressed? Either way, you feel better.

But how to account for the combination group's unexpected results? Blumenthal assumed those who exercised and took Zoloft would show the best results, but they had the worst relapse rates. He speculated that some of the participants didn't like the idea of antidepressants, especially considering they had expressly signed up for a study to test the effects of exercise on depression. Several were disappointed when they found out they would also receive medication, and during the trial some said they thought the medication was *interfering* with the benefits of exercise. That's unlikely from a physiological standpoint, but it's possible that taking medication undercut the sense of self-mastery that exercise confers. As

Blumenthal explained in the study, 'Instead of incorporating belief 'I was dedicated and worked hard with the exercise progr it wasn't easy, but I beat this depression,' patients might incorpo the belief 'I took an antidepressant and got better.'"

THE BEST FIX

When we talk about depression, we don't use the word *cure* beca we only have subjective measures for behavior and emotions. Abo a third of patients with depression achieve full remission of th symptoms with antidepressants. Another third feel much better wi medication, but they may continue to have problems with motiv tion, lethargy, and fatigue. The bad thoughts are gone, but eve though they're able to get out of bed, they're still not knocking o the door for a new job or doing what they should be doing. They' feeling less than well, lingering in the shadows of depression. Th current *Diagnostic and Statistical Manual of Mental Disorders* lis nine symptoms for depression, and you need to have six to receiv a diagnosis of depression. Say you can't concentrate, can't sleep, fee worthless, and aren't interested in anything. That's four. Technicall you aren't depressed. What are you, then? Just miserable? My point is, with any degree of depression, you need to snuff it out completely. And exercise is starting to be taken very seriously in this regard.

Madhukar Trivedi, a clinical psychiatrist who is the director of the Mood Disorders Research Program at the University of Texas Southwestern Medical School, has been researching the effectiveness of using exercise to augment antidepressants. In 2006 he published a pilot study showing that patients who weren't responding to antidepressants lowered their scores on a common depression test by 10.4 points on a 17-point scale—a huge drop—after twelve weeks of exercise. All seventeen patients were deeply depressed and had been taking antidepressants for at least four months. The drugs weren't working, but they stayed on them for the experiment.

Trivedi enlisted the Cooper Institute to set up the exercise protocol. Then they let the patients, who were inactive to begin with, exercise at home, either walking or riding a stationary bike as often and as rigorously as they wanted. The only requirement was that they expend a certain amount of energy each week. Most chose to walk an average of fifty-five minutes three days a week. Nine people dropped out, which isn't unusual, but of the eight who completed the exercise program, five achieved full remission. Even the patients who stuck with the program for only a few weeks showed an improvement in symptoms.

The numbers were small, but Trivedi's results were huge. At least for some people, when drugs don't work, exercise does. It begs the question, why wouldn't you add exercise right from the beginning? Especially if you're playing trial and error with a host of other medications? But the allure of the magic pill is powerful, and it takes a long time to overturn attitudes. Just ask T. Byram Karasu, who was in charge of the American Psychiatric Association's work group on major depressive disorders. He pushed to get the APA to formally adopt exercise in its treatment guidelines for depression and suggested that psychiatrists tell every patient to walk three to five miles a day or do some other type of vigorous exercise. The APA balked, presumably because, while most doctors acknowledge the anecdotal evidence that exercise improves mood, they say there isn't enough scientific evidence. In this age of deconstructing the brain and unlocking the secrets of the life and death of cells, it's difficult for psychiatrists to consider such a holistic strategy as exercise a treatment.

Any doctor will tell you that the worst patients are other doctors. Imagine the difficulty, then, in convincing a patient of mine with a medical degree to exercise for her depression. Grace, who has a history of mild bouts of depression, also happens to be a psychiatrist with a sophisticated knowledge of medicine. Even so, we

haven't been able to find an antidepressant without side effects. SSRIs seem to work best for her, but she would try one and then stop because they all made her gain weight very quickly. She's brilliant and well aware of the biology of exercise—at least some of it—but she just didn't do it.

Last summer, she injured her back and ended up immobilized in bed for a period. Purely for physical rehabilitation, she started swimming. It was the only thing she could do, and it felt good because the water supported her body and eased her pain. She enjoyed it and began exercising in the pool for three hours a day. Not only did her pain decrease, but she also started seeing some long-lost muscle tone and she felt good about herself.

Then the pool shut down for the winter, her back flared up, and her mood took a dive again. This time she also got angry. Flat on her back with few options for exercise, she started lifting weights—just three-pound dumbbells that she would pump up and down fast enough to get her heart rate up several times a day. Even that minor activity helped, and more important, the experience sparked a shift in her brain and in her mind. I've been seeing Grace for years, but there was something about this situation that put exercise into perspective for her.

She's worked out the kinks in her back and has been very steady with her swimming routine. She says she's able to think and write more creatively, and she feels a newfound sense of vigor that hasn't gone unnoticed by her family and friends. Not that it should have surprised her: She recalled that when she was training every day for the tae kwon do team in college, she did some of her best work. Then as a young doctor in Boston, she got into running marathons. Like many people, it was only after she started a family that she fell out of the habit of exercise. "I just got too busy and forgot the benefits of working out," she says. "Now I feel like I have my brain back."

It wasn't until we were able to take good pictures of the brain that we really began to understand how various medical treatments and exercise break us out of the bonds of depression. In the early 1990s, using MRI, we noticed bright patches on brain scans of certain depressed patients. The hyperintensities, as they are known, appeared in the white matter, the portion of densely bundled axons that connect neurons in the gray matter of the cortex. Zooming in further, we found differences in the volume of the cortex—the gray matter was physically shrunken. Gray matter is the thin, wrinkly covering of the brain made up of cells that direct all of our complex functions such as attention, emotions, memory, and consciousness. The MRI scans pointed toward a radical notion: that chronic depression may cause structural damage in the thinking brain.

Related research showed that depressed patients also had measurable changes in the amygdala and the hippocampus, crucial players in the stress response. We knew the amygdala was central to our emotional life, but we were just discovering that the memory center was also involved in stress and depression. In 1996 Yvette Sheline of Washington University in St. Louis compared ten patients with depression to ten healthy controls of the same physical stature and educational background, and she found that the hippocampus of depressed patients was up to 15 percent smaller compared to that of the controls. She also found evidence that the degree of shrinkage was directly related to the length of depression, and this was news. It might explain why so many patients with depression complain of learning and memory trouble, and why mood deteriorates in Alzheimer's, the neurodegenerative disease that begins with erosion of the hippocampus.

High levels of the stress hormone cortisol kill neurons in the hippocampus. If you put a neuron in a petri dish and flood it with cortisol, its vital connections to other cells retract. Fewer synapses develop and the dendrites wither. This causes a communication breakdown, which, in the hippocampus of a depressed brain could partly explain why it gets locked into thinking negative thoughts—it's recycling a negative memory, perhaps because it can't branch out to form alternative connections.

Neuroimaging has cracked open an entirely new view of the biology of depression. The picture was—and still is—crude and fuzzy, but now positron-emission tomography (PET) scans and functional MRI (fMRI) scans allow scientists to go beyond snapshots and see the brain at work. At the same time, we learned that new nerve cells are born every day in the hippocampus and possibly in the prefrontal cortex—two areas shriveled in depression. The new tools and new discoveries led to a reformulation of the neurotransmitter theory.

Our understanding hasn't junked the old theory, just expanded it. Now we see depression as a physical alteration of the brain's emotional circuitry. Norepinephrine, dopamine, and serotonin are essential messengers that ferry information across the synapses, but without enough good connections in place, these neurotransmitters can only do so much. As far as the brain is concerned, it's job is to transfer information and constantly rewire itself to help us adapt and survive. In depression, it seems that in certain areas, the brain's ability to adapt grinds to a halt. The shutdown in depression is a shutdown of learning at the cellular level. Not only is the brain locked into a negative loop of self-hate, but it also loses the flexibility to work its way out of the hole.

Redefining depression as a connectivity issue helps explain the wide range of symptoms people experience. It's not just a matter of feeling empty, helpless, and hopeless. It affects learning, attention, energy, and motivation—disparate systems that involve different parts of the thinking brain. Depression also affects the body, shutting down the drive to sleep, eat, have sex, and generally look after ourselves on a primitive level. Psychiatrist Alexander Niculescu

sees depression as a survival instinct to conserve resources in an environment void of hope—"to keep still and stay out of harm's way," he wrote in a 2005 article in *Genome Biology*. It's a form of hibernation: When the emotional landscape turns wintry, our neurobiology tells us to stay inside. Except that it can last much longer than a season. It's as if our entire being has said, there's nothing out there for me, so I may as well quit. Thus the atrophy, the halt of neuroplasticity, neurogenesis, and overall lack of connectedness. It's no wonder we haven't been able to define depression as a single problem.

THE UNDERLYING CONNECTION

If depression is primarily a communication breakdown, or a loss of the brain's ability to adapt, it's very good news for the value of exercise. We learned in the early 1990s that brain-derived neurotrophic factor (BDNF) protects neurons against cortisol in areas that control mood, including the hippocampus. It is the fertilizer that encourages neurons to connect to one another and grow, making it vital for neuroplasticity and neurogenesis. While extremely high levels of cortisol can decrease BDNF, antidepressants and especially exercise do just the opposite. BDNF is the rope in a tug of war between chronic stress and adaptability. The Miracle-Gro molecule became the new serotonin, and we began measuring, blocking, increasing, and otherwise tweaking it in every way imaginable, to see how it affected mood in mice and men.

We can't ask a rodent if it's depressed, but we can see how it reacts to inescapable stress. If its feet are shocked, does it attempt to escape or does it freeze? This is the experimental model for learned helplessness, a popular way of describing human depression that implies an inability to cope with adversity and to take the action necessary to survive and flourish. If the lab animal gives up, it's considered to be depressed.

In one such experiment, when BDNF was injected directly into the hippocampi of mice, they were much quicker to try to escape than their nontreated counterparts. The injection seemed to have the same effect as exercise and antidepressants on the animals' behavior. Conversely, scientists have bred mice with genes that produce 50 percent less BDNF and found that they don't respond well to antidepressants, suggesting it's a necessary ingredient for the drugs to work. Such mice were significantly slower to try to escape stress than their peers with normal BDNF function.

In people, scientists are limited to measuring BDNF in the bloodstream, which, at best, offers a rough approximation of levels in the brain. One study of thirty depressed patients showed that every one of them had lower than normal BDNF levels. In another, antidepressants restored depressed patients' BDNF levels to normal, and yet another showed that higher levels correspond to fewer symptoms. In postmortem studies of people with depression who died of suicide, their brains had significantly decreased levels of BDNF. Even in healthy people, low BDNF levels have been correlated with personality traits that make them more vulnerable to depression, such as neuroticism and hostility.

Exercise boosts BDNF at least as much as antidepressants, and sometimes more, in the rat hippocampus. One study showed that combining exercise with antidepressants spiked BDNF by 250 percent. And in humans, we know that exercise raises BDNF, at least in the bloodstream, much like antidepressants do.

As with norepinephrine in the 1960s, BDNF may be the tip of the iceberg. Today, research focuses on BDNF as well as vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF-2), insulin-like growth factor (IGF-1), and all the related chemicals involved in encouraging neuroplasticity and neurogenesis. At the same time, pharmaceutical companies are funding research to mark and measure all these factors, and to map the genes affected by them, so they can figure out how to mimic their actions. BDNF

...than serotonin, closer to the source. Ultimately, the genes have to turn on the flow.

The shift from the neurotransmitter hypothesis to the connectivity theory is a move from outside to inside the nerve cell. In addition to working at the synapse, as serotonin does, BDNF turns on genes to produce more neurotransmitters and neurotrophins, puts the brakes on self-destructive cellular activity, releases antioxidants, and provides the proteins used as building material for axons and dendrites. These gene-regulated adaptations of BDNF might go a long way toward explaining the delayed effect of antidepressants.

It can often take three weeks for antidepressants to work. Is it merely a coincidence that the process of neurogenesis—from the time a stem cell is born in the hippocampus until it plugs into the network—takes approximately the same amount of time? Many researchers think not. The latest twist to the connectivity theory is that a shutdown of neurogenesis might be a factor in depression. Some have shown that blocking neurogenesis in rats negates the effect of antidepressants, so it's a possibility. This could provide an even stronger link between exercise and its antidepressant effect, given that exercise clearly elevates BDNF and its sibling growth factors, and that these are essential to the building process of neurogenesis. Nobody has shown that a deficit of BDNF *causes* depression, but it's not for lack of trying. In 1997 Yale psychiatrist Ronald Duman published "A Molecular and Cellular Theory of Depression" in the *Archives of General Psychiatry*, and ever since he and others have been unraveling the story of BDNF. In 2006 he charted how various treatments influence BDNF, including all available antidepressants, as well as less common forms of treatment such as electroconvulsive therapy (ECT) and transcranial magnetic stimulation (TMS). They all boost BDNF in the hippocampus, and Duman pointed out that ECT, the most effective treatment, raises it by 250 percent.

But how is it that a blunt physical manipulation such ECT—which sends an electrical current through the brain induce a seizure—can work like drugs, therapy, and exercise think ECT provides a useful metaphor. If we look at depressi as a sort of brain lock, then we can see a common thread betwe these approaches: They are all shocks of a sort. They send spa flying to change the dynamic in the brain. Some parts are stuck a constant whir, and others are locked in place. The key, I think, to wake up the brain and the body so you can pull yourself out the downward spiral. What makes aerobic exercise so powerful that it's our evolutionary method of generating that spark. It ligh a fire on every level of your brain, from stoking up the neuron metabolic furnaces to forging the very structures that transm information from one synapse to the next.

BREAKING THE BONDS

While molecular scientists approach brain lock with tools to picl it, Emory University neurologist Helen Mayberg wants to smash i wide open. Several years ago, she tested a radical therapy called deep brain stimulation (DBS), in which she inserted an electrode into the subgenual cortex in a half dozen severely depressed patients, for whom every other form of medical treatment had failed. "These people are stuck," Mayberg says. "They have an inability to put thought to action because they're not in gear. We need to find a way, metaphorically, to unstick them." She used voltage, and the results were profound: All six patients spontaneously described sensations such as a "disappearance of the void" right there on the operating table, the second the electrodes were switched on. Four of them eventually achieved full remission.

The area of the brain Mayberg targeted is the tip of the anterior cingulate, which is the major way station between information coming down from prefrontal cortex and up from limbic system—the

well. As the seat of executive function, it prioritizes what you pay attention to and indirectly regulates the limbic system, integrating cognitive and emotional signals. If it fails to shift attention from the negative, in the case of depression, you can't think about anything else. "Not being able to initiate, not being able to think clearly, not being able to care about your family—those are secondary to something that your attention is directed to internally, but that is a false signal," Mayberg explains. "You can fix it, and then other problems may be more accessible."

The real goal of DBS is to bring the prefrontal cortex back online so you can use your executive function to tackle the core issues. It frees you up to attack the problem rationally—*I'm not a bad person; my children love me; and my life is not beyond repair.* This is also one of the effects of exercise. In 2003 a group of German neuroscientists did an experiment with twenty-four people with mild depression who were being treated with drugs, and ten healthy controls. They all took a battery of neuropsychological tests designed to gauge executive function. They repeated the tests after thirty minutes of stationary cycling at 40 percent of their maximum heart rate, then again at 60 percent—low enough to keep lactic acid out of the equation. The depressed patients showed significant improvement on two of the four tests at both intensity levels, which demonstrated that exercise immediately improves the highest form of thinking. Even one bout of exercise was enough to influence the prefrontal cortex. The ten nondepressed volunteers didn't show much improvement, but then, there was nothing to fix.

Mayberg would be the first to say that executive function is still only part of the story. In comparing PET scans of patients who responded to antidepressants against others who responded to cognitive behavior therapy, she found that the two approaches change activity levels of the limbic system from opposite directions. Antidepressants seem to work through a bottom-up chain of events, meaning the activity begins in the brain stem and ripples through the limbic system until it reaches the prefrontal cortex. This might explain why antidepressants relieve the physical effects first—we feel more energetic before we feel less sad. With cognitive behavioral therapy and psychotherapy, we feel better about ourselves before we feel better physically. Therapy works from the prefrontal cortex down, to modify our thinking so we can challenge the learned helplessness and spring ourselves out of the hopeless spiral.

The beauty of exercise is that it attacks the problem from both directions at the same time. It gets us moving, naturally, which stimulates the brain stem and gives us more energy, passion, interest, and motivation. We feel more vigorous. From above, in the prefrontal cortex, exercise shifts our self-concept by adjusting all the chemicals I've mentioned, including serotonin, dopamine, norepinephrine, BDNF, VEGF, and so on. And unlike many antidepressants, exercise doesn't selectively influence anything—it adjusts the chemistry of the entire brain to restore normal signaling. It frees up the prefrontal cortex so we can remember the good things and break out of the pessimistic patterns of depression. It also serves as proof that we can take the initiative to change something. This paradigm holds true for exercise's effect on mood in general, regardless of whether we're depressed or coping with some nagging symptoms. Or even if we're just having a bad day.

The idea of attacking depression from both directions couldn't be more important. "Even with the brain stimulation patients, once you get the system back online, they need mental rehabilitation," Mayberg says. "Part of the initial rehab is simply to get them doing something. The best kind of behavioral therapy is to just go outside, go for a walk. Do something. It doesn't require you to have elaborate planning. It doesn't put you into a negative spin. If you do a physical activity, it's very quickly reinforcing because, before, you couldn't initiate to make yourself do something."

[illegible] it, and exercise is the perfect tool. You begin to look at the world differently, and you see trees instead of a barren wasteland. When you see yourself moving, that alone is an achievement—proof that you can help yourself.

OUT OF THE TUNNEL

Science has come a long way since our search for the single culprit began, and the decades' worth of research generated from the monoamine hypothesis has taught us volumes about the biology of emotions. The closer we get to the cause of depression, the more complex it appears. When we began, everyone was fairly certain that the problem was an imbalance of neurotransmitters at the synapses. Now we know for certain that it's not so simple.

Ironically, I think this is precisely why exercise has yet to be embraced as a medical treatment. It doesn't simply raise serotonin or dopamine or norepinephrine. It adjusts all of them, to levels that, we can only presume, have been optimally programmed by evolution. The same goes for exercise's effect on BDNF, IGF-1, VEGF, and FGF-2, which provide the building material and oversight for the construction of new connections and neurons. In short, exercise affects so many variables in the brain that its nigh impossible to isolate its effect as we'd like—in the name of hard science. But the evidence is there, from the action of microscopic molecules to massive surveys of tens of thousands of people over the years. Yes, exercise is an antidepressant. But it is also much more.

Still, it's not surprising that about half the patients in studies on exercise and depression drop out. Perhaps because most of them are inactive to begin with, getting started is that much more challenging. This is critical for doctors to keep in mind when recommending exercise. With people who are already feeling hopeless, it's important to keep expectations reasonable, so as not to reinforce

the negative. On the other hand, studies have shown that e people who find the act of exercise inherently unpleasant hav positive mood swing the minute the workout is over. If you kn what's on the other side, it's easier to push through the difficulty

Human beings are social animals, so if you're depressed, it wo be ideal to choose a form of exercise that encourages making co nections and that can take place outside or in some environme that stimulates the senses. Asking someone to join you in exerc and putting yourself in a new setting will give those newly hatch neurons a powerful reason for being; new connections need to formed to represent the sensory stimulation. Breaking out of t emptiness that the brain has been locked into provides a sen of purpose and self-worth that evokes a positive future. Once yc develop the positive feeling, you need to devote it to somethin Then you get the bottom-up motivation and physical boost com bined with the top-down reevaluation of yourself. By motivatin the body to move, you're encouraging the mind to embrace life.

THE PRESCRIPTION

The first question people ask me when I suggest exercise as therapy is "How much should I do?" There is no firm answer—especially given depression's wide spectrum of symptoms and severity—but Madhukar Trivedi has drawn some conclusions about the amount necessary to be effective. By quantifying exercise as a dose, he hopes to cast treatment in terms the medical profession might accept. This is all-important, because doctors have to spend time with their patients to figure out what sort of regimen is likely to keep them moving.

In one study, Trivedi and Andrea Dunn divided eighty depressed patients into five groups, four with exercise protocols of different intensities and frequencies and one control group assigned to supervised stretching only (to see if social interaction with the

ody weight as the "dose" measure. The high-intensity groups ıed an average of 1,400 calories (eight calories per pound) dur- the course of either three or five sessions per week. At the end ıree months, regardless of frequency, the high-intensity groups :heir depression scores in half. Practically speaking, their symp- s dropped significantly. The low-intensity groups burned an av- e of 560 calories (three calories per pound) and lowered their es by a third, about the same as the stretching group—and ıt as effective as a placebo. What this means for us is what I al- s tell people: some exercise is good, more is better (to a point). Trivedi and Dunn based the high dose on public health recom- ıdations for exercise, which suggest thirty minutes of moderate)bic activity on most days. If you weigh 150 pounds, that would ıslate into about three hours at moderate intensity per week. : low dose would be equivalent to eighty minutes per week. Just multiply your body weight by eight to figure out how much should be burning for the high dose, and then head to the gym ind out how many calories you burn during a given workout)st of the aerobic machines track this for you). If you weigh 150 nds and burn 200 calories in thirty minutes on the elliptical ner, you'd want to do six sessions a week to meet the high dose. I would recommend at least this much exercise for anyone), like my patient Bill, has a shadow syndrome of depression. :se are people who aren't clinically depressed but who tend to k at life with a primarily pessimistic attitude, or who have the)ression that nobody in the world, themselves included, meets ir high standards. For Bill, when he got into running and lifting ghts, he met up with a group of regulars at the gym every morn- for a workout and coffee. His performance and relationships at :k improved, and he felt like he didn't look at every new project h knee-jerk resentment. He almost welcomed new challenges, ich completely changed his wife's view of him.

to have low self-esteem, partly because they're so uncertain about how they'll feel from day to day—crabby or buoyant? One of my patients, Jillian, fit this description. We started exploring this issue after she got engaged to the man of her dreams and began feeling blue and irritable more often than not. Naturally, I had been pushing her to exercise, and she finally joined a gym near her office. Wisely, she teamed up with a co-worker who was already going, and they supported each other to work out every day at lunchtime. After a few months, she was pleased with herself, and she expressed how stabilizing the daily activity was. It helped her maintain a rhythm in her life, which added to her sense of stability.

Some people who have mood swings may describe themselves as manic-depressive or bipolar, but that's a problem of an entirely different magnitude. I haven't discussed bipolar disorder primarily because so little research has been done on exercise's effects on it. Recently, though, a preliminary study clearly showed that hospitalized patients with bipolar disorder who participated in a walking program suffered fewer depressive symptoms and less anxiety compared to those who were unwilling or unable to participate. And giving bipolar patients a stable social routine has been shown to improve long-term outcomes. It's only been very recently that exercise started making its way into treatment protocols for bipolar disorder.

In some ways, exercise is even more important for prevention than it is for treatment. One of the first symptoms of depression, even before your mood drops to new lows, is sleep disturbance. Either you can't get up or you can't get to sleep or both. I think of it as sleep inertia—trouble starting or stopping. First you lose your energy, then your interest in things. The key is to get moving immediately. And do not stop. Set up a schedule for a daily walk, run, jog, bike ride, or dance class. If you can't sleep, go for a walk in the dawn light and do it every day. Take the dog out, change your

schedule—run from the depression. Burn those 1,400 calories as if your life depended on it, and nip it in the bud.

If you have severe depression, you may feel like you're at the bottom of a pit, in a state of slow death, and that it's almost impossible to make it outside or go to the gym or even think about moving. First, see your doctor about medication and get yourself some omega-3 supplements, which are proven to have antidepressant effects. This will, I hope, loosen up the brain lock enough for you to at least go for a walk. Ask for help. Get a friend or family member to agree to come by every day, at the same time if possible, to escort you outside and around the block. In England and Australia, walking groups for patients with depression have been popular for years, and now they're starting to make their way to the United States, so check the Internet to see if there is one in your area. If that's not an option, and you have the means, set up a regular time with a personal trainer. This might sound crazy, I realize, if you feel like you can't even lift yourself off the couch, but if that's the case, activity is all the more urgent.

Exercise is not an instant cure, but you need to get your brain working again, and if you move your body your brain won't have any choice. It's a process, and the best strategy is to take it one step—and then one stride—at a time. Start slowly and build on it. At its core, depression is defined by an absence of moving toward anything, and exercise is the way to divert those negative signals and trick the brain into coming out of hibernation.

6
Attention Deficit
Running from Distraction

"I SUSPECT THE first time I realized I was not like my peers was at the early age of three when I discovered no one else in my family or neighborhood was forced to wear a child leash," wrote Sam, a thirty-six-year-old venture capitalist who came to me in hopes of understanding his lifelong disorder, which was starting to manifest itself in his young son. "I have always been known in the family as the troublemaker and spent most of my childhood in the doghouse and the 'dunce' corner. My teachers felt that I had the ability to be a good student but never fully applied myself. I am able to express myself well and organize my thoughts, but often procrastinate."

Sam is no dunce, but like so many others with attention-deficit/hyperactivity disorder (ADHD), his erratic behavior led everyone around him to label him as stupid or stubborn or spoiled. He didn't want his son to suffer the same shame, and he was now seeking help at the encouragement of his business partner and his wife. "Neither quite understands how I can function with so much chaos in my life," he told me.

Chaos, high drama, deadline pressure—acute stress of any form acts like a drug for Sam's brain. His letter to me outlining his history acknowledges that he had disciplinary problems because he