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Drink Like a Lawyer: The Neuroscience of Substance Use and its Impact on Cognitive Wellness

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Drink Like a Lawyer: The Neuroscience of Substance Use and its Impact on Cognitive Wellness

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DRINK LIKE A LAWYER: THE NEUROSCIENCE OF SUBSTANCE USE AND ITS IMPACT ON COGNITIVE WELLNESS

Debra S. Austin*

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INTRODUCTION

Lawyers make powerful contributions to American society within the legal system—by participation in government, and through representation in the news, media, and entertainment businesses. But do they do so while impaired? Lawyers suffer from higher levels of anxiety and depression than the rest of the population,¹ but most do not enter law school with these mental health issues.² Lawyers rank fourth in professions with the most suicides according to the Centers for Disease Control.³ Lawyer suicides are likely linked to stress, anxiety, depression, and/or substance abuse.

Law students, lawyers, and judges are vulnerable to substance abuse. One study concluded that at least 15 percent of lawyers are alcoholics compared to approximately 10 percent in the general population.⁴

Disciplinary actions against attorneys involve substance abuse 50 to 75 percent of the time.⁵ These problems may start in law school where school events may teach students “to drink like a lawyer.”⁶

Neuroscience research has shown that both the brain and the genes enjoy the power of plasticity, which means that personal choices and environments shape the development of lawyers throughout their lives.⁷ Major obstacles to legal education reform, enumerated in *The Carnegie Report*, are also significant sources of chronic stress for law students:

- The competitive classroom climate,
- The competitive atmosphere of most law schools, and
- The grade curve.⁸

¹ See NANCY LEVIT & DOUGLAS O. LINDER, *THE HAPPY LAWYER: MAKING A GOOD LIFE IN THE LAW* 6–8 (2010); DOUGLAS LITOWITZ, *THE DESTRUCTION OF YOUNG LAWYERS: BEYOND ONE L* 16–26 (2006); ANDREW J. MCCLURG, *1L OF A RIDE: A WELL-TRAVELED PROFESSOR’S ROADMAP TO SUCCESS IN THE FIRST YEAR OF LAW SCHOOL* 315–17 (2009); REBECCA NERISON, *LAWYERS, ANGER, AND ANXIETY: DEALING WITH THE STRESSES OF THE LEGAL PROFESSION* 15–39 (2010); Lawrence S. Krieger, *Institutional Denial About the Dark Side of Law School, and Fresh Empirical Guidance for Constructively Breaking the Silence*, 52 J. LEGAL EDUC. 112, 113–15 (2002); Corie Rosen, *The Method and the Message*, 12 NEV. L.J. 160, 161 n.8 (2011).

² LITOWITZ, *supra* note 1, at 21; Krieger, *supra* note 1, at 114.

³ Rosa Flores & Rose Marie Arce, *Why Are Lawyers Killing Themselves?*, CNN (Jan. 20, 2014, 2:42 PM), <http://www.cnn.com/2014/01/19/us/lawyer-suicides/index.html>; see also Patrick Krill, *Why Lawyers Are Prone to Suicide*, CNN (Jan. 21, 2014, 10:15 AM), <http://www.cnn.com/2014/01/20/opinion/krill-lawyers-suicide/index.html>.

⁴ Patrick J. Schiltz, *On Being a Happy, Healthy, and Ethical Member of an Unhappy, Unhealthy, and Unethical Profession*, 52 VAND. L. REV. 871, 876–77 (1999).

⁵ Betty Reddy & Ruth Woodruff, *Helping the Alcoholic Colleague*, 3 PROF. LAW, May 1992, at 1, 4.

⁶ MCCLURG, *supra* note 1, at 321.

⁷ NORMAN DOIDGE, *THE BRAIN THAT CHANGES ITSELF* 240 (2007); SCOTT BARRY KAUFMAN, *UNGIFTED: INTELLIGENCE REDEFINED* 10–11 (2013).

⁸ WILLIAM M. SULLIVAN ET AL., *EDUCATING LAWYERS: PREPARATION FOR THE PROFESSION OF LAW* 27–29 (2007) (providing the most recent critique of legal education suggesting reforms based on a three apprenticeship model); see NERISON, *supra* note 1, at 68.

Stressors continue in law practice and include workload, adversarial environments, and win-lose high stakes outcomes.⁹ A common response to chronic stress is self-medication because drugs of abuse can make a lawyer feel less stressed.¹⁰

The brain can be divided into three functional areas that evolved from the top of the spine up: the primitive brain, the emotional brain, and the thinking brain.¹¹ The emotional and thinking brains have a complex relationship, and both are engaged in the learning process¹² and in the use of substances.¹³ Information moves through the brain in an electrical-chemical-electrical process via communication nerve cells called neurons (electrical) and neurotransmitters between neurons (chemical).¹⁴ Substance use impacts both the neurons and the neurotransmitters in the brain.¹⁵

Legal educators need a better understanding of what aspects or characteristics of legal education contribute to the decline in mental health of law students, lawyers, and judges, and neuroscience developments may provide this insight. Law students, lawyers, judges, and law professors will benefit from the knowledge of how stress and substance use effect the lawyer brain. This article describes the neurobiology of learning; explains the brain's reward system; examines the neural impact of stress; details the transformational conditions of neuroplasticity, neurogenesis, and epigenetics; discusses lawyer addiction; and illustrates how medications such as antidepressants, and substances such as alcohol, nicotine, marijuana, caffeine, cocaine, study drugs, and opiates impact brain function. The article concludes with neuroscience-based recommendations for law students, lawyers, judges, law schools, and law firms to optimize brain health and lawyer wellness.

I. THE LAWYER BRAIN

The brain is the key tool of the law student, lawyer, judge, and law professor. There are many forms of intelligence. Daniel Goleman wrote about the power of deploying emotional intelligence ("EQ") to improve personal and professional relationships.¹⁶ Physical health can be improved using the three pillars

⁹ LEVIT & LINDER, *supra* note 1, at 7.

¹⁰ ROBERT M. SAPOLSKY, *WHY ZEBRAS DON'T GET ULCERS* 346 (3d ed. 2004).

¹¹ JUDITH HORSTMAN, *THE SCIENTIFIC AMERICAN: DAY IN THE LIFE OF YOUR BRAIN* 4–6 (2009) [hereinafter HORSTMAN, *DAY IN THE LIFE*].

¹² RITA CARTER ET AL., *THE HUMAN BRAIN BOOK* 159 (2009) [hereinafter CARTER ET AL., *HUMAN BRAIN*]; JOHN MEDINA, *BRAIN RULES: 12 PRINCIPLES FOR SURVIVING AND THRIVING AT WORK, HOME AND SCHOOL* 103, 109 (2008).

¹³ CARTER ET AL., *HUMAN BRAIN*, *supra* note 12, at 128.

¹⁴ JOSEPH LEDOUX, *SYNAPTIC SELF: HOW OUR BRAINS BECOME WHO WE ARE* 47 (2002).

¹⁵ MICHAEL S. SWEENEY, *BRAIN: THE COMPLETE MIND* 198–203 (2009).

¹⁶ *See generally* DANIEL GOLEMAN, *EMOTIONAL INTELLIGENCE* (10th ed. 1995); DANIEL GOLEMAN, *SOCIAL INTELLIGENCE: THE NEW SCIENCE OF HUMAN RELATIONSHIPS* (2006).

of body intelligence (“BQ”): awareness, knowledge, and engagement.¹⁷ Awareness calls for attending to your body’s signals.¹⁸ Knowledge is the development of health literacy and engagement is commitment to an action plan that evolves over time to help one thrive.¹⁹

What is good for the lawyer’s body is also good for the lawyer’s brain. An emerging component of BQ is brain health literacy, the knowledge of how to cultivate cognitive fitness. Brain health literacy or neuro-intelligence (“NQ”) is a critical competency for lawyers, judges, law faculty, and law students because lawyering is a cognitive profession. The optimistic phenomenon of plasticity in the lawyer brain and genes demonstrates that personal choices, environments, and cultures shape the development of the lawyer’s brain throughout life. NQ can improve the cognitive fitness of individual lawyers and law students, and enhance the cognitive wellness of the lawyering profession.

The lawyer brain is the size of a coconut,²⁰ and is the consistency of Jell-O²¹ or tofu.²² It weighs only three pounds,²³ yet it uses 20 percent of the body’s oxygen, 25 percent of its calories, and 25 percent of its blood flow.²⁴ It developed from the top of the spine upward into three functional areas: the primitive brain, the emotional brain, and the thinking brain.²⁵

The primitive brain is located above the spine and governs the basic motor functions of breathing, digestion, and heartbeat.²⁶ Major components of the primitive brain include the brain stem and cerebellum.²⁷ The emotional brain is wrapped around the primitive brain and is in charge of emotions, hunger, circadian rhythm, sex hormones, and addiction.²⁸ The major parts of the emotional brain are the amygdala, hippocampus, thalamus, and hypothalamus, and emotional brain components in the reward system are the ventral tegmental area

¹⁷ Jim Gavin & Margaret Moore, *Body Intelligence: A Guide to Self-Attunement*, 7 IDEA FITNESS J., Nov. 2010, available at <http://www.ideafit.com/fitness-library/body-intelligence-a-guide-to>.

¹⁸ *Id.*

¹⁹ *Id.*

²⁰ RITA CARTER, MAPPING THE MIND 14 (2010) [hereinafter CARTER, MAPPING THE MIND].

²¹ DAVID M. EAGLEMAN, INCOGNITO: THE SECRET LIVES OF THE BRAIN 2 (2011).

²² DANIEL G. AMEN, CHANGE YOUR BRAIN CHANGE YOUR BODY: USE YOUR BRAIN TO GET AND KEEP THE BODY YOU HAVE ALWAYS WANTED 18 (2010).

²³ GERALD M. EDELMAN, WIDER THAN THE SKY: THE PHENOMENAL GIFT OF CONSCIOUSNESS 15 (2004).

²⁴ AMEN, *supra* note 22, at 17.

²⁵ BARRY J. GIBB, THE ROUGH GUIDE TO THE BRAIN 6–8 (2d ed. 2012); JUDITH HORSTMAN, THE SCIENTIFIC AMERICAN: BRAVE NEW BRAIN 3–4 (2010) [hereinafter HORSTMAN, BRAVE NEW BRAIN]; HORSTMAN, DAY IN THE LIFE, *supra* note 11; DAVID PERLMUTTER & ALBERTO VILLOLDO, POWER UP YOUR BRAIN: THE NEUROSCIENCE OF ENLIGHTENMENT 16–21 (2011).

²⁶ GIBB, *supra* note 25, at 37; HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 3.

²⁷ GIBB, *supra* note 25, at 36–37.

²⁸ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 53; GIBB, *supra* note 25, at 38–39; HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 4; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 4.

(“VTA”), nucleus accumbens (“NAC”), and ventral pallidum (part of the globus pallidus).²⁹ The thinking brain is the walnut-shaped top section³⁰ comprised of two hemispheres and four lobes.³¹ The frontal lobe is devoted to reasoning, planning, and language; the occipital lobe is in charge of vision; the temporal lobe controls hearing; and the parietal lobe mediates movement, taste, temperature, and touch.³² The thinking brain is responsible for executive function, reasoning, and higher-order thinking.³³

The primitive, emotional, and thinking sections of the lawyer brain are comprised of two types of brain cells: neurons, which are in charge of communication; and glial cells, which insulate neurons and support their information transport function.³⁴ Neurons are shaped like trees with branches at the top called dendrites, a cell body with a nucleus, and a trunk called an axon.³⁵ Information flows one direction through neurons, from the dendrites at the top of the tree-like structure to the roots of the axon, called the axon terminal.³⁶ Dendrites are the input channels of the neuron, and axon terminals are the output channels.³⁷

The communication site where the information output axons meet the input dendrites is called the synapse.³⁸ Information moves through the neuron as an electrical impulse.³⁹ Some neurons move information with purely electrical pathways,⁴⁰ but most neurons transport information across the synapse via chemicals called neurotransmitters.⁴¹ Information travels as an electrical impulse within the neuron and by chemical neurotransmitter between neurons.⁴² Each brain cell connects with up to 10,000 neighboring cells.⁴³

Chemical synaptic transmission is how information flows from neuron to neuron throughout the lawyer brain.⁴⁴ Neurotransmitters leave the axon termi-

²⁹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 53–54, 64–65, 128; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 4–5; NEIL V. WATSON & S. MARC BREEDLOVE, THE MIND’S MACHINE: FOUNDATIONS OF BRAIN AND BEHAVIOR 39 (2012).

³⁰ CARTER, MAPPING THE MIND, *supra* note 20.

³¹ SWEENEY, *supra* note 15, at 20–21.

³² CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 66; CARTER, MAPPING THE MIND, *supra* note 20; GIBB, *supra* note 25, at 40; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 6.

³³ PERLMUTTER & VILLOLDO, *supra* note 25, at 19–20.

³⁴ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 69; SWEENEY, *supra* note 15, at 10–11.

³⁵ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 69; SWEENEY, *supra* note 15, at 10–11.

³⁶ GIBB, *supra* note 25, at 33–35.

³⁷ LEDOUX, *supra* note 14, at 40–41.

³⁸ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 69; LEDOUX, *supra* note 14, at 40–42.

³⁹ GIBB, *supra* note 25, at 34; SWEENEY, *supra* note 15, at 14.

⁴⁰ Steven A. Siegelbaum & Eric R. Kandel, *Overview of Synaptic Transmission*, in PRINCIPLES OF NEURAL SCIENCE 177, 178 (5th ed. 2013).

⁴¹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 73; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 6.

⁴² LEDOUX, *supra* note 14.

⁴³ CARTER, MAPPING THE MIND, *supra* note 20, at 16.

⁴⁴ Siegelbaum & Kandel, *supra* note 40, at 177.

nal of the first neuron, move across the tiny synaptic gap, and interact with the dendrite of the next neuron.⁴⁵ A neurotransmitter can either excite or inhibit activity in the receiving neuron.⁴⁶ The brain's major excitatory neurotransmitter is glutamate,⁴⁷ which aids learning and memory by strengthening neural networks.⁴⁸ The main inhibitory neurotransmitter is gamma-aminobutyric acid ("GABA"),⁴⁹ which suppresses messages from your brain to your muscles during sleep to help you to remain still,⁵⁰ and slows the brain by decreasing the electrical impulses between neurons.⁵¹ In addition to action initiated by neurotransmitters, the synapse is the location where psychoactive drugs and toxins create their impact.⁵²

The lawyer brain continuously deploys critical neurotransmitters in chemical synaptic transmissions.⁵³ Table 1 provides a snapshot of the major neurotransmitters and their functions.⁵⁴

Three key principles of lawyer brain function are neuroplasticity, neurogenesis, and epigenetics. The brain is a "self-assembled system, wiring up its own synaptic connections."⁵⁵ Neuroplasticity is the brain's ability to reshape its neural circuits in response to new experiences.⁵⁶ It can be harmed by illnesses and poor treatment, healed by healthful conditions, and amplified by stimulating environments.⁵⁷ The brain evolves and adapts when exposed to new learning and skills, redesigning the hundreds of millions of connections between neurons.⁵⁸ The neural network is known as the connectome⁵⁹ and each lawyer's system of neural pathways is unique.⁶⁰

⁴⁵ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 73.

⁴⁶ *Id.*; CARTER, MAPPING THE MIND, *supra* note 20, at 16.

⁴⁷ CARTER, MAPPING THE MIND, *supra* note 20, at 29; Steven A. Siegelbaum et al., *Synaptic Integration in the Central Nervous System*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 210, 213.

⁴⁸ SAPOLSKY, *supra* note 10, at 209–10.

⁴⁹ James H. Schwartz & Jonathan A. Javitch, *Neurotransmitters*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 289, 294.

⁵⁰ HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 165.

⁵¹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 125; GIBB, *supra* note 25, at 155.

⁵² WATSON & BREEDLOVE, *supra* note 29, at 94–97.

⁵³ MARK F. BEAR ET AL., NEUROSCIENCE: EXPLORING THE BRAIN 122 (4th ed. 2015).

⁵⁴ WILLIAM J. BROAD, THE SCIENCE OF YOGA: THE RISKS AND THE REWARDS 99–100 (2012); CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 105, 223, 231; CARTER, MAPPING THE MIND, *supra* note 20, at 29; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 8, 34; DALE PURVES ET AL., PRINCIPLES OF COGNITIVE NEUROSCIENCE 389 (2012); JOHN J. RATEY WITH ERIC HAGERMAN, SPARK: THE REVOLUTIONARY NEW SCIENCE OF EXERCISE AND THE BRAIN 37, 64, 117, 121 (2008); SWEENEY, *supra* note 15, at 15, 213, 221; Christof Koch, *The Neuroscience of Consciousness*, in FUNDAMENTAL NEUROSCIENCE 1090, 1095 (2013).

⁵⁵ Sebastian Seung & Rafael Yuste, *Appendix E: Neural Networks*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 1581, 1591.

⁵⁶ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 193; SWEENEY, *supra* note 15, at 17.

⁵⁷ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 193; SWEENEY, *supra* note 15, at 13.

⁵⁸ DOIDGE, *supra* note 7, at 45–47.

TABLE 1: MAJOR NEUROTRANSMITTERS AND THEIR FUNCTIONS

Neurotransmitter	Post-Synaptic Effect	Function
Acetylcholine	Mostly Excitatory	Attention, wakefulness, learning, and memory. Alzheimer's Disease is linked to low levels of acetylcholine.
Dopamine	Excitatory and Inhibitory	Reward experiences, focuses attention, motivation, and meaning.
Endorphins	Inhibitory	Block pain by docking in opiate receptors. Levels are elevated by exercise.
Gamma-aminobutyric Acid ("GABA")	Inhibitory	Quiets neurons, promotes calm, and reduces anxiety. GABA is increased by yoga. Depression is linked to low levels of GABA.
Glutamate	Excitatory	Learning, memory, and increase of synaptic strength.
Norepinephrine	Mostly Excitatory	Regulates moods, blood pressure, heartbeat, and arouses attention.
Oxytocin	Inhibitory	Released during intercourse and pregnancy and promotes social bonds and trust.
Serotonin	Inhibitory	Regulates mood, emotion, appetite, and sleep. Low serotonin produces irritability from hunger. Many antidepressants work by addressing low levels of serotonin.

The lawyer brain evolves continuously from birth to death. Neurogenesis is the birth of new brain cells in the hippocampus and olfactory bulbs,⁶¹ structures in the emotional brain.⁶² Until 1998, scientists believed that the human brain could not grow new brain cells.⁶³ Research has shown that neurogenesis can be stimulated by exercise, learning, and antidepressants,⁶⁴ but it can also be suppressed by stress.⁶⁵

⁵⁹ SEBASTIAN SEUNG, *CONNECTOME: HOW THE BRAIN'S WIRING MAKES US WHO WE ARE*, at xiii (2012); Steven A. Siegelbaum & Eric R. Kandel, *Prefrontal Cortex, Hippocampus, and the Biology of Explicit Memory Storage in PRINCIPLES OF NEURAL SCIENCE*, *supra* note 40, at 1524.

⁶⁰ MEDINA, *supra* note 12, at 66.

⁶¹ HORSTMAN, *BRAVE NEW BRAIN*, *supra* note 25, at 8, 10.

⁶² CARTER ET AL., *HUMAN BRAIN*, *supra* note 12, at 64.

⁶³ SWEENEY, *supra* note 15, at 293–94.

⁶⁴ *Id.* at 294.

⁶⁵ SAPOLSKY, *supra* note 10, at 217–18.

At birth, a lawyer's genome is inherited deoxyribonucleic acid ("DNA").⁶⁶ Epigenetics is the study of how gene regulation is impacted by environments.⁶⁷ Environmental influences change the way genes are expressed (switched on or off)⁶⁸ via proteins called the epigenome.⁶⁹ Epigenetics clarifies the impact of nurture on nature because the DNA template lawyers are born with is changed by a transcription function where environments, actions, thoughts, and feelings impact gene expression.⁷⁰ The interaction of genetic makeup and personal experience changes the lawyer brain.⁷¹

Neuroplasticity, neurogenesis, and epigenetics reveal the brain's capacity to change and heal throughout the life of the lawyer. Armed with the power of NQ, law students, lawyers, law professors, and judges can make decisions that improve cognitive function. Law schools, law firms, and court systems can enhance their cultures to make cognitive wellness a priority.

II. MOTIVATION AND REWARD IN THE LAWYER BRAIN

For lawyers, cognition involves a complex interaction between the emotional brain and the thinking brain.⁷² Learning requires the acquisition of new information and the storage of that information in memory.⁷³ There are three stages of developing memories for later use: encoding, consolidation, and retrieval.⁷⁴ Information enters the thinking brain, is encoded by the senses,⁷⁵ and moves through the emotional brain in two processes.⁷⁶ The "quick and dirty" route speeds information through the emotional brain's panic button, the amygdala, to assess it for life-threatening potential.⁷⁷ The amygdala aids rapid decision-making where survival is at risk and it is part of memory-storage for emotionally-charged experiences.⁷⁸ The slower analytical route moves information

⁶⁶ HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 13.

⁶⁷ JOHN J. RATEY & RICHARD MANNING, GO WILD: FREE YOUR BODY AND MIND FROM THE AFFLICTIONS OF CIVILIZATION 235 (2014).

⁶⁸ DOIDGE, *supra* note 7, at 220; Cornelia I. Bargmann & T. Conrad Gilliam, *Genes and Behavior*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 42.

⁶⁹ DOIDGE, *supra* note 7, at 220; HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 13.

⁷⁰ DOIDGE, *supra* note 7, at 220–21; HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 8; Bargmann & Gilliam, *supra* note 68, at 41.

⁷¹ Bargmann & Gilliam, *supra* note 68, at 63.

⁷² CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 154–57; SWEENEY, *supra* note 15, at 240–43.

⁷³ SWEENEY, *supra* note 15, at 236.

⁷⁴ *Id.* at 239–40, 246.

⁷⁵ *Id.* at 248.

⁷⁶ CARTER, MAPPING THE MIND, *supra* note 20, at 83.

⁷⁷ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 125; SUSAN GREENFIELD, THE PRIVATE LIFE OF THE BRAIN: EMOTIONS, CONSCIOUSNESS, AND THE SECRET OF THE SELF 18 (2000).

⁷⁸ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 158–59; SWEENEY, *supra* note 15, at 242.

through the memory-processing hippocampus in the emotional brain to access prior knowledge and support thoughtful decision-making.⁷⁹

New information is processed by the emotional and thinking brains before it becomes fully consolidated in long-term memory.⁸⁰ Consolidation requires a neural communication loop from senses in the thinking brain, to the hippocampus, and back again.⁸¹ Memory consolidation takes up to two years, mostly while lawyers sleep, and thus information is recalled from this emotional-thinking brain neural loop for an extended period of time.⁸² Eventually, fully consolidated memories reside in the thinking brain of the lawyer in the knowledge network connectome.⁸³ The process of encoding, consolidation, and retrieval conducted by the emotional and thinking brains is a learning system.

Eric R. Kandel, who won the Nobel Prize for research on the molecular foundations of learning,⁸⁴ discovered that learning takes place through the synaptic interaction of neurotransmitters and receptors.⁸⁵ Synaptic transmission, where an electrical spike travels through a neuron and triggers the release of neurotransmitters that disperse across the synaptic cleft and bind to receptors, is strengthened with use.⁸⁶ When neurons fire repeatedly, the process is called long-term potentiation (“LTP”).⁸⁷ LTP is the basis of synaptic plasticity,⁸⁸ and Canadian psychologist Donald Hebb described LTP as the process where “[c]ells that fire together wire together.”⁸⁹ The lawyer brain is continuously re-wired by experiences, thoughts, and feelings.⁹⁰

Motivation and reward also involve a complicated interplay between the lawyer’s emotional and thinking brain.⁹¹ The amygdala helps determine whether an experience is pleasurable and to be repeated, or unpleasant and avoided.⁹²

⁷⁹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 125; CARTER, MAPPING THE MIND, *supra* note 20, at 83–84.

⁸⁰ CARTER ET AL., HUMAN BRAIN, *supra* note 12; CARTER, MAPPING THE MIND, *supra* note 20, at 164.

⁸¹ CARTER ET AL., HUMAN BRAIN, *supra* note 12; SWEENEY, *supra* note 15, at 252.

⁸² CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 159.

⁸³ *Id.*; MEDINA, *supra* note 12, at 140–41; SEUNG, *supra* note 59, at 79; Daniel L. Schacter & Anthony D. Wagner, *Learning and Memory*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 1441, 1448.

⁸⁴ SWEENEY, *supra* note 15, at 236.

⁸⁵ SWEENEY, *supra* note 15, at 236.

⁸⁶ DAVID J. LINDEN, THE COMPASS OF PLEASURE 54–55 (2011).

⁸⁷ CARTER, MAPPING THE MIND, *supra* note 20, at 160; LINDEN, *supra* note 86, at 55; SWEENEY, *supra* note 15, at 248.

⁸⁸ Joseph R. Manns & Elizabeth A. Buffalo, *Learning and Memory: Brain Systems*, in FUNDAMENTAL NEUROSCIENCE, *supra* note 54, at 1029, 1029–30.

⁸⁹ LEDOUX, *supra* note 14, at 79.

⁹⁰ RATEY, *supra* note 54, at 36.

⁹¹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 128.

⁹² HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 77.

The hippocampus begins to store the memories associated with the experience, including where it took place and who was present.⁹³

The neurotransmitter most associated with reward seeking is dopamine, which is released by the ventral tegmental area (“VTA”) in the emotional brain.⁹⁴ Long neurons in the VTA extend their axon trunks to several target areas in the emotional brain: the nucleus accumbens (abbreviated as “NAC”, this structure relates to pleasure and reward); the amygdala and anterior cingulate cortex (emotional centers); the dorsal striatum (habit learning); and the hippocampus (memory processing).⁹⁵ The VTA axons also extend to the prefrontal cortex (reasoning, judgment, and planning) in the thinking brain.⁹⁶ The specialized VTA axons deliver dopamine to these emotional and thinking brain targets.⁹⁷

The VTA neurons are excited into dopamine release by neurons in the medial forebrain bundle (“MFB”), made up of the amygdala, NAC, septum, and prefrontal cortex.⁹⁸ The MFB axons dispatch the neurotransmitter glutamate to activate dopamine release in the VTA.⁹⁹ The evolutionary purpose of the mesolimbic dopamine system (hereinafter dopamine system) in the lawyer brain is to drive eating and procreation to ensure survival.¹⁰⁰

Synaptic transmission in the lawyer brain has four stages: 1) synthesis and storage of the transmitter in the pre-synaptic neuron; 2) transmitter release; 3) interaction of the transmitter with the receptors on the post-synaptic neuron; and 4) removal of the transmitter from the synaptic cleft.¹⁰¹ VTA neurons specialize in synthesis, storage, and release of dopamine.¹⁰² After release, transmitters bind to receptors and initiate changes in the post-synaptic cell.¹⁰³ Neurotransmitters are inactivated when they are removed from the synaptic cleft by reuptake back into the pre-synaptic neuron, or they are broken down by enzymes.¹⁰⁴

⁹³ *Id.*

⁹⁴ DOIDGE, *supra* note 7, at 106; *see* LINDEN, *supra* note 86, at 3–4. Dr. Arvid Carlsson of the University of Gothenburg in Sweden discovered dopamine as a neurotransmitter in 1957. He received the 2000 Nobel Prize for Medicine for his discovery. Vikram K. Yeragani et al., *Arvid Carlsson, and the Story of Dopamine*, 52 INDIAN J. PSYCHIATRY 87–88 (2010), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2824994/>.

⁹⁵ GIBB, *supra* note 25, at 176–77; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 77; LINDEN, *supra* note 86, at 16–18; SWEENEY, *supra* note 15, at 177.

⁹⁶ GIBB, *supra* note 25, at 176–77; LINDEN, *supra* note 86, at 16–18.

⁹⁷ HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 77.

⁹⁸ *Id.*; LINDEN, *supra* note 86, at 18.

⁹⁹ LINDEN, *supra* note 86, at 18.

¹⁰⁰ GIBB, *supra* note 25, at 176.

¹⁰¹ Schwartz & Javitz, *supra* note 49, at 289.

¹⁰² LINDEN, *supra* note 86, at 15–16.

¹⁰³ JERROLD S. MEYER & LINDA F. QUENZER, PSYCHOPHARMACOLOGY: DRUGS, THE BRAIN, AND BEHAVIOR 27 (2d ed. 2013).

¹⁰⁴ *Id.* at 85–86.

In order to complete communication between neurons, transmitters must bind to receptors on the dendrites of the target neurons.¹⁰⁵ Receptors have the ability to recognize specific molecule shapes of transmitters, or of drugs that mimic transmitters.¹⁰⁶ Transmitters or drugs that have the best chemical fit attach to the receptors and activate them.¹⁰⁷ These activating transmitters or drugs are called agonists.¹⁰⁸ Drugs that fit into and bind to receptors, but that do not activate them, are called receptor antagonists.¹⁰⁹ Antagonists block the receptor and prevent activation.¹¹⁰

Scientists used to describe balance in the body systems as homeostasis.¹¹¹ The newer equilibrium model, allostasis, acknowledges that the body is comprised of numerous interacting systems including the nervous, endocrine, circulation, immune, and digestive systems.¹¹² The brain coordinates body-wide changes in these systems to maintain allostasis.¹¹³ This allows energy transfer between systems, to deal with changing conditions and to build capacity for future challenges.¹¹⁴ The systems can also recalibrate in order to adjust to changes within them.¹¹⁵ Receptors respond to an absence of agonists (activator transmitters) by increasing their numbers and this receptor growth process is called up-regulation.¹¹⁶ When receptors are chronically activated by the presence of too much transmitter, they respond by reducing their numbers, known as down-regulation.¹¹⁷

One example of a recalibration is when poor diet causes persistent elevation of blood glucose and the body responds with increased insulin production.¹¹⁸ When the endocrine system learns to expect high insulin levels, it reduces the number of insulin receptors.¹¹⁹ The down-regulation of insulin receptors causes insulin resistance.¹²⁰

High levels of dopamine in the lawyer brain cause a recalibrating down-regulation of the dopamine system.¹²¹ Down-regulation of dopamine receptors

¹⁰⁵ LINDEN, *supra* note 86, at 16–17.

¹⁰⁶ MEYER & QUENZER, *supra* note 103, at 27–28.

¹⁰⁷ *Id.*

¹⁰⁸ *Id.* at 28.

¹⁰⁹ *Id.*

¹¹⁰ *Id.*

¹¹¹ RATEY & MANNING, *supra* note 67, at 233.

¹¹² *Id.*

¹¹³ SAPOLSKY, *supra* note 10, at 9.

¹¹⁴ RATEY & MANNING, *supra* note 67, at 233–36.

¹¹⁵ *Id.* at 235.

¹¹⁶ MEYER & QUENZER, *supra* note 103, at 28.

¹¹⁷ *Id.*

¹¹⁸ RATEY & MANNING, *supra* note 67, at 235–36.

¹¹⁹ *Id.* at 236.

¹²⁰ *Id.*

¹²¹ *Id.* at 237.

is caused by repeated use of drugs that artificially activate the dopamine system.¹²²

Scientists have learned about the dopamine system from studying the behavior of rodents and their willingness to forgo food, breeding, and their offspring to self-stimulate this system using electrodes or psychoactive drugs.¹²³ Extensive parallel discoveries have been made in brain research involving rodents and humans, and thus findings on the rodent dopamine system help to explain lawyer willingness to self-medicate with, and in some cases become addicted to, substances that activate the dopamine system.¹²⁴

Neuroscientist Peter Sterling described the brain's role in allostatic recalibration as a series of sticks (pain) and carrots (pleasure) that motivate adaptation.¹²⁵ Dopamine was thought to be the pleasure carrot that motivates behavior, but motivation, reward seeking, and reinforcement have a much more complex story.¹²⁶

Desire is yearning for a reward you believe will result in pleasure or satisfaction.¹²⁷ Reward seeking has three elements: motivation, learning, and pleasure.¹²⁸ Motivation is the desire to obtain an incentive, and learning is the process that occurs when action leads to acquisition of the incentive.¹²⁹ Pleasure is the subjective feeling when a reward is enjoyed.¹³⁰

Achieving a reward involves both the thinking-emotional brain learning system and the dopamine system (release of dopamine by the VTA in the NAC),¹³¹ which together forms a motivation control system.¹³² When a lawyer feels a need, such as hunger, it is registered in the VTA, which releases dopamine in the NAC.¹³³ The NAC responds by releasing an opioid-like peptide transmitter in the VTA, which forms a circuit.¹³⁴ NAC neurons prompt motor activity in the basal ganglia causing an increase in physical activity.¹³⁵ The dopamine system promotes arousal, effort, and activity to help the lawyer respond

¹²² MEYER & QUENZER, *supra* note 103, at 28.

¹²³ LINDEN, *supra* note 86, at 20; Peter B. Shizgal & Steven E. Hyman, *Homeostasis, Motivation, and Addictive States*, in *PRINCIPLES OF NEURAL SCIENCE*, *supra* note 40, at 1095, 1104.

¹²⁴ LEDOUX, *supra* note 14, at 220; LINDEN, *supra* note 86, at 16; MEYER & QUENZER, *supra* note 103, at 248.

¹²⁵ RATEY & MANNING, *supra* note 67, at 236–37.

¹²⁶ WILLIAM A. MCKIM & STEPHANIE D. HANCOCK, *DRUGS AND BEHAVIOR: AN INTRODUCTION TO BEHAVIORAL PHARMACOLOGY* 115 (7th ed. 2013).

¹²⁷ CARTER ET AL., *HUMAN BRAIN*, *supra* note 12, at 128; PURVES ET AL., *supra* note 54, at 472.

¹²⁸ MEYER & QUENZER, *supra* note 103, at 250.

¹²⁹ *Id.*

¹³⁰ *Id.*

¹³¹ MCKIM & HANCOCK, *supra* note 126, at 115–19.

¹³² *Id.* at 115.

¹³³ *Id.*

¹³⁴ *Id.*

¹³⁵ *Id.*

to his hunger.¹³⁶ At the same time, the learning system is receiving information from the senses, which is sorted by the thalamus and directed to the amygdala and hippocampus.¹³⁷ Here, memory and past action are considered in evaluating stimuli for potential in acquiring the incentive.¹³⁸

If the lawyer was a young hungry coyote, the activation of his dopamine system would prompt an increase in motor activity.¹³⁹ Increased activity might result in finding an unsuspecting prairie dog by accident.¹⁴⁰ When the coyote happens upon the prairie dog, the VTA releases more dopamine in the NAC and the location of the prairie dog field is registered in the thinking-emotional brain (cortex-hippocampus) memory loop.¹⁴¹ The prairie dog field acquires incentive salience for the coyote, which means that future hunger-inspired dopamine system activation will prompt a return visit to the prairie dog field.¹⁴² If the coyote wanders by the field when he is not hungry, the dopamine system will be activated not by hunger, but by the prairie dog field stimulus.¹⁴³

The role of dopamine, once thought to be responsible for the subjective feeling of pleasure,¹⁴⁴ has been challenged by experiments that manipulate dopamine.¹⁴⁵ In rodent studies, genetically altered dopamine-deficient mice are able to learn stimulus-reward associations and mice that have prolonged dopamine in the synapses do not learn any faster than normal mice.¹⁴⁶ Parkinson's patients, who suffer from a lack of dopamine, do not report less pleasure when eating sweet foods; nor does administering dopamine to their NAC increase their feelings of pleasure.¹⁴⁷ And experienced cocaine users, who were treated with a dopamine-depleting drug prior to cocaine use, reported reduced craving for cocaine, but no reduction in cocaine-induced euphoria.¹⁴⁸ The role of dopamine appears to be a consequence of associative learning, responsible for motivation, but not sensations of pleasure.¹⁴⁹

Current research indicates that pleasure in the rodent motivation control system seems to be located in tiny hedonic hotspots in the NAC and the ventral

¹³⁶ *Id.*

¹³⁷ *Id.*

¹³⁸ *Id.*

¹³⁹ *Id.* at 116.

¹⁴⁰ *Id.*

¹⁴¹ *Id.* at 116–17.

¹⁴² *Id.* at 117.

¹⁴³ *Id.*

¹⁴⁴ MEYER & QUENZER, *supra* note 103, at 250–51.

¹⁴⁵ MCKIM & HANCOCK, *supra* note 126, at 117; MEYER & QUENZER, *supra* note 103, at 251.

¹⁴⁶ MCKIM & HANCOCK, *supra* note 126, at 117.

¹⁴⁷ *Id.* at 118.

¹⁴⁸ MEYER & QUENZER, *supra* note 103, at 251.

¹⁴⁹ MCKIM & HANCOCK, *supra* note 126, at 117; MEYER & QUENZER, *supra* note 103, at 251.

pallidum.¹⁵⁰ In rodents, facial responses to sweet (rewarding) flavors can be enhanced by opioids and cannabinoids in these hot spots.¹⁵¹ Stimulation of the hotspots is not dependent on dopamine.¹⁵² Dopamine appears to be critical to motivation and the reinforcing effects of rewards,¹⁵³ while opioid receptors in the ventral pallidum seem to stimulate pleasure in the lawyer brain.¹⁵⁴ The lawyer brain produces naturally-occurring opioids and endocannabinoids, which would explain the subjective pleasure experience associated with drug use.¹⁵⁵

III. THE STRESSED LAWYER BRAIN

Americans suffer from overwork. Research shows that 37.8 percent of professional men and 14.4 percent of professional women work more than fifty hours a week.¹⁵⁶ Overwork is a practice that lawyers likely adopt during law school, where success is defined in terms of grades, class rank, and selection to serve on journals.¹⁵⁷ Lawyers suffer from higher levels of anxiety and depression than the rest of the population,¹⁵⁸ but most do not enter law school with these mental health issues.¹⁵⁹ Depression is an emotional state of having too few positive emotions along with too many negative ones, often coupled with feelings of intense grief and guilt.¹⁶⁰ According to the Centers for Disease Control, lawyers rank fourth in professions with the highest number of suicides.¹⁶¹ Lawyer anxiety, depression, and suicide rates are likely linked to overwork and chronic stress.¹⁶²

The six primary emotions recognized by psychologists are fear, anger, sadness, disgust, surprise, and joy.¹⁶³ Emotions are automatic and unconscious physiological responses to stimuli that cause physical changes like blushing,

¹⁵⁰ MCKIM & HANCOCK, *supra* note 126, at 118; MEYER & QUENZER, *supra* note 103, at 251; Kyle S. Smith et al., *Ventral Pallidum Roles in Reward and Motivation*, 196 BEHAV. BRAIN RES. 155, 156–57 (2009).

¹⁵¹ MCKIM & HANCOCK, *supra* note 126, at 118–19; MEYER & QUENZER, *supra* note 103, at 251.

¹⁵² MCKIM & HANCOCK, *supra* note 126, at 118.

¹⁵³ *Id.* at 119.

¹⁵⁴ Smith et al., *supra* note 150, at 158.

¹⁵⁵ MCKIM & HANCOCK, *supra* note 126, at 318; MEYER & QUENZER, *supra* note 103, at 407.

¹⁵⁶ JULIAN FORD & JON WORTMANN, HIJACKED BY YOUR BRAIN: HOW TO FREE YOURSELF WHEN STRESS TAKES OVER 18–19 (2013).

¹⁵⁷ LEVIT & LINDER, *supra* note 1, at 125.

¹⁵⁸ See LEVIT & LINDER, *supra* note 1; LITOWITZ, *supra* note 1; MCCLURG, *supra* note 1; NERISON, *supra* note 1; Krieger, *supra* note 1; Rosen, *supra* note 1.

¹⁵⁹ Krieger, *supra* note 1, at 114.

¹⁶⁰ SAPOLSKY, *supra* note 10, at 273.

¹⁶¹ Flores & Arce, *supra* note 3.

¹⁶² LEVIT & LINDER, *supra* note 1, at 7.

¹⁶³ SWEENEY, *supra* note 15, at 208.

sweaty palms, and increased heart rate and blood pressure.¹⁶⁴ Feelings are conscious perceptions of automatic emotional responses.¹⁶⁵ Stress involves some combination of negative emotions.¹⁶⁶

The autonomic nervous system works with the lawyer's brain to maintain allostatic balance.¹⁶⁷ The two divisions of the autonomic system maintain this stability: the sympathetic nervous system ("SNS") and the parasympathetic nervous system ("PNS").¹⁶⁸ The SNS is commonly known as the fight-or-flight system and the PNS as the rest-and-digest system.¹⁶⁹ The fight-or-flight arousal system is the body's accelerator and the rest-and-digest calming system is its brake.¹⁷⁰

The lawyer fight-or-flight SNS was designed to produce rapid response when escape from predators was a priority.¹⁷¹ This system is activated during acute stress, which is short-lived and potentially helpful in dealing with short-term challenges, such as a court hearing or a 5K run.¹⁷² However, the SNS can be perpetually activated during long-term chronic stress, such as job loss, financial problems, troubled family relationships, or possibly law school.¹⁷³

The fight-or-flight SNS response starts in the lawyer's emotional brain, and is ignited by the brain's "panic button," the amygdala.¹⁷⁴ The amygdala initiates the quick and dirty route to SNS activation by signaling the thalamus to focus attention and the hypothalamus to release stress hormones.¹⁷⁵ The two primary stress hormones are adrenaline (also known as epinephrine) and glucocorticoids (the main glucocorticoid is cortisol).¹⁷⁶ Stress hormones elevate heart

¹⁶⁴ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 124; GIBB, *supra* note 25, at 96; SWEENEY, *supra* note 15, at 208; Joseph E. LeDoux & Antonio R. Damasio, *Emotions and Feelings*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 1079, 1079.

¹⁶⁵ LeDoux & Damasio, *supra* note 164.

¹⁶⁶ See CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 232; CARTER, MAPPING THE MIND, *supra* note 20, at 96–97.

¹⁶⁷ Terry L. Powley, *Central Control of Autonomic Functions: Organization of the Autonomic Nervous System*, in FUNDAMENTAL NEUROSCIENCE, *supra* note 54, at 729, 729; see John P. Horn & Larry W. Swanson, *The Autonomic Motor System and the Hypothalamus*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 1056, 1066–67.

¹⁶⁸ PURVES ET AL., *supra* note 54, at 326.

¹⁶⁹ PURVES ET AL., *supra* note 54, at 326.

¹⁷⁰ LINDA GRAHAM, BOUNCING BACK: REWIRING YOUR BRAIN FOR MAXIMUM RESILIENCE AND WELL-BEING 201 (2013).

¹⁷¹ Byron A. Campbell et al., *Origins of Orienting and Defensive Responses: An Evolutionary Perspective*, in ATTENTION AND ORIENTING: SENSORY AND MOTIVATIONAL PROCESSES (Peter J. Lang et al. eds., 2011).

¹⁷² PERLMUTTER & VILLOLDO, *supra* note 25, at 59.

¹⁷³ PERLMUTTER & VILLOLDO, *supra* note 25, at 59; see LITOWITZ, *supra* note 1, at 10, 19.

¹⁷⁴ RATEY, *supra* note 54, at 62.

¹⁷⁵ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 125; GREENFIELD, *supra* note 77, at 18; RICK HANSON, BUDDHA'S BRAIN: THE PRACTICAL NEUROSCIENCE OF HAPPINESS, LOVE, & WISDOM 52 (2009).

¹⁷⁶ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 232; MEDINA, *supra* note 12, at 174; PERLMUTTER & VILLOLDO, *supra* note 25, at 60; SWEENEY, *supra* note 15, at 40; Andrea C.

rate, raise blood pressure, mobilize energy, slow digestion, and suppress the immune system.¹⁷⁷ Fight-or-flight SNS activation is meant to help solve short-term problems, like the evasive action required to escape harm.¹⁷⁸ Chronic stress causes long-term elevated levels of glucocorticoids, causing the following serious health problems: a compromised immune system; increased appetite and body fat; decreased muscle mass and bone density; and increased irritability, anger, anxiety, and depression.¹⁷⁹

Lawyer stress can be described as a physiological response to a stressor: 1) that is measurable by another party, 2) that the lawyer perceives as negative, and 3) that the lawyer feels she has no control over.¹⁸⁰ *The Carnegie Report*, the most recent critique of legal education, states that chronic law student stress is caused by the grade curve and competitive learning atmosphere.¹⁸¹ Stressors continue in law practice and include tight deadlines, adversarial environments, and win-lose high stakes outcomes.¹⁸² Bar examiners frequently ask about mental health and addiction issues on bar applications, and three states (Georgia, Minnesota, and North Carolina) suggest that lawyer stress starts in law school.¹⁸³

Damage from chronic stress extends to the lawyer brain. Activation of the fight-or-flight SNS causes circular damage in the emotional brain because cortisol causes the amygdala to produce additional cortisol and the hippocampus,

Gore, *Neuroendocrine Systems*, in *FUNDAMENTAL NEUROSCIENCE*, *supra* note 54, at 799, 804; Steven E. Hyman & Jonathan D. Cohen, *Disorders of Mood and Anxiety*, in *PRINCIPLES OF NEURAL SCIENCE*, *supra* note 40, at 1402, 1409. The pituitary gland and the hypothalamus in the emotional brain tell the adrenal gland sitting atop the kidneys to release adrenalin and glucocorticoids. CARTER ET AL., *HUMAN BRAIN*, *supra* note 12, at 232; PERLMUTTER & VILLOLDO, *supra* note 25, at 60.

¹⁷⁷ PERLMUTTER & VILLOLDO, *supra* note 25, at 60; Gore, *supra* note 176, at 804; *see* SAPOLSKY, *supra* note 10, at 13.

¹⁷⁸ MEDINA, *supra* note 12, at 175.

¹⁷⁹ *See* SAPOLSKY, *supra* note 10, at 13; SHAWN M. TALBOTT, *THE CORTISOL CONNECTION* 22 (2007).

¹⁸⁰ MEDINA, *supra* note 12, at 173–74 (Jeansok Kim and David Diamond developed this three-part definition of stress).

¹⁸¹ SULLIVAN ET AL., *supra* note 8, at 31.

¹⁸² LEVIT & LINDER, *supra* note 1, at 7.

¹⁸³ Rebecca S. Mick, *Making the Mark—Character and Fitness for Admission to the Bar*, GA. B. ADMISSIONS 8, <https://www.gabaradmissions.org/making-the-mark> (last visited Apr. 12, 2015) (“The Board understands that law school and life in general can be stressful and may result in an applicant’s seeking counseling or other treatment.”); *Character and Fitness for Admission to the Bar*, MINN. ST. BD. L. EXAMINERS (Oct. 2014), <http://www.ble.state.mn.us/resource-center/character-and-fitness-for-admission-to-bar.aspx> (“Bar examiners recognize that the stresses of law school and other life factors may result in an applicant suffering from psychological problems.”); *Character and Fitness Guidelines*, N.C. BD. L. EXAMINERS, <http://www.ncble.org/character-fitness/> (last visited Apr. 12, 2015) (“Board members recognize that the stresses of law school, as well as other life factors, frequently result in applicants seeking psychiatric or psychological counseling.”).

normally responsible for calming the amygdala, is suppressed.¹⁸⁴ The amygdala is over-sensitized and the hippocampus is compromised.¹⁸⁵

Where the SNS stimulates arousal, defense, and escape, the rest-and-digest PNS promotes nourishment, procreation, and the return to allostatic balance after SNS activation.¹⁸⁶ The PNS slows the heart rate, lowers blood pressure, promotes digestion, and suppresses the release of adrenaline.¹⁸⁷ Law students and lawyers benefit from learning how to enhance the rest-and-digest PNS.

Many law students and lawyers spend months and years in fight-or-flight SNS overdrive, believing their performance is improved by the adrenalin rush.¹⁸⁸ This plight may be fueled by caffeine and other substances.¹⁸⁹ Neuroscientists have proven that cognitive performance is diminished during flight-or-flight SNS arousal.¹⁹⁰ The impact of stress on lawyer brain cognition includes degradation of the following functions: concentration, memory, problem-solving, language processing, and math performance.¹⁹¹

The hippocampus, the brain's memory processor, is highly susceptible to stress because it has abundant glucocorticoid receptors.¹⁹² Glucocorticoids damage and kill cells in the hippocampus and weaken synaptic connections.¹⁹³ Damage to the hippocampus can create a harmful cycle where greater glucocorticoid release produces additional hippocampal atrophy.¹⁹⁴ Brain scans show hippocampi shrink in size in people who experience post-traumatic stress disorder, major depression, and repeated jet lag, and right hippocampus volume is reduced in young chronic cannabis users.¹⁹⁵ A common response to chronic stress is self-medication because drugs of abuse can make a lawyer feel less stressed.¹⁹⁶

¹⁸⁴ HANSON, *supra* note 175, at 52–53; RATEY, *supra* note 54, at 66–67.

¹⁸⁵ HANSON, *supra* note 175, at 57.

¹⁸⁶ David G. Amaral & Peter L. Strick, *The Organization of the Central Nervous System*, in *PRINCIPLES OF NEURAL SCIENCE*, *supra* note 40, at 337, 353; Horn & Swanson, *supra* note 167, at 1066.

¹⁸⁷ BROAD, *supra* note 54, at 90; SWEENEY, *supra* note 15, at 41; Powley, *supra* note 167, at 734.

¹⁸⁸ GAYATRI DEVI, *A CALM BRAIN: HOW TO RELAX INTO A STRESS-FREE, HIGH-POWERED LIFE* 6 (2012).

¹⁸⁹ *Id.* at 15–16.

¹⁹⁰ HANSON, *supra* note 175, at 52–60; RATEY, *supra* note 54, at 67–71; *see* DEVI, *supra* note 188, at 83–86.

¹⁹¹ MEDINA, *supra* note 12, at 178.

¹⁹² *Id.* at 177; Gore, *supra* note 176, at 804.

¹⁹³ SANDRA AAMODT & SAM WANG, *WELCOME TO YOUR BRAIN* 86 (2008); AMEN, *supra* note 22, at 248; DOIDGE, *supra* note 7; MEDINA, *supra* note 12, at 179; DAVID A. SOUSA, *HOW BRAIN SCIENCE CAN MAKE YOU A BETTER LAWYER* 25 (2009); Gore, *supra* note 176, at 804.

¹⁹⁴ SAPOLSKY, *supra* note 10, at 387.

¹⁹⁵ MCKIM & HANCOCK, *supra* note 126, at 322; SAPOLSKY, *supra* note 10, at 221.

¹⁹⁶ SAPOLSKY, *supra* note 10.

IV. THE SELF-MEDICATED LAWYER BRAIN

The broadest definition of the term drug is “any substance that alters the physiology of the body,” which can include food.¹⁹⁷ Some drugs serve as nutrients, some treat disorders, and some are consumed to produce a high.¹⁹⁸

Lawyers suffer from anxiety and depression at rates that are higher than the rest of the population.¹⁹⁹ Some of them turn to antidepressants for relief and others may self-medicate with drugs of abuse or food.

A. Antidepressants

Antidepressants improve depression in approximately two-thirds of people who try them.²⁰⁰ A study of the placebo effect in fifty-one people with major depression found that 52 percent receiving antidepressant medications and 38 percent receiving placebos responded positively to treatment.²⁰¹ Factors that improve the placebo expectancy effect include a good relationship with the doctor, positive expectations of the medicine, and a desire to feel better.²⁰² The results suggest that depression can be improved in multiple ways because placebo-takers showed an increase in thinking brain prefrontal cortex activity, while the drug-takers showed decreased activity in the same region.²⁰³ Because antidepressants reduce anxiety in depressed patients, they are also being prescribed to treat anxiety that is unrelated to depression.²⁰⁴

Monoamine transmitters are synthesized from amino acids and include norepinephrine, dopamine, and serotonin.²⁰⁵ Scientists believe that depression is linked to low levels of serotonin.²⁰⁶ When neurotransmitters are released, the presynaptic neuron keeps the communication signal clean and precise by clearing excess transmitter from the synaptic gap.²⁰⁷ Removal and recycling of the transmitter is called reuptake, when the transmitter is returned to the presynaptic neuron via a transporter.²⁰⁸ Neurotransmitters can also be degraded and

¹⁹⁷ MCKIM & HANCOCK, *supra* note 126, at 1.

¹⁹⁸ *Id.*

¹⁹⁹ See LEVIT & LINDER, *supra* note 1, at 6; MCCLURG, *supra* note 1; Krieger, *supra* note 1; cf. Rosen, *supra* note 1, at 162 (commenting on how lawyers' depression appears to originate in law school).

²⁰⁰ GIBB, *supra* note 25, at 187; MEYER & QUENZER, *supra* note 103, at 563.

²⁰¹ GIBB, *supra* note 25, at 188.

²⁰² *Id.*

²⁰³ *Id.*

²⁰⁴ MEYER & QUENZER, *supra* note 103, at 564.

²⁰⁵ George B. Richerson et al., *The Modulatory Functions of the Brain Stem*, in PRINCIPLES OF NEURAL SCIENCE, *supra* note 40, at 1038, 1040.

²⁰⁶ MEYER & QUENZER, *supra* note 103, at 554–56.

²⁰⁷ GIBB, *supra* note 25, at 189.

²⁰⁸ NEIL R. CARLSON, PHYSIOLOGY OF BEHAVIOR 58 (11th ed. 2013).

flushed out.²⁰⁹ Proper disposal of neurotransmitters is critical to neuron communication.²¹⁰

All antidepressants increase the function of serotonin or norepinephrine, or both.²¹¹ Selective serotonin reuptake inhibitors (“SSRIs”) work by leaving more serotonin in the synapse.²¹² Monoamine oxidase A (“MAO-A”) is a protein that degrades serotonin, norepinephrine, and dopamine in the synapse, thus monoamine oxidase A inhibitors (“MAOIs”) work by blocking the destruction of serotonin, norepinephrine, and dopamine.²¹³

SSRIs block the reuptake of serotonin by the releasing neuron, making more serotonin available to stimulate the post-synaptic neuron.²¹⁴ It takes several weeks for the brain to adapt to the new serotonin levels, but once it does, the lawyer’s mood improves.²¹⁵ Long-term treatment with SSRIs increases neurogenesis, the birth of new brain cells in the hippocampus, which may be influential to mood enhancement.²¹⁶ It also increases production of brain-derived neurotrophic factor (“BDNF”), an important neurotrophin that regulates neuron survival and synaptic changes in the lawyer brain.²¹⁷ Antidepressants prevent stress-related BDNF reduction and neuron damage.²¹⁸

With all the SSRIs except Prozac, there is an increased risk of suicide.²¹⁹ They also cause sexual dysfunction, and withdrawal from SSRIs can cause headaches, diarrhea, and aggression.²²⁰

MAOIs increase serotonin, norepinephrine, and dopamine in the synapse by reducing transmitter extinction.²²¹ MAOIs have serious side effects including insomnia, weight gain, and elevated blood pressure.²²² MAOIs impact all systems in the body, thus some foods (certain cheeses, meats, and pickled products) and other drugs (cold medications, aspirin, amphetamine, alcohol, cocaine, opioids, and barbiturates) must be avoided because they could cause life-threatening high blood pressure or stroke.²²³

²⁰⁹ SAPOLSKY, *supra* note 10, at 278.

²¹⁰ *Id.*

²¹¹ MEYER & QUENZER, *supra* note 103, at 553.

²¹² GIBB, *supra* note 25, at 190; MEYER & QUENZER, *supra* note 103, at 556.

²¹³ GIBB, *supra* note 25, at 189–90.

²¹⁴ *Id.* at 190–91.

²¹⁵ *Id.*

²¹⁶ *Id.*

²¹⁷ MEYER & QUENZER, *supra* note 103, at 559.

²¹⁸ *Id.*

²¹⁹ GIBB, *supra* note 25, at 190.

²²⁰ *Id.* at 191.

²²¹ MEYER & QUENZER, *supra* note 103, at 564–65.

²²² *Id.* at 564.

²²³ *Id.*

B. *Drugs of Abuse*

The National Survey on Drug Use and Health (“NSDUH”) interviews about seventy thousand randomly-selected Americans who are twelve years or older each year.²²⁴ In 2012, 82 percent of Americans reported having used alcohol, 62 percent reported having smoked cigarettes, and 42 percent reported having used marijuana or hashish at least once.²²⁵

Illicit drug use is increasing among Americans. In the 2010 NSDUH study, approximately 22.6 million Americans age twelve or older had used an illicit drug in the last month.²²⁷ That represents 8.9 percent of Americans in that age range.²²⁸ In the 2012 NSDUH study, approximately 23.9 million Americans, or 9.2 percent of the U.S. population age twelve and older, used an illicit drug in the last month.²²⁹ Table 2 shows the percentage of 2012 NSDUH study participants who have used a drug of abuse at least once.

Lawyer goal-directed behavior involves the evaluation of the risks, costs, and benefits of any activity.²³⁰ Motivation and reward-seeking have a neural basis in the emotional and thinking brains.²³¹ Motivation is a desire to obtain a

TABLE 2: DRUG USE STATISTICS²²⁶

Type of Drug	% Reporting Use
Alcohol	82.30
Cigarettes	61.90
Smokeless Tobacco	17.70
Marijuana/Hashish	42.80
Pain Relievers	14.20
Stimulants	8.30
Cocaine	14.50
MDMA	6.20
Methamphetamine	4.70
Heroin	1.80

²²⁴ *About the Survey*, NAT’L SURV. ON DRUG USE & HEALTH, https://nsduhweb.rti.org/respweb/project_description.html (last visited May 7, 2015).

²²⁵ CTR. FOR BEHAVIORAL HEALTH STATISTIC & QUALITY, RESULTS FROM THE 2012 NAT’L SURVEY ON DRUG USE & HEALTH, <http://archive.samhsa.gov/data/NSDUH/2012SummNatFindDetTables/DetTabs/NSDUH-DefTabsSect1peTabs1to46-2012.htm#Tab1.1B> (last visited May 29, 2015); CTR. FOR BEHAVIORAL HEALTH STATISTIC & QUALITY, RESULTS FROM THE 2012 NAT’L SURVEY ON DRUG USE AND HEALTH, <http://archive.samhsa.gov/data/NSDUH/2012SummNatFindDetTables/DetTabs/NSDUH-DefTabsSect2peTabs1to42-2012.htm#Tab2.1B> (last visited May 29, 2015).

²²⁶ *Nat’l Survey of Drug Use and Health*, NAT’L INST. ON DRUG ABUSE, <http://www.drugabuse.gov/national-survey-drug-use-health> (last visited May 7, 2015).

²²⁷ CTR. FOR BEHAVIORAL HEALTH STATISTIC & QUALITY, U.S. DEP’T OF HEALTH & HUMAN SERVS., PUB. NO. (SMA) 11-4658, RESULTS FROM THE 2010 NAT’L SURVEY ON DRUG USE AND HEALTH: SUMMARY OF NAT’L FINDINGS 11 (2011), *available at* <http://www.oas.samhsa.gov/NSDUH/2k10NSDUH/2k10Results.pdf>.

²²⁸ *Id.*

²²⁹ CTR. FOR BEHAVIORAL HEALTH STATISTIC & QUALITY, U.S. DEP’T OF HEALTH & HUMAN SERVS., PUB. NO. (SMA) 13-4795, RESULTS FROM THE 2012 NAT’L SURVEY ON DRUG USE AND HEALTH: SUMMARY OF NAT’L FINDINGS 13 (2013), *available at* <http://www.samhsa.gov/data/NSDUH/2012SummNatFindDetTables/NationalFindings/NSDUHresults2012.pdf>.

²³⁰ Shizgal & Hyman, *supra* note 123, at 1103.

²³¹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 128.

reward.²³² A reward is an object, stimulus, or activity that has positive value and gives a lawyer pleasure.²³³ Some law students, law faculty, lawyers, and judges self-medicate with drugs of abuse as a response to stress, anxiety, and/or depression. The lawyer should understand what is known about the actions and impacts of mixed action drugs (alcohol, nicotine, cannabis), stimulants, opiates, sedatives, hallucinogens, and some foods on the lawyer brain.

Drugs that bind to and activate receptors are called agonists and drugs that bind to and block receptors are called antagonists.²³⁴ Some drugs are agonists because they mimic naturally-occurring endogenous substances in the lawyer brain.²³⁵ Neuroscientists are in the early stages of understanding the impacts of psychoactive drugs and various addictions.²³⁶ Most addictive substances hijack the dopamine system.²³⁷ The artificial activation of the dopamine system, and intense euphoria that accompanies use of drugs of abuse, can become the basis of addiction.²³⁸ Table 3 provides a taxonomy of the major classes of addictive drugs and their endogenous natural analogs.

1. *Mixed Action Drugs*

Mixed action drugs produce several different effects in the lawyer brain, and they include alcohol, nicotine, and cannabis.²³⁹

a. *Alcohol*

Alcohol is the second most widely used drug in America, surpassed only by caffeine.²⁴⁰ William Burroughs called alcohol “[o]ur national drug” in his 1959 novel *Naked Lunch*,²⁴¹ and it is our most abused drug.²⁴²

The active ingredient in all alcoholic beverages is ethanol and its effects on the lawyer brain are quite complex.²⁴³ Alcohol is a mixed action drug because at low doses it begins as a stimulant that results in mild euphoria and has an anxiolytic impact, reducing the discomfort of anxiety.²⁴⁴ Later it has a depressant effect on neural activity.²⁴⁵ In higher amounts, it causes incoordination and sedation.²⁴⁶

²³² MEYER & QUENZER, *supra* note 103, at 250.

²³³ Shizgal & Hyman, *supra* note 123, at 1104; *see* LINDEN, *supra* note 86, at 18.

²³⁴ PURVES ET AL., *supra* note 54, at 25.

²³⁵ *See* LINDEN, *supra* note 86, at 44.

²³⁶ MEYER & QUENZER, *supra* note 103, at 250.

²³⁷ DOIDGE, *supra* note 7, at 106.

²³⁸ LINDEN, *supra* note 86, at 20.

²³⁹ LINDEN, *supra* note 86, at 41.

²⁴⁰ MEYER & QUENZER, *supra* note 103, at 266.

²⁴¹ WILLIAM S. BURROUGHS, *NAKED LUNCH* 224 (2001).

²⁴² MEYER & QUENZER, *supra* note 103, at 266.

²⁴³ GIBB, *supra* note 25, at 184–85.

²⁴⁴ CARLSON, *supra* note 208, at 631; WATSON & BREEDLOVE, *supra* note 29, at 95.

²⁴⁵ WATSON & BREEDLOVE, *supra* note 29, at 95.

²⁴⁶ CARLSON, *supra* note 208, at 631.

TABLE 3: DRUG TAXONOMY²⁴⁷

Drug	Source	Endogenous Analogs
<i>Mixed Action Drugs</i>		
Alcohol		
Nicotine	Tobacco	Acetylcholine
Marijuana	Cannabis	Endocannabinoids
<i>Stimulants</i>		
Caffeine	Trimethylxanthine	Adenosine
Cocaine	Coca Leaf	
Amphetamine	Synthetic	
<i>Opiates</i>		
Morphine, Methadone, Oxycodone, Heroin	Opium Poppy	Endorphins
OxyContin, Fentanyl	Synthetic	
<i>Sedatives</i>		
Barbiturates	Synthetic	
Benzodiazepines	Synthetic	
<i>Hallucinogens</i>		
LSD		
Phencyclidine (“PCP”)	Synthetic	

Humans have been producing alcoholic beverages for over five thousand years²⁴⁸ using fermentation, a process where microscopic yeast cells fall from the air onto any substance containing sugar, such as honey, fruit, or grains.²⁴⁹ Fermentation of grapes yields wine, rice produces sake, and grains create beer.²⁵⁰ Fermentation continues until the yeast dies and the alcohol content is about 15 percent.²⁵¹ Distillation is required to reach a higher alcohol content.²⁵² The fermented mixture is heated and the alcohol vapor is cooled in a still and

²⁴⁷ LINDEN, *supra* note 86, at 40–41, 44; Shizgal & Hyman, *supra* note 123, at 1106.

²⁴⁸ GIBB, *supra* note 25, at 185.

²⁴⁹ MEYER & QUENZER, *supra* note 103, at 267; WATSON & BREEDLOVE, *supra* note 29, at 95.

²⁵⁰ MEYER & QUENZER, *supra* note 103, at 267.

²⁵¹ *Id.*

²⁵² *Id.*

collected as distilled spirits or hard liquor such as whiskey, rum, and tequila.²⁵³ Alcohol concentrations of hard liquor typically consumed in the United States are 40 percent to 50 percent.²⁵⁴

The impact of alcohol is described in terms of blood alcohol concentration (“BAC”).²⁵⁵ The Centers for Disease Control and Prevention describes impacts on driving by BAC level:

- BAC at 0.02 percent
 - Decline in visual tracking of moving objects
 - Decline in the ability to perform two tasks at once
- BAC at 0.05 percent
 - Reduced coordination
 - Trouble steering
 - Reduced ability to track moving objects
 - Reduced response to emergency driving situations
- BAC at 0.08 percent
 - Impaired perception, concentration, and information processing
 - Impaired judgment and reasoning
 - Less control over speed
 - Short-term memory loss.²⁵⁶

The National Transportation Safety Board has proposed lowering the legal driving limit from a BAC of 0.08 percent to 0.05 percent to reduce alcohol-related accidents and deaths.²⁵⁷

It has been thirty years since Congress passed the National Minimum Age Drinking Act, which prohibits people under twenty-one years of age from purchasing or possessing alcohol, and withholds federal highway funding from any state that does not conform to the minimum drinking age.²⁵⁸ The U.S. Department of Transportation estimates that 525 lives were saved in 2012 because of the minimum drinking age law.²⁵⁹ Impaired driving in 2012 caused more than ten thousand deaths, which is a rate of one death every fifty-one minutes.²⁶⁰

²⁵³ *Id.*

²⁵⁴ *Id.*

²⁵⁵ *Id.* at 268.

²⁵⁶ *Effects of Blood Alcohol Concentration (BAC)*, CTRS. FOR DISEASE CONTROL & PREVENTION, http://www.cdc.gov/Motorvehiclesafety/Impaired_Driving/bac.html (last updated Jan. 13, 2015).

²⁵⁷ Mike M. Ahlers, *Tougher Drunk-Driving Threshold Proposed to Reduce Traffic Deaths*, CNN (May 15, 2013, 6:36 AM), <http://www.cnn.com/2013/05/14/us/ntsb-blood-alcohol/>.

²⁵⁸ 23 U.S.C. § 158 (2012).

²⁵⁹ NAT'L HIGHWAY TRAFFIC SAFETY ADMIN., U.S. DEP'T OF TRANSP., LIVES SAVED IN 2012 BY RESTRAINT USE AND MINIMUM DRINKING AGE LAWS 1 (2013), *available at* <http://www-nrd.nhtsa.dot.gov/Pubs/811851.pdf>. Estimate of lives saved in 2011 is 535, in 2010 is 552, in 2009 is 626, and in 2008 is 716. *Id.*

²⁶⁰ *Impaired Driving*, NAT'L HIGHWAY TRAFFIC SAFETY ADMIN., <http://www.nhtsa.gov/Impaired> (last visited May 7, 2015).

The National Institute on Alcohol Abuse and Alcoholism defines binge drinking as a pattern of drinking that brings the BAC up to 0.08 percent within two hours, which is about four drinks for women and five drinks for men.²⁶¹ From 2010 to 2012, the national average for people ages twelve to twenty of alcohol binge use was 15.9 percent within thirty days of the survey, but the range from different geographical areas of the U.S. was from 9.17 percent to 46.32 percent.²⁶² About four out of five college students drink, and two out of five participate in binge drinking.²⁶³ Students who drink risk injury, academic problems, health problems, and death.²⁶⁴ Other students are impacted by drinkers when they become victims of assault or sexual abuse.²⁶⁵

Binge drinking practices from undergraduate life may follow students into law school and law practice. One study found that where minimum drinking ages were lower than twenty-one years, adult males had significantly higher rates of alcohol use and were involved in more traffic fatalities.²⁶⁶ Binge drinking has been correlated with general anxiety disorders, especially for men.²⁶⁷ Binge drinking and alcohol overdose can be lethal, as demonstrated by the recent alcohol poisoning death of English singer Amy Winehouse in 2011.²⁶⁸ Drinking problems may begin in law school, where school events may teach students “to drink like a lawyer.”²⁶⁹ Lawyers may drink to relieve stress and one study estimated that 15 percent of lawyers are alcoholics.²⁷⁰

Alcohol is bioavailable, readily absorbed from the gastrointestinal (“GI”) tract, distributed by the blood, and introduced into the brain.²⁷¹ The neurochem-

²⁶¹ *Drinking Levels Defined*, NAT’L INST. ON ALCOHOL ABUSE & ALCOHOLISM, <http://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking> (last visited May 7, 2015).

²⁶² CTR. FOR BEHAVIORAL HEALTH STATISTICS & QUALITY, U.S. DEP’T OF HEALTH & HUMAN SERVS., *THE NSDUH REPORT: UNDERAGE BINGE ALCOHOL USE VARIES WITHIN AND ACROSS STATES 2* (2014), available at <http://www.samhsa.gov/data/2K14/NSDUH199/sr199-underage-binge-drinking-2014.pdf>.

²⁶³ *College Drinking*, NAT’L INST. ON ALCOHOL ABUSE & ALCOHOLISM, <http://www.niaaa.nih.gov/alcohol-health/special-populations-co-occurring-disorders/college-drinking> (last visited May 7, 2015).

²⁶⁴ *Id.*

²⁶⁵ *Id.*

²⁶⁶ Robert Kaestner & Benjamin Yarnoff, *Long-Term Effects of Minimum Legal Drinking Age Laws on Adult Alcohol Use and Driving Fatalities*, 54 J.L. & ECON. 365, 367 (2011).

²⁶⁷ James A. Cranford et al., *Substance Use Behaviors, Mental Health Problems, and Use of Mental Health Services in a Probability Sample of College Students*, 34 ADDICTIVE BEHAVS. 134, 142 (2009).

²⁶⁸ MCKIM & HANCOCK, *supra* note 126, at 152.

²⁶⁹ MCCLURG, *supra* note 1, at 321.

²⁷⁰ Bridget A. Maloney, Note, *Distress Among the Legal Profession: What Law Schools Can Do About It*, 15 NOTRE DAME J.L. ETHICS & PUB. POL’Y 307, 320 (2001).

²⁷¹ MEYER & QUENZER, *supra* note 103, at 268.

ical effects of alcohol are complex because alcohol acts on multiple neurotransmitters: GABA, glutamate, dopamine, and endorphins.²⁷²

Alcohol binds to neurons that allow the amplification of the neurotransmitter GABA.²⁷³ Alcohol is an indirect agonist at the GABA receptors, increasing the post-synaptic response to GABA.²⁷⁴ The calming and sedating effects of alcohol are due to increased GABA,²⁷⁵ which is an inhibitory transmitter responsible for slowing or stopping synaptic firing.²⁷⁶ GABA is the synaptic transmission brake.²⁷⁷ The result is some degree of drunkenness producing reduced anxiety, social disinhibition, poor motor control, and sensory problems.²⁷⁸

Alcohol also interferes with the transmission of glutamate.²⁷⁹ Glutamate is a major excitatory transmitter.²⁸⁰ Alcohol acutely inhibits glutamate by reducing its effectiveness, even at a social drinker BAC level of 0.03 percent.²⁸¹ Alcohol, like other glutamate antagonists, impairs learning and memory, and disrupts long-term potentiation.²⁸² Alcohol also prevents neurogenesis, the birth of new neurons, in the hippocampus.²⁸³ Alcohol reduces glutamate release in the hippocampus, weakening spatial memory.²⁸⁴ When the brain experiences a reduction in glutamate due to regular alcohol use, it responds by up-regulating, or increasing the number of receptors.²⁸⁵ When alcohol use is reduced, the increased glutamate activation at the additional receptors is responsible for the dangerous seizures associated with alcohol withdrawal and the cell death responsible for brain damage in alcoholics.²⁸⁶

Like other addictive drugs, alcohol increases the release of dopamine in the NAC.²⁸⁷ Alcohol also triggers the release of the body's endogenous opioids, endorphins.²⁸⁸ Dopamine is responsible for the reinforcing effects, and opiate

²⁷² MCKIM & HANCOCK, *supra* note 126, at 306; MEYER & QUENZER, *supra* note 103, at 283–90.

²⁷³ BROAD, *supra* note 54, at 202.

²⁷⁴ CARLSON, *supra* note 208, at 631–33.

²⁷⁵ CARLSON, *supra* note 208, at 633.

²⁷⁶ CARLSON, *supra* note 208, at 122.

²⁷⁷ *Id.*

²⁷⁸ WATSON & BREEDLOVE, *supra* note 29, at 95.

²⁷⁹ CARLSON, *supra* note 208, at 632.

²⁸⁰ MCKIM & HANCOCK, *supra* note 126, at 141; MEYER & QUENZER, *supra* note 103, at 285.

²⁸¹ MEYER & QUENZER, *supra* note 103, at 285.

²⁸² CARLSON, *supra* note 208, at 632; MCKIM & HANCOCK, *supra* note 126, at 141; MEYER & QUENZER, *supra* note 103, at 285.

²⁸³ MCKIM & HANCOCK, *supra* note 126, at 141.

²⁸⁴ CARLSON, *supra* note 208, at 632.

²⁸⁵ MCKIM & HANCOCK, *supra* note 126, at 141; MEYER & QUENZER, *supra* note 103, at 285.

²⁸⁶ MEYER & QUENZER, *supra* note 103, at 285.

²⁸⁷ CARLSON, *supra* note 208, at 632; MEYER & QUENZER, *supra* note 103, at 287.

²⁸⁸ CARLSON, *supra* note 208, at 633; MCKIM & HANCOCK, *supra* note 126, at 147; MEYER & QUENZER, *supra* note 103, at 289.

receptors are critical to the rewarding impacts of alcohol.²⁸⁹ The opiate receptor blockers naloxone and naltrexone prevent the effects of alcohol in rats, monkeys, and humans.²⁹⁰

Research has linked chronic alcohol consumption to shrinkage of brain volume.²⁹¹ Heavy alcohol use causes a deficiency in vitamin B1 (thiamine), which is critical for brain glucose metabolism, and low levels of thiamine cause brain cell death.²⁹² Those who abstain from alcohol were found to have greater brain volume than former drinkers and moderate drinkers.²⁹³ Alcoholics have brain shrinkage in the following areas:

- Frontal lobes, causing apathy, disinhibition, and diminished executive function;
- Hippocampus, causing memory problems; and
- Cerebellum, causing incoordination.²⁹⁴

A lawyer does not have to drink heavily for years to induce impairment of cognition and damage to the brain.²⁹⁵ A study of college students, age eighteen to twenty years old, who periodically participated in binge drinking showed verbal memory deficits, indicating damage to the hippocampus.²⁹⁶

b. *Nicotine*

Nicotine is derived from the tobacco plant and it is the third most widely used recreational drug behind caffeine and alcohol.²⁹⁷ Tobacco was smoked, chewed, and brewed in tea by the indigenous peoples of the Americas, and Christopher Columbus brought it from the Bahamas to Europe in 1492.²⁹⁸ The British brought tobacco to its Virginia colony giving birth to the American tobacco industry, and in 1884 the first cigarette machine was built.²⁹⁹ Modern technology produces 4,000 to 8,000 cigarettes per minute.³⁰⁰

Smoking is an extremely efficient method of drug delivery and nicotine is delivered to the brain in seven seconds via cigarette smoke.³⁰¹ Nicotine enters the lawyer's lungs on tiny hydrocarbon tar particles, in a chemical mixture con-

²⁸⁹ CARLSON, *supra* note 208, at 632–33; MCKIM & HANCOCK, *supra* note 126, at 147; MEYER & QUENZER, *supra* note 103, at 287, 289.

²⁹⁰ CARLSON, *supra* note 208, at 633.

²⁹¹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 44; MCKIM & HANCOCK, *supra* note 126, at 154; MEYER & QUENZER, *supra* note 103, at 277; SWEENEY, *supra* note 15, at 200.

²⁹² MCKIM & HANCOCK, *supra* note 126, at 154; MEYER & QUENZER, *supra* note 103, at 276.

²⁹³ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 44.

²⁹⁴ MCKIM & HANCOCK, *supra* note 126, at 154; MEYER & QUENZER, *supra* note 103, at 277.

²⁹⁵ MCKIM & HANCOCK, *supra* note 126, at 154.

²⁹⁶ *Id.*

²⁹⁷ GIBB, *supra* note 25, at 194; MEYER & QUENZER, *supra* note 103, at 374.

²⁹⁸ GIBB, *supra* note 25, at 194; MEYER & QUENZER, *supra* note 103, at 373.

²⁹⁹ MEYER & QUENZER, *supra* note 103, at 374.

³⁰⁰ *Id.* at 375.

³⁰¹ *Id.*

taining known carcinogens.³⁰² An average cigarette delivers ten puffs of nicotine and a smoker who consumes one and a half packs per day gets about 300 hits of nicotine daily.³⁰³ Nicotine stays in the lawyer's system for about two hours; thus, withdrawal symptoms keep the lawyer smoking at regular intervals.³⁰⁴

Nicotine impacts cognitive processes including attention and memory.³⁰⁵ Nicotine impacts mood and appetite, and suppresses the REM sleep cycle.³⁰⁶ It is a mixed action drug because it produces varying behavioral and physiological effects for smokers, and differences in smokers and non-smokers.³⁰⁷ In non-smokers, nicotine can increase anxiety, dizziness, and nausea.³⁰⁸ It can also improve cognitive and motor function.³⁰⁹ For smokers, nicotine administration is thought to relieve withdrawal symptoms, but it can also improve attention.³¹⁰

Nicotine engages the dopamine system from the VTA to the NAC, and thus is highly addictive.³¹¹ Other drugs that activate the dopamine pathway include alcohol,³¹² cannabis,³¹³ cocaine and amphetamine,³¹⁴ and heroin.³¹⁵ Research has shown that humans, non-human primates, rats, mice, and dogs will all self-administer nicotine to receive its rewarding effects.³¹⁶ Nicotine is an acetylcholine receptor agonist.³¹⁷ It binds to the neurotransmitter acetylcholine receptors, which excite the glutamate receptors in the VTA, causing dopamine release in the NAC.³¹⁸

Nicotinic acetylcholine receptors activate parts of both the fight-or-flight SNS and the rest-and-digest PNS.³¹⁹ In activating the SNS, nicotine is a potent

³⁰² *Id.*

³⁰³ *Id.*

³⁰⁴ *Id.* at 376.

³⁰⁵ PURVES ET AL., *supra* note 54, at 23, 25.

³⁰⁶ HORSTMAN, *DAY IN THE LIFE*, *supra* note 11, at 170; PURVES ET AL., *supra* note 54, at 23, 25.

³⁰⁷ MEYER & QUENZER, *supra* note 103, at 377.

³⁰⁸ *Id.*

³⁰⁹ *Id.* at 377–79.

³¹⁰ *Id.* at 377, 379.

³¹¹ BEAR ET AL., *supra* note 53, at 568–69; MCKIM & HANCOCK, *supra* note 126, at 191; MEYER & QUENZER, *supra* note 103, at 379–80; SWEENEY, *supra* note 15, at 201; George F. Koob et al., *Reward, Motivation, and Addiction*, in *FUNDAMENTAL NEUROSCIENCE*, *supra* note 54, at 871, 886.

³¹² CARLSON, *supra* note 208, at 632.

³¹³ *Id.* at 633.

³¹⁴ BEAR ET AL., *supra* note 53, at 568–69; CARLSON, *supra* note 208, at 627; SWEENEY, *supra* note 15, at 201.

³¹⁵ BEAR ET AL., *supra* note 53, at 569; CARLSON, *supra* note 208, at 625; SWEENEY, *supra* note 15, at 201.

³¹⁶ MEYER & QUENZER, *supra* note 103, at 379.

³¹⁷ BEAR ET AL., *supra* note 53, at 150; Koob et al., *supra* note 311.

³¹⁸ LINDEN, *supra* note 86, at 44, 46; WATSON & BREEDLOVE, *supra* note 29, at 95; Koob et al., *supra* note 311.

³¹⁹ MEYER & QUENZER, *supra* note 103, at 381.

stimulant,³²⁰ increasing alertness, heart rate, blood pressure, and digestion.³²¹ This activation can increase a smoker's risk for heart disease and stroke.³²² It constricts blood vessels in the skin, likely causing the skin to wrinkle and age faster than that of nonsmokers.³²³ Nicotine increases stomach acid and muscle contraction in the bowels, which can cause stomach ulcers, diarrhea, and irritable bowel.³²⁴

Nicotine is highly toxic and nicotine poisoning can occur if tobacco is swallowed or exposed to skin in tobacco fields.³²⁵ Nicotine poisoning causes nausea, vomiting, diarrhea, headaches, dizziness, confusion, hearing and vision problems, weakness, drop in blood pressure, breathing difficulty, and collapse.³²⁶ Victims of nicotine poisoning are treated for shock, induced to vomit, given charcoal to absorb nicotine in the stomach, and helped to breathe.³²⁷

More people in the United States die of smoking-related lung cancer each year than from colon, breast, and prostate cancers combined.³²⁸ Smoking cigarettes increases the risk of cancer, heart disease, stroke, hardening of the arteries, emphysema, and chronic bronchitis.³²⁹ Nicotine is so addictive, many people refuse to stop smoking, even after the onset of serious illness.³³⁰ Sigmund Freud, a doctor who knew smoking caused a cancer that resulted in the removal of most of his jaw, did not quit smoking.³³¹

When smokers quit, they experience deterioration in mood for three to four weeks, when mood then returns to the pre-cessation state.³³² But mood improves during the next ten weeks, becoming better than during smoking.³³³

c. *Cannabis*

Marijuana and *hashish* (cannabis) are produced from the *Cannabis sativa* plant, also known as the flowering hemp.³³⁴ Marijuana and hashish can be smoked, or eaten in edibles such as cookies, brownies, or candy.³³⁵ Cannabis is

³²⁰ Richerson et al., *supra* note 205, at 1041.

³²¹ MEYER & QUENZER, *supra* note 103, at 381; WATSON & BREEDLOVE, *supra* note 29, at 94.

³²² MEYER & QUENZER, *supra* note 103, at 381.

³²³ MCKIM & HANCOCK, *supra* note 126, at 191.

³²⁴ *Id.*; MEYER & QUENZER, *supra* note 103, at 381.

³²⁵ MEYER & QUENZER, *supra* note 103, at 381.

³²⁶ *Id.*

³²⁷ *Id.*

³²⁸ WATSON & BREEDLOVE, *supra* note 29, at 100.

³²⁹ MCKIM & HANCOCK, *supra* note 126, at 201–03; MEYER & QUENZER, *supra* note 103, at 390.

³³⁰ CARLSON, *supra* note 208, at 628.

³³¹ *Id.*

³³² MCKIM & HANCOCK, *supra* note 126, at 192.

³³³ *Id.*

³³⁴ GIBB, *supra* note 25, at 215; MEYER & QUENZER, *supra* note 103, at 402.

³³⁵ MEYER & QUENZER, *supra* note 103, at 402.

a mixed action drug because it is a depressant, but can act as a mild hallucinogenic and stimulant, occasionally causing paranoia.³³⁶

Synthetic cannabinoids have been produced to treat anorexia, weight loss in AIDS patients, and chemotherapy-related nausea in cancer patients.³³⁷ Several synthetic cannabinoids produced for recreational use, such as Spice and K2, are illegal.³³⁸

Cannabis likely originated in Asia and was used for medical and religious purposes in China, India, and the Middle East.³³⁹ Hashish made its way to France via Napoleon's soldiers, or the French doctor Jacques-Joseph Moreau, who helped found a hashish club for artists and writers that included Victor Hugo, Alexandre Dumas, and Charles Baudelaire.³⁴⁰ Hemp was grown in the U.S. colonies, perhaps most famously by George Washington, but not consumed for intoxication.³⁴¹ The practice of smoking cannabis was probably influenced by immigrants from Mexico and the Caribbean in the early 1900s.³⁴²

Cannabis is a controversial drug because it is gradually being decriminalized and inconsistent research results have made conclusions about potential harm difficult to interpret.³⁴³ The consumption of cannabis for recreational purposes has been approved in Colorado,³⁴⁴ Washington,³⁴⁵ Alaska,³⁴⁶ Oregon,³⁴⁷ and Washington, DC,³⁴⁸ but these state laws conflict with federal law, which makes cannabis consumption illegal.³⁴⁹ Twenty states and Washington, D.C. allow consumption of cannabis for medical purposes.³⁵⁰

Delta-9-tetrahydrocannabinol ("THC") gives cannabis products their psychoactive properties.³⁵¹ Another group of chemical compounds called cannabinoids may also contribute to the drug effects of cannabis.³⁵² When marijuana is smoked, it is readily absorbed through the lungs, which absorb about 20 per-

³³⁶ LINDEN, *supra* note 86, at 41; WATSON & BREEDLOVE, *supra* note 29, at 96.

³³⁷ MCKIM & HANCOCK, *supra* note 126, at 314–15.

³³⁸ *Id.* at 315.

³³⁹ MEYER & QUENZER, *supra* note 103, at 403.

³⁴⁰ *Id.*

³⁴¹ *Id.*

³⁴² *Id.*

³⁴³ MCKIM & HANCOCK, *supra* note 126, at 327.

³⁴⁴ COLO. CONST. art. XVIII, § 16.

³⁴⁵ WASH. REV. CODE §§ 69.50.360, 69.50.4013 (2014).

³⁴⁶ ALASKA STAT. § 17.38.010 (2015).

³⁴⁷ Oregon Control, Regulation, and Taxation of Marijuana and Industrial Hemp Act, 2014 Ballot Measure 91 (Or. 2015).

³⁴⁸ D.C. CODE § 48-904.01 (2014).

³⁴⁹ 21 U.S.C. § 812 (2012).

³⁵⁰ *Marijuana Resource Center: State Laws Related to Marijuana*, OFFICE OF NAT'L DRUG CONTROL POLICY, <http://www.whitehouse.gov/ondcp/state-laws-related-to-marijuana> (last visited May 8, 2015).

³⁵¹ GIBB, *supra* note 25, at 215; MEYER & QUENZER, *supra* note 103, at 402.

³⁵² MCKIM & HANCOCK, *supra* note 126, at 314.

cent to 30 percent of the THC.³⁵³ THC levels in the blood decline fairly rapidly, but complete elimination of the drug is much slower because it persists in fat tissue where it can be detected in urine tests for more than two weeks after a single marijuana use.³⁵⁴

Administration of THC to mice results in reduced activity, decreased body temperature, reduced pain sensitivity, and spatial learning deficits.³⁵⁵ When lawyers smoke marijuana, they can experience effects ranging from lightheadedness, euphoria, disinhibition, and relaxation, to hallucinations.³⁵⁶ THC increases blood flow to the skin causing warmth or flushing, increases heart rate, and stimulates appetite.³⁵⁷ High doses of cannabis can cause agitation, a feeling of being separated from oneself, a sense that the world is unreal, and paranoia.³⁵⁸ Cannabis also interferes with normal stages of sleep.³⁵⁹

Cognitive problems caused by cannabis include deficits in verbal, spatial, time estimation, and reaction time tasks.³⁶⁰ Cannabis interferes with all aspects of memory processing: encoding, consolidation, and retrieval.³⁶¹ It disrupts hippocampus function, important to memory formation.³⁶² One study showed high levels of cannabis use in adolescence resulted in a reduction of volume of the right hippocampus.³⁶³ Long-term impacts of cannabis use are still under investigation, but studies show executive function is impaired in long-term users for two to three weeks after cessation.³⁶⁴ Executive function can be compared to a switch operator controlling the pattern of tracks at a busy railroad.³⁶⁵ Executive functions are supervisory or regulatory, such as planning, processing options, guiding sensory information flow, initiating motor action, and inhibiting inappropriate responses.³⁶⁶ Psychomotor performance is impaired under demanding task conditions like driving following a moderate or high dose of cannabis, or when a low dose of cannabis is combined with alcohol.³⁶⁷

Researchers are very interested in the impact of cannabis on educational performance. A study of approximately 1,000 New Zealanders who were re-

³⁵³ MEYER & QUENZER, *supra* note 103, at 404.

³⁵⁴ *Id.*

³⁵⁵ *Id.* at 406.

³⁵⁶ MCKIM & HANCOCK, *supra* note 126, at 319; MEYER & QUENZER, *supra* note 103, at 413–14.

³⁵⁷ MCKIM & HANCOCK, *supra* note 126, at 319; MEYER & QUENZER, *supra* note 103, at 414.

³⁵⁸ MCKIM & HANCOCK, *supra* note 126, at 320, 328; MEYER & QUENZER, *supra* note 103, at 414.

³⁵⁹ MCKIM & HANCOCK, *supra* note 126, at 319.

³⁶⁰ MEYER & QUENZER, *supra* note 103, at 415.

³⁶¹ MCKIM & HANCOCK, *supra* note 126, at 328; MEYER & QUENZER, *supra* note 103, at 415.

³⁶² CARLSON, *supra* note 208, at 635; WATSON & BREEDLOVE, *supra* note 29, at 96.

³⁶³ MCKIM & HANCOCK, *supra* note 126, at 322.

³⁶⁴ MEYER & QUENZER, *supra* note 103, at 416.

³⁶⁵ PURVES ET AL., *supra* note 54, at 437.

³⁶⁶ MEYER & QUENZER, *supra* note 103, at 367; PURVES ET AL., *supra* note 54, at 431.

³⁶⁷ MEYER & QUENZER, *supra* note 103, at 416.

cruited at age three were subject to neuropsychological testing at age thirteen prior to cannabis use, and again at age thirty-eight.³⁶⁸ At age thirty-eight, the amount of cannabis use and dependence were significantly associated with cognitive impairment, including lower IQ, even after controlling for education level.³⁶⁹ Amotivational syndrome, which includes symptoms such as apathy, aimlessness, decreased productivity, reduction of long-range planning, and loss of achievement motivation has been found in chronic cannabis users in thirty years of research.³⁷⁰ Cannabis use has been correlated with reduced educational achievement, and it has been established that cannabis impacts attention and memory.³⁷¹

Brain imaging studies indicate abnormalities in the hippocampus and anterior cingulate cortex of chronic cannabis users.³⁷² Chronic use has also been linked to the development of schizophrenia, chronic cough, bronchitis, and pre-cancerous cells in bronchial biopsy samples.³⁷³ Activation of cannabinoid receptors suppresses immune function and interferes with the reproductive systems in both men and women.³⁷⁴ Marijuana smoke contains 50 percent to 70 percent more carcinogens than tobacco smoke and marijuana smoke is typically held longer in the lungs.³⁷⁵ Although the median age for developing cancer is between fifty-five and sixty-five years, one study of lung cancer patients under forty-five found that nearly all of them smoked both marijuana and tobacco.³⁷⁶

Endogenous means produced from within,³⁷⁷ and the brain produces endogenous cannabinoids.³⁷⁸ Endogenous cannabinoids modulate pain perception and play a role in hunger.³⁷⁹ Cannabinoid receptor antagonists that block the receptors reduce food consumption in animals and humans.³⁸⁰ Thus, the cannabinoid system is a target of pain control and obesity research.³⁸¹

Cannabinoid CB₁ receptors in the brain are in the cerebral cortex, hippocampus, cerebellum, striatum, and basal ganglia.³⁸² Cannabinoid CB₂ receptors have been discovered in the central nervous system, the immune system, the GI tract, bones, and fat cells.³⁸³ Activation of cannabinoid receptors inhibits the

³⁶⁸ *Id.* at 422.

³⁶⁹ *Id.*

³⁷⁰ *Id.*

³⁷¹ MCKIM & HANCOCK, *supra* note 126, at 329.

³⁷² MEYER & QUENZER, *supra* note 103, at 423.

³⁷³ *Id.*

³⁷⁴ *Id.*

³⁷⁵ MCKIM & HANCOCK, *supra* note 126, at 330.

³⁷⁶ *Id.*

³⁷⁷ CARLSON, *supra* note 208, at 124.

³⁷⁸ MCKIM & HANCOCK, *supra* note 126, at 318; MEYER & QUENZER, *supra* note 103, at 407.

³⁷⁹ MEYER & QUENZER, *supra* note 103, at 409.

³⁸⁰ *Id.*

³⁸¹ *Id.*

³⁸² MCKIM & HANCOCK, *supra* note 126, at 317; MEYER & QUENZER, *supra* note 103, at 405.

³⁸³ MCKIM & HANCOCK, *supra* note 126, at 317; MEYER & QUENZER, *supra* note 103, at 405.

release of the inhibitory neurotransmitter GABA and the excitatory neurotransmitter glutamate.³⁸⁴ Cannabinoids can also inhibit the release of acetylcholine, serotonin, norepinephrine, and dopamine.³⁸⁵

THC has a stimulating effect on dopamine neurons, causing release of dopamine in the NAC.³⁸⁶ Opioid systems appear to play a role in the reward and feelings of pleasure associated with THC use because studies have found that opioid agonists enhance THC self-administration and opioid antagonists decrease THC self-administration in animals.³⁸⁷ Research suggests that the endocannabinoid system, via CB₁ and CB₂ receptors, may play a significant role in reinforcement, dependence, and/or relapse for nicotine, alcohol, opioids, and cocaine.³⁸⁸

2. Stimulants

Stimulants increase wakefulness and enhance mental function.³⁸⁹ Some common stimulants are caffeine, cocaine, and amphetamines (including Adderall and Ritalin).

a. Caffeine

Caffeine, one of the most commonly used drugs in the world, is present in coffee beans, tea leaves, soda, energy drinks, and chocolate.³⁹⁰ Caffeine increases arousal, improves concentration, and enhances sports performance.³⁹¹ When the lawyer starts her day with coffee, caffeine blocks adenosine receptors in her brain.³⁹² Adenosine is a neurotransmitter responsible for inducing sleep.³⁹³ Caffeine is an antagonist that sits on the adenosine receptor, blocking it and keeping it from promoting sleep.³⁹⁴ Caffeine has a complex relationship with dopamine, which involves interaction with adenosine, dopamine, and cannabinoid receptors.³⁹⁵ It stimulates dopamine release in the brain,³⁹⁶ and that impacts glutamate and acetylcholine.³⁹⁷ Recently, three substances have been identified in chocolate that are similar to the endogenous cannabinoids in the

³⁸⁴ SWEENEY, *supra* note 15, at 200.

³⁸⁵ MEYER & QUENZER, *supra* note 103, at 406.

³⁸⁶ MCKIM & HANCOCK, *supra* note 126, at 318; MEYER & QUENZER, *supra* note 103, at 417.

³⁸⁷ MEYER & QUENZER, *supra* note 103, at 417.

³⁸⁸ MCKIM & HANCOCK, *supra* note 126, at 319; MEYER & QUENZER, *supra* note 103, at 417.

³⁸⁹ LINDEN, *supra* note 86, at 40.

³⁹⁰ MEYER & QUENZER, *supra* note 103, at 393–94.

³⁹¹ *Id.* at 395.

³⁹² GIBB, *supra* note 25, at 194; MCKIM & HANCOCK, *supra* note 126, at 216; MEYER & QUENZER, *supra* note 103, at 397, 399.

³⁹³ GIBB, *supra* note 25, at 194; MEYER & QUENZER, *supra* note 103, at 399.

³⁹⁴ BEAR ET AL., *supra* note 53, at 671; GIBB, *supra* note 25, at 194.

³⁹⁵ MCKIM & HANCOCK, *supra* note 126, at 217.

³⁹⁶ GIBB, *supra* note 25, at 194; MCKIM & HANCOCK, *supra* note 126, at 216.

³⁹⁷ MCKIM & HANCOCK, *supra* note 126, at 217.

brain.³⁹⁸ The presence of endocannabinoids may explain the pleasure lawyers get from both coffee and chocolate.³⁹⁹

The therapeutic benefits of caffeine include its ability to improve the effectiveness of over-the-counter analgesics like Anacin and Excedrin and to treat newborn babies who stop breathing.⁴⁰⁰ When the lawyer over-consumes energy drinks or coffee, excess caffeine can cause agitation, insomnia, tremors, seizures, gastrointestinal distress, heart palpitations, respiratory problems, and cardiac arrest.⁴⁰¹

b. Cocaine

Cocaine is derived from the South American coca plant and was chewed or brewed in tea by indigenous people to ease pain, improve mood, and alleviate hunger.⁴⁰² In the 1880s, cocaine was used as an anesthetic and added to the original Coca-Cola soda.⁴⁰³ Sigmund Freud, a cocaine user, promoted cocaine to help people work longer and harder without tiring and to treat alcoholism and depression.⁴⁰⁴

If a lawyer ingested cocaine, it would interact with three neurotransmitters in the lawyer brain: dopamine, serotonin, and norepinephrine.⁴⁰⁵ Cocaine binds to the transporters for these three transmitters and prevents them from reuptake by the pre-synaptic neuron.⁴⁰⁶ Transporters are responsible for clearing the transmitters from the synapse for recycling by the presynaptic neuron.⁴⁰⁷ This leaves dopamine, serotonin, and norepinephrine to linger in the synaptic cleft and activate the receptors on the post-synaptic neuron.⁴⁰⁸

Cocaine does not interact with the three transporters equally.⁴⁰⁹ It has the highest affinity (binds most strongly) with the serotonin receptor, followed by the dopamine and finally the norepinephrine transporters.⁴¹⁰ But cocaine does more than block the reuptake of dopamine, it also stimulates the release of dopamine in the NAC and amygdala.⁴¹¹ When dopamine receptors in the NAC

³⁹⁸ *Id.*

³⁹⁹ *Id.*

⁴⁰⁰ MEYER & QUENZER, *supra* note 103, at 397.

⁴⁰¹ GIBB, *supra* note 25, at 194; MEYER & QUENZER, *supra* note 103, at 398.

⁴⁰² GIBB, *supra* note 25, at 197–98; WATSON & BREEDLOVE, *supra* note 29, at 97.

⁴⁰³ GIBB, *supra* note 25, at 198; WATSON & BREEDLOVE, *supra* note 29, at 97.

⁴⁰⁴ GIBB, *supra* note 25, at 198; MEYER & QUENZER, *supra* note 103, at 339–41.

⁴⁰⁵ MEYER & QUENZER, *supra* note 103, at 343.

⁴⁰⁶ MCKIM & HANCOCK, *supra* note 126, at 233; MEYER & QUENZER, *supra* note 103, at 343; SWEENEY, *supra* note 15, at 48; Shizgal & Hyman, *supra* note 123, at 1106.

⁴⁰⁷ BEAR ET AL., *supra* note 53, at 158.

⁴⁰⁸ GIBB, *supra* note 25, at 32; MCKIM & HANCOCK, *supra* note 126, at 233; MEYER & QUENZER, *supra* note 103, at 343.

⁴⁰⁹ MEYER & QUENZER, *supra* note 103, at 344.

⁴¹⁰ *Id.*

⁴¹¹ BEAR ET AL., *supra* note 53, at 547; MCKIM & HANCOCK, *supra* note 126, at 235; Koob, *supra* note 311, at 884.

and amygdala are blocked by antagonists, rats will stop self-administration of both cocaine and amphetamine.⁴¹² Thus, the acute reinforcing effects of cocaine are due to dopamine.⁴¹³ Because naltrexone, an opioid antagonist, reduces craving and use for several amphetamines, the subjective pleasure from cocaine use likely involves opioid and/or endocannabinoid receptors.⁴¹⁴

Cocaine use in lawyers causes euphoria and suppresses hunger and fatigue.⁴¹⁵ It increases alertness and self-confidence.⁴¹⁶ It is a sympathomimetic drug, meaning that it produces symptoms of fight-or-flight SNS activation.⁴¹⁷ Cocaine use causes increased heart rate and blood pressure, elevated body temperature, and narrowing of blood vessels.⁴¹⁸ Low cocaine doses are usually not harmful, but the risks of heavy cocaine use include seizures, heart failure, stroke, and brain hemorrhage.⁴¹⁹ It is possible for a single cocaine dose to trigger a stroke or seizure.⁴²⁰

Recent rodent research has shown that the narrowing of blood vessels during cocaine administration can significantly interrupt blood flow to the brain and this impact is intensified with repeated drug use.⁴²¹ Cognitive deficits in cocaine-dependent lawyers include thinking brain weaknesses in inhibitory control and evaluation of consequences.⁴²² Imaging studies in monkeys demonstrate increased prefrontal cortex thinking brain cognitive deficits with repeated cocaine exposure as compared to initial dosing.⁴²³ Human imaging studies show decreased volume in some areas of the thinking brain, as well as abnormalities in both gray and white matter in the cerebral cortex.⁴²⁴

⁴¹² Koob et al., *supra* note 311, at 883.

⁴¹³ *Id.*

⁴¹⁴ MCKIM & HANCOCK, *supra* note 126, at 252.

⁴¹⁵ Koob et al., *supra* note 311, at 883.

⁴¹⁶ BEAR ET AL., *supra* note 53, at 546.

⁴¹⁷ *Id.* at 547; MCKIM & HANCOCK, *supra* note 126, at 235; MEYER & QUENZER, *supra* note 103, at 346; Koob et al., *supra* note 311, at 883.

⁴¹⁸ BEAR ET AL., *supra* note 53, at 547; MEYER & QUENZER, *supra* note 103, at 346.

⁴¹⁹ MEYER & QUENZER, *supra* note 103, at 346.

⁴²⁰ *Id.* at 358. The most famous case of a seizure and death from cocaine-related cardiac arrest is that of college basketball star Len Bias in 1986. Keith Harriston & Sally Jenkins, *Maryland Basketball Star Len Bias Is Dead at 22: Traces of Cocaine Found in System*, WASH. POST (Jun. 20, 1986), <http://www.washingtonpost.com/wp-srv/sports/longterm/memories/bias/launch/bias1.htm>.

⁴²¹ MEYER & QUENZER, *supra* note 103, at 346.

⁴²² *Id.* at 353.

⁴²³ *Id.*

⁴²⁴ *Id.* at 358.

c. *Amphetamine (including methamphetamine, mephedrone, Ritalin, and Adderall)*

Amphetamine is a synthetic stimulant with a chemical structure that closely resembles dopamine.⁴²⁵ Two similar plant compounds are khat, an evergreen shrub found in East Africa and the Arabian peninsula, and ephedrine from the herb *Ephedra*.⁴²⁶ Weight loss supplements contained ephedrine, but it produces sympathomimetic symptoms including increased blood pressure and risk of stroke or heart attack.⁴²⁷ Ephedrine was used in asthma medicine because it also widens bronchial airways.⁴²⁸ Some decongestants contain pseudoephedrine and they are closely regulated because they can be synthesized to create methamphetamine.⁴²⁹

Amphetamine was synthesized in 1887 because doctors were seeking a synthetic substitute for ephedrine.⁴³⁰ Amphetamine was used to relieve nasal congestion and to treat narcolepsy, and by the military to keep pilots awake on long flights.⁴³¹

Methamphetamine has a more powerful impact on the nervous system than amphetamine,⁴³² but it produces similar neurochemical and behavioral effects.⁴³³ Other members of the amphetamine family are 3,4-methylenedioxymethamphetamine (“MDMA”, “Ecstasy”), 3,4-methylenedioxyamphetamine (“MDA”), and 3,4-methylenedioxy-N-ethylamphetamine (“MDE”).⁴³⁴ This article will use the term “amphetamine” as a label for this group of drugs.

Amphetamine works by blocking the reuptake of norepinephrine, serotonin, and dopamine, and by stimulating the release of dopamine.⁴³⁵ Amphetamine uses the dopamine transporter, intended to remove and recycle dopamine by the presynaptic neuron, to enter the neuron and stimulate the release of addi-

⁴²⁵ *Id.* at 362.

⁴²⁶ *Id.*

⁴²⁷ *Id.* at 362–63. Baltimore Orioles pitcher Steve Bechler collapsed and died at spring training in 2003. After it was discovered that he had been taking high doses of a weight loss supplement containing ephedra, the FDA banned sales in 2004. *Id.* at 363.

⁴²⁸ *Id.* at 363.

⁴²⁹ *Id.* at 363 n.4.

⁴³⁰ GIBB, *supra* note 25, at 196; MEYER & QUENZER, *supra* note 103, at 363.

⁴³¹ BEAR ET AL., *supra* note 53, at 547; GIBB, *supra* note 25, at 196–97; MEYER & QUENZER, *supra* note 103, at 363.

⁴³² MEYER & QUENZER, *supra* note 103, at 364.

⁴³³ *Id.* at 363.

⁴³⁴ *Id.* at 362; *Methylenedioxymethamphetamine (MDMA, Ecstasy)*, NAT'L HIGHWAY TRAFFIC SAFETY ADMIN., <http://www.nhtsa.gov/people/injury/research/job185drugs/methylenedioxymethamphetamine.htm> (last visited May 11, 2015).

⁴³⁵ BEAR ET AL., *supra* note 53, at 547; MCKIM & HANCOCK, *supra* note 126, at 235; Shizgal & Hyman, *supra* note 123, at 1107.

tional dopamine into the synapse via the transporter.⁴³⁶ Thus, the effects of norepinephrine and dopamine at the synapses are prolonged and intensified.⁴³⁷ Because naltrexone, an opioid antagonist, reduces craving, pleasure, and use of amphetamine, it is likely that the endogenous opioid system is responsible for the euphoria created by amphetamine.⁴³⁸

If a lawyer uses amphetamine, it stays in her system for seven to thirty hours, producing a longer-lasting high than she can achieve with a single administration of cocaine.⁴³⁹ She will experience considerable sympathomimetic symptoms from the norepinephrine that are similar to those caused by cocaine (elevated heart rate, blood pressure, and temperature).⁴⁴⁰ While she may feel more alert, awake, and confident,⁴⁴¹ she is exposing her brain to damage of the dopamine axons and terminals in the thinking and emotional brains and long-lasting dopamine reduction.⁴⁴² Chronic amphetamine use results in deficits of dopamine and serotonin in the brain; elevated blood pressure; and increased risk of stroke, heart attack, hardening of the arteries, and death.⁴⁴³

Mephedrone is a stimulant derived from the substance in the khat plant, known as bath salts or plant food, that inhibits the reuptake of dopamine and serotonin and increases the levels of these transmitters in the NAC.⁴⁴⁴ It was sold legally until 2011 and early research results indicate risks of use include compromised serotonin and dopamine systems in the brain, excessive and prolonged sympathomimetic activity, agitation, seizures, and death.⁴⁴⁵

ADHD medications, used to treat attention deficit hyperactivity disorder (“ADHD”), include Adderall (mixed amphetamine salts) and Ritalin (methylphenidate).⁴⁴⁶ Children with ADHD exhibit inattentiveness, impulsivity, and excessive movement.⁴⁴⁷ Between 3 percent and 7 percent of children, and three times more boys than girls, are diagnosed with ADHD.⁴⁴⁸ One theory is that dopamine transporters do not reuptake the right amount of dopamine in brains impacted by ADHD, so there is less dopamine than necessary to produce a clean signal for transmission between neurons.⁴⁴⁹ Reduced dopamine levels result in a compromised reward signal that lacks appropriate timing con-

⁴³⁶ MCKIM & HANCOCK, *supra* note 126, at 233; MEYER & QUENZER, *supra* note 103, at 364–65; Shizgal & Hyman, *supra* note 123, at 1106.

⁴³⁷ BEAR ET AL., *supra* note 53, at 547.

⁴³⁸ MCKIM & HANCOCK, *supra* note 126, at 252.

⁴³⁹ MEYER & QUENZER, *supra* note 103, at 364.

⁴⁴⁰ *Id.* at 346, 365.

⁴⁴¹ *Id.* at 365.

⁴⁴² *Id.* at 368.

⁴⁴³ *Id.* at 369.

⁴⁴⁴ *Id.* at 370.

⁴⁴⁵ *Id.*

⁴⁴⁶ LINDEN, *supra* note 86, at 40; MEYER & QUENZER, *supra* note 103, at 366.

⁴⁴⁷ MEYER & QUENZER, *supra* note 103, at 366.

⁴⁴⁸ GIBB, *supra* note 25, at 164.

⁴⁴⁹ CARLSON, *supra* note 208, at 599; GIBB, *supra* note 25, at 165.

nection to the stimulus or produces no effect.⁴⁵⁰ Low doses of ADHD stimulants calm over half the children impacted by ADHD.⁴⁵¹

Ritalin and Adderall have been used as study drugs to increase concentration and reduce fatigue.⁴⁵² Increased wakefulness and a reduced need for sleep are caused by significant synaptic increase in dopamine and norepinephrine.⁴⁵³ Up to 25 percent of college students are self-medicating to get an edge on exams or fuel marathon study sessions.⁴⁵⁴ Ritalin is a dopamine agonist that works by blocking dopamine transporters and preventing reuptake, leaving more dopamine in the synaptic gap.⁴⁵⁵ Because Ritalin alleviates ADHD symptoms, scientists believe the disorder is caused by deficiencies in dopaminergic transmission.⁴⁵⁶ Research shows that a moderate dose of Ritalin improves attention and working memory, and increases the responsiveness of neurons in the thinking brain, but a high dose profoundly suppresses neural activity.⁴⁵⁷ Adderall, an amphetamine, causes the release of both dopamine and epinephrine into the synaptic cleft through their transporters, and their lingering impact in the cleft is due to the blocked transporters.⁴⁵⁸

Risks of these medications include sympathomimetic increases in heart rate and blood pressure, as well as abuse potential because administration via snorting or injection can cause euphoria.⁴⁵⁹ Animal research has shown that Ritalin and Adderall can alter brain structure and function that results in greater anxiety, gloomy moods, and cognitive deficits, similar to impairment caused by cocaine (poor impulse control, inability to appreciate consequences, decreased brain volume).⁴⁶⁰

Law and medical students are using Ritalin and Adderall to gain an edge in education systems where students face “steep grading curves and declining job prospects.”⁴⁶¹ Some students procure these drugs with prescriptions and others purchase them on the black market.⁴⁶² Surveys of university students indicate that somewhere between 6.9 percent and 60 percent of students have used prescription stimulants for cognitive enhancement.⁴⁶³ Ritalin and Adderall are

⁴⁵⁰ HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 91.

⁴⁵¹ MEYER & QUENZER, *supra* note 103, at 366.

⁴⁵² *Id.* at 142–43.

⁴⁵³ *Id.* at 143.

⁴⁵⁴ HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 23.

⁴⁵⁵ CARLSON, *supra* note 208, at 598; GIBB, *supra* note 25, at 165.

⁴⁵⁶ CARLSON, *supra* note 208, at 598–99.

⁴⁵⁷ *Id.* at 599–600.

⁴⁵⁸ See MEYER & QUENZER, *supra* note 103, at 368.

⁴⁵⁹ *Id.* at 366.

⁴⁶⁰ HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 27–28; MEYER & QUENZER, *supra* note 103, at 353, 358; Kelline R. Linton, *Scholastic Steroids: Is Generation Rx Cognitively Cheating?*, 39 PEPP. L. REV. 989, 997 (2012).

⁴⁶¹ Linton, *supra* note 460, at 1003.

⁴⁶² *Id.* at 1002–03.

⁴⁶³ *Id.* at 1002.

Schedule II drugs because they have a high potential for abuse or dependence.⁴⁶⁴ Side effects of these drugs include dizziness, headaches, and insomnia, and more serious reactions are blurred vision, seizure, stroke, and death.⁴⁶⁵ As with all amphetamines, use of Ritalin and Adderall can cause a down-regulation in dopamine and norepinephrine receptors, reducing the normal function of dopamine and norepinephrine in the lawyer brain.⁴⁶⁶ Animal studies show that when they are trained under the influence of amphetamine, they cannot fully remember what they learned once the amphetamine is out of their system.⁴⁶⁷

3. Opiates

Opiates are sedatives derived from the opium poppy. They include 1) the natural opiates: opium, morphine, codeine, and thebaine; 2) the semisynthetic opiates: heroin (which converts to morphine in the brain), Dilaudid, and oxycodone; and 3) the synthetic opiates: OxyContin, fentanyl, Demerol, Darvon, Talwin, and methadone.⁴⁶⁸ The active ingredients in opium are morphine, codeine, and thebaine.⁴⁶⁹

Opium was used in ancient Egypt and Greece for medicinal and recreational purposes, and eating or smoking opium was common in Turkey and Iran where alcohol was banned.⁴⁷⁰ In 1680, English doctor Thomas Sydenham introduced laudanum, which was wine laced with opium, saffron, cinnamon, and cloves.⁴⁷¹ Laudanum was consumed by many English and American women, and it was an ingredient in infant teething and muscle ache products.⁴⁷² German chemist Friedrich Wilhelm Sertürner isolated morphine from opium in 1805, a pharmacology milestone as the first isolation of an active ingredient from a medicinal plant.⁴⁷³ Morphine was used to ease pain in wounded soldiers in the Civil War.⁴⁷⁴ The Bayer drug company introduced heroin, a derivative of morphine, in 1898 as a non-addictive pain killer.⁴⁷⁵ Heroin is approximately ten times more potent than morphine.⁴⁷⁶ Opiates were not regulated in the United States until the 1910s.⁴⁷⁷

⁴⁶⁴ *Id.* at 1013.

⁴⁶⁵ *Id.* at 1012.

⁴⁶⁶ MEYER & QUENZER, *supra* note 103, at 369.

⁴⁶⁷ MCKIM & HANCOCK, *supra* note 126, at 242.

⁴⁶⁸ GIBB, *supra* note 25, at 213–14; LINDEN, *supra* note 86, at 40–41; MEYER & QUENZER, *supra* note 103, at 307–08 fig.11.4.

⁴⁶⁹ MCKIM & HANCOCK, *supra* note 126, at 255.

⁴⁷⁰ MEYER & QUENZER, *supra* note 103, at 306.

⁴⁷¹ *Id.*

⁴⁷² *Id.*

⁴⁷³ LINDEN, *supra* note 86, at 48; MEYER & QUENZER, *supra* note 103, at 307.

⁴⁷⁴ LINDEN, *supra* note 86, at 49.

⁴⁷⁵ *Id.*; MEYER & QUENZER, *supra* note 103, at 307.

⁴⁷⁶ MCKIM & HANCOCK, *supra* note 126, at 258.

⁴⁷⁷ MEYER & QUENZER, *supra* note 103, at 306.

Opiates are rapidly bioavailable via smoking, snorting, subcutaneous injection under the skin, and intravenous (“IV”) injection.⁴⁷⁸ Opiates produce profound pain relief, sedation, mood changes, mental clouding, nausea, vomiting, and constipation.⁴⁷⁹ Rapid penetration into the brain via IV or inhalation produces euphoria.⁴⁸⁰ In high doses, opiates result in coma and death due to respiratory failure.⁴⁸¹ Opium has been used throughout history as a poison, which sheds light on how some users die from accidental overdose.⁴⁸² The receptor antagonists naloxone and nalorphine can prevent or reverse the effects of opioids because of their ability to occupy opioid receptor sites, which means they can save the lives of overdose victims, seconds after administration.⁴⁸³

The brain’s opiate receptors were discovered in the early 1970s⁴⁸⁴ and its endogenous opioids, endorphins, were discovered in the mid-1970s.⁴⁸⁵ Activation of opiate receptors inhibits the release of glutamate, GABA, norepinephrine, acetylcholine, and dopamine.⁴⁸⁶ Activation of opioid receptors can also change gene expression in neurons, causing longer-term alterations in neural transmission.⁴⁸⁷

The effects of opiates are more complex than other drugs because opiates produce reward and reinforcement in the brain in two ways: the dopamine system and via opioid receptors.⁴⁸⁸ Opioid receptors are found in the primitive brain—in the brain stem and spinal cord where they modulate pain and impact respiration—which is why opiate overdose can cause respiratory arrest.⁴⁸⁹ Opioid receptors are also found in the thinking brain (cortex) and the emotional brain (hippocampus, amygdala, thalamus, hypothalamus, VTA, and NAC).⁴⁹⁰ Opioid receptors in both the VTA and NAC⁴⁹¹ create reward and reinforcement independent of the dopamine system, but opioids also cause release of dopamine from the VTA to the NAC.⁴⁹²

⁴⁷⁸ *Id.* at 308.

⁴⁷⁹ BEAR ET AL., *supra* note 53, at 448; CARLSON, *supra* note 208, at 625; MEYER & QUENZER, *supra* note 103, at 308–09.

⁴⁸⁰ MEYER & QUENZER, *supra* note 103, at 308–09.

⁴⁸¹ *Id.* at 305, 308–09.

⁴⁸² MCKIM & HANCOCK, *supra* note 126, at 270.

⁴⁸³ *Id.* at 261; MEYER & QUENZER, *supra* note 103, at 307.

⁴⁸⁴ MCKIM & HANCOCK, *supra* note 126, at 259; RATEY, *supra* note 54, at 117. The receptors were discovered by Sol Snyder and Candace Pert. LINDEN, *supra* note 86, at 44.

⁴⁸⁵ GIBB, *supra* note 25, at 214; LINDEN, *supra* note 86, at 44; MCKIM & HANCOCK, *supra* note 126, at 259; MEYER & QUENZER, *supra* note 103, at 312.

⁴⁸⁶ MCKIM & HANCOCK, *supra* note 126, at 259.

⁴⁸⁷ *Id.* at 260.

⁴⁸⁸ Shizgal & Hyman, *supra* note 123, at 1107.

⁴⁸⁹ *Id.*

⁴⁹⁰ MCKIM & HANCOCK, *supra* note 126, at 260.

⁴⁹¹ *Id.*

⁴⁹² CARLSON, *supra* note 208, at 625; MCKIM & HANCOCK, *supra* note 126, at 261; MEYER & QUENZER, *supra* note 103, at 324–25; Shizgal & Hyman, *supra* note 123, at 1108.

If a lawyer used opiates, he would experience analgesia (pain relief), euphoria, respiratory depression, sedation, decreased blood pressure, hypothermia, constipation, urination problems, pupil constriction, suppression of the vomiting and coughing centers, and reduced sex drive.⁴⁹³ He would also experience insomnia because he would spend more time in lighter sleep stages and less time in slow-wave and REM sleep stages.⁴⁹⁴ Opiate use also diminishes both male and female fertility.⁴⁹⁵ Although opiate use causes an initial high, those feelings are replaced with adverse moods.⁴⁹⁶ An infrequent opiate user would experience lethargic performance on tasks, with greater impairment of motor functions than of cognitive ones.⁴⁹⁷ An opiate-dependent lawyer would show little impairment as a result of tolerance for the drug, unless drug administration is interrupted, throwing him into withdrawal.⁴⁹⁸ Withdrawal from opiates is not life-threatening, but is extremely uncomfortable, like a harsh case of the flu lasting about a week.⁴⁹⁹

Endogenous opioids are involved in the reinforcement of natural rewards, such as food and water.⁵⁰⁰ They likely play a role in the reward and reinforcing impact of some addictive drugs, as well as some foods, because naloxone which blocks opiate receptors reduces the reinforcing effects of alcohol.⁵⁰¹

Opiates are highly reinforcing for two reasons: 1) they activate the dopamine system⁵⁰² and 2) when opiates activate opioid receptors, they also generate a rewarding effect.⁵⁰³ This greatly increases the likelihood that opiates will be used again.⁵⁰⁴

4. Sedatives

Anxiety disorders are the most common psychiatric problems, and women are twice as likely as men to suffer from anxiety.⁵⁰⁵ If a lawyer has generalized anxiety disorder, she experiences “excessive and relentless worry,” and she is irritable, fatigued, and distracted.⁵⁰⁶ Her thinking brain (prefrontal cortex) is suppressed and her emotional brain panic button (amygdala) is hyperactive.⁵⁰⁷

⁴⁹³ MCKIM & HANCOCK, *supra* note 126, at 261–62; MEYER & QUENZER, *supra* note 103, at 326 tbl.11.2.

⁴⁹⁴ MCKIM & HANCOCK, *supra* note 126, at 262.

⁴⁹⁵ *Id.*

⁴⁹⁶ *Id.* at 263.

⁴⁹⁷ *Id.* at 264.

⁴⁹⁸ *Id.*

⁴⁹⁹ *Id.* at 267.

⁵⁰⁰ See CARLSON, *supra* note 208, at 625.

⁵⁰¹ *Id.*

⁵⁰² MEYER & QUENZER, *supra* note 103, at 324–25.

⁵⁰³ *Id.* at 315, 324.

⁵⁰⁴ *Id.* at 325.

⁵⁰⁵ MCKIM & HANCOCK, *supra* note 126, at 162.

⁵⁰⁶ *Id.*

⁵⁰⁷ *Id.*

Sedatives are tranquilizers (anxiolytics) used to calm anxiety, but they can cause incoordination, drowsiness, and slow reaction times.⁵⁰⁸ They also reduce neuron excitability.⁵⁰⁹ Benzodiazepines are prescribed currently, and barbiturates were used in the past.⁵¹⁰

Barbiturates and *Benzodiazepines* (including Valium and Librium) are indirect agonists of GABA receptors.⁵¹¹ GABA is the most important transmitter responsible for inhibiting synapse activity.⁵¹² Without GABA, the neurons in the brain would be firing uncontrollably and the brain would be unstable.⁵¹³

Barbiturates are calming in low doses, but in high doses they impair walking and talking, and result in unconsciousness, depressed respiration, coma, and death.⁵¹⁴ The risk of lethal overdose is the reason for the shift away from prescribing barbiturates for anxiety, and instead toward prescribing benzodiazepines.⁵¹⁵

Benzodiazepines are used to treat anxiety and to aid sleep.⁵¹⁶ They result in sedation, muscle relaxation, anxiety reduction, and mood regulation.⁵¹⁷ Their addictive potential relates to GABA reduction.⁵¹⁸ Benzodiazepines reduce GABA in the VTA, allowing glutamate to inspire dopamine production.⁵¹⁹ The dopamine system from the VTA to the NA is engaged by benzodiazepines, and a single dose given to lab mice changes the neuroplasticity of the glutamate that drives dopamine production.⁵²⁰

The disadvantage of benzodiazepines in controlling anxiety is the undesirable concurrent sedation.⁵²¹ High doses of benzodiazepines cause extreme sedation, but are not life-threatening.⁵²² Since they engage the dopamine system, benzodiazepines are addictive.⁵²³

⁵⁰⁸ MCKIM & HANCOCK, *supra* note 126, at 163; MEYER & QUENZER, *supra* note 103, at 531.

⁵⁰⁹ MEYER & QUENZER, *supra* note 103, at 531.

⁵¹⁰ MCKIM & HANCOCK, *supra* note 126, at 163.

⁵¹¹ CARLSON, *supra* note 208, at 123; MCKIM & HANCOCK, *supra* note 126, at 164; MEYER & QUENZER, *supra* note 103, at 286.

⁵¹² See CARLSON, *supra* note 208, at 123.

⁵¹³ *Id.*; MCKIM & HANCOCK, *supra* note 126, at 167.

⁵¹⁴ MCKIM & HANCOCK, *supra* note 126, at 168–69; see CARLSON, *supra* note 208, at 123.

⁵¹⁵ MCKIM & HANCOCK, *supra* note 126, at 169.

⁵¹⁶ CARLSON, *supra* note 208, at 123.

⁵¹⁷ MCKIM & HANCOCK, *supra* note 126, at 168.

⁵¹⁸ *Id.* at 169.

⁵¹⁹ *Id.*

⁵²⁰ *Id.*

⁵²¹ *Id.* at 168.

⁵²² *Id.*

⁵²³ *Id.* at 169.

5. *Hallucinogens*

Hallucinogens, such as lysergic acid diethylamide (“LSD”), phencyclidine (“PCP”), and ketamine, disrupt perception via distortion of senses.⁵²⁴ LSD is a powerful psychedelic drug that causes sensory overload by exciting serotonin receptors.⁵²⁵ PCP, also known as angel dust, and ketamine are dissociatives that result in sensory deprivation.⁵²⁶ PCP and ketamine are antagonists of glutamate receptors.⁵²⁷ The cerebral cortex and hippocampus have many glutamate receptors and blocking them likely contributes to cognitive impairments caused by PCP and ketamine.⁵²⁸ PCP and ketamine are addictive because they activate the dopamine system.⁵²⁹ Ketamine also appears to stimulate opioid receptors.⁵³⁰

6. *Foods: Sugar, Fat, and Carbohydrates*

Between 1960 and the present, the average weight of an American adult has increased by approximately twenty-six pounds.⁵³¹ This is because restaurants and food producers serve large portions of foods that thoroughly activate the dopamine system.⁵³² The lawyer brain is hardwired to like sugar and fat because they were rare in our ancestral diet and when available, were consumed in large quantities to store for times of famine.⁵³³

Dopamine and endogenous opioids have a role in the rewarding effects of food.⁵³⁴ There are two phases in the reward cycle: hedonic pleasure (liking) and incentive motivation (wanting).⁵³⁵ Both dopamine and opioids contribute to enjoyment of and motivation to eat sweet or fatty foods.⁵³⁶

Diets that incorporate a lot of sugar or fat generate an increase in dopamine signals.⁵³⁷ These foods also cause the release of serotonin.⁵³⁸ The more sugar or fat that is consumed, the more dopamine release, resulting in a craving (wanting) for additional sugar or fat.⁵³⁹

⁵²⁴ LINDEN, *supra* note 86, at 40.

⁵²⁵ BEAR ET AL., *supra* note 53, at 546; GIBB, *supra* note 25, at 209–10; MEYER & QUENZER, *supra* note 103, at 432.

⁵²⁶ GIBB, *supra* note 25, at 211.

⁵²⁷ MEYER & QUENZER, *supra* note 103, at 444.

⁵²⁸ *Id.*

⁵²⁹ *Id.* at 445.

⁵³⁰ GIBB, *supra* note 25, at 211; MEYER & QUENZER, *supra* note 103, at 445.

⁵³¹ LINDEN, *supra* note 86, at 82–83.

⁵³² *Id.* at 83.

⁵³³ *Id.*

⁵³⁴ MEYER & QUENZER, *supra* note 103, at 249, 314.

⁵³⁵ *Id.* at 314.

⁵³⁶ *Id.* at 249, 314.

⁵³⁷ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 113.

⁵³⁸ SWEENEY, *supra* note 15, at 198.

⁵³⁹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 113; LINDEN, *supra* note 86, at 83–84; SWEENEY, *supra* note 15, at 198.

Sugar, fats, and carbohydrates increase endogenous opioids in rat studies.⁵⁴⁰ Opioid antagonists reduce consumption of these highly palatable foods, while reinstating consumption of standard rat chow.⁵⁴¹ Administration of additional opioids, such as morphine, increases consumption of chocolate cookies, fat, and carbohydrates.⁵⁴²

Rapid and generous pleasure signals are the most rewarding and addictive.⁵⁴³ The combination of fat and sugar produces a larger reward signal than either of them independently, making that concoction uber addictive.⁵⁴⁴

A number of pleasurable activities activate the dopamine system and trigger the release of dopamine: playing video games, having sex, gambling, shopping, and exercise.⁵⁴⁵ Consumption of fat, sugar, carbohydrates, and drugs of abuse engage the dopamine system, which was designed to motivate repeated behavior.⁵⁴⁶ If repeated enough, this could lead to an addiction.

V. THE ADDICTED LAWYER BRAIN

Even if the lawyer is not worried about the impacts of casual drug use, there is always the potential that recreational drug use could become addiction. The dopamine system is intimately involved in reward-based learning.⁵⁴⁷ The reinforcing effects of dopamine are involved in the addictive properties of drugs.⁵⁴⁸ Drugs that powerfully activate the dopamine system (cocaine, amphetamine, and heroin) carry the greatest risk of addiction, while drugs that weakly activate it (alcohol and cannabis) present a lower risk.⁵⁴⁹ Drugs that do not activate the dopamine system (LSD and SSRI anti-depressants) carry little to no risk of addiction.⁵⁵⁰

Hedonism is the pursuit of pleasure and anhedonia or dysphoria is the inability to feel pleasure.⁵⁵¹ Early drug use often involves wanting the rush, euphoria, or high.⁵⁵² Transition to addiction occurs when drug use evolves from drug wanting and motivation for the drug reward, to drug needing and how bad the user feels without the drug.⁵⁵³

⁵⁴⁰ MEYER & QUENZER, *supra* note 103, at 314.

⁵⁴¹ *Id.*

⁵⁴² *Id.*

⁵⁴³ LINDEN, *supra* note 86, at 84.

⁵⁴⁴ *Id.*

⁵⁴⁵ DOIDGE, *supra* note 7, at 309; MEYER & QUENZER, *supra* note 103, at 234–35.

⁵⁴⁶ LINDEN, *supra* note 86, at 83–84.

⁵⁴⁷ MCKIM & HANCOCK, *supra* note 126, at 116.

⁵⁴⁸ *Id.* at 119.

⁵⁴⁹ GIBB, *supra* note 25, at 192; LINDEN, *supra* note 86, at 47.

⁵⁵⁰ GIBB, *supra* note 25, at 202; LINDEN, *supra* note 86, at 47.

⁵⁵¹ SAPOLSKY, *supra* note 10, at 272.

⁵⁵² *Id.* at 345.

⁵⁵³ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 128; SAPOLSKY, *supra* note 10, at 345.

Addiction is a form of learning,⁵⁵⁴ and dopamine is responsible for motivating behavior.⁵⁵⁵ When a lawyer needs a drug, the dopamine system is activated.⁵⁵⁶ The dopamine pathway begins in the VTA and the long neurons extend to the NAC where dopamine is released.⁵⁵⁷ The NAC connections provide coordination of motivation, emotional content, and motor responses.⁵⁵⁸ The first release of dopamine in the NAC is in response to the need, and it causes the lawyer to seek the drug.⁵⁵⁹ Information about the location of the drug is recalled from the lawyer's thinking-emotional brain loop where it is stored.⁵⁶⁰ The dealer or friend who provides the drug has incentive salience.⁵⁶¹ Incentive means the lawyer is attracted to the supplier and salience means the supplier commands the lawyer's attention in the future.⁵⁶² The more the lawyer procures the drug this way, the greater the incentive salience is attached to the drug supplier.⁵⁶³ Soon, dopamine release will be prompted by the supplier (stimulus), rather than the drug (reward).⁵⁶⁴ All the rituals associated with the lawyer's drug use will have incentive salience; they are readily perceived, attention-grabbing, and promote drug wanting.⁵⁶⁵

There is a difference between the lawyer's drug wanting (motivation or craving) and drug liking (euphoria, high, or pleasure).⁵⁶⁶ Drug wanting is mediated by the dopamine system when incentive salience drives the lawyer to seek the drug.⁵⁶⁷ Dopamine release in the NAC evolves a neutral stimulus, such as a drug-supplying friend or drug use location, into a desire stimulus via the establishment of incentive salience.⁵⁶⁸ Researchers have discovered dopamine's role as a reinforcer by blocking its action in the brain.⁵⁶⁹ Blocking dopamine receptors or limiting dopamine release from destroyed axons does not stop an animal from eating a sweet food reward, but it does decrease motivation and attempts to obtain it.⁵⁷⁰ Mice with increased dopamine are more motivated to acquire sucrose, but show normal levels of liking the sweet solution.⁵⁷¹ Liking seems to

⁵⁵⁴ LINDEN, *supra* note 86, at 50.

⁵⁵⁵ MCKIM & HANCOCK, *supra* note 126, at 119.

⁵⁵⁶ *Id.* at 116.

⁵⁵⁷ MEYER & QUENZER, *supra* note 103, at 287.

⁵⁵⁸ *Id.*

⁵⁵⁹ *See* MCKIM & HANCOCK, *supra* note 126, at 116–17.

⁵⁶⁰ *See id.* at 117–18.

⁵⁶¹ *See id.* at 117.

⁵⁶² *See id.*

⁵⁶³ *See id.*

⁵⁶⁴ *See id.*

⁵⁶⁵ *See id.*

⁵⁶⁶ MEYER & QUENZER, *supra* note 103, at 251.

⁵⁶⁷ MCKIM & HANCOCK, *supra* note 126, at 119.

⁵⁶⁸ *Id.* at 119.

⁵⁶⁹ *Id.*

⁵⁷⁰ *Id.*

⁵⁷¹ *Id.*

be driven by the presence of endogenous opioids or endocannabinoids⁵⁷² or opiate or cannabinoid drugs,⁵⁷³ in small areas of the NAC and ventral pallidum.⁵⁷⁴

Motivation in the complex motivation control system is mediated by dopamine.⁵⁷⁵ Its role is to ensure the lawyer takes actions, again and again, that guarantee her survival.⁵⁷⁶ It was meant to reinforce natural rewards, and these reinforcers have satiation mechanisms that maintain allostasis.⁵⁷⁷ Drugs access the brain in high concentrations soon after dosing, creating intense dopamine surges that appear to have no limits on reinforcing capacity.⁵⁷⁸ Concurrent surges of reinforcing dopamine with pleasure-inducing opioids and cannabinoids (endogenous or artificial) guarantee the lawyer will be motivated to seek and use drugs that cause this activation of the motivation control system.⁵⁷⁹

Stress hormones have a unique relationship with the dopamine system. Stress hormones activate the dopamine system, causing release of dopamine in the NAC, and repeated exposure to stress sensitizes the dopamine system.⁵⁸⁰ Stress hormones also amplify the reinforcing effects of drugs.⁵⁸¹ Thus, a stressed lawyer who self-medicates with a drug may be more vulnerable to addiction.

The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders ("DSM-5") designates a substance use disorder as "a maladaptive pattern of substance use, over at least a 12-month period, that has led to significant impairment or distress by clinical standards."⁵⁸² Drugs of abuse (nicotine, alcohol, amphetamine, cocaine, morphine, and heroin) activate the dopamine system, causing dopamine release in the NAC.⁵⁸³ The dopamine enhances the salience of drug-related stimuli and reinforces the impacts of the drug.⁵⁸⁴ Characteristics of addiction include: tolerance, a reduced drug response after repeated doses,⁵⁸⁵ and dependence, when drug abstinence produces withdrawal symptoms.⁵⁸⁶

⁵⁷² *Id.*

⁵⁷³ See MEYER & QUENZER, *supra* note 103, at 249.

⁵⁷⁴ MCKIM & HANCOCK, *supra* note 126, at 118; MEYER & QUENZER, *supra* note 103, at 249.

⁵⁷⁵ MCKIM & HANCOCK, *supra* note 126, at 119; MEYER & QUENZER, *supra* note 103, at 251–52.

⁵⁷⁶ MCKIM & HANCOCK, *supra* note 126, at 119.

⁵⁷⁷ *Id.* at 120.

⁵⁷⁸ *Id.*

⁵⁷⁹ See *id.*

⁵⁸⁰ *Id.*

⁵⁸¹ *Id.*

⁵⁸² MEYER & QUENZER, *supra* note 103, at 233.

⁵⁸³ MCKIM & HANCOCK, *supra* note 126, at 125; MEYER & QUENZER, *supra* note 103, at 249.

⁵⁸⁴ MCKIM & HANCOCK, *supra* note 126, at 125.

⁵⁸⁵ *Id.* at 40; MEYER & QUENZER, *supra* note 103, at 32–33.

⁵⁸⁶ MCKIM & HANCOCK, *supra* note 126, at 44; MEYER & QUENZER, *supra* note 103, at 242.

Chronic overstimulation of the dopamine system creates an adjustment or down-regulation of the system.⁵⁸⁷ The result is a decrease in dopamine release in the NAC when drugs of abuse are stopped.⁵⁸⁸ Withdrawal from alcohol, nicotine, amphetamine, cocaine, and heroin decreases the activity of the dopamine system and the release of dopamine in the NAC.⁵⁸⁹ Decreased activation of the dopamine system may cause a lack of interest in natural rewards such as social events or recreational activities.⁵⁹⁰ Chronic drug use may impair thinking brain functions such as decision-making, attention, and impulse control.⁵⁹¹

Neuroplasticity, neurogenesis, and epigenetics ensure there is always a path to strengthen the lawyer brain.

VI. HEALING THE LAWYER BRAIN

NQ reveals that lawyers have a great deal of control over cognitive power, and that legal cultures can inspire or compromise the cognitive capacities of constituents. The lawyer brain is continuously evolving with each experience, thought, and feeling. Choices lawyers make can imperil or amplify cognitive function because the lawyer brain can grow new brain cells in the hippocampus (neurogenesis); activate and deactivate genes (epigenetics); and make new connections in its vast networks (neuroplasticity).⁵⁹²

Results from a 2011 meta-analysis (1,603 articles) of research on the relationship between exercise and cognition show that exercise can both prevent cognitive deterioration and heal cognitive damage.⁵⁹³ Exercise improved minor memory loss as well as the effects of Alzheimer's Disease.⁵⁹⁴ The key advantage was attributed to improved neurogenesis and neuroplasticity, facilitated by neurotrophins.⁵⁹⁵ A critical finding was that exercisers developed "significantly larger hippocampal volumes" and more robust synaptic connections.⁵⁹⁶

A study of 1.2 million Swedish boys who entered the military between 1950 and 1976 measured cardiovascular fitness and cognitive abilities.⁵⁹⁷ Car-

⁵⁸⁷ BEAR ET AL., *supra* note 53, at 569 box 16.5; GIBB, *supra* note 25, at 192.

⁵⁸⁸ BEAR ET AL., *supra* note 53, at 569 box 16.5; SWEENEY, *supra* note 15, at 202.

⁵⁸⁹ CARLSON, *supra* note 208, at 624.

⁵⁹⁰ MCKIM & HANCOCK, *supra* note 126, at 126.

⁵⁹¹ *Id.*

⁵⁹² CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 193; HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 8; SWEENEY, *supra* note 15, at 17; Bargmann & Gilliam, *supra* note 68; *see* DOIDGE, *supra* note 7, at 45–47.

⁵⁹³ RATEY & MANNING, *supra* note 67, at 105–06. Sedentary lifestyle has been associated with a decline in cognitive skills and the following diseases: heart failure, heart attack, coronary artery disease, hypertension, stroke, type 2 diabetes, asthma, obstructive pulmonary disease, arthritis, immune dysfunction, and cancers of the breast, colon, prostate, and pancreas. *Id.* at 104.

⁵⁹⁴ *Id.* at 106.

⁵⁹⁵ *Id.* at 107.

⁵⁹⁶ *Id.*

⁵⁹⁷ *Id.* at 109.

dio fitness was a better predictor of cognitive ability and IQ than familial history, and those with higher fitness scores had better education, greater life satisfaction, and higher socioeconomic standing as adults.⁵⁹⁸ Exercise has improved standardized test scores for students at schools in Illinois⁵⁹⁹ and California.⁶⁰⁰

A. *Exercise and Brain Health*

Nike's ubiquitous meme "Just Do It"⁶⁰¹ is the right advice for the lawyer who wishes to enhance or heal her brain. "[E]xercise is the single most powerful tool you have to optimize your brain function."⁶⁰² Aerobic exercise—that which increases the lawyer heart rate for an extended period⁶⁰³—benefits the lawyer brain in three ways:

- (1) it increases blood and oxygen flow;
- (2) it elevates and balances important neurotransmitters; and
- (3) it stimulates the release of neurotrophins.⁶⁰⁴

When a lawyer raises her heart rate with aerobic exercise, her blood vessels produce nitric oxide, which allows blood to flow deeper into body and brain tissues.⁶⁰⁵ The lawyer brain requires 25 percent of the body's blood flow, 20 percent of its oxygen, and 25 percent of its calories.⁶⁰⁶ Greater blood flow maximizes the distribution of oxygen and nutrient resources to the brain, and improves elimination of waste.⁶⁰⁷ One study put a group of volunteer participants on a three-month exercise regimen, and brain scans showed a 30 percent increase in their hippocampal capillaries.⁶⁰⁸ Exercise increases blood volume in the hippocampus, keeping it hearty and well-functioning.⁶⁰⁹

Normal brain function requires complex synaptic transmission with the aid of a delicate concoction of neurotransmitters. No activity beats aerobic exercise for taming transmitters and creating neurochemical harmony in the brain. Signaling in the brain is largely the responsibility of glutamate (excites and activates neurons) and GABA (calms and inhibits neurons).⁶¹⁰ Regulating brain processes, such as signaling, is the duty of serotonin, norepinephrine, and do-

⁵⁹⁸ *Id.*

⁵⁹⁹ RATEY, *supra* note 54, at 9–12.

⁶⁰⁰ *Id.* at 21–22.

⁶⁰¹ *Just Do It*, NIKE, http://www.nike.com/us/en_us/c/justdoit (last visited May 15, 2015).

⁶⁰² RATEY, *supra* note 54, at 245.

⁶⁰³ AMEN, *supra* note 22, at 110.

⁶⁰⁴ *Id.*; HORSTMAN, BRAVE NEW BRAIN, *supra* note 25, at 29; MEDINA, *supra* note 12, at 22; PERLMUTTER & VILLOLDO, *supra* note 25, at 87–97; RATEY, *supra* note 54, at 38.

⁶⁰⁵ MEDINA, *supra* note 12, at 21.

⁶⁰⁶ AMEN, *supra* note 22, at 17.

⁶⁰⁷ MEDINA, *supra* note 12, at 21–22.

⁶⁰⁸ RATEY, *supra* note 54, at 246.

⁶⁰⁹ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 65; MEDINA, *supra* note 12, at 22; Amara & Strick, *supra* note 186, at 349.

⁶¹⁰ BROAD, *supra* note 54, at 99; RATEY, *supra* note 54, at 37.

pamine.⁶¹¹ Most drugs aimed at improving mental health issues target these regulating neurotransmitters.⁶¹² Serotonin affects mood, emotion, appetite, and sleep.⁶¹³ Norepinephrine governs attention, arousal, heartbeat, and blood pressure.⁶¹⁴ Dopamine manages motivation and reinforcement.⁶¹⁵ Aerobic exercise has the unique power to both elevate and balance these key regulating transmitters.⁶¹⁶

While neurotransmitters ensure communication between neurons, a group of proteins called neurotrophins are responsible for cultivating brain cells.⁶¹⁷ The neurotrophins are brain-derived neurotropic factor (“BDNF”),⁶¹⁸ insulin-like growth factor (“IGF-1”), vascular endothelial growth factor (“VEGF”), and fibroblast growth factor (“FGF-2”).⁶¹⁹ These neurotrophins are all elevated by aerobic exercise.⁶²⁰

BDNF was discovered in 1990 to nourish brain cells in the hippocampus like a fertilizer,⁶²¹ making it especially important to learning and memory.⁶²² BDNF is essential to neurogenesis because it helps create new neurons, and it is crucial to neuroplasticity because it facilitates synapse formation.⁶²³ Vital also to epigenetics, the gene that boosts BDNF production is switched on by exercise and intellectual stimulation.⁶²⁴ Nutrition plays a role in the acquisition of BDNF. Excess sugar decreases BDNF, while food rich in folate, Vitamin B12, curcumin (the active ingredient in turmeric), and omega-3 fats increase BDNF.⁶²⁵

BDNF causes the growth of new dendrites, the neuron branches that receive information from the presynaptic axon.⁶²⁶ It binds to the receptors on the

⁶¹¹ RATEY, *supra* note 54, at 37.

⁶¹² *Id.* at 37–38.

⁶¹³ CARTER ET AL., HUMAN BRAIN, *supra* note 12, at 231; CARTER, MAPPING THE MIND, *supra* note 20, at 28; HORSTMAN, DAY IN THE LIFE, *supra* note 11, at 8, 34; RATEY, *supra* note 54, at 37; SWEENEY, *supra* note 15, at 15, 221.

⁶¹⁴ SWEENEY, *supra* note 15, at 15, 221.

⁶¹⁵ CARTER, MAPPING THE MIND, *supra* note 20, at 29.

⁶¹⁶ MEDINA, *supra* note 12, at 16–17; RATEY, *supra* note 54, at 38.

⁶¹⁷ RATEY, *supra* note 54, at 38.

⁶¹⁸ *Id.*

⁶¹⁹ *Id.* at 51.

⁶²⁰ DOIDGE, *supra* note 7, at 255; RATEY, *supra* note 54, at 50–53.

⁶²¹ AMEN, *supra* note 22, at 110; MEDINA, *supra* note 12, at 22; RATEY, *supra* note 54, at 38–39.

⁶²² RATEY, *supra* note 54, at 39.

⁶²³ AAMODT & WANG, *supra* note 193, at 89; DEEPAK CHOPRA & RUDOLPH E. TANZI, SUPER BRAIN: UNLEASHING THE EXPLOSIVE POWER OF YOUR MIND TO MAXIMIZE HEALTH, HAPPINESS, & SPIRITUAL WELL-BEING 35 (2012); DOIDGE, *supra* note 7, at 80; PERLMUTTER & VILLOLDO, *supra* note 25, at 87.

⁶²⁴ MEDINA, *supra* note 12, at 22; PERLMUTTER & VILLOLDO, *supra* note 25, at 88–92.

⁶²⁵ PERLMUTTER & VILLOLDO, *supra* note 25, at 93; RATEY & MANNING, *supra* note 67, at 93.

⁶²⁶ RATEY, *supra* note 54, at 40.

postsynaptic dendrites, improving the signal strength of the information.⁶²⁷ Inside the neuron, BDNF activates the genes that produce serotonin and increase BDNF production.⁶²⁸ BDNF behaves like a brain cell fertilizer because it grows, strengthens, and protects neurons.⁶²⁹

Scientists have learned about the importance of BDNF through rodent studies because rodents naturally enjoy activity.⁶³⁰ Mice who ran on a wheel increased their hippocampal BDNF over sedentary control-group mice, and the more they ran, the higher the BDNF increases.⁶³¹ BDNF increases the rate of learning.⁶³² Mice are not fond of water, so one experiment tested their ability to remember a platform escape route from their rodent swimming pool.⁶³³ Wheel-runners remembered the escape route and swam for it immediately.⁶³⁴ Couch potato mice struggled to figure out how to exit the pool, and when dissected, wheel-runners had twice the new hippocampal neurons as couch potatoes.⁶³⁵ In 2007, researchers showed that humans learned vocabulary words 20 percent faster after exercise than before exercise, and that the learning rate correlated directly with levels of BDNF.⁶³⁶

The three other neurotrophins (IGF-1, VEGF, and FGF-2) are elevated by exercise and work with BDNF to build and maintain brain cell circuitry.⁶³⁷ During exercise, IGF-1 is released by muscles when they sense a need for glucose.⁶³⁸ Glucose is the sole source of energy for the brain and the major source for muscles, and IGF-1 works with insulin to deliver glucose to the cells.⁶³⁹ BDNF and IGF-1 collaborate to empower learning because during exercise, BDNF increases the uptake of IGF-1.⁶⁴⁰ IGF-1 then activates glutamate and serotonin release, and encourages BDNF receptor growth, enhancing the synaptic connection necessary for memory consolidation.⁶⁴¹ Glucose empowers learning,⁶⁴² new blood vessels are needed to deliver glucose to the cells, and VEGF builds more capillaries in both the body and brain when exercise signals

⁶²⁷ *Id.*

⁶²⁸ *Id.*

⁶²⁹ *Id.*

⁶³⁰ *Id.* at 44.

⁶³¹ *Id.*

⁶³² *Id.* at 45.

⁶³³ *Id.* at 50.

⁶³⁴ *Id.*

⁶³⁵ *Id.*

⁶³⁶ *Id.* at 45.

⁶³⁷ LEDOUX, *supra* note 14, at 81, 153; RATEY, *supra* note 54, at 51–53.

⁶³⁸ RATEY, *supra* note 54, at 52.

⁶³⁹ *Id.*

⁶⁴⁰ *Id.*

⁶⁴¹ *Id.*

⁶⁴² *Id.*

low oxygen.⁶⁴³ FGF-2 aids cell growth and facilitates long-term potentiation (“LTP”).⁶⁴⁴

As lawyers age, the natural production of the neurotrophins is reduced.⁶⁴⁵ This depresses the rate of neurogenesis.⁶⁴⁶ Aerobic exercise increases BDNF, IGF-1, VEGF, and FGF-2 in the lawyer brain, so neural degeneration can be curtailed or delayed.⁶⁴⁷

A busy lawyer who is now convinced that she should either start or increase aerobic exercise to empower her brain might wonder how much exercise is necessary. Thirty minutes of exercise two or three times per week can improve cognitive function.⁶⁴⁸ Daily exercise elevates BDNF more than on alternate days.⁶⁴⁹ The bottom line is the more fit the body, the greater the cognitive benefit.⁶⁵⁰ Our genes are coded for consistent activity, from a time when we had to worry about locating and acquiring food.⁶⁵¹ To fully engage the endurance metabolism, lawyers should do a low or moderate-intensity activity every day and a high-intensity activity a couple of times per week.⁶⁵² Interval training increases BDNF and norepinephrine production.⁶⁵³ Should a lawyer get sick or injured and have to suspend exercise for a short period, the hippocampus has a “molecular memory” for producing BDNF, and even if exercise is paused for two weeks, it takes just two days of exercise to return the hippocampus to BDNF fighting form.⁶⁵⁴

A lawyer who enjoys strength training might wonder about its benefits. When weight lifters train, they engage in a process known as hormesis.⁶⁵⁵ Hormesis is a response to a low dose of a stressor, such as exercise, that damages muscles due to overuse and then repairs them at bigger and better strength.⁶⁵⁶ Hormesis develops resilience in the body and brain.⁶⁵⁷ One study showed that older adult weight lifters increased levels of IGF-1, VEGF, and FGF-2 to levels of a thirty-year-old, rather than their average age of sixty-five years.⁶⁵⁸ Yoga practitioners are able to increase their GABA levels, even when

⁶⁴³ *Id.*

⁶⁴⁴ LEDOUX, *supra* note 14, at 153; RATEY, *supra* note 54, at 52–53.

⁶⁴⁵ AMEN, *supra* note 22, at 161–62; RATEY, *supra* note 54, at 52–53.

⁶⁴⁶ RATEY, *supra* note 54, at 53.

⁶⁴⁷ *Id.* at 51–53.

⁶⁴⁸ *Id.* at 55.

⁶⁴⁹ *Id.* at 261.

⁶⁵⁰ *Id.* at 245–51.

⁶⁵¹ *Id.* at 248.

⁶⁵² *Id.* at 248–50.

⁶⁵³ *Id.* at 257.

⁶⁵⁴ *Id.* at 261–62.

⁶⁵⁵ RATEY & MANNING, *supra* note 67, at 103.

⁶⁵⁶ *Id.*

⁶⁵⁷ *Id.*

⁶⁵⁸ RATEY, *supra* note 54, at 258.

they are new to the practice, although more experienced yogis show the greatest GABA increases.⁶⁵⁹

Complex movement, that which requires skills greater than mindless treadmill running or stationary bike pedaling, provides different beneficial effects in the brain.⁶⁶⁰ Rats trained on an obstacle course had a 35 percent increase in BDNF in the cerebellum (largely responsible for movement), where wheel-running rats had none.⁶⁶¹ Beneficial practices such as yoga, dance, Pilates, karate, and CrossFit can build robust brain resources that can be coopted for cognition.⁶⁶²

B. Exercise and Stress, Anxiety, and Depression

“Evolution has made provisions for our happiness, but to take advantage of them you’ve got to move.”⁶⁶³

The hippocampus is “obsessed with the stress,”⁶⁶⁴ and it is extremely sensitive to the stress hormone cortisol.⁶⁶⁵ A small amount of cortisol helps memory formation, but too much suppresses memories.⁶⁶⁶ The fight-or-flight SNS stress response begins when cortisol and norepinephrine boost glutamate production, which speeds up the flow of information in the hippocampus.⁶⁶⁷ Cortisol activates the genes that produce neurotrophins, which generate additional dendrites and receptors, and forge more powerful synapses.⁶⁶⁸ These new brain resources vigorously secure the stress-based survival memory, while buffering the neurons in that circuit from other memories.⁶⁶⁹ Thus, memories that are not related to the stressor are obstructed during the fight-or-flight response.⁶⁷⁰ This explains why it is hard to learn new material, and also why studies show it is difficult to retrieve existing memories, when the lawyer brain is exposed to chronic stress.⁶⁷¹ The neurons in the hippocampus have boosted glutamate and diverted neurotrophins to shut out the stimuli that are not related to survival.⁶⁷² When subjected to stress hormones, the hippocampus shrinks and the extent of

⁶⁵⁹ BROAD, *supra* note 54; RATEY, *supra* note 54, at 259.

⁶⁶⁰ RATEY, *supra* note 54, at 55–56; RATEY & MANNING, *supra* note 67, at 119–22.

⁶⁶¹ RATEY, *supra* note 54, at 55.

⁶⁶² *Id.* at 56; RATEY & MANNING, *supra* note 67, at 120–22.

⁶⁶³ RATEY & MANNING, *supra* note 67, at 119.

⁶⁶⁴ RATEY, *supra* note 54, at 68.

⁶⁶⁵ *Id.* at 66–67.

⁶⁶⁶ *Id.* at 67.

⁶⁶⁷ *Id.*

⁶⁶⁸ *Id.*

⁶⁶⁹ *Id.*

⁶⁷⁰ *Id.* at 68.

⁶⁷¹ *Id.*

⁶⁷² *Id.*

hippocampal reduction and memory loss is directly proportional to the rise in cortisol.⁶⁷³

BDNF, the premier neurotrophin, can protect against stress hormones as long as there are sufficient amounts in the brain.⁶⁷⁴ Exercise increases all the neurotrophins, helping to inoculate the brain against stress.⁶⁷⁵ Exercise raises the fight-or-flight response threshold, subdues cortisol release, and increases and balances the regulatory transmitters serotonin, norepinephrine, and dopamine.⁶⁷⁶

Exercise is helpful in both treating and preventing anxiety and depression.⁶⁷⁷ Scientists believe the reason it is ameliorative and preventative is because it elevates and rebalances levels of serotonin, norepinephrine, and dopamine in the brain.⁶⁷⁸ In 2010, the American Psychiatric Association listed exercise as a proven depression treatment.⁶⁷⁹

Stress and depression can cause a drop in neurotrophin levels and a corresponding reduction in neurogenesis.⁶⁸⁰ Exercise increases BDNF, IGF-1, VEGF, and FGF-2, so it is a powerful prescription for relieving stress and improving depression.⁶⁸¹

Getting treatment for anxiety and depression can reduce stress and reverse hippocampal damage.⁶⁸² Research shows that exercise significantly mitigates symptoms of anxiety disorders.⁶⁸³ Exercise increases GABA, the calming transmitter that is a primary target for anti-anxiety medications.⁶⁸⁴ It also elevates serotonin, which helps the prefrontal cortex calm the panicky amygdala.⁶⁸⁵

When the lawyer increases her respiration and heart rates with exercise, she is training her brain to understand that the symptoms of fight-or-flight activation can be beneficial.⁶⁸⁶ Several large studies have concluded that exercisers are less anxious and depressed, less stressed and more socially outgoing.⁶⁸⁷ In addition to exercise, lawyers could add a restorative practice like yoga, mind-

⁶⁷³ *Id.* at 76.

⁶⁷⁴ MEDINA, *supra* note 12, at 179.

⁶⁷⁵ RATEY, *supra* note 54, at 72–73.

⁶⁷⁶ *Id.* at 78–79.

⁶⁷⁷ MEDINA, *supra* note 12, at 16–17; RATEY, *supra* note 54, at 37–38.

⁶⁷⁸ MEDINA, *supra* note 12, at 16–17; RATEY, *supra* note 54, at 37–38.

⁶⁷⁹ RATEY & MANNING, *supra* note 67, at 110.

⁶⁸⁰ RATEY, *supra* note 54, at 53.

⁶⁸¹ *Id.*

⁶⁸² *See id.*

⁶⁸³ *Id.* at 92.

⁶⁸⁴ *Id.*

⁶⁸⁵ *Id.* at 107.

⁶⁸⁶ *Id.*

⁶⁸⁷ *Id.* at 119.

fulness, meditation, or gratitude, shown to empower the rest-and-digest PNS and reduce the impacts of stress.⁶⁸⁸

People with depression suffer from low levels of BDNF.⁶⁸⁹ Antidepressant medications increase the rate of neurogenesis.⁶⁹⁰ Exercise increases BDNF at least as much, and sometimes more, than antidepressants.⁶⁹¹ Research shows that exercise augments antidepressants in some patients,⁶⁹² and for others it is equally or more effective compared to the medication.⁶⁹³ Perhaps the most significant finding is that the more the patient exercises, the greater the improvement in mood.⁶⁹⁴ This may be due to the power of exercise to balance neurotransmitters and increase neurotrophins, the building blocks of brain cells.⁶⁹⁵

C. Exercise and Substance Use

All the things a lawyer could become addicted to—caffeine, nicotine, alcohol, drugs of abuse, or food—augment dopamine levels in the NAC.⁶⁹⁶ While having sex increases dopamine levels from 50 percent to 100 percent, cocaine inflates the dopamine rate a mind-boggling 300 percent to 800 percent above normal levels.⁶⁹⁷ The powerful incentive salience of drugs tricks the user brain, via the reinforcing capacity of dopamine, into thinking that this form of reward seeking is a matter of life and death.⁶⁹⁸ Drugs activate the dopamine system in unnatural ways.⁶⁹⁹

Exercise can change brain chemistry and rewire connections harmed by drug use or addiction.⁷⁰⁰ Damaged brain cells can grow back.⁷⁰¹ Research, demonstrating the hope that neurogenesis can inspire, suggested that chickadees which learn new songs every spring sprout a significant number of new brain cells in the hippocampus in the process.⁷⁰² Simply providing a running

⁶⁸⁸ See Debra S. Austin, *Killing Them Softly: Neuroscience Reveals How Brain Cells Die From Law School Stress & How Neural Self-Hacking Can Optimize Cognitive Performance*, 59 LOY. L. REV. 791, 837–47 (2013).

⁶⁸⁹ RATEY, *supra* note 54, at 131.

⁶⁹⁰ Hyman & Cohen, *supra* note 176, at 1413.

⁶⁹¹ RATEY, *supra* note 54, at 131.

⁶⁹² *Id.* at 125–26.

⁶⁹³ *Id.* at 122–24.

⁶⁹⁴ *Id.* at 124.

⁶⁹⁵ *Id.* at 132.

⁶⁹⁶ *Id.* at 170.

⁶⁹⁷ *Id.*

⁶⁹⁸ *Id.* at 171.

⁶⁹⁹ *Id.*

⁷⁰⁰ *Id.* at 180–81.

⁷⁰¹ *Id.* at 48.

⁷⁰² *Id.* at 49.

wheel for a rodent results in profound cell birth.⁷⁰³ Lawyers can grow several thousand new brain cells in the hippocampus every day.⁷⁰⁴

For addicts, exercise can blunt withdrawal symptoms by calming the amygdala and increasing dopamine production.⁷⁰⁵ A 2004 study showed that a mere ten minutes of exercise could reduce cravings in alcoholics.⁷⁰⁶ Exercise can improve self-regulation.⁷⁰⁷ Students on a two-month exercise program were given psychological tests every two weeks and they recorded their daily habits in journals.⁷⁰⁸ Tests showed they improved their self-control, smoked less, drank less caffeine and alcohol, ate a healthier diet, lost their tempers less often, and increased their gym visits.⁷⁰⁹

Research on the phenomenon of the runner's high has been focusing on endocannabinoids such as anandamide and 2-arachidonoylglycerol ("2-AG").⁷¹⁰ Both of these endocannabinoids are produced in the body and brain during exercise.⁷¹¹ When endocannabinoid receptors are strongly activated, the result is a feeling of euphoria, much like that produced from drug use.⁷¹² Endocannabinoid receptors are activated by cannabis, exercise, and chocolate.⁷¹³

Engaging the lawyer endurance metabolism by doing a combination of low to moderate intensity activity every day and a high-intensity activity two to three times per week, maximizes cognitive and physical benefit.⁷¹⁴ Pushing exercise limits, even for short periods of time, increases transmitters, endorphins, and endocannabinoids.⁷¹⁵ When fit male college students ran on treadmills or spun on stationary bikes for fifty minutes, at 70 percent to 80 percent of their maximum heart rate, they increased the endocannabinoid anandamide levels in their blood by nearly 100 percent.⁷¹⁶ Interval training is a good way to work on this, and small intensity-level increases elevate BDNF and norepinephrine levels.⁷¹⁷ One study showed that interval training improved learning when treadmill runners incorporated two three-minute sprints over a forty-minute run.⁷¹⁸

⁷⁰³ *Id.*

⁷⁰⁴ CHOPRA & TANZI, *supra* note 623, at 34.

⁷⁰⁵ RATEY, *supra* note 54, at 179, 182.

⁷⁰⁶ *Id.* at 177.

⁷⁰⁷ *Id.* at 188.

⁷⁰⁸ *Id.*

⁷⁰⁹ *Id.*

⁷¹⁰ *Id.* at 182–83.

⁷¹¹ *Id.* at 183.

⁷¹² *Id.*

⁷¹³ *Id.*

⁷¹⁴ *Id.* at 248.

⁷¹⁵ *Id.* at 256–57.

⁷¹⁶ *Id.* at 183.

⁷¹⁷ *Id.* at 257.

⁷¹⁸ *Id.*

Exercise elevates dopamine levels as well as endorphins.⁷¹⁹ This improves attention and motivation, and it is reinforcing for any activity that raises the lawyer heart rate, such as running, swimming, cycling, rowing, or CrossFit.⁷²⁰ It also releases endocannabinoids in the lawyer brain, making him feel better during and after exercise.⁷²¹

D. *Exercise and Culture*

Neuroendocrinologist Bruce S. McEwen and researcher Linn Getz describe individuals by their tolerance for environmental challenges as dandelions who can thrive anywhere, and orchids who are much more vulnerable.⁷²² Armed with NQ, lawyers can improve resilience and build brain capacity, moving closer to the dandelion end of the scale.⁷²³

An environment that combines learning, exercise, and social contact causes neurons to propagate new dendrites, form more synaptic connections, and improve signaling because the axons have thicker myelin coating.⁷²⁴ The lawyer brain can grow several thousand hippocampal brain cells each day, and this process is hastened by exercise, intellectual stimulation, and social connectedness.⁷²⁵ Cultures in which law students are educated and lawyers are at work should engender these values. Law schools, law firms, and court systems should facilitate workout groups rather than happy hours.

Now that we know dopamine is responsible for reward reinforcement⁷²⁶ and endogenous opioids and/or cannabinoids are responsible for experiencing pleasure,⁷²⁷ exercise is the activity lawyers should all be engaging in to:

- build brain resources,
- extinguish the impacts of stress,
- protect against anxiety and depression,
- prevent cognitive decline, and
- replace substance use.

Exercise should be elevated from a “Just Do It”⁷²⁸ position to a “Just Do It Again” imperative, because dopamine is the “Do-It-Again” neurotransmitter.⁷²⁹

⁷¹⁹ *Id.* at 121.

⁷²⁰ *Id.*

⁷²¹ *Id.* at 257.

⁷²² RATEY & MANNING, *supra* note 67, at 238.

⁷²³ *Id.* at 239.

⁷²⁴ RATEY, *supra* note 54, at 47.

⁷²⁵ CHOPRA & TANZI, *supra* note 623, at 34–35.

⁷²⁶ MCKIM & HANCOCK, *supra* note 126, at 119; MEYER & QUENZER, *supra* note 103, at 251–52.

⁷²⁷ MCKIM & HANCOCK, *supra* note 126, at 119.

⁷²⁸ *See supra* note 601 and accompanying text.

⁷²⁹ MCKIM & HANCOCK, *supra* note 126, at 119.

CONCLUSION

Starting in law school, the legal profession's continuous stress on the lawyer brain can lead law students, lawyers, law professors, and judges to depression, alcoholism, and drug abuse. This stress impairs the cognitive function of the lawyer brain. However, if law schools, law firms, and court systems support healthier lifestyles to improve cognitive wellness, such as exercise and diet, it will have a positive impact on the culture of the legal profession.