

Factors in Neurotoxicity in Adolescents

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Introduction

Our four-year study on adolescent mood and emotion biochemistry completed its first year in March 2002. The study is investigating the impact of essential amino acids and fatty acids on neurological neurotransmitter and neuropeptide functions. The study utilizes plasma amino acid levels taken at four-month intervals and looks at deficiencies and/or excesses of specific amino acids and their impact on ion channel function. We are looking at predictor molecules such as those amino acids which impact conventional transmitter development as in GABA and acetylcholine. We are investigating environmental impacts on developing adolescent brains.

The study examines neurotransmitters and the chemical languages of the brain. Neurotransmitters may be either excitatory or inhibitory, and they have a major impact on ion channels. In each nerve cell there is a need for strict control of the properties and localization of the channels. For example, the glutamate sensitive channels need to be localized at synaptic junctions where glutamate is released from axonal terminals. Transmitters can have two effects at synapses: one is to open or close ionic channels; the other is to trigger a change in the rate of a chemical reaction. Conventional transmitters such as glutamate, GABA or acetylcholine usually act as fast transmitters triggering the opening or closing of a channel and a subsequent change in the electrical potential of the receiving cell.¹ Change in reaction is regulated by enzymatic reactions. This often occurs when there is a co-regulation of conventional and peptide transmitters.

The purpose of our neuro-chemical inquiry is to correlate conventional trans-

mitter function with amino acid and neurotransmitter levels and to look at a rapid firing excitatory response, such as from glutamate, in relation to functional neurotransmitter availability in a given subject. For example, among adolescents who have been labeled hyperactive we have detected a pattern of high levels of glutamate, and low potentiation of inhibitory substances such as GABA, and often, glycine.

The study focuses on average teenagers. If one looks at a bell curve of teenagers today in the USA, only a relatively small percentage of them are really thriving. Based on our observations, we estimate that less than 20% of adolescents today are doing really well. At the far end of the bell curve are the adolescents with serious neurological/psychiatric issues: suicidal depression, schizophrenia, bipolar and violent behaviors. The teenagers we are investigating comprise the 65 to 75% of the average kids today that are just not thriving. Essentially we are looking at moody, sullen girls and angry, hostile boys.

We are in a unique position to study this issue because we integrate our research with a clinical practice where people come to talk about their anxiety and depression and learn how their biochemistry impacts their lives and their moods. We also work with a number of physicians and psychiatrists around the country.

We always begin by looking at the subject's amino acid levels, through plasma testing, because these are the building blocks from which all the neurotransmitters and neuropeptides are made.

The Clinical Functions of Methionine and Tryptophan

Methionine: We find methionine deficiencies in many depressed adolescents, more so in boys than in girls. We increase the amount of a given amino acid in ratio to the other amino acids when custom

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developing amino acid protocols for a given child. After the imbalance is corrected, if the depression remains, which is not common, we then utilize larger molecules such as SAmE (S-adenosyl-methionine) in low doses. Often we start at 100mg, bid. Methionine metabolism disorders have a diverse symptomology and the pathways are dependent on vitamins B₆, B₁₂, and folate. Sub-clinical symptoms include allergic tendencies, headaches, muscle weakness and depression.²

Tryptophan: This is an extraordinarily important amino acid and it has great value both in the treatment of depression and in pain control. The molecule itself is safe; there were problems in the early 1990's when a short-cut in its synthesis was used and the culture became contaminated. Now we primarily use 5-HTP (5-hydroxy-tryptophan) the intermediary metabolite of tryptophan to serotonin. 5-HTP is extracted from the Griffonia seed through a multi-step process. The seeds come from an African tree grown mostly in Ghana and the Ivory Coast. 5-HTP is not as effective in pain control, but can help depression of the type associated with anger, which the SSRI psychotropic drugs can benefit, at least as band-aids.

Increases or decreases in tryptophan levels lead to concomitant changes in serotonin levels because it is likely that tryptophan is the single most important rate-limiting factor in the synthesis of serotonin.³ The increased brain levels of tryptophan and serotonin as a consequence of protein inadequacies can best be articulated as the amounts of free plasma tryptophan needed to be available to the brain at the expense of the periphery.

From a teleological viewpoint, the increases in brain serotonin and 5-hydroxy-indoleacetic acid may be only the consequence of a need to assure the availability for adequate brain protein synthesis and development early in life.⁴ Tryptophan serves as a precursor to niacin and serot-

onin (approximately 10% of dietary protein is used for serotonin synthesis). Tryptophan has been shown to help induce sleep due to the production of serotonin in the brain stem.⁵ Tryptophan has been used extensively to help aggression, and depression associated with rage; it may potentate the therapeutic action of certain anti-depressants. Our current research at The University of Denver supports the premise that the 5-HTP molecule is compatible with the SSRI class of drugs according to the molecular spectra, as seen in Fourier Transform Infrared Spectra. This means that the molecules are safe to be used congruently, that the mechanism of action of the allopathic, psychotropic drugs is probably enhanced by increased levels of tryptophan, and conversely, there is less need for the medication in many individuals as they attain optimal levels of these neuro amino acids

Psychotropic Medication Use in Children

The increasing use of psychotropic medication on children in the United States is alarming. Five million American children are taking psychiatric drugs today. For ages 6 and under the usage is up 580%; for ages 7 to 12 it is up 151% since 1991.^{6,7} We are not anti-drug, but we find it alarming that so many children are being given psychotropic drugs, this is in addition to the those who are taking stimulants such as Adderall® (a mixture of four different amphetamine salts) and Concerta® (a reformulation of Ritalin for sustained delivery) and Ritalin.® We will utilize psycho-pharmaceuticals as band-aids when seeing somebody who is in a dangerous mental state, suicidal for example. We strive to wean them off the medications using a safe integration of biochemical protocols. These drugs are alarming in their neuro-chemical implications and many children and adolescents we see have been on them for a long time.

Parents often ask if children's moods are getting worse and we think that they are. We want to provide a biochemical con-

jecture for why this might be so. There is a complex interplay between the neuronal chemistry and neuronal development of children and adolescents, especially in the prefrontal cortex of the developing brain, which is the seat of reason and logic. Adolescent boys do not have a fully developed prefrontal cortex until about 25 years of age, while for girls it's about 18 months younger. We conjecture that exposure of the undeveloped prefrontal cortex to xenobiotic chemicals directly or indirectly affects the plasticity of the brain. If you expose a vulnerable child's brain—meaning a child who's vulnerable to dopaminergic excess—and you combine that with an undeveloped prefrontal cortex, you have a person who will go from anger to revenge without an intermediary process of reason and logic. This is, simply, a brain too young for good judgment. We expect our kids to show good judgment when they don't have the neuronal capacity to do so.⁸

Part of teaching children how to develop well is to teach them to anticipate breakdowns in life. Breakdowns are constitutive of life, of course, but learning to anticipate breakdowns is a capacity that the developing brain has to learn. Many of the adolescents who show high-risk behavior have a tendency to dopaminergic excess, or an attraction to dopaminergic chemicals, such as cocaine, and they go hunting for this adrenaline rush.

In our research we have a section where we're looking at the biochemical proclivity to certain substances. Adolescents who are deficient in dopamine precursors tend to like stimulant molecules, and those who are deficient in serotonin are much more attracted to hallucinogens or marijuana. If you look at the structure of psilocybin, a hallucinogen, and serotonin, the molecules are remarkably similar. A person deficient in serotonin is likely to be attracted to these hallucinogenic drugs if they tend to have addictive behavior. The problem is that when they take the altered sub-

stance, their brains get an overload of serotonin-like messages, and they lose the boundaries between reality and perception. That's an especially difficult dance neurochemically.

Moody, Sullen Girls

Our recent four-year study on women's mood biochemistry⁹ looked at women at peri-menopause from ages 47 to 51 and found that a lot of women are in a "bad mood." In correlating this data on irritable midlife women to the bad moods of their adolescent daughters, the data infer they often have a similar hormonal make-up. The irritable ones tend to be estrogen dominant, and the sullen ones lean more toward depression and low levels of hormones as they mature. How this coordinates with ionic channel function is also important in that all neurochemical impacts have an effect on the manner in which neurons fire.

In our experience the girls are much more difficult to deal with than the boys. In general the girls are meaner than boys and often they are either herd-oriented in cliques, or are fierce and aggressive. The girls are often blame-oriented and irritable¹⁰ and they're hard on the boys. The despair that we're hearing about from boys is a pervasive issue. It's no longer okay to say, "Well, kids will just be kids." It's simply not true. There are children who really need to be reigned in; they can be dangerous to others; it's children abusing children. Adolescents will try on different personalities, different ways of behaving, the same way they try on different clothes. Unfortunately, a lot of them start experimenting with chemical substances, either the abuse of prescription drugs, or illicit substances, thus altering their experience of life because they have a chemical experience instead of a natural experiential one. If they're given pharmaceuticals at too young an age they don't learn how to process problems properly; they're experiencing problem-solv-

ing through a chemical matrix.

From our study on women's biochemistry, we can conjecture backward from the midlife women and see how their daughters are presenting in adolescence. Contrary to some of the popular literature in nutrition and hormones today there is a lot of diversity in how hormones impact the brain. When we started our four-year study, we thought progesterone was the answer to everything and we learned, it really is not, rather there is a dance between progesterone and estrogen that differs significantly depending upon whether a woman is estrogen dominant or estrogen deficient. Our study identified two important groups of women: women prone to anxiety and women prone to depression. The women prone to anxiety and irritability felt much better on natural micronized progesterone. They were in peri-menopause, so they were still cycling. As they got closer to menopause, a lot of those women actually needed estrogen, but used progesterone to deal with their anxiety. The women in our other group had more of a flat-affect kind of depression, and responded beautifully to estrogen. The beta-estradiol and estriol, which is the Tri-Est[®] that we work with from the compounding pharmacies, had a beneficial impact on depression.

Adolescent girls tend to be estrogen-dominant; they are having an-ovulatory cycles, they're not ovulating properly and their hormone chemistry often mimics that of their midlife mothers. In the latter part of their cycle these irritable girls tend to have pronounced PMS when this should be the progesterone-dominant part of the cycle. We postulate that xenobiotics and foreign estrogen-mimicking chemicals disrupt the accessibility to the progesterone receptors.

Angry, Hostile Boys

The herd mentality is predominant in our culture more and more, thus the prescribing of stimulant drugs to children to try to fit the round peg children into square

holes of a structured existence. Something we observed a couple of years ago in Aspen, Colorado, where outdoor education is almost a holy thing, is a poignant example of this pigeon holing. In our clinic, we have had a number of children tell us how they hate going on trips away from home with lots of other kids. It comes as kind of a revelation when you start hearing these stories. A couple of years ago, a seven year old who was the mascot for the Aspen Volunteer Fire Department had three aggressive third grade teachers standing over him, badgering him, saying, "Why don't you want to go on Outdoor Ed? You're going to have fun; and, it's going to be great!" In situations like this, boys will do just about anything to save face, so this little guy said: "Well, you know, I'd really like to help you out but I'm with the fire department and there's going to be a fire that night, and I'm on call." Boys try so hard to look good and to not deal with their emotional worlds. Twenty years later their wives are in our office saying; "My husband won't talk about his feelings." They learned not to a long time ago. Men need to learn how to talk about difficult issues, and women need to learn to contain their emotions; just because they "feel it" doesn't mean it requires expression all the time, which exhausts men. Of course, the stereotypes of boys don't fit for a lot of our children. They get told: "if you don't like team sports you're a jerk," and "if you don't do this you're an idiot." This is a form of abuse.

In November, 2001, we began studying boys 8-12 year-olds, because so many parents were telling us that their boys were having emotional issues well before adolescence. In addition to the "moody, sullen girls" and "angry, hostile boys" in our adolescence study we included an 8-12 year-old group of children that's getting quite big. We have divided them into three categories: victims, bullies, and odd boys. Odd boys are often the most creative, interesting boys if they survive adolescence. We

are looking at their neurotransmitter chemistry and their fatty acid chemistry, and are testing for neuro-chemical similarities in these different categories of kids. Teenage suicide is on the rise. A lot of this has biochemical implications, which we are investigating. We think in terms of biochemistry, how biology and the psyche mirror one another. If a child has one adult who mirrors who he or she is, they will have a chance.^{11,12}

The Neurochemical Basis of Mood Biochemistry

Dendrites are the gossips of the neuron. They stand guard on the nerve cells, deciding whether a message is worth sending on to the brain. If an action potential is created, then the message gets sent down the axon hillock. A message enters the nervous system as an electrical impulse. The message travels down the axon from the axon hillock, and then crosses the synapse as chemical energy. We all experience our world through chemistry, but the message enters initially as an electrical potential. We studied neuronal inhibition, how the molecules that inhibit the over-firing in the amygdala - the emotional seat of the brain - work. GABA is an important molecule that is poorly understood in the pharmaceutical world. The benzodiazepine drugs, Valium® and all its grandchildren, work by up-regulating GABA (which is both a neurotransmitter and an amino acid).¹³ One of these grandchildren is Xanax® (alprazolam), which is the most widely prescribed drug in the world today.

The root of anxiety is simply that the brain is over-firing messages. If there is not enough neuro-inhibitory function at the synapse (i.e. insufficient neuro-inhibitors), the brain will over-fire. People prone to anxiety appear to have a unique biochemistry which tends towards this over-firing of messages, often as a result of insufficient neuro-inhibitors. For example, when a child who has a problem with over-sequencing sees

a bully, or someone he perceives to be a bully, his brain can jump straight into overdrive without being able to process the actual level of threat. In our clinic we have seen kids as young as five years old who have been prescribed anti-anxiety pharmaceuticals. If they were just given the proper neurotransmitter inhibitors it could change their lives.

An important part of our research is how to help people safely get off or reduce their dependence on these strong, highly addictive benzodiazepines. At our clinic, we are experimenting by carefully titrating increased levels of GABA in kids and having positive results. GABA is the most widely distributed inhibitory neurotransmitter in the brain, and is one of the most important regulators of brain function.¹⁴ Prolonged periods of stress and psychopharmaceutical use severely deplete GABA levels.¹⁵ We postulate that if the CNS has enough of the correct neuro-inhibitors (GABA being foremost, though there are others as well) in the synapses, then they will not over-fire and the tendency to anxiety will be reduced. There are many variations on the quantitative aspects of using these molecules and we are investigating these. Our understanding of these pathways comes largely from the research on the functional characteristics of GABA-A receptors, and their selective receptor agonists. One of the distinctive characteristics of GABA receptors is their widespread distribution and concentration throughout the CNS.

GABA is formed as part of the glutamine – glutamate metabolic pathway, but the nervous system cannot necessarily restore healthy levels. Physiologic and psychological stresses deplete the neuro-inhibitory transmitters. Glutamine has many functions, but if a person has an inability to process the metabolic pathway to making GABA, then the glutamine can metabolize into a first cousin, monosodium glutamate. It is important to start any treatment protocol with a good understanding of the biochemistry of the individual.

Our clinic treats a lot of children who could be greatly helped if their synaptic chemistry were improved. There are genetic tendencies, not even mutations, in families that go on for generations.

Environmental Chemistry: Hexane Soot, Dust Storms and Children's Brains

We obtained polyaromatic hydrocarbons by burning normal hexane at our lab in Denver to make hexane soot. This is what diesel fumes are made of, and the air is filled with these fumes. Jet fuel, which burns at high pressure and high temperature, and then falls off at low pressure, has some of these similar molecules, but it's not nearly as toxic. It doesn't have the additives, except when it's military jet fuel in storage tanks. There are highly reactive areas in the molecular sequencing where there are unpaired electrons which can produce a lot of damage.

In the polyaromatic hydrocarbons we see places which can easily cleave off and hydrolyze molecules that then become toxic and interfere with the uptake of critical neurotransmitters in a vulnerable child's brain. So if you think your teenage boy's brain is on fire, it probably is. This is the stuff they're breathing, which has an impact on dopaminergic chemistry.

At our laboratory, we are also looking into the neurochemical implications of the work of two senior scientists at the U.S. Geological Survey¹⁶ involving dust storms. Dust storms have become a major issue in environmental medicine. A dust storm came off the coast of Africa in February, 2000, crossed the North Atlantic, and went through the Caribbean onto the coast of southern Florida. Significant is what's in the dust. Because of the 30-year drought in the Sahara, tens of thousands of tons of dust annually blow off the Sahara and travel across the North Atlantic. We used to think the oceans were an effective barrier, but they're not. One of the scientists found a horde of African grasshoppers on the deck of his sailboat in the Caribbean. That's the visible contamination. On a

summer day in southern Florida virtually half the particulates in the air, including fungi, bacteria and viruses, come from Africa.

There are also dust storms coming from the other side of the world. In April 1998, a huge dust storm swept across the Gobi Desert, and then across Japan, the North Pacific and into the United States. We had a major impact from it in Colorado and it went all the way across the United States to New England and eastern Canada. The stockpiles of organophosphates and other toxic pesticides that we sent to developing countries are coming back to us in these storms.¹⁷ Due to the global transport of environmental toxins, there's no such thing as a perfectly organic farm anymore, but eating as much organic food as possible is the best approach.

We advocate a plant-based diet, because the lower you eat on the food chain, the more hydrolysis takes place and the easier it is to wash off these toxins. These toxic elements have a high affinity for fat and all animal food, including free-range chicken and beef, is loaded with fat. Sixty percent of your brain is fat and these toxic elements have a high affinity for the fatty tissue in animals. The conclusion of this is that nobody's safe unless we're all safe.

Case Study: Jay

Jay, was an eight-year old with a serious rage disorder. He was brought to us after he had punched his babysitter, which led to police involvement. The son of parents who had his life mapped out for him, Jay had a different agenda and his biochemical tendency toward anger conflicted with the very high expectations his family placed on him. He had been on Ritalin since he was five years old and at the time of his first visit he was on three psychotropic medications: Prozac® (Fluoxetine), Pamelor® (Nortriptyline), and Ritalin® (Methylphenidate). This combination of drugs is hazardous because nortriptyline over-enhances the activity at the receptors of methylphenidate. We tested Jay's amino acid chemistry and fatty acids. Children with

rage disorder often have very low docosahexaenoic acid and high arachidonic acid levels. His results confirmed that he was deficient in all the essential building blocks for making his own neurotransmitters. We began treatment by custom formulating a free-form amino acid powder that provided his brain with amino acids it hadn't had before. Jay's diet consisted largely of steak and french fries, and one might think that someone who ate a high-protein diet might have more amino acids, but he didn't. This is because a meat and potatoes diet provides mostly bound amino acids and the body needs free-form amino acids to alter the chemistry.

Jay started to improve. Over time, he was able to get off the Prozac and Pamelor, but for another 18 months he was still on Ritalin, because his parents insisted that he stay at his prestigious school. Finally they realized it was not working and when they decided to change schools we were able to get him off the Ritalin, because he was finally in a more suitable environment. It has been a difficult case and there seems to be a familial component to this problem—we are treating a cousin of Jay's who has also had a lot of similar neurochemical issues.

This is a good example of why it is imperative to start with the essential amino acids. The literature shows that even though there have been good results giving adults direct intermediary molecules, specifically tyrosine for depression, it's generally not a good idea with children.¹⁸ They often overreact to it and can become quite agitated. Adults can better tolerate that intermediary crossover.¹⁹

The Drug Message and its Impact on Children

In the United States, with the lessening of the commercial restrictions, kids are seeing drug ads on television for Zoloft, Paxil and Prozac. These ads are designed to say to them, "You're deficient in some way; you need this substance." Consider

for a moment the messages children are getting by being exposed to the use of psychotropic medications. Of course the effect is not limited to children; we frequently encounter people who go from doctor to doctor for more and different medications. People we interview tell us that they think, "Well, my Prozac isn't working that well, I'll try this other doctor." Most will not tell the second doctor that they're already taking an SSRI drug. When the second doctor recommends a different drug they start combining them. We have also had patients who go into the health food store saying, "Well, I saw this St. John's Wort stuff on TV, I'll take this, too." Never mind the fact that St. John's Wort isn't probably effective—there have been two European studies that found it does have some effect on mild depression and a major U.S. study saying it does nothing.²⁰ Irrespective of that debate, there is a poor understanding of the complex chemistry of most of the herbal remedies because they're really multifaceted structures. I've never seen any good data on whether an herb like St. John's Wort is working as an MAO inhibitor or an SSRI, and you never combine MAO inhibitors with SSRI medications. So people like this who think their serotonin uptake inhibitor isn't working, often start combining things, and some of these drug/drug or drug/herb combinations can be very negative.

Pharmaceutical Biochemistry

Dr. Joseph Glenmullen, M.D. of the Harvard Department of Psychiatry proposes that SSRI drugs stop working because of a backlash effect.²¹ Our research gives us a slightly different perspective. If you take 100 people who are depressed and you give them all Prozac, what you will see is that one third of them will do somewhat better. A third of them will do better initially when the pharmacology of the drug kicks in, and then back off. They often will be worse than they were before. The last third of them will do worse initially. This is determined by

their biochemical individuality. What happens is that in the first group, those people have available serotonin to give the drug something to work on. In the second group, they are not making enough serotonin. A number of people find, when given titrated doses of tryptophan or HTP, the drug actually works better than it did before. We've been able to then use it as a bridge, with the goal to get them off it as soon as possible. The third group, that does worse, does so because they're not deficient in serotonin at all; they're actually deficient in dopamine. Serotonin and dopamine have a synergistic effect on each other. It is, in our opinion, not so much dependent upon time of the day—although that's somewhat true—but it's much more important to understand the biochemical proclivity of a particular individual. Or, conversely, a small percent may be highly sensitive to serotonin and develop neurotoxins to it quite readily. This is perhaps what occurs with hallucinogenic drugs.

We have patients with severe tendencies toward anxiety who can't tolerate dopamine. They get agitated from any increase in their dopaminergic chemistry. Those people are not attracted to stimulants. But, definitely, there's a tendency for people to want more dopamine in the morning. That's why people can't wait to have coffee in the morning. We live in a very turned-on world. People want that hit. Why, then, are these SSRI's so tremendously popular? There is a seesaw effect between serotonin and dopamine. If you introduce a new element into this synergistic system, there's going to be some major fallout.

The placebo effect of SSRI drugs is well documented, yet the pharmaceutical world would have you believe that these are the nirvana of drugs—the end of the road—in terms of drug protocols. If you look at the history of psychopharmacology, it's fraught with disasters and major near-misses. Where those drugs are useful is when somebody is really in bad shape and you

need some bridge until they can get the help they need. We don't think they're effective for long-term use for most people, and the side effects are numerous.^{22,23}

The molecular structure of Prozac® is very interesting. In developing such a drug, a team of psycho-pharmacologists is given the task to go into the lab and solve the problem of how to anchor serotonin in people's brains so that they won't be depressed. The question is missing something important, which is that serotonin is only one factor and one type of depression. Serotonin is helpful when somebody has a rage-type of depression and is very angry. It is not usually helpful when somebody has a flat-affect kind of depression. The structure of Prozac® is such that it is remarkably stable, with an aromatic benzene ring anchored to a trichloromethyl group, like a tent pole anchoring it to the ground. It strongly reinforces the neuronal sequencing on the mechanism of action of serotonin. A person taking these drugs has an increased serotonin capacity. For somebody who doesn't need serotonin Prozac can make them feel very strange.²⁴

Cocaine and amphetamines target the ventral tegmental area of the brain and connect to the pleasure circuit. The proponents of the stimulant drugs have said to me that when kids, who they think need them, don't take stimulant drugs, they have more of a tendency to be attracted to illicit drugs as they get older. That has not been our experience. We have boys in our study who have been on Adderall, Concerta, or Ritalin and their brains have learned to like that pleasure circuit. They tell us, "I like cocaine!" Their brains have turned on to that for a long time.^{25,26}

Opiates also target the structures that cocaine does, as well as the regions activated by the natural opiates. Alcohol affects all the pleasure circuits in the brain. That's why alcohol is so insidious. When people say alcohol and pot rarely lead to serious addiction they are wrong. We have

never known a kid who had a hard drug problem who didn't start there. Furthermore, drugs dramatically alter the experience of growing up. The tragedy is that the neuronal chemistry will actually change if there's significant exposure to these drugs, and the personality of the child is marked forever. Cocaine works by blocking the molecule that allows dopamine to go back to its proper receptor sites. It stays in circulation for too long. It's analogous to what Prozac does to serotonin, except that cocaine is a very dangerous drug. When dopamine cannot return to its proper sites the craving intensifies over time.

In our research into HTP and Prozac® we are again working on molecular integration and the compatibility of these molecules. This is similar for Zoloft® and Paxil.® We are learning how to safely titrate up levels of HTP while we're weaning someone off the SSRI drugs.

We find it interesting how similar the spectra of Ritalin and Xanax are. These molecules are amazingly alike in their structural tendencies, yet one is intended to speed you up, and the other to calm you down. One works by articulating the plasticity of dopamine to get kids more focused, and the other works by supposedly up-regulating GABA sequencing and calming them down. There are better ways to do both.

Conclusion

Every time we hear about a new wonder drug, we wonder what the side-effects will be. There are good drugs and bad drugs. We prefer drugs that have been around for a while, like aspirin. That can be considered a good drug. There is a significant overuse of pharmaceutical psychotropic drugs driven largely by product development and it seems that real research is not done until after the side-effects show up.

In our work with children and adolescents, and we say this for adults as well, we are looking at how to create a context of balance. We believe that the brighter the

light the darker the shadow, and so if an something seems too good go be true, it probably is. Teaching our youth how to appreciate staying with the uncertainty of that gray zone of life, where there can be great joy and growth, is fundamentally important. They need to be coached on how to engage their true feelings, fears and anxieties without reaching for a psychopharmaceutical crutch. That doesn't mean they shouldn't take risks. They should be encouraged to take rational risks, but not to go to extremes that are obviously damaging and destructive.

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