May 2013 Issue | Joel Robertson, PharmD Robertson Health

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Welcome to *Functional Medicine Update* for May 2013. We have a really extraordinary opportunity this issue to speak with and hear from a most remarkable person who will give us a perspective on what I would call lifestyle intervention, environmental modulation, and its relationship to mental health affect and overall physiological function. This is Dr. Joel Robertson. Dr. Robertson is quite a remarkable individual, having developed, over the years, many programs that are involved with facilitating improved mental health, physical health, and physiological health. He's recorded tens of thousands of patient records and data points. You can hear from him directly as to his story, but I think you'll find it fits beautifully within the matrix and concepts that we've been developing in *Functional Medicine Update*. Dr. Robertson also brings, I think, a very interesting point of perspective that has been a theme of ours, and that is, what is the role of lifestyle intervention utilizing the functional medicine matrix for improving function in areas where there's not an acute disease, so we can't fall into a tidy little diagnostic code, but there's also not the presence of what we consider high-functioning health. It's kind of this grey area that we've talked about in many issues of *Functional Medicine Update*. I think Dr. Robertson's book, which talks about "Natural Prozac," in other words, activating the body's own reticular immune neurological system with multiple features of a healthy lifestyle, is a very, very powerful concept and certainly represents a treatment modality that may be most beneficial in terms of both safety and effectiveness in those conditions that fit in between optimal function and what we might call a very distinct pathological disease (a mental health problem).[1] In that grey area is where so many people reside in terms of their dysphoria, depression, lack of motivation low energy, fatigue—things that are interrelated to arousal. Dr. Robertson will tell you about the breakthroughs that he's made in understanding these types and facilitating improved function.

We are witnessing a very dramatic, continued theme, which I think most of us are seeing in the media every day, be it either the medical press or the general press, which is a rising tide of chronic illness and a reduced sense of overall health within our culture. This seems paradoxical to most of us because this is a time where we have more high tech medical therapies, and more clinical options, and more pharmaceutical intervention agents than ever before, yet the overall health of the country seems to be in peril. And when we look at why that is, we recognize it's not just solely a consequence of our society growing older. In fact, the data would say that children born in the United States today are being found to have more atopic disorders than ever before: allergies, asthma, and various types of what we might consider cognitive affective disorders like attention deficit disorders. So we might raise the question: If these conditions that we call chronic illness conditions are not just reserved for the older population but are being seen in our younger population, what is the reason for it? Are our genes suddenly changing to be more prevalent to these conditions? Well, that's not likely, at least not the genetic structure in and of itself. Our epigenetics might be changed in certain respects, but certainly not mutational changes

occurring at the core of our genome. Therefore there must be other events that are influencing the expression of our genes that are creating the increased prevalence of these conditions that we consider to be chronic illnesses.

Atopy and Asthma Rates Among Children Appear to Be Increasing, Especially in the United States

In fact, it was very interesting that just recently a study which surveyed 80 thousand children in the United States was looking at the presence and prevalence of conditions like asthma and allergies.[2]According to Dr. Ruchi Gupta, who is at Northwestern University, Feinberg School of Medicine, in Chicago, there is definitely something clinically happening that is increasing the relative frequencies of these conditions, and it's most likely something related to the environment in which these children are found. Then we say, "Well, what does that mean?" Does it mean environmental pollution? Does it mean environmental stress? Does it mean environmental nutritional changes with poor quality nutrition, or are there a myriad of other factors? Low grade radiation? I mean, one could conjure up many, many variables. Or could it be components of all of those that are working on the genetics of the individual that are creating the increased prevalence of these conditions?

In fact, it's interesting to note that when we compare children in the United States to that of children born outside the United States, that these atopic disorders of allergy and asthma are increasing much more rapidly in the United States than in other countries. It might suggest that there are certain environmental variables in the United States culture that seem to be more prevalent in producing the outcome called these conditions than in other places. So this is a medical detective story. How do we tease this apart? How do we understand it? It's not just finding new drugs to treat allergies and asthma. It's finding the causative agents. In fact, Dr. Sidney Baker, in a wonderful quote that he's been credited with many times, when asked, "What is the treatment for chronic illness?" he said, "Well, it's very simple." Number one is you find out those things that the child is exposed to at too high a level and you take those away. And then you find out those things that the child is not getting enough of and you give them. So it's taking away the bad things and it's giving the good things. That's the basic philosophy rather than just suppressing the symptoms by giving a medication that manages the outcome—the symptoms—without managing the cause and effect.

So I think that's an interesting watchword to what we're going to hear from Dr. Robertson as it pertains to how the functional medicine model as an operating system can play a role. The same thing holds true, obviously, for other conditions like autistic spectrum disorders, where we're seeing such a rising prevalence. One might say this rising prevalence is a consequence of just better diagnosis and it's sensitivity in our culture to it now, so we're suddenly recognizing it, but it's probably more than just better diagnosis. There's something else going on here that's leading to a rising prevalence of these conditions, which is not just suddenly the genes of children started to be autistic. There is something in the environment—the complex nature of interaction with genes that probably are susceptible genes, that give rise to the outcome that we give a name to called autistic spectrum disorders.

Recent Article on the Etiopathogenesis of Austistic Spectrum Disorders

In fact, a very interesting paper was just published in the journal *Medical Hypotheses* talking about etiopathogenesis of autistic spectrum disorders, trying to fit the pieces of the puzzle together. This appeared in the April 24th issue in 2013. And the authors go on to say that when we look at this particular

constellation of data that relates to autistic spectrum disorder, we're led to understand that there's something going on in the immune system of children that seems to trigger these neurocognitive behavioral problems.[3] The genes were always sitting there in wait of the information from the environment, and what their getting—probably starting in utero and going on in infancy—are messages that can trigger certain types of phenotypes that we call autistic spectrum disorders. I think it's a very interesting observation, and in fact they conclude that to confirm this hypothesis we need new research approaches that look at these things from a different perspective: the interface between genetics and environment to chronic infections and nutritional deficiency like vitamin D, the presence of immune system dysregulation—all of these things become part of a broader footprint upon which we would rest our observations to determine whether a child has certain things that we can either take away or add to ameliorate their condition that we call autistic spectrum disorder, or ASD. So it's another example, I think, of the fact that we cannot account for these rising prevalences of certain disorders strictly on the basis of a traditional model and say, "Well, they have the genes for the condition and that's why they got it." Now we have to say, "Well, the genes have always been there; why are more children experiencing this condition?"

It's Never Water Under the Bridge: Lifestyle Can Impact Health at All Ages

That leads us into a question of lifestyle. If environment and lifestyle are intimately interrelated, which obviously they are, does lifestyle play any role in the amelioration, prevention, and/or management of these conditions? And does it cut across multiple ages? I often hear, when I speak to groups in which the people that are listening are principally seniors, they say, "I wish I would have known this information when I was younger. I might have been able to do something about it, but now, you know, it's water under the bridge. I'm too old and I really can't do much about it now. I mean, I'm 75 years old or 80 years old, and it's too late." But if we start looking at the data that's been developed over the last couple of years we see that it's never too late. Yes, it is true the earlier the better, however there is plasticity that appears in physiology throughout all of our lives, up to the last breath we take. So there is still functional capability that is untapped in individuals if they take away the things that are creating the problem and add the things back that are necessary for function.

How do I know about that? Let's take an example from a recent paper that appeared in the *American Heart Journal*. This was in May of 2013, page 785, in which the investigators were looking at the benefits and costs of an intensive lifestyle modification program.[4] These are in older age individuals with symptomatic coronary artery disease. These were all Medicare beneficiaries, so they fit into this category of people that I was talking about who are often attendees at lectures that I might give to seniors. What they showed in this multi-site demonstration project that was conducted between the years 2000 and 2008, that those individuals that were engaged and successfully participated in an intensive lifestyle modification program with activity, diet, and stress management were found to have a very significant reduction in need for medical services, a very significant improvement in cardiac function as well as overall physiological function, and that the cost per individual for medication and medical management was less than that of the people of the same age and the same condition who didn't engage in the intensive lifestyle intervention.

These are examples, I think, of how important these variables are that we've often relegated to a second tier in medicine because we said, "Well, that's public health, and public health, that's not medicine. That's somebody else's business." But actually these concepts as applied through the lens of functional

medicine and personalizing intervention through diet, lifestyle, and environment, are very, very powerful primary tools for ameliorating these conditions that we call diseases of the 21st century. What we often know is that these conditions first are seen as kind of diffuse symptomatologies. They don't often present as acute illness, and so they are more difficult to get our arms around in medicine. They may be things like chronic pain, chronic fatigue, depression, low energy. I mean, these are like a constellation of symptoms that cut across almost every diagnosis and so people say, "Well, gee, that's so confusing. Let's just wait until they get sick enough that I can really get a better handle on what they've got, so I'll just manage the symptoms with sleeping pills or anti-inflammatories, or anti-depressants until they get more serious." However, what we're starting to recognize is that by the use of arrays of various types of biomarkers we're able to examine functional changes in individuals that precede the onset of disease that are associated with these altered states of physiology—what we might call disturbed physiology or disturbed metabolism—that are tracking a trajectory towards becoming acute disease. So it gives us the tools, for the first time, to start looking earlier at these types of things that will become later more significant and more pathologic. Earlier intervention not only saves years of declining function, but also it prevents what might be a much more costly, expensive, and difficult disease by putting it off into the future or maybe eliminating it entirely. This is Dr. James Fries' concept of compressing morbidity and increasing functional capacity. And you might recall that that concern was raised all the way back since 1980 when we started talking about this compression of morbidity improving function throughout life.[5]

Assessment Protocols: Diagnostic Markers versus Prognostic Evaluative Tools

When we start looking at this from an assessment protocol, we're not so much looking at diagnostic markers as we're looking for prognostic evaluative tools. And what's the difference between a diagnostic marker and a prognostic marker? A diagnostic marker is one that we would look for—something that you can taste, touch, feel, see—that is related to a pathology (an end-organ pathology). Where a cell, or a tissue, or an organ can be seen under a microscope, or with radiological examination, or with specific types of biochemical markers to be a specific indication of a disease, like you might have with troponin in a person who has suffered a heart attack. You would say, "Oh, that's an indicator of a heart attack. It's a pathological indicator." There are, however, a myriad of other newer biomarkers that are being employed to try to look at trajectory of function, that map the domain of physiological function. Any one of those by themselves is probably not as valuable as when they are taken as a family of different assessment tools, like using data from an oral glucose tolerance test with fasting insulin, with glycosylated hemoglobin, CEF peptide, and glucose determinations all together to get a better understanding of the domain of insulin sensitivity and glucose tolerance. That's why we often in research will use what's called a compiled glucose determination that actually uses a variety of variables, including things like fasting glucose to our postprandial glucose, fasting insulin to our postprandial insulin, to start to look at insulin sensitivity well before one gets to a frank state of diabetes. Some people call this pre-diabetes, or insulin resistance, or metabolic syndrome, and it has its own sequelae of presentation symptoms and signs that give us early assessment opportunities to evaluate before the onset of frank diabetes.

So these circulating biomarkers become very important for predicting overall trajectories towards later stage risk, and we can see virtually every organ system has its own unique defining biomarker portfolio. This raises a very interesting question, and that is: How many things do you need to test? That's still open for a lot of debate. I think the first point is, if one starts without an exhaustive profile of tests, to do a good physical history on the patient and to really understand the antecedents and triggers that that patient has experienced that are giving rise to their signs and symptoms. As one starts to dig through the

detective work using the functional medicine model, however, you may require certain extended types of evaluative tools—other biomarkers that help us to understand the nature of that individual's own metabolic disturbance or physiological dysfunction. That's where certain biomarkers become predictive and become prognostic in their evaluation.

There's a very nice paper that recently appeared in *PLoS One Medicine* in April of 2013 that really looked at various types of markers for disturbances that are associated with cardiometabolic disease.[6]—things like fibrinogen, which we might think of as a clotting factor but it's also related to inflammatory potential; or apolipoprotein B, which is the nascent lipoprotein for low density lipoprotein's atherogenic dense particles; or apolipoprotein A-1, which is a very important carrier for the HDL component that leads to cholesterol efflux and atherosclerotic regression. We recognize even inflammatory markers like C-reactive protein and Interleukin-6 become important assessment tools and we can even go into metabolic tools like uric acid, which tracks not just for gout, but also tracks for cardiovascular risk as well (cardiometabolic risk). So all of these become tools that are not focused on diagnosis of a specific disease, but looking at patterns of metabolic disturbance that drift toward, or has a trajectory towards, disease.

We can tie these together, obviously, with specific types of genotypic analysis, and here's where SNP analysis—so called single nucleotide polymorphism analysis—becomes useful as well. You can't change your genes, but you can understand something about what you genes deliver to you in terms of potential, either strengths and/or susceptibilities. Let's use an example: the apoE gene. That, as you know, exists in three polymorphs: the apoE 3, apoE 4, and apoE 2 polymorphs. These particular genotypes, knowing in a haplotype we might have one gene coming, obviously, from our mother that could be an apoE 2, and another from our father that could be apoE 3, but if you happen to have the apoE 4 from your mother and also from your father (the double apoE 4 allele), that is associated with a very high risk and incidence of cardiovascular disease and Alzheimer's. So some people say, "Well, I don't want to really know my apo genotype because, you know, there's nothing I can do about it, and woe is me if I just got the bad luck of the draw and got an apoE 4." But what we're recognizing now is that actually you can do something about it. You can't change your apoE genotype, but you can change the environment in which the apoE genotype is exposed to characteristics that might lead to the expression of an adverse outcome. So in the case of an apoE 4, what would that be? That would be an individual who probably needs to be very careful not to consume excessive amounts of saturated animal fat. They need to be very cautious to manage their oxidant-antioxidant balance, meaning proper antioxidant, high phytonutrient content foods (flavonoids and xanthophylls and polyphenols), because these are individuals that have a much higher oxidative stress risk, and individuals that are much more saturated fat sensitive. Rather than saying, "Well, I don't really want to know my apoE genotype because there's nothing I can do about it," in this case what we're really saying is we do want to know these characteristics because it helps us to modify our personalized environment in such a way as to create a more likely positive health outcome.

By the way, this was discussed very nicely in another article that appeared in *PLoS One Medicine* in 2013 in the April 17 issue, in which the authors, at the Department of Genetics, at the University of Oslo Medical School and Hospital in Norway, and what they were looking at were apoE genotypes in relationship to cardiovascular disease and rheumatoid arthritis, and found that there were correlations between apoE 4 and both cardiovascular disease and rheumatoid arthritis that were associated with oxidative-prone (or let's call it inflammatory-prone) phenotypes.[7] So in that case, these individuals need to obviously balance their lifestyle and their diet and their intake of substances very carefully to be

on a low inflammatory program.

Metabolic Flexibility: Multiple Pathways Lead to Plasticity and Health

What we are really talking about is use of specific personalized lifestyle intervention based on sieving through the functional medicine model that would improve metabolic flexibility. I think this is a very interesting term when we talk about metabolic flexibility. You recall that years ago when we were designing the formalism of functional medicine, we talked about the importance of metabolic redundancy, or metabolic degrees of freedom, or making sure that there were multiple pathways in our network that were open and available for substances to move from starting materials to endpoints—that the more redundancy, the more sophisticated, the more plasticity, the more healthy the individual. So this is what we call metabolic flexibility. You see it with heart rhythm if you're measuring what you might call chaotic flexibility in heart rhythm; we know that the healthier an individual is, the more flexibility they have in heart rhythms in the fine structure of their electrocardiogram, and so that's a measure of these physiological degrees of freedom.

I think that these are closely associated with decreased biological aging. There's this inverse relationship between increased metabolic degrees of freedom and that of biological aging. Higher freedom, lower biological aging. This was actually talked about in a very nice commentary in the journal *Nutrition and Metabolism*, in which they were really looking at the interface or the interrelationship between genetic and environmentally determined function.[8] On the proper interface between an individual's genome and their environment, you get maximum mitochondrial bioenergetics capability, maximum metabolic degrees of freedom, and decreased biological aging principles. This cuts across many animal studies and different species. The objective, obviously, in the approach that we're describing and the implementation of a functional medicine intervention system into a personalized lifestyle management program is to match up these genotypes with the environment in such a way as to produce maximum metabolic flexibility, reduce biological aging, improve organ reserve, and reduce the risk to later stage disease.

Research Continues to Link Nutrition to Cognitive Function and Mental Health

How does that relate to things like cognitive function and mental health? Well, there is tremendous work going on in this area that is very, very exciting. You'll hear more about this from Dr. Robertson. But I'm reminded of a very interesting paper that appeared just recently in the *Journal of Nutritional Biochemistry* in 2013 titled "Nutritional Modulation of Cognitive Function and Mental Health" by investigators from the University of South Australia in Adelaide. In this particular paper they showed that the dietary risk factors for cognitive dysfunction and mental health are now becoming fairly clear.[9] Things that enhance insulin resistance. Things that increase inflammatory burden. Things that increase cellular mitotic activity. All of those types of principals in our diet are those principals that are associated with—over time—decreasing cognitive and increasing issues related to mental health. So there is a very important strong relationship that's appearing from good research that ties diet and lifestyle intervention tightly together with the ever increasing prevalence in older age populations of cognitive dysfunction and of diseases like Alzheimer's. In fact, there's a very nice review paper that appeared just recently that describes the role of nutrition and diet in Alzheimer's disease.[10]

I think what we're starting to see is a very, very science-based, supporting system for the sensibility that the lifestyle of the individual plays a very important role in experiencing the expression of their genes, which gives rise to their phenotype, which is their health and disease patterns. That model is a very powerful model from which the functional medicine operating system delivers, I think, the interventions that relate to personalization of cause rather than just amelioration of effects as is often the case with pharmaceutical intervention programs that are tied only to symptom amelioration.

With that in mind as a prelude, let's go to our Researcher/Clinician of the Month, Dr. Joel Roberson, who will really give us the news to use.

INTERVIEW TRANSCRIPT

Researcher of the Month

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We're so excited, once again, to be at that portion of Functional Medicine Update where I'm introducing our researcher of the month. You know, we've been so privileged, haven't we, over the last many years, to have some of the top opinion leaders in the world who have been sharing what I think will become the nature of the way medicine and health care evolves in the years to come. It's kind of at that leading edge that we often get treated with, to hear what's happening—no surprise—this month we have a similar circumstance with our clinician/researcher of the month, Dr. Joel Robertson, in Michigan.

Dr. Robertson is a PharmD. He is chairman and CEO of a widely recognized leader in the area of brain chemistry. I think you're going to find his work just fascinating. You probably know if you've been reading the literature over the last few years that he has a best-selling book called Natural Prozac, which we'll let him tell us about. His background is that which has really developed, through the Robertson Research Institute, which is a nonprofit organization dedicated to enhancing lives and advancing the knowledge of healthcare professionals, this whole nature of the lifestyle/brain/environment/function interconnection, and he's done it in a way that, as you'll hear from him, is very novel, very unique, very innovative, and I think very pace-setting for 21st century medicine, because it's really built on a systems biology approach, which, as we've talked about over the last few years, is where health care is really moving. It is moving away from disease as a single entity to moving to the interaction and understanding of the network of systems that interface with function and ultimately give rise to such complex things as high-order thinking and cognitive ability.

Dr. Robertson, wonderful to have you as a leader for us here on Functional Medicine Update, and thank you very much for making yourself available and talking to us about global health solutions. Tell us a little bit about how a PharmD made this extraordinary transition and became the leader that you are in this field?

JR: First of all, I appreciate the opportunity to share, and talk, and all the great work that you've been doing, and like you say, moving medicine to a different level. It's kind of funny because I do a lot of speaking in front of pharmacists, and they say, "How did you make that transition?" And I say, "Well, I

never really practiced as a pharmacist. When I came out of school, I actually contracted with taking over emergency room laboratory, pharmacy, and physical therapy departments (to run those), and in doing that of course pharmacology—and my specialty is neuropharmacology—I guess the best way to say it is, 'Why would I do what I don't want to do?'"

Emergency Room Observations Lead to Theories of Neuropharmacology

You know, I'd see people come in in the emergency rooms and they'd have a heart attack or something, and give them a new way of changing and they would come back and they hadn't made changes. From my perspective, with all the observing of the addictions, and the compulsions, and the behavior, you know, it just began to start the theory of maybe it has something to do with neuropharmacology. So that PharmD aspect came in very handy when I started looking at pharmacology, how drugs interact, how they affect us, and how they affect behavior, and you could correlate it back into the whole main chemical make up.

JB: One of the things that has struck me of the many that you've accomplished in your work is your ability to make your information very accessible to individuals that I think have impact on many others, like your work with General Motors, and Fuji, and Dow, and United Airlines, and the Department of Defense. You've really been able to make this news to use. Is this a gift that you've always had, or is this something that you worked at in developing systems that could translate this information into a way that would be user-friendly for a variety of different levels of organizations?

Behavioral Emergencies: National Treatment Protocols Were Needed for ER Personnel

JR: I was asked by the government to write the national treatment protocols for the emergency medical services when I was only 27. And when you're talking about behavioral emergencies, and you're talking about emergency room physicians, you have a disconnect. You have a lot of information with a lot of technical diagnostic training needed, and yet you have physicians who really don't want to deal with them. So that whole ability to say, "How do I learn to take information to people who may not understand it, and yet make it useful for them?" So in many ways that challenge of taking to the emergency room physician how do you handle these difficult-to-handle patients, was a great stepping stone for me to say, only in a different fashion, "How do you take complex topics and complex interaction, make them something that is simple and yet useable and that I can verify and give immediate feedback to the effectiveness of what they are doing?" That's probably one of those things where opportunity knocks. I was thrown into a situation, and had to learn how to do that, because I, like a lot research guys, love to write in the technical world, which isn't very practical.

JB: Yes. So when I read your book, which by the way is very, very well-written and very user-friendly, Natural Prozac, which has as a subtitle "Learning How to Release Your Body's Own Anti-depressants." I was taken back to all the work that's been done over the last few decades about addiction and the serotonin receptor and how we have these genes for melancholy and how certain types of the serotonin 5 receptor polymorphisms lead certain individuals into higher risk of additive behavior. When I read your book, it sprung me free from this kind of determinism or our genes to there are things that we can do, and I think that's a very empowering concept. I bet you've been confronted with people who say, "Oh, I'm sorry addiction and depression and so forth…it's in our genes; there's little we can do about it." Can you tell us about what kinds of things you experience in getting this message out and giving people much

more options for how they regulate these functions?

Treating Before You Know What's Broken

JR: Absolutely. You know, I think what people fail to realize is when you think in terms of the nerves of the synapse, even researchers don't stop and think, "I have this electrical energy that turns to chemical energy, and then it has to go back to electrical energy." That's essentially pre-synaptic, to synaptic, and then post-synaptic. So when someone says they have a genetic problem, it's not all three areas. It might be pre-synaptic so that I don't have enough, and then you say, "Well, let's figure out how to create it." And it might be, for example, serotonin—I don't have enough of the enzyme (you know, tryptophan and dehydrogenase) to convert tryptophan into 5-HTP. Well then you say, "Let's skip them, and let's go right to 5-HTP." Or maybe it's a post-synaptic receptor gene transport issue. Great, we know how to do some things. I think the issue probably lies with our whole concept of how we diagnose behavioral medicine. When I look at depression, that's a diagnosis, and yet I look at it and I'm going, "I could have low serotonin, I could have low dopamine, I could have low norepinephrine, I could have high GABA, or I could have a combination of all four." And so we throw it in the one system, and treat it, and as I've always said, you're treating before you know what's broken. And that's one of the difficult things that I see when we're dealing with behavioral medicine. We can really pinpoint now as to where (if there is) a genetic issue, where (if there is) an environmental issue, and how do we reprogram the brain to work around it, or stimulate areas that might be able to stimulate it. So it all comes into recognizing what's broken in order to fix it or enhance it.

JB: One of the many things that you've done, I think, that makes this very useable is the way you've approached assessment. I'd like you to talk about how you develop your assessment instrument and tools, and what that does, because I think it really grounds the whole approach that you've got in a very measureable system.

Neurohormonal Assessment: Arousal, Satiation, or Combination

JR: Absolutely. For one of my first jobs I actually ran an alcohol and drug rehab center. One of the first things that I felt we needed was to answer a question: what will I do versus what should I do? The vast majority of medicine in which you're asking people to change behavior, you tell them that they should do it, instead of asking what they will do. So one of the first things that we said is that we need to know three things about our brain, and the first one is what is our reward center? What will we do? Why is it that feels good for us? For example, if you want me to relax, I'm going to be one of these guys that climbs mountains, white water rafts. You know, I do things—I bike, I run. That relaxes me. You want to stress me then ask me to curl up by the fire and read a book. So essentially we're taking this complicated brain, and we're talking about our neurohormonal dynamic, but we made it kind of simple for people and we say either you are arousal, or you are satiation, or you are combination. So that was the first thing that we said we needed to assess, because then I can prescribe activity, diet, and using different things based upon what a person will do, so we can have compliance. The second thing is I had to look at what are the behaviors that I will tend to do when I get out of balance? So you take a guy like me, and I'm high dopamine, high gas pedal chemical, and the behaviors that I do and I personally consider these post-Pavlovian bell because it's like when my dopamine goes up, I become impulsive, my listening skills go down, I want to make a decision even if I don't have the information, and I'm not very patient. Probably I even get task oriented. These are behaviors that are done automatically when people's brain chemistry

changes. So I thought I need to figure out what behaviors are because if I'm doing excitatory behavior but I'm depressed I'm probably trying to treat myself with my behaviors, or if I'm doing depressing things, then I'm doing it in response to my brain chemistry. So the second one is very important for what behaviors are connected. And we know things such as cocaine addiction, sex addiction, risk taking—all the same neurotransmitter issue, they just show up in different behaviors, so it's very helpful. The third thing, which was the biggest challenge, was how do you know what brain chemical is off. I think the greatest insight that we had as we did all the research by doing blood studies and CSF (cerebral spinal fluid studies), all that sort of stuff to try to find the correlation, and we couldn't find the correlation. Then we finally decided that—and I think this is the key thing that people need to understand when they are talking about the brain—there is no normal serotonin, but there is an optimal for an individual. There is no normal dopamine, but there is an optimal. So in our particular case, now my pharmacology background came in and said, "Let's look at what are the symptoms that we might have of low serotonin? What are the symptoms that we might have of high serotonin? And a lot of that is drug side effects. Drugs that enhance certain chemicals and drugs that reduce certain chemicals. And let's ask physiological questions and try to correlate. And when you correlate, what you can do is really say, "Alright, I've got a series of symptoms that I can measure, and so might say I have five low serotonin symptoms, so that's my hypothesis." Now I can go in and say, "Well, I know, for example, if you put me on a treadmill (or a person on a treadmill) and do a short burst of exercise, and check their dopamine, you're going to have tyrosine hydroxylase turnover and burn up the dopamine." So you can prescribe it to those activities, diets, behaviors to your hypothesis, and what should happen? Symptoms get better and behavior changes. That's how we did the research on over 25,000 people.

JB: So when I look at how you've described that complexity in a very understandable way in your writings, you talk about, as you mentioned earlier, the satiation type and the arousal type, and then that directs into specific types of therapeutic interventions that then modulate those networks (those biological networks). I'm fascinated, looking at your blogosphere, at how people have talked about how they have responded and how their personalized approach that you've described out of this assessment have led them to have remediation of problems that they've had for some time; even on medication they weren't successfully able to be balanced, but on the program that you've described, they are starting to get tremendously positive outcomes. It seems that this ability to take this complexity and to summarize it down into these categories has a very, very valuable outcome in terms of making it accessible to people who will get better outcomes.

JR: That's the key thing, I think, with a lot of medicine: how can somebody own it, measure it, feel it, understand it? We started out originally looking at the area of what we would say unhealthy brains trying to get them better, whether they were unhealthy because of compulsions, additions, depression, things of that nature. And then that's an "ah-ha" into the area where it became performance enhancement, and so I've taken on professional sports—wrestling, NASCAR—and corporate executives and saying, how do we make you perform better by A) eliminating unwanted behaviors that are caused by your brain chemistry, and B) just teaching you to perform better because different brain chemicals are more effective when you want to be creative that they are when you are wanting to be detailed and budgetary, etc.? So really it's a matter of when a person understands the symptoms that relate to their brain chemicals, and they understand they are already changed their brain chemistry, whether they had a cup of coffee, whether they had a piece of red meat, or whether they had pasta, they are changing their brain chemistry. Now it's just a matter of teaching them enough to say, "How do I hit the behavior, the music, the things that change brain chemistry, and use them specifically to be able to enhance or inhibit the release of the brain

chemicals that I need to do in my particular case?" People really grasp that. We have great compliance and great effectiveness, because like you said, I like to refer to it profoundly simple.

Self Health: The Mobilization of the Individual

JB: I think what you've done, and I really commend you for this, is you've made this system available so that a person can help themselves. There is a self health component here that's very, very valuable so that it doesn't build a dependency on a therapist for the solution, because this, in the end, is the person's own life, and their own solution is inherent in the way they manage that life. I think you've done a very good job of balancing professional health with the mobilization of the individual to become the activist for themselves, to be involved with self health.

JR: That's one of the things, I think. I ran a behavioral medicine clinic, and our motto was "The less you see of us, the better off you are." And I really believe that empowering people with knowledge so that they can do it on their own and , and then our mental health and our behavioral medicine systems are dealing with those that need more support but don't have family support or systems, and can give them more time, more energy, and get more outcomes. So definitely that was a model of behavior that we began right out of the gate saying, "I need to equip people to be able to not see us, versus see us."

JB: To make this more understandable for our listeners because they don't have the benefit of sitting with you here and talking this though, could you maybe give us a case history or two as to how this program works in people with, say, something like ADHD, or some kind of complex behavioral issue?

JR: Yes, absolutely. The first thing that we do is of course we run through a series of tests like this, and then we teach them what I'm going to call the yellow flags. I believe this is the only behavioral medicine program that looks pre-Pavlovian, meaning once the bell rings you don't have a choice. So if you do something—let's say a bell rings that says, "I feel like I'm going to drink"—and that bell rings and a person may drink or they may go to AA, but the bell is still rung. Our goal is to say, "How do we know the bell's going to ring?" Let's use an example of ADHD. You take something like that and we teach the person to say, "Let's define the yellow flag that says the bell is going to ring." It might be with ADHD or with dopamine, you might say, "The ciliary muscle there in your eye begins to twitch. Your jaw begins to tighten up. Your heart rate goes up a little bit. Your palms get a little sweaty. You find yourself not able to focus." Those are what we call yellow flags that say, "If you stay within that environment and don't do anything, the bell's going to ring and you're out of control." So we say to them and we teach them, so you realize in your case that everybody's different, we might say to them, "We want you to use CBS—chicken, beans, and salad. Get off of the red meat, back down from caffeine, stop the Mountain Dews, stop the energy drinks, because those are all contributing to the high dopamine. If you can go out and go for a brisk walk or a run go do that. Take music—listen to slow, consistent beat music. And those are things all that are decreasing dopamine." And what happens is immediately they can begin to feel the ciliary muscle stop twitching. They get immediate feedback that says, "I'm getting what I need to do because I know when it spikes I'm going to be impulsive, and all of these behaviors that we might have identified." So, we really begin to teach them how to identify the yellow flags, and as you well know, one of the difficulties in medicine is teaching people how to listen to their body. And we just teach them techniques to listen to what's going on in the brain chemistry so that they can make a response, do something, to correct before it gets to the point that they are out of control.

JB: I think that is so amazingly consistent with the functional medicine model. It's very exciting to hear how there's a confluence or a convergence, really, of the way that you've approached this and the way functional medicine has talked about. We would call those early warning signs that are related to change in function like involuntary reactions in smooth muscle, and then we would say, "What triggers are related to the change in those mediators that associate with those functional changes?" So our language in functional medicine might be a little bit different, but it sounds very similar to what you are doing—moving back on the timeline of pathology towards functional changes that are associated with triggers and mediators that ultimately arrive at a pathology, but you want to get it much earlier than when you're in the acute state. It sounds like you've done it in such a way that it really makes it understandable to the patient. They don't have to be neurophysiologists to understand how they are traveling down that road of progression.

JR: Yes, and you know, there are different types of people. We always take our assessment and we say, "We're going into three categories." The first one is some people, and I'm not going to be stereotypic, but some of my hockey players just say, "Tell me what to do. I just want to perform better." So we give them that. And the next group will say, "Tell me what to do and tell me why you're seeing things change"—like you said, the genetics, the developmental time of the brain when that might have been impacted. And then there's the third group that says, "Give me the research." So we like to hit all three categories, including those who say, "All I want to know is how to do it, fix it, and etc." And a lot of times we have some really tough people and tough environments in South Africa. Some of these guys are working in 140 degree temperatures up on scaffolding in these electrical power plants, and we're just teaching them how to learn the feedback and know what to do so that they don't get out of control. That might be, "Just tell me what to do." And then we'll expand it out depending on the personality. And again, it's always trying to say, "Where is that person at now? How do I get them to do what they need to do to get better, because once they get better they're going to take it a next step to even begin to move into prevention?" So kind of like this transition of health is where you try to take people.

An Epidemic of Sleep Disorders

JB: Yes, I can see that. That's really a positive feedback reward system, that a person is building confidence and they're getting positive response, which then builds more activation and more commitment to the program. Let me shift over to another problem that I know is increasing prevalence in our society and one that you deal with as a constellation of issues within the behavioral medicine field, and that's sleep and its association—or let's call it sleep disturbance associated with apnea and what appears to be almost an epidemic of sleep disorders. Tell us a little bit about how your program interrelates with these sleep disturbances.

JR: I think that's really one of the key things, that you've got the cyclical nature. Sleep, of course, is necessary—especially REM sleep—for us to be able to balance our brain chemistry back end, and if we don't get it, it kind of gets into a cycle, and then if you get out of balance then you don't sleep. Just as we do in everything, we kind of think in terms of algorithms. When we look at sleep we'll say, "Let's define and figure out what is happening as to why we're not sleeping." And I'm going to give you an example of what I think is beginning to happen in our society, and why we're starting to see so much sleep apnea. First of all, not to simplify, but I think that most studies are going to show that serotonin is related to compulsive disorders, and compulsive disorders can go anywhere from addiction (drinking alcohol, or it can be obesity, or it can be perfectionism, whatever), so many of them are not "pathological" in the

medical field, but they are in lifestyle, and that appears to be a dominant characteristic. So you start into this whole cycle of thinking which then can create stress—stress in relationships, stress in your self-esteem, which can cause dopamine to go up. So now you have a double whammy happening: I have low serotonin because that's what I was born with; I have high dopamine because of the way I'm thinking because of it, and then the brain kicks in and says, "I'm going to try to compensate with too much gas by pulling on GABA." So you've got two dynamics going on. That is the most common issue—that combination—that I see with sleep disturbances. Because you have this thought process that is compulsive in thinking so a person can't shut off their brain, they have their anxiety issues going so they can't get to sleep, and then they have GABA there fighting it so when they go to sleep it goes quickly and they come back out. So basically when you look you have to say, "If that's my pattern, I have to attack low serotonin/high dopamine, and I can't cover it up with something hypnotic because that won't do it. When you're dealing with sleep, you've got to go back to the root cause, and there really are about 9 or 10 different combinations with sleep that are very different in how you approach them—different, even if there is medication, how you would use medication. Our main goal is to do it as natural as possible as much as possible. Because you're absolutely right, you get into this cycle and the cycle just keeps extending and makes it worse, and then typically, tack on all the sedative hypnotics which interfere with the natural sleep rhythms. It's just a bizarre sort of thing that happens and it needs to be attacked at the root cause.

JB: Clearly, everyone that's listening is saying, "Wow, I need to tap into Dr. Robertson's program and become much more understanding of it. Where would you suggest a person start to become more familiar with your work and how it can be applied?

JR: Go to the website, which is www.robertsonhealth.com and you'll see three different categories there. The Robertson Global Health Solutions is our global diagnostics, so that's not what we've been talking about. When you come into Robertson Wellness, that's where you'll come in and actually there are three different types of approaches that different listeners may want. If you're a clinician and you're looking at using this with your patients, we have what we call a Blaine Chemistry Optimization Program Certification. We teach them how to use it with their patients. The second one a consumer can tap in on, and that's called the Behavioral Medicine Self Care. And then, of course, we have the corporate work where we work with our professional athletes and executives, and that often involves a medical person because it's almost like a concierge medicine, but then we do have those that are based on coaches and people who have corporate clients.

JB: I can say that what you have done over your many years of service and practice is just truly remarkable, Joel. It's amazing to see the scope and the span of your activities. It also suggests to me that there couldn't be a better time in our history than now for people to really take a hard look at what you've amassed in your 20,000-plus case history experiences, because to me this is one of the greatest needs that we have—this whole behavioral medicine and how it interrelates with personalized lifestyle medicine and how that interrelates with the functional approach towards neurocognitive behavioral outcomes. I really want to applaud and celebrate what you've accomplished. It's quite amazing.

JR: Jeff, I appreciate it. I just wanted my life to make a difference. As I began I said to my kids, "I don't think what I do will ever be appreciated, but I believe that I'll at least challenge people to think differently." I feel really blessed that not only are people embracing it, but also my kids have gone into the field and joined me. So it's kind of a great opportunity and exciting that as a family we pushed toward

not really a new way of thinking but a new way of communicating. And it is exciting and making a difference in peoples' lives is the most rewarding thing that you can do.

JB: Thank you. That's certainly very high advocacy and I think we share that goal between us. I want to thank you for sharing with our listeners this extraordinary work you've done over the many years.

JR: I thank you so much, it's always about people working together. Together we can make much more impact.

JB: Dr. Joel Robertson, thank you so, so much, and I think you gave us some real great news to use, some places that people can get started. Thank you so much for being with you today.

JR: Thank you. I appreciate it.

JB: You take care,

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