

April 2011 Issue | Garry Egger, MPH, PhD Southern Cross University, Australia

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Welcome to Functional Medicine Update for April 2011. It has been quite a busy start for the 2011 year. Things are going on with healthcare financing, and with new biological discoveries and changes in the pharma industry. A global rising tide of chronic age-related diseases continues. These western diseases are not just isolated to the shores of western countries, but rather can be exported as a consequence of the lifestyle and environment. In the east, the rising tide of diabetes has become almost a hockey-stick increase in prevalence.

Global Chronic Disease Trends are Changing

In 1984 or 1985, when I had a chance to first visit Hong Kong as an invited lecturer at Hong Kong University Medical School, I gave my presentation on type 2 diabetes. After my lecture the dean came up to me and said, “Dr. Bland that was a very interesting lecture. We really appreciated how well you prepared and how well you presented that, but really we don’t have type 2 diabetes here in China, so it’s not of a very high clinical relevance.” It’s ironic or interesting, I think—maybe it’s even a sign of the times—that in 2006, when I was invited back to speak (this was a new dean at the same medical school; probably many of the faculty were new), the topic that they asked me to speak on was type 2 diabetes. In the intervening period of 20-plus years, a condition that was not seen with any clinical importance suddenly was of major importance, and their interest was, “Could you tell us a little bit about the origin of type 2 diabetes? How environment interacts with genes to induce and produce a phenotypic expression of insulin resistance and hyperglycemia and all the secondary untoward consequences of that?”

I think this illustrates a changing tide and a changing complex environment globally. We are starting seeing the age of first infirmity actually going down to a younger age. We saw the landmark paper that appeared in the New England Journal of Medicine some five years ago that described how children born today will have, on average, a mean life expectancy less than that of their parents (the first time in the history of our country—the United States—where that’s occurred).^[1]

These are all very dramatic, big picture changes/transitions/observations that illustrate the fact that we’ve got an issue that requires a new approach—an approach that is different than treating the symptom when it develops. Rather we need to be digging deep into understanding the origin of these complex, age-related chronic diseases and treating their cause and not just their effect. We need to understand the metabolic disturbance that later goes on to become a diagnosable disease with an ICD-9 code. The metabolic disturbance is a consequence of some relationship the environment has to genetic expression that is unique to that individual’s response to their environment.

This month in Functional Medicine Update we're privileged to have an expert in lifestyle medicine, Dr. Garry Egger from Southern Cross University in Sydney, Australia. Dr. Egger and his colleagues have authored what I think may be the first textbook in the area of lifestyle medicine that has recently been revised into a new edition, which I believe starts to codify, specifically, the components of a lifestyle-based medical intervention program.^[2] We'll talk about the standard of identity and standard of care as it relates to lifestyle medicine.

Lifestyle is a Patient's Environment

What I often tell people when they say, "This sounds interesting but I really don't do lifestyle medicine or nutrition in my practice," is, "Lifestyle is shared among every patient. In fact, there is not a single patient who doesn't have a lifestyle. They don't have an environment that is not without impact upon their genes and how they express their function." If a person is practicing scientific medicine, which we all say that we are here in the 21st century, then they would want to control as many variables in producing reproducible outcome in their patients as possible, and one of the wild cards that can influence the outcome of any therapy is that of lifestyle and environment. If you don't control that wild card—that variable called lifestyle—then you are at some kind of risk, I believe, relative to the reproducibility of outcome and relative to the therapy that is chosen. That therapy could be pharmacology, it could be structure/function type, it could be structural, it could be acupuncture, it could be nutritional—all of these various interventions are modified, in part, in outcome into the phenotype as a consequence of the lifestyle of that individual patient and how it interrelates with their genetic uniqueness.

Some people call this concept genetic susceptibility. I think that term is a little bit misleading because it suggests that all of the factors that are in our genes somehow make us susceptible to some disease. If you really think about it, we're not susceptible; we're unique in our response. Some of those things that we think are susceptibilities really may have been evolutionarily advantageous at an earlier period in our cultural history, under a specific set of environmental circumstances. However, when they are expressed in a disadvantageous way, we label them as a disease.

The term "disease," in and of itself, somewhat has bias in it, has some bigotry in it, has some discrimination in it. Because once you say a person has a disease--be it diabetes, or cancer, or heart disease, or arthritis, or whatever name you would like to put on their condition--they then become that disease. They become Mrs. Jones, the diabetic, or Mr. Smith, the coronary heart disease patient. As a consequence they then fulfill those criteria of that specific set of diseases.

Even though we recognize each patient is unique to their own condition, we tend to lump them into a descriptive term, calling them a disease. That would suggest, in the mind of that person, that they are flawed, they are less capable, they have some kind of inherent weakness. It was the luck of the draw. Rather than that kind of a model, we might say that actually their genes are unique to their own specific situation, and they are actually selected for value in an environment that goes way back to the early legacy of that person's genealogy.

So what we consider a disease today is really, I believe, a story that can be reframed. And that story is that individuals—all of us, every one of us—has our own unique response to our environment, and the environment for one to maximize their function may be vastly different than the environment for another. The body is responding, as it should, to that specific environmental set of conditions based upon its own unique genetic background.

We could say, “Hyperlipidemia is not just the genes for heart disease, it’s the genes that relate to an exposure of a certain environment that then induces—in that person’s genetic expression—the biosynthesis of cholesterol, or triglycerides, or lipoproteins.” Similarly, arthritis or systemic lupus erythematosus or rheumatoid arthritis is not just a consequence of flawed genes, it’s a consequence of a specific immunological response to the environment, internal and external, that gives rise to that particular set of autoantibodies that we then associate with that particular disease. Cancer is a response to a proliferative cell type of signaling that occurs from that set of gene responding to that environmental message.

You can’t change the genes, but you can change the environment to match an individual’s own unique set of characteristics. That takes away the burden of saying, “I’m flawed, I’m injured, I’m imperfect, I have a disease,” to saying, “Actually I need to find the environment to modulate my function so that I’m not displaying an alarm reaction, which we see in the physiology as a pre- or post-pathology process.” I think this is a very different concept and context of looking at dysfunction and how it interrelates with what we later call disease than saying a person is flawed, imperfect, and they have something wrong with them that makes them have heart disease or makes them have arthritis.

Why do I believe this is even a worthwhile conversation to bring up? Because it has a much more optimistic plasticity associated with it. It is much more able to be modified by engaging the right kind of activity, rather than this deterministic model that says, “Well, you’re diseased. You have this problem. It’s probably because you have bad genes: your father had heart disease, your uncle had heart disease, and you should just expect to get it too.” I think we’re into a different kind of frame shift as it pertains to how we describe, and how we model, and ultimately how this translates into therapeutics, and prevention, personalized medicine, and functional improvement in the individual in 21st century medicine.

That is the context from which is born this view of lifestyle medicine. We’re fortunate in living today, where the tools are available to start pulling together the root origin of cellular disturbances and how those metabolic disturbances translate into alarm responses that later become tissue pathologies that we call disease. Lifestyle and environment interface with genetic expression patterns and signal transduction so that they ultimately regulate what we see as our function over time--over the decades of living--and ultimately smooth our function and change our body shape and how we actually respond to the world, whether it is responding physiologically in an alarm state, which could be inflammation, it could be dyslipidemia, it could be hyperglycemia, it could be glycation, it could be oxidative stress. All these things are alarm responses of our genes to a specific environmental set of circumstances. Lifestyle medicine is recontextualizing itself to be married to the concepts of personalized medicine.

As I mentioned, we’re very fortunate to have as our guest today, one of the experts in this area, one of the coauthors of a textbook on lifestyle medicine. He is an individual who I think brings a tremendous perspective related to the emergent application of the gene-environment connection in the clinic and ultimately into patient management. With that, let’s move to our Clinician/Researcher of the month, Dr.

Garry Egger

INTERVIEW TRANSCRIPT

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What does the future look like? What are the landscape and topography areas that are shaping health care? And how can we really be more successful in managing this burden of chronic disease that has been a rising tide in the developed world for the last decade or so?

Our Clinician/Researcher of the Month today has a very remarkable background and productivity record.

His name is Garry Egger. Garry is currently a Professor of Lifestyle Medicine and Applied Health Promotion at Southern Cross University. It is an esteemed university I'm familiar with in Australia. He has spent most of his life as a consultant in epidemiology and health promotion throughout Asia and the South Pacific and Australia.

Years ago--I was actually familiar with this—he innovated a concept called “GutBusters,” which was the world's first men's weight loss program. He is an advisor to the World Health Organization on chronic disease. He was instrumental in establishing the Australian Lifestyle Medical Association (similar to the American College of Lifestyle Medicine that I'm a member of here in the United States), and he is currently investigating the relationship between obesity, climate change, environment, and economic growth. He is the author of more than 30 books, including six texts, and more than 150 peer-reviewed articles.

For me, this is a very interesting closing-of-a-circle. About six months ago I was searching for a good text on lifestyle medicine for some of teaching, and I happened onto this book called Lifestyle Medicine. The authors are none other than Andrew Bynes, Stephan Rossner, and our own Garry Egger. Closing this loop and now having the chance to talk with Dr. Egger, I recognize that there are so many areas where we share a similar theme and these fit nicely into the concepts we've discussed in Functional Medicine Update over the last couple of decades.

One of Dr. Egger's recent papers, co-authored with Dr. John Dixon from Stanford, really got my attention because it is titled “Should Obesity Be the Main Game? Or Do We Need an Environmental Makeover to Combat the Inflammatory and Chronic Disease Epidemics?”[3] It is a very provocative title, and a very interesting article that appeared in Obesity Reviews.

Dr. Egger, welcome to Functional Medicine Update and thanks for being available today all the way down in Australia. How did you go down the path of being an expert in lifestyle medicine? What took you in this direction?”

The Relationship Between Economic Growth and Chronic Disease

GE: Thanks, Jeffrey, for that introduction. It's a big introduction—I can't wait to hear what I've got to say now after that introduction.

I've been working in this area for 40 years. I come from a bio-behavioral background and an epidemiological background. I've always been interested in what is the cause of the causes, in Geoffrey

Rose's terms. When I look at the epidemiology of a particular problem, I like to go back and span out, if you like, and look at the big picture. Let's talk about obesity and type 2 diabetes, for example, as the big chronic diseases of the era. You could say that the immediate and proximal causes are an individual's behavior, particularly what they eat, how active they are, whether they get good sleep, whether they are highly stressed—all of these sorts of things. But these are really just the immediate causes. They are causes of those causes. And then there are causes of those causes of those causes.

If you track these causes back, it becomes very interesting because it leads not just to the individual behavior and an individual response, but it's the environment that drives that response. And when you take it back, the big driver of all of this is the modern environment in which we live, which is really economic growth. There is no doubt that growth and development in all countries has tremendous advantages up to a point, but beyond the point you have to get negative returns when you've got exponential growth in anything. None of the early economists would ever admit that this can go on forever, and now we're starting to see the consequences of that, particularly in obesity, which is probably a warning sign that there are other things going on in the community that are not healthful. I don't think obesity in itself, after many years of working in that area, is necessarily all that important. I think in some cases it is, but I think in many cases it's a warning sign; it's a canary in a coal mine, if you like, that there are other things going on in society that we need to look at. That's basically where the whole lifestyle medicine approach comes in.

JB: You said some things just in that introduction that I think are extraordinarily interesting and provocative. In the states, as I'm sure it is in Australia, we're often told in the medical world that the cause of all these chronic diseases is obesity, and therefore just applying the first law of thermodynamics of calories in versus calories expended is really the solution to the problem: reduce the calories in and increase the calories expended and we're going to solve the problem. But when I look at China, which has a rapidly rising prevalence of type 2 diabetes and heart disease, I find that the BMIs of the Chinese are actually not that large. They are not above 30. They are starting to have problems with BMIs that are in the mid-20s, that we would consider (in the states) to be still in the acceptable range. It suggests that there is something about the environmental relationship with those genotypes that spreads itself out to increase the risk of chronic disease related to these lifestyle changes that may make these individuals more of a canary in a coal mine (to use your analogy) than in the Western world with our sand pile genotypes. I think this obesity concept of "Is it the cause or the effect?" (the question you are raising) is very, very important, because I think we've always assumed it's the cause, but I think what I'm interpreting is that maybe you are suggesting it is the effect. Am I on the right track?

Growth Beyond Maturity is Either Obesity or Cancer

GE: Yes, you are. With the Chinese it is a slightly different suggestion because as you're aware, Asians genetically are more prone to metabolic diseases at a lower proportion of, particularly, abdominal fat. We don't know the reasons, but it's probably an evolutionary thing that they are more prone to that. What we are seeing in China is virtually a microcosm of what's happening in the rest of the world, but it's happening so fast in China because of development.

My belief is that it's not so much the obesity that is leading to the diseases, it's the things that lead to the obesity, which is the change in diet, and the change in activity, and other aspects of the industrial environment. We see in China, as we see in all other developing countries, a point at which you get a

crossover between the infectious diseases and the chronic diseases, which is called the epidemiological transition. This occurs through natural development in developing countries. India is about to go through it as well. At that point, because the infectious diseases start to go down dramatically as a result of development and as a result of hygiene, in particular, and changes in lifestyle (not just the availability of antibiotics and medications and so on), you're also getting a rise in chronic diseases as a result of lifestyle, and the chronic diseases start to take over from the infectious diseases.

It seems like that is the sweet spot in development. Beyond that point we start to get diminishing returns on investment, as the economists would call it. And any good economist knows that when you invest money in any project, that project starts to yield enormous returns to start off with, but as you continue that investment you start to get diminishing returns on your investment. That's exactly what's happening with development throughout the world.

This is very difficult for medical scientists to get their heads around because it involves an understanding of macroeconomics. This growth train that we've been on started after the Second World War. It was instigated, initially, 100 to 150 years ago, by the early economists. I It was really trains that put us on track for this modern growth phenomenon.

One of your well-known physicists over there made the point that growth beyond maturity is either obesity or cancer. I think that sums it up quite brilliantly because I think we've got to that point where we've got growth beyond maturity. The Turkish prime minister made an inadvertent slip about democracy once when he said, "Democracy is a tram that you ride until you get to your destination, but then you get off." I think if we paraphrase that and say that economic growth is a tram you ride until your destination and then you get off, it really sums up the modern medical conundrum: we've gotten to that destination, and we really have to change it if we want to continue to improve our health and well being.

JB: I think that's a very provocative comment—very interesting. Were you a friend to John Cameron, the physicist? Because I know he did a lot of work in cancer and that area.

GE: No, no. I was referring to John Maynard Keynes. He was the great economist that started the revival after the Great Depression in the 1930s. The physicist that I referred to was Al Bartlett, who is from Colorado, who just made that statement. It came out in the Colorado daily press, I think it was. It has been quoted ever since that growth beyond maturity is either obesity or cancer.[4] I think it is a great statement.

JB: Yes, it is. It's uncontrolled growth, isn't it? It's unregulated growth.

GE: Exactly. Uncensored growth, basically. One of the interesting things that we've been doing here—Boyd Swinburne, my co-author on a new book we've got out, and myself—has been looking at not just obesity in developing countries and developed countries, but looking at the differences between those developed countries. We've actually found (if you look at this just from a desktop epidemiology perspective) that what happens when a country that is quite poor starts to become richer is you start to get a healthy increase in obesity and you get a decrease in infectious diseases, but you start to get a rise in chronic diseases. Beyond a certain point you get a split and obesity starts to flatten out in most countries, but in some countries (countries we've called the hard capitalist countries, and that includes Australia, the United States, the United Kingdom, New Zealand, and Canada, in particular), obesity continues to increase and it is greater than other countries that we call soft capitalist countries, such as Sweden,

Norway, Germany, France, and even Japan. We're trying to now tease out what it is within those different types or different forms of governance, if you like, in countries that would lead to obesity as a signal that chronic diseases are getting worse in those countries.

JB: That's very interesting. In the article that you authored with John Dixon (the one I mentioned earlier, "Should Obesity Be the Main Game?"), you have a proposed model (this is one of the figures in the paper) on the effects of lifestyle on metabolic outcome. And I think that model is a testable model. It has to do with lifestyle influencing various metabolic functional statuses, such as oxidative stress, inflammatory response, and insulin resistance. It's like a dog chasing its tail; once it starts it becomes self-replicating. Could you describe that model you have developed?

Explaining "Meta-Inflammation"

GE: Yes. Inflammation—or meta-inflammation, rather than just inflammation—is a relatively newly discovered phenomenon. It's a low-grade systemic form of inflammation that seems to run throughout the body, particularly through the epithelial tissue, but even through other tissue and through glial tissue, which is even more interesting for reasons which I'll point out in a moment. Our theory is testable and I'll talk about some testing that we've done on it.

Human beings have evolved in an environment in which they have had hundreds of thousands of years of consistency. The human body—the immune system—develops a sort of friendship with that consistency, such that there is no adverse reaction. In other words, if we are eating fruits and vegetables as we have been doing for the last hundred thousand years, the immune system perceives this is something that is quite natural. It's like a bacterial virus that we've evolved over hundreds of thousands of years. And we have several of these. Of course, we call them our "little friends"; they are very friendly bacteria and microorganisms that we've grown with, and in fact we've come to depend on them.

The sudden change that occurred around the time of the industrial revolution—let's say mid-19th century—has meant that our lifestyle has changed dramatically so that we're eating processed foods that haven't evolved with us over those hundreds of thousands of years. We've become much, much less active as a result of technology. The stress levels have changed dramatically. Our sleep levels have changed because we've got lights and electronics and so on. As a result, the body has reacted—the immune system has reacted—in this low grade, systemic, meta-inflammation response. It has been called meta-inflammation because it is inflammation of the metabolic system.

The theory is that the immune system is reacting to our lifestyle. Not to microorganisms as it has done over the past and with which we are very familiar (we've known this for 2000 years). The response is to inflammation. Now we are saying that it is reacting—it's responding—to our lifestyle. Nobody wants to give up the modern lifestyle, of course, because this is the spectral progression and we've gotten enormous advantage out of the modern industrial way of life, but there are obviously disadvantages and the immune system just hasn't had time to adjust to these.

A Pilot Trial on Immune System Reaction to Diet

We put this to the test in a study that has just been published in the British Journal of Nutrition, where we actually got people here to eat—and this is a random crossover trial—a lean form of meat, which was

kangaroo.[5] Aboriginal people, here, of course, have been eating kangaroo for perhaps hundreds of thousands of years, and hence you would expect the body to have adjusted to that and to not respond in any immune way to the type of meat that is kangaroo meat. We're not saying that all humans have evolved with kangaroo, but they've evolved with that type of low saturated fat, high mono unsaturated fat meat. We compared that with a meal of what's called Wagyu beef. I'm sure in the US you are familiar with that, as we are here. It has only been around for about 30 years. It came out of Japan. It is a high-grade form of beef. It is a very fatty, saturated fat form of beef, and humans haven't evolved with a high level of saturated fat.

What we found is that after eating the kangaroo, there is no reaction by the immune system. It just carries on as if this is a normal type of thing that happens to human beings. After the Wagyu beef, on the other hand, we get this huge rise in what are called inflammatory markers—things like CRP, interleukin-6, and TNFalpha. These are measures of the immune reaction, which is the early stages of inflammation, which we then know leads on to (or possibly even comes from) oxidative stress, which then leads on in a course or chain to insulin resistance and other forms of chronic disease. It supports the hypothesis. There's a lot more work to be done, obviously.

This whole area of meta-inflammation I find is both useful in itself and it's useful as a metaphor for what else is going on in the environment. If we talk about inflammation of the environment, you can see that chronic disruption (chronic change, as it is often called, but I think it is probably preferable to talk about chronic disruption) is a bit similar to insulin resistance in the body, in the climate. It's virtually carbon resistance in the atmosphere, which leads to a lack of sequestration of this carbon, which leads to the potential problems through carbon buildup. That's a big step to take and it's a huge step for most medical researchers to take because it is stepping right outside of their comfort zone. I think all of us are pretty uncomfortable in doing it, but we think that it's got to be done because we think that health is such an important issue it has to be considered in the broader environment.

JB: I very much like the model you've proposed in your review. I think you have a table in there that discusses proinflammatory and anti-inflammatory loads that would contribute to meta-inflammation, including things like nutrition, obesity, inactivity, smoking, and exposure to oxidant stress in the atmosphere and so forth. I think we're subjected to a total load of all these environmental factors that are picked up by receptor mechanisms at our cellular level and transmitted into genes and into an alarm reaction that we call meta-inflammation. Metabolic distortion is a very, very interesting new model of the etiology of chronic disease.

GE: Yes, and I'm fascinated to talk to you about this because you're obviously dealing very intensively with the end stages—the cellular processes—that are involved in this. That's certainly not my expertise, but I think the combination of what you're doing there and what we're proposing (and I say proposing because it really is in a hypothesis stage) is the starting process of all this, the change in the environment that is brought about by things like the agrarian revolution and the industrial revolution. It's a great combination to have, because you can follow the slowest route right from the environment through to the cellular level. I think that's the issue that's got to be presented to medical scientists to convince them to look at the bigger picture, rather than to just deal with the disease at its end point.

Lifestyle and Gastric Bypass Surgery

JB: I know John Dixon, your co-author on this paper, is an expert in the bariatric surgery area. We've come to recognize that when you take people that are morbidly obese and you put them through Roux-en-Y gastric resection surgery, their metabolic distortion (like their diabetes and their cardiac arrhythmias and their dyslipidemias) correct without major weight loss. How does this all fit?

GE: It fits beautifully, actually, and it was a bit of a revelation, I think, to John, who has been dealing with—at that end—looking at the reasons for why this happens. When I presented this idea to him we discussed it, and together with Boyd Swinburne we had long and arduous conversations about this, I can assure you. There is no sort of set pattern in what we are doing because we think we are stepping outside the mold, if you like. In many instances—John being much more of a clinician than I am—he feels a little bit more uncomfortable doing that than I do.

What you say is perfectly true. We don't know why the gastric banding in the Roux-en-Y surgery and so on works. We know that there is some connection there with hormonal influences. We do know that what it does is change the causes of the obesity so that people don't get hungry and they don't eat the types of foods that they were eating before. Now the big question is: If you didn't have the surgery but you just were able to change the lifestyles that lead to the obesity, would that have the same effect? It's really difficult to test that. It would seem that is the case. The work that has been done on that suggests that it is the case: that it's not so much the obesity, it's the lifestyle that leads to the obesity which is causing the metabolic problems. You change the lifestyle and you change the metabolic problems.

JB: Tell us a little bit about this book that you have just recently co-authored with Boyd Swinburne, *Planet Obesity*.^[6] It sounds like it takes this theme to the next level.

GE: Yes, and we're hoping to get this published in the United States shortly, because obviously that's the big market that would create a bigger interest in this environmental approach, I think. The book is basically about the influence—the distal causes—of obesity as being economic growth, and looking—as we have talked about—at meta-inflammation, and meta-inflammation as an underlying factor associated with obesity. But the fact that you can change meta-inflammation by changing lifestyle and by changing environment without actually changing obesity—that obesity itself is probably a warning sign to the rest of the world that we're going down the wrong path. We've gone too far along that path, and now we have to start reconsidering what the alternative is.

There's no economist in the world—there's nobody in the world, surely—who would say that anything can continue to increase exponentially as economic growth has done over the last 100 years. Even with the early economists, none of them suggested that was sustainable over the long-term, and nobody would be foolish enough to suggest that. But economists aren't thinking about (at this stage) what the alternative is because we're still in a gray phase, which is rewarding, in many respects, for most people. Our standard of living has improved up until this point. It is starting to decrease in the western countries, but nobody wants to talk about that. They all want to talk about the continued need to grow. It's not until we get the economists on board, and we are starting to do that.

There are some very good and free-thinking and outward-thinking economists who are starting to say, "Well, maybe we've got to rethink this whole growth system." We've got medical scientists now who are starting to think, "Well, maybe we've got to bring in the economists, we've got to look at the economic side of things as well, in terms of health." And it is once we start to get those two groups of

people together that we'll start to see a major change in thinking. Hopefully it can happen before anything dramatic occurs in growth, such as what occurred in the 2008 financial crisis. If you just look at that for a moment, it didn't actually go long enough for us to be able to tell accurately whether there was a change in obesity during that period.

Does an Economic Recession Increase or Decrease Obesity Rates?

Some analysts actually suggest that if we have difficulties in the economy, if we do get another recession like that, we'll get increases in obesity. I'm of the opposite ilk. I think it's exactly the opposite, as we've seen in Cuba, for example. When the Russians left Cuba in 1989 and left them without fuel, the Cubans had to adjust, and during the period from 1989 to 2004 there was some very good epidemiological work done, and they actually decreased their obesity level by about 50%. Their heart disease rates went down by about 18%. Everything went down. Diabetes went down. Everything went down except cancer, and that's probably because there wasn't enough time for that to decrease.

Another example is Nauru, which is a little island in the Equator where I've worked with the World Health Organization. They have the highest rate of type 2 diabetes in the world and also the highest rate of obesity (about 90% of the population are obese). But that is based on the fact that they are a very wealthy little pacific island country with a genetic predisposition to get obese very quickly. Once you add those two things—a genetic predisposition plus the wealth that they got from superphosphates that they used to send down to Australia, particularly to put on farms—then the obesity rate increased dramatically and their diabetes rate (they had 50% diabetes in the country). They ran out of superphosphate. There's only a certain amount of superphosphate; it's a finite resource, as is petroleum. Once they ran out, they went broke. And when they went broke, their health improved (it didn't decrease, it improved). Their diabetes rate has dropped; we think it's about 30% now, doing those measurements with WHO. The obesity rate is dropping dramatically. All of the individual efforts, if you like, to get people to eat less and to move more—to change obesity at the individual level—have been superseded by this national environmental accounts that dropped obesity and the chronic diseases back dramatically as a result of changing the big picture, if you like.

JB: It's really interesting for you to be talking about this because it brings back a memory that I had kind of put in dormancy in my own life. In 1973 I was a professor at the university and I was teaching a course on environmental science, and I had Dennis and Donella Meadows come speak to us, the authors of *Limits to Growth*.^[7] They did some of the early computer modeling about growth models. As you probably recall, they were not received with a lot of enthusiasm by much of the world economic community and they were considered to be kind of antithetical to good thinking.

GE: Exactly.

JB: I think if you go back and review their book and their work closely you'll find it mirrors very nicely. This is 38 years ago, but it is very comparable to what you are talking about today—the results or the outcome of not having looked at some of the things they said seriously.

GE: And it has been revised, of course, that book. There is a new edition that came out in 2004.^[8] Interestingly one of our scientists out here at the CSIRO actually has checked the predictions from the 1974 edition and found that we are very much on track for the business-as-usual scenario they predicted.

The business-as-usual scenario is quite a dangerous one, because if you continue with business as usual, they predicted that between 2010 and 2020 there will be major disruptions to the way we live because of big oil and a whole range of other factors.

Overview of Lifestyle Medicine Textbook

JB: That's very, very fascinating. Let's just touch upon this book that really got me connected to you through the literature, and that's Lifestyle Medicine. You tell me there's a new edition that just came out in December of 2010. I've got to pick that up because I'm still working with the first edition. I find the chapters to be very interesting. Not only did you introduce lifestyle medicine and its epidemiology and relationship to chronic disease, but you went on to talk about prescription for health with physical activity, about nutrition, about fitness, fatness, and body composition (the difference between visceral adipose tissue—so-called being VAT versus being fat), how stress plays a role, depression as an outcome, understanding addictions, sleep. It's a very, very nice overview of the complex nature of how people's experiences in their environment interact with their genetic pluripotential to give rise to an outcome that we call epidemiology. What kind of feedback have you gotten on the book?

GE: We got great feedback from the US, surprisingly. The new version is actually 2011; it's only just come out. There's an American version. We've had to change a few of our expressions, as your listeners will understand from listening to me. There are probably things that I've said that are very Australian and they don't quite understand, so we've changed some of the expressions. The feedback so far has been extremely good. We've got very good relationships with the American Lifestyle Medicine Association. We have run two Australian Lifestyle Medicine Association conferences out here in the last two years. We've got one hopefully in November again in Sydney this year, if there is anybody interested in coming down. Don't forget to pay your carbon offsets if you do, of course (if you fly down).

We've added extra chapters now, and we've done one on the meta-inflammation which explains that it is the underlying basis of just about all chronic diseases, if not all chronic diseases (in my view anyway).

And we've added another chapter on chronic pain because we think chronic pain, like other lifestyle causes of disease, is actually a disease in itself and can be considered in terms of lifestyle. I mentioned before the glial hypothesis. We know that glia has a connection to the neural system, and we know that glia is also associated with chronic pain. We're beginning to put together a picture of lifestyle and lifestyle influences on glia in chronic pain. We're about to do some research—we've just applied for money to do some research—looking at changes in lifestyle to see if this affects chronic pain and inflammatory markers as much (or more than) just the linear approach to chronic pain, which is “You've got pain therefore take a medication.”

What we are talking about here in lifestyle medicine is really systems theory approach. It is getting away from the linear notion of, “Yes, you've got a disease. Here's the treatment.” You've got to go back and look at the systems and what leads to what, and then what leads on to disease. So you go back and look at the cause of the causes, as I pointed out before.

JB: Yes, as I said in our introduction, this marries itself beautifully with the whole construct of functional medicine, as we defined it over 20 years ago. The Institute for Functional Medicine is now certifying doctors in this whole area. It sounds like there is a very nice tie-together in systems biology and medicine between the functional medicine activity and the lifestyle medicine activity.

Teaching Lifestyle Medicine in Australia

Let me just ask one final question, and I thank you for all the time you have given us on this discussion, by the way. What's your experience in Australia, and maybe in your other contacts with the American Lifestyle Medicine College, in changing doctors' thoughts about integrating this within their practices and seeing the value of it as it relates to beating back the rising tide of chronic disease? Are we getting stickiness, in your perspective?

GE: I have to be honest with you, Jeffrey, that is the big gap at the moment. In a lot of the work that I am doing here I actually teach GPs in this area. We run lifestyle medicine courses around the country. And, in fact, we do not only lifestyle medicine courses in a classroom (I run a course out of Southern Cross University), but we take doctors on adventures where we teach them lifestyle medicine on the run, so to speak. We have a saying, and I hope I can use it on your program, that teaching lifestyle medicine in the classroom is a bit like teaching sex in a nunnery. So we take them out to take them pedaling. We have a trip organized to the Swedish archipelago where Stephan Rossner is our connection in mid-July this year. We go bush walking out in the central Australian desert. We actually sit around the campfire and do a course in lifestyle medicine at night. We find the doctors that get the experiential experience like that actually are able to put it into practice with their patients.

Doctors have come into a classroom (and there is a lot of them who do), and for many of them there is a period of awareness where they get it. Where they actually get it and they realize that because 70% of their patients are there because of chronic diseases, they have to change their whole paradigm in dealing with these patients. It's a difficult process, though, because that involves understanding motivation. This is something that medical practitioners haven't had wide experience in: to understand and apply motivation to the patient, so that the patient becomes the self-manager. Self-management is one of the other chapters in our new book. You have to be able to understand how to get the patient involved in their own self-management. You cannot treat chronic disease with a drug. Surely you can add a drug as an adjunct, but you cannot treat it solely with a drug. It has to be self-management of the patient over the long term, working with the doctor in combination. So it's got to be a partnership arrangement, not only with the doctor but with other allied health professionals as well.

Readiness to Change: Doctors and Patients

JB: I think you've really set a beautiful and important theme there that has been kind of a theme through our last 29 years of doing this, and that is this readiness to change: where is the patient in their readiness to change? And where is the doctor in their readiness to change model?

GE: Exactly, yes.

JB: And then Albert Bandura, from Stanford, and his self-efficacy model, which is really part of what you're talking about. And then lastly Halsted Holman, who is also at Stanford Medical School (a Professor Emeritus there) who has talked about the need for a new model for chronic disease management because nearly 80% of expenditures go for treatment of chronic disease and yet we are still teaching, almost exclusively, an acute disease model to medical students.

GE: Exactly.

JB: It sounds like you've got all these things wrapped beautifully into your work. I want to really compliment you. This is an advocacy that the time has come for. I think you're doing a great job in getting this information out. I love your article, and I think this textbook is something that should be in everybody's hands. Thank you for your tireless efforts.

GE: Thank you very much for listening. It's a fascinating area, but I must admit we're on the cusp, here. There's not a lot of understanding so it's great to have somebody like yourself and your program to allow us to expand on this much more. Thanks very much.

JB: Thank you. Keep up the great work and we'll be in touch.

Bibliography

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