April 2014 Issue | James Fries, MD Stanford University School of Medicine

http://seattlewebd.com/testpage/knowledgebase/april-2014-issue-james-fries-md/

DOWNLOAD SUMMARY NOTES |

Welcome to *Functional Medicine Update* for April 2014. Oh boy, do I have a treat in store for you this month. You know, sometimes my own zeal gets the best of me, but this is, to me, one of those very special moments because we have a chance to do a back-to-the future issue. We're going back to really the start of functional medicine and origin. What were the fundamental groundings that led me to even come up with the concept of functional medicine as a term, knowing that it had been used for some time for geriatric medicine, or for psychosomatic medicine? Why would I choose a term that had already seemed to get tainted by other definitions? The reason I did that was because of the impression of the article that was authored by our clinician/researcher of the month this month in the April issue of *Functional Medicine Update*, Dr. James Fries. You probably recall he authored what I consider a luminary paper in the *New England Journal of Medicine* in 1980. A few years—in fact a decade—before we started the Institute for Functional Medicine. From the day that article was read by me in 1980 until the origin of the Institute for Functional Medicine and functional medicine as a concept, it was the driving force of that article, "Aging, Natural Death, and the Compression of Morbidity" that really set me on this path.[1]

We're now very fortunate. As you know it's a few years since 1980. You can do the math: 34 years. With that length of time, a lot has happened in Dr. Fries' life as well in the life of the Institute for Functional Medicine. Let's move into our discussion with Dr. James Fries, Professor Emeritus, Stanford University School of Medicine, in the immunology and rheumatology department.

INTERVIEW TRANSCRIPT

Clinician/Researcher of the Month
James Fries, MD
Professor of Medicine (Immunology and Rheumatology) Emeritus
Stanford University School of Medicine
Palo Alto, CA
https://med.stanford.edu/profiles/james-fries

Well here we are once again at that point of Functional Medicine Update that gives me the greatest sense

of enjoyment and I hope you as well as a listener, and that is our opportunity to visit with someone that we consider to be really treating the medicine of the 21st century. It couldn't be more appropriately forecast that this particular issue will go down as a legacy issue, because we're tracing back with the individual who I think, if I was to review my 35-to-40 years in the field, probably has been the seminal impactful person in terms of my thinking, and that's Dr. James Fries, Professor Emeritus at Stanford University Medical School, immunology and rheumatology. Those of you who have heard me on Functional Medicine Update over the years or in other venues or read my writings, probably recognize that the most cited article that I have used over those years has been the seminal paper that Dr. Fries authored in 1980 in the New England Journal of Medicine, "Aging, Natural Death, and the Compression of Morbidity." When that article was published it was like a mega lumen bulb went off for me and has lit a path forward that ultimately led to the birthing of the Institute for Functional Medicine.

The Relationship Between Organ Reserve and Chronological Aging

In that particular paper, Dr. Fries talks about this concept that really was a term that I believe he formulated called "compression of morbidity," which seems so logical to me. He related it to things like organ reserve—that as animals undergo chronological aging that they tend to lose organ reserve that they had as a more youthful animal, and that then makes them more at risk to environmental perturbations that may exceed their reserve and cause what we call a catastrophic event, maybe even a life-threatening event. So the concept was holding onto organ reserve and compressed morbidity in the last phase of a life, which hopefully leads out to the limits of your biologically determined lifespan as an organism.

That model seemed so extraordinarily logical to me that I was just overwhelmingly surprised when I saw the letters to the editor that followed that article that appeared in the New England Journal of Medicine in which people were vehemently criticizing this model, which I couldn't even believe how any intelligent person would criticize what seemed so logical and so well laid out. But their criticisms were things like, well, this argument would just keep people alive longer and they would require more medical expenditures because we know that the older you get the more medical expenditures and the more medical services you require, so this was going to not save money, it was going to cost money to the healthcare system. And then other people said, well, you know, this concept will take focus off disease care and it will prevent us from really developing our disease care model as effectively as we should because there is no proof that prevention really pays dividends. So you had all these outlier opinions that sometimes were in such a language that didn't sound, to me, anything other than polemical, as if people were just into self-protection and were not really engaged in the appropriate type of dialogue about this very important concept.

With that in mind, it seems like a very, very appropriate time to come back and revisit with Dr. Fries what all has gone on in his more than 300 publications and seminal contributions to the field over these many years.

Dr. Fries, thanks so much for visiting us at Functional Medicine Update. I think maybe the first question is, what led you—in 1980—to the classic paper in the New England Journal of Medicine, and were you as surprised as I with some of the criticisms to what seemed like such a sensible proposition?

Gerontology and the Failure-of-Success Paradigm

JF: Yes, I was. I'll give you a little bit of background for it. It was amazing prior to this set of reasoning and development that people had thought about aging in such a basically nonsensical way, and it led them

into all of these contradictions. The idea was called the failure-of-success paradigm and it was leading the field of gerontology, and it was that the more that science extended life, the worse the health of the population was going to be. So what you thought were successes in that they affected diseases, were in fact not; they actually contributed to more disability, morbidity, than you'd had before. And it was very strange, but the science of gerontology at that time was referred to colloquially as the "science of drawing downwardly sloping lines." There was no way, given that paradigm, that one could improve human health. You were locked in to the fact that you were going to lose every time you thought you had gained. There were some metric problems and some other issues that were contributing kind of underneath the radar. One was that there was not very much multidisciplinary research into human aging. There was limited scientific research, but there was not the kind of thing that brought historians and psychologists and clinicians and athletes and trainers and all of the things that have something to bear on the phenomenon that we call human aging. They didn't talk with each other. The concept of gerontology should be to improve human health and human life, and this had exactly the opposite connotation, and it had an abiding faith that molecular science was going to give you things which it hadn't yet done.

The metric that I think failed the field at that time was that there was pretty good evidence on mortality, but there was no evidence on morbidity, so that one couldn't actually find out how impaired and how disabled the human populations were, what the risk factors were for them getting disabled more seriously or more earlier. You couldn't do that because you didn't have any data. Now after the compression of morbidity hypothesis came out we began to get data about two years later because there were funded long-term studies, which we'll probably talk about later in this period. But the metric which said that you knew that people were living longer and longer but you didn't have any data on how sick they were. There was a whole point in the life course which had not been recognized, and I would argue that it's the most important point in the life course if you're talking about health, and it's the age at which you first begin to get chronically ill and impaired. That date was unknown, but it was implicitly assumed that it was unchangeable.

So you had a paradigm which was built upon the fact that people were living longer and longer, but you didn't know what was happening to their health as they proceeded through. It was that and there was a lot of dogma, and there was a lot of self-serving behavior. People were trying to get NIH money, and there were ways in which they could get that and ways in which they couldn't. They couldn't talk about soft sciences, like what's the role of exercise and so forth. It was a discouraging time, and there were not a lot of brilliant people studying aging back in the 70s and 80s.

Changing the Paradigm from a Disability Focus to a Functional Focus
JB: I think one of the things that struck me immediately when I read your 1980 paper, and it stuck with me—there has probably been an epigenetic change in my neuronal genome ever since—was your changing the definition from that of a disability focus to a functional focus (to measuring function), which is really like changing the paradigm from a disease focus to a health focus. I thought that was a very, very profound—subtle, but very profound—altered perspective, going from everything built on disability to a model built around relative range of function. That seemed like an amazing change in thinking.

JF: Well, we started saying, all right, let's take a new metric; let's study disability, which was what, as a rheumatologist, I studied. The broader term is morbidity. So, thinking about the quality of life and the things that affect that as more important, potentially, than the length of life, was a radical change, and there have been many, many shifts from the preceding paradigm to what we have now, which is still

evolving. But it has changed and essentially reversed its own side, because it really got into the concept of prevention being the only way to improve human health, that is, the only really meaningful, really powerful way to improve human health because things that happened after you'd passed that point in life when you were now disabled to some degree and going to get worse, that was ill health ahead. So the key concept was to go into a longitudinal life-type of thinking and talk about how to postpone disease. Not talking about preventing disease or curing disease. Postponing disease is by far the strongest approach that you have, and we now know that. We know quite well what exercise does. We know quite well what smoking did. And we're improving rather dramatically in a lot of these ways. But it required getting the observation point and the paradigm that you were trying to quantify in an entirely different fashion.

JB: It was very interesting to me because naysayers are always easy to come by because it doesn't require any discipline for proof other than being a critic. You then, later, with Anthony Vita and your group, came back in 1998 in the New England Journal of Medicine with what I consider really a very profound proof of principle paper on Penn alumni, on aging, health risk, and cumulative disabilities.[2] Can you tell us a little bit about that work and whether it helped change the naysayer opinions at all?

JF: Sure. We were studying risk factors and diseases with the Framingham study before we started thinking seriously—I started thinking seriously—about these issues. And there was pretty good data, as I indicated, there. What we didn't have with risk factor models, though, they were directed at cardiovascular death and learning more about that. But in fact they didn't have anything to do with the trajectories of illness, and there were not longitudinal studies other than those which had their endpoints as mortality or disease-specific morbidities. We knew that you had to change those endpoints. It isn't always connected with our work, but a big area of work that I have also done, and they had connection in the way in which the thinking developed, was that we had to have a better metric.

We developed a health assessment questionnaire, which had a lot of distinctive features, but it measured disability/disablement in a quantitative way. It used a series of dimensions, and it used a quantitative scale, and it gave you a score. And you could go to people every six months and get that score for them. You could tell when they first began to be subnormal in some area of behavior, and you could see what progressed from that point on. So you had to have a metric, and the Health Assessment Questionnaire disability index is now the most widely used scale. It and the SF36, which came on a little bit later on, and they have changed the way in which people have looked at disease.

Two Long-Running Longitudinal Studies Continue to Collect Data

I recognized in 1980, actually, that we had to get longitudinal data on aging, so we began two longitudinal studies—the Runner's Study began in 1984 and the University of Pennsylvania study that you were alluding to began in 1986.[3] We have studied those people and continue to study them now 30 years later. We have been able to follow people through the period of time when they were aging, and dying, and their disability was directing and we were able to pay attention with what happened to the speed of progression of illness with age. That is, the development of disability or the development of morbidity with age, because you had longitudinal studies. And then with the longitudinal studies, we found out quite early on, that people were moving too fast between their providers and between their towns and their jobs and everything else to keep track of them in a normal medical follow-up technique. Framingham, for example, used a small town in Massachusetts, which had very little mobility and you could follow people for a long time, but they still lost a third of the people that were under their follow-up.

The concept that we had for our longitudinal studies was that we would look at a subgroup of people physically in person, but we would follow people with what are now called patient-reported outcomes—levels of disability and so forth by mail and telephone. And we would get people enlisted and we would keep them enlisted a very, very long time and we did that. The findings have been reported about every five years for each of the studies and the follow-up has gotten longer, the subjects have gotten older, until now we're studying 90-year-olds, and we're continuing to learn that the inputs—the major interventions which people take for themselves and many of the other ones that your organization looks at as well—the major interventions are things that improve your physical fitness. They are aerobic exercise, they are intellectual exercise, they are things that challenge the limits and maintain the vitality of the individual, and they really do that, and now we can look at what actually happens when people do this for a lifetime and what are the major risk factors, or in this instance, the major health factors? Major health factors are exercise, absence of cigarettes and other forms of tobacco, and obesity. Those are the risk factors that make a large change, so the question is, how large was this change?

Studies like Framingham indicated that you could detect differences of these kinds of risk factors, particularly cigarette smoking when they were looking at it. And they didn't have exercise variables at all. Cardiologists at the time Framingham began did not believe in exercise as a preventive force, but we now know, after 30 years, that the exercise variable accounts for as much as 16 years of slowed aging. So if you're looking at aging as a morbidity phenomenon, and it has a tendency to progress from midlife to the end of life, then that morbidity variable can be postponed by up to 16 years by regular vigorous physical exercise, for example. That was the Runner's study.

The University of Pennsylvania study was a study of alumni from the University of Pennsylvania classes of 1939 and 1940. They were looked at for three variables: the exercise variable, the smoking variable, and the obesity variable (the BMI variable). Again, the differences were very large between people who had good health risks on these dominant variables and the people that did not, and the differences were on the order of eight-to-ten years in postponement. So notice we don't talk about cross-sectional differences anymore; we try to talk about how much have we postponed the onset of disability? Postponement is a term that should take precedence over prevention, because things are generally not prevented; they are postponed, and the postponement is the variable that is most explanatory.

JB: You know, it's interesting, as I recall the way you stratified exercise in the Penn Alumni study, was the top exercising group was greater than 60 minutes a week of exercise, which seems, in context today (in your Runner's study) to be fairly low-level commitment to exercise, but yet it had a profound effect on the outcome of compressing morbidity.

JF: Yes, but the Runner's people ran between a thousand and two thousand miles a year when they started, and their exercise throughout life was there. So a good number of them were way beyond the physical fitness goals that people had, so a lot of people were running marathons, they were exercising and running a lot of miles a week. (As was I, at the time.) We all knew how much it was improving.

JB: But wasn't it true that in the Penn Alumni study, as contrasted to the Runner's study, that the way you stratified exercise there was greater than 60 minutes a week of exercise was considered your top group?

JF: Yes. Let me explain a little bit about how you build a hypothesis into a study. In this instance we

wanted to be careful, in the design of a longitudinal study of morbidity, that we didn't get trapped by the non-biological variables that we knew were profoundly important. They were important to morbidity and they were important to mortality. You had to study populations that were favored populations because otherwise education, lack of education, and poverty were profound affectors—so socioeconomic class, independent of any of the biological risk factors was a determinant. So if we really wanted to look at aging by itself we had to study people who were favored people—they had good educations, they had good family histories, they had adequate incomes. Those people we found at the University of Pennsylvania, for example, because those were the attributes that people who were attending the University of Pennsylvania—a prestigious university—had. And they also were more reliable. They key was we didn't want to get into a position where we were confounded with socioeconomic factors and couldn't determine what was happening on the biologic side.

The Runner's study was our first concept in this area and in it we took elite runners—running devotees. They were fanatics about exercise, and they put a lot of time and effort into it. And they did extremely well as a result of that. But at the same time, they were nonsmokers, they were fit and not obese, and all of these other things were contributing as well. But you had people that all had 16 years of education, for example. You had to have people that the social factors could not confound the results, and then you had to compare potentially confounding variables, as you do with any longitudinal study. The key was looking at a group that was going to demonstrate a phenomenon if it were there. Those were the people who were already free and had all the health advantages and how much could they get? Well, it turned out the runners could get 16 years additional benefit when they were compared with controls that also were very favored.

Exercise Shown to be Most Effective Variable in Preserving Health and Improves Joint Health
JB: I think that's just absolutely profound. There is an interesting…there are many interesting papers that you have published, but another one that kind of follows on from that is the study that you published on vigorous physical activity and disability development in healthy overweight versus normal weight seniors, which was part of this 13-year longitudinal study, and finding—probably against a lot of people's bias—that exercise even in people with elevated BMI had a very protective effect against premature disability. There is this view that if you are high BMI maybe you shouldn't be running or you shouldn't be exercising because it could be damaging to your joints and you're just going to get arthritis. But it appeared as if your data really spoke to the opposite of that.[4]

JF: Yes, and that's been the general lesson—that if you had to pick a single anti-aging remedy of all of the ones which are touted and talked about by some, the one that is most effective in preserving health is exercise. I'm talking to a group of people who understand things very well now. It works through strengthening the physical body, increasing the organ reserve. It turns out in retrospect, because we even thought the opposite when we started studying the effect on joints, but it turns out that exercise improves the joints. We have a more recent study in which, when we are looking at the runners after many years, the number of destroyed and the controls, about 500 of each, and the runners had four destroyed joints out of the 500 people—destroyed knee joints that either were bone-on-bone or had knee replacements. And the control group had twelve.[5] So the thesis that pain-free exercise is almost inevitably good for you went against the people who had not studied the renewal aspects of exercise.

The joints, for example, contain living cells, but they have no excretory system, and they have no oxygenating system. There is no blood flow to the cells that are in the articulate cartilage and the

articulate cartilage is what degenerates in osteoarthritis. In fact, you can get osteoarthritis accelerated in joints that are casted. The self-lubricating design is really an amazing one because as you compress the cartilage when you put weight on the joints you squeeze out water with waste products and it goes into the joint fluid and then it gets removed, and you do the opposite with oxygen, so that oxygen is brought in and fertilizes and allows the cells to live even though they have no blood supply. So there are a whole lot of reasons that are now understood pretty well as to why we don't see the deleterious effects of exercise that were postulated at the time we began our study. We were afraid of them, also, but it turns out if you are doing pain-free exercise activity, and you have to sometimes search around to find the activity that you can do most comfortably, but if it is pain-free then it is good for you. If it's a joint which has been subjected to sideways trauma, for example, or a variety of things that make it function abnormally, then there are potentially problems of overuse, but there aren't for pain-free exercise.

Evaluative Tools: The NIH PROMIS Program

JB: That's really good news-to-use. As I have watched the evolution of your work over the years I've recognized that as you've started to focus on terms like "healthy aging" and "successful aging" and "increasing health span," that it led you more and more into looking at how you assess the range of functions from those like in your Runner's study, that may be more elite (all), to those that are fairly disabled with a disease like rheumatoid arthritis, where they might have significant limitations in their function. I know you've been expanding your assessment program, this NIH PROMIS Program (Patient-Reported Outcome Management Information Systems). Tell us a little bit about how you're broadening the range of kind of psychometric and evaluative tools that are used for evaluating function.[6]

JF: Well, I told you a few minutes ago that the HAQ which I created was a real advance in being able to assess people's morbidity over time. That metric has been now improved again—probably another order of magnitude—by the development of some new measurement sciences so that there is item response theory where you move from a questionnaire to a bank of items and computerize adaptive testing, which allows you to more quickly and more accurately assess the level that a person is in. These are better rulers, and it's important when you get the rulers in that they develop new hypotheses as you get along.

To get to the point you were sort of alluding to, the WHO, in 1948, came out with a prescient definition, which is that health is not merely the absence of disease. It is total, physical, mental and social well-being. Now, that's been ignored in the breach, through most of the time since 1948, but it continues to be the WHO definition of health. And now we get to a point in which we begin to look at the implications of that for…let's take the most-studied dimension of morbidity: physical function. We've called it disability, and we did that because we were docs when we were starting and we saw enough illness as the thing that we were after, but in fact to define "normal" as the center of a population, on a scale which starts at zero for being normal (which means being average), goes against the fulfillment implicit in the WHO definition of health, because you can have better-than-average health. In fact, even if you have rheumatoid arthritis you can sometimes have better-than-average health. It means that you don't accept return to normality (you know, the average of a population) as success. No, it may be getting the patient with scleroderma to complete a marathon. There are many things than can be done that can raise people who have disability or morbidities to where they have less of it, and quite frequently it will take them up to a scale that didn't exist that is better than normal, because normal was defined as zero.

Now you have to have physical function with two scales (scales going in both directions) because you want to be able to applaud and be happy with having someone who is already very fit become even fitter.

Today the Olympics is testing this thesis out at some scale. People can get really good when they practice and seek ideals and are competitive about it even, or when they are just doing it for fun. But the whole idea is that you change the entire vision of medicine around. Medicine is not just trying to get rid of disease. It's not very good at that, anyway. But it's total physical, mental, and social well-being. So that's a fundamental paradigm shift. PROMIS has now adopted a physical function scale which absorbs the old HAQ dimension and the old SF36 dimension, and allows you to go both directions. It is fundamentally extremely important that we have a scale that measures the entire system. We called what we had floor effects and ceiling effects, and ceiling effects are that if you go to a population of fairly "normal" people, then half of them will be above normal, and half of them will be below normal. Now if you call zero normal, then everybody is either zero or lower, so there is a ceiling effect that is present because your scale won't measure certain kinds of health, and they are very important kinds of health.[7] So PROMIS now is a group, and because it's been well-funded and there are a lot of very good people in it, it's gradually moving this over to where we can talk about health as well as sickness.

JB: Yes, and I really want to applaud your contribution because I think that over these years, the impact of your model, in this compression of morbidity model, has really had its impact on things like the Patient-Reported Outcome Management Information System as to what questions need to be asked. Unless you know the questions to ask, you're never going to get any of the answers, right? That's a simple philosophical conundrum. I really want to applaud the origin of much of this discussion back to your 1980 article. I think the work that you have done helps to give these instruments that have the broader breadth of functional capability so we can measure, as you said, both ceiling effects and floor effects. As a rheumatologist and an immunologist, obviously you've watched the development of disease-modifying, anti-rheumatic drugs over the years, and the effects that they have on morbidity and on function. You probably—I know you have because you've published papers on this—looked at some of the pharmacogenomics and how different people respond to these different drugs. What do you think the role of genomics will be in this whole area of lifestyle medicine as we move forward? Do you think that we're going to get more tailored approaches to help individuals achieve their optimal function?

Will Genomics Disappoint Us?

JF: Yes. That's a big subject area because I think that we're going to be overall—if you take the dreams of the past—a little disappointed with genomics, because the environment and lifestyle approaches (psychological as well as physical) to improving health operate pretty broadly against most people. Sometimes you need very targeted things, but I would put a different criticism on, let's say, rheumatology and the search for disease-modifying drugs. We're anxious to use these when they are there and to move people as well as we can from sickness into health, or from disability into normal or above-normal functional levels. But we have to do that in conjunction with the broader environment, and we have to recognize the narrowness and the limits of the tailoring. Ultimately, genomics is disappointing to many people right now, certainly compared with ten years ago because it has had a limited ability to do breakthrough things. We're learning in rheumatology, for example…we have these very, very expensive drugs which have come in and they appear to be much better than what we had before. But the concept that we were trying to turn off the disease process entirely was present all along, and now we know, for example, that almost always you can do just as well with three of the old drugs as with one of the new drugs. So there is a combination targeting which is not exactly genomics, and it's not exactly at the molecular level, but it is using several different approaches to reducing an inflammatory load and finding out that maybe for ten cents on the dollar you can get the same kind of result using older drugs which are less of a nuisance to administer and much less expensive for society. The things that are sort of readily

available to all of us that impact on health are extraordinarily important and I think will remain the most important and then we'll be trying to pick out particular sense of disease mechanisms which are dominant in a particular patient and then targeting those correctly. So you have to do both, but I think to move away from the foundations of health would result in much more problem than gain.

JB: Thank you. That's very insightful. Your paper that you had published in 2011 titled "Compression of Morbidity, 1980 to 2011: A Focused Review of Paradigms and Progress" I think is a really great seminal work that ties together so much of what you're thinking has related to in terms of advancing the process of how this gets applied in practice.[8] The concept that you had mentioned earlier of downsloping curves—this presumption that we had early on in gerontology that it was kind of genetically preordained that we're just going to be on a downsloping curve of function over time with increasing disability, so those curves would kind of be mirror images of one another—is a very deterministic model of an outcome which requires more and more crisis care because there is nothing you can do about it other than be there when a person starts falling apart. Your model of multiple curves I think is a very, very important part that people, working with enlightened health providers can find a little bit what curve they want to be on, and they're not relegated to the predestined downsloping curve. Is that a fair assessment of some of the takeaways?

The Concept of Diagnosis Can Hold Back Thinking in the Field of Aging Research

JF: Yes, and it takes me to sort of a related point. We've probably been held back in our serious thinking by the concept of diagnosis. Diagnosis—when I was in medical school—it was the linchpin of everything. That is, you gathered a lot of data on the patient, and you made a diagnosis, and then you looked up in the book what the treatment was, and you gave the treatment, and then you got the cure. But it turns out that when one is talking about the phenomenon of aging and morbidity and so forth, that the diagnosis is not really very helpful.

I can take people with rheumatoid arthritis who are healthy and people with rheumatoid arthritis who are not. I can take heart attacks, let's say, or coronary artery disease, and say, "That's a disease. We have an entity there." But the lifetime morbidity curve of people is very, very different. There are actually identifiable trajectories. Let's take the person with coronary artery disease who has a fatal heart attack at age 40. Now, there's very little morbidity associated with that, but there is a tragedy in that the life is terminated in what seems very, very early. Then you've got people that have multiple heart attacks, and congestive heart failure, and go on with illness for 20 years and then finally die at an average age. That's an entirely different trajectory. It has many, many times the morbidity of the sudden death trajectory, yet they are all the same diagnosis, but there are at least four or six major trajectories of coronary artery disease. And then if you start combining those with the concomitant diseases—the emphysema, and the peripheral vascular disease, and other things…well, I guess the thing is we're now trying to say that we need to study trajectories, not diagnoses, because we'd like to change the distribution of the different trajectories if we want to get the overall diagnosis of behaving better than it was. This is another paradigm shift in which the different levels of disability of morbidity within a particular diagnostic category is huge, and it goes from nothing to very severe. So clearly what you want to do is to be moving people from some trajectories to other trajectories and that requires a different kind of thinking than that which we had when we labeled people all with diagnoses.

JB: I think that's absolutely wonderfully stated, and actually you relate that very nicely in one of your recent paper titled "The Theory and Practice of Active Aging" that was published in Current Gerontology

and Geriatrics Research in 2012.[9] It's ironic because I have just authored a book, which unbeknownst to me until I read your more recent papers, the title of the book really relates indirectly—maybe even directly—to what you're speaking of. The title of the book is Disease Delusion, and it's built on this very same theme—that we've had the sine qua non in medicine of the diagnosis of a disease, and that had very great utility so I don't want to throw the baby out with the bathwater, but it also can become a limiting concept as it relates to the promotion of a healthy population. I think your model is absolutely prescient for where we are right now in the evolution of our healthcare system.

Let me ask one last quick question. You have a colleague—you have many great colleagues there at Stanford, but one that I've had the privilege of getting to know a little bit who has some very shared common interests in the themes that we've been discussing, and that's Dr. Halsted Holman, who is a Professor Emeritus there and has done a lot on self-care and the need for a new clinical education in medical school. Have the two of you had discussions or collaboration at all, because it seems like you're working off the same tapestry?

JF: Oh, yes, and his office is two doors from mine. I've known Hal for a long time. We've co-authored books together, which made some conceptual changes. He's been very interested in the social side of medicine and the way in which care is organized. There are so many things that are wrong with the current approaches to maintaining and improving health that it's hard to know where to start. But partly you could say we need to start at the training, or we need to start at the paradigms, but we have to get to a place where our measurement terms are the appropriate ones, and our study techniques and methods are the appropriate ones. Hal has been a very broad contributor to the broadening of concepts and the use of multidisciplinary groups of people in the care process. He's just been a very effective contributor.

JB: Yes, I can see the two of you as being great thought leaders, that's for sure. With your kind of senior perspective over the years as to what's been going on in health care and how the system is evolving and knowing the challenges that it now has as it relates to the rising cost of health care and the burgeoning of this chronic disease epidemic globally, what's your forecast? What's your scenario look like as you look out over the future?

JF: There are still many things that need to be rethought. We're going right now through a debate about a particular approach to health and health care which is based on the financing of health care. It's not going well, and it is almost certain not to go well over time. That is, not as well as it could have because it is designed to be very expensive and to do a variety of things that I actually consider a little bit immoral. I think that taking the money from the young people and using it on the old people is not a good thing to do. We all remember when we were starting out, and it was not the time when we had excess funds to support everybody else. I'm afraid that if you were really going to organize a system that was going to give better care, it would have to start with self-care and with education and how to take care of yourself and how to deal with most minor things by yourself. Capability in the family, in the friends network that you have to get some mutual assistance with things that are often low-tech but of tremendous power. Getting those things organized in a really effective way, as compared with trying to throw money at the problem, or change the social structure of the country.

I just don't think that any of the things at the systems level—that is, the health plan level—are improvements. We have, for example, a lot of single payer systems. We have the VA, we have Medicare, we have government employees, we have a number of single payer systems, and they are okay. But they

are not fundamentally better than things which are paid for by thousands of insurance companies, because that's not the big deal. This last year I went from a fee-for-service system to Medicare. Now, when you are going to say which is better, there's not very much difference. There's a lot of difference in the polemic, but not in the health results. I would like to see something that starts with self-care and ends with outcome improvement. I've had a saying that the two things that you have to have if you're going to change the health plan is that it's got to cost less, because ours is way too expensive by any national thing, and it robs a variety of other needs in the society, and it's got to get better outcomes, so that nobody who is currently involved in the debate on any side says of Obamacare, for example, or any other alternative, "We're going to get better health. We're going to have a metric in and we're going to have a healthier society as a result of this." Now if you work backwards from how you get a healthier society, it's going to mainly do prevention. It's going to have to keep people healthy longer. When people make a lot out of a change and it doesn't have the ability to lower costs and it doesn't have the ability to improve outcomes, I don't see this as an advance. I see this as a wasted effort in large part.

JB: What certainly hasn't been a wasted effort I think is the impact that your 1980 article in the New England Journal of Medicine has had on the changing dialogue, discussion, and ultimately research and development within health care. I think moving from a disease-based model to a functional-based model focused on compression of morbidity is a way that we're going to get on top and ultimately manage the epidemic rise in chronic illness in our society. I just want to thank you for all of your years of contribution and service, and as just one of literally probably millions of people that have been positively impacted by your thinking, to thank you, and on behalf of the Institute for Functional Medicine, really for the germ seed that led to its origin in 1990.

JF: Thank you, and I, in turn, am tremendously impressed with what you've been able to accomplish along this line and with the thinking and contributions of your group. We can have a mutual admiration society, here, and maybe we can come out with a world that's healthier.

JB: Thank you so much. The best to you and your family and I look forward to talking again soon. Thanks so much, Dr. Fries.

JF: Thank you, Jeff.

Bibliography

[1] Fries JF. Aging, natural death, and the compression of morbidity. *N Engl J Med*. 1980 Jul 17;303(3):130-5.

[2] Vita AJ, Terry RB, Hubert HB, Fries JF. Aging, health risks, and cumulative disability. *N Engl J Med*. 1998 Apr 9;338(15):1035-41.

[3] Lane NE, Bloch DA, Wood PD, Fries JF. Aging, long-distance running, and the development of musculoskeletal disability. A controlled study. *Am J Med*. 1987 Apr;82(4):772-80.

[4] Bruce B, Fries JF, Hubert H. Regular vigorous physical activity and disability development in healthy

overweight and normal-weight seniors: a 13-year study. *Am J Public Health*. 2008 Jul;98(7):1294-9.

[5] Chakravarty EF, Hubert HB, Lingala VB, Fries JF. Reduced disability and mortality among aging runners: a 21-year longitudinal study. *Arch Intern Med*. 2008 Aug 11;168(15):1638-46.

[6] Fries JF, Bruce B, Cella D. The promise of PROMIS: using item response theory to improve assessment of patient-reported outcomes. *Clin Exp Rheumatol*. 2005 Sep-Oct;23(5 Suppl 39): S53-7.

[7] Bruce B, Fries J, Lingala B, Hussain YN, Krishnan E. Development and assessment of floor and ceiling items for the PROMIS physical function item bank. *Arthritis Res Ther*. 2013 Oct 3;15(5):R144.

[8] Fries JF, Bruce B, Chakravarty E. Compression of morbidity 1980-2011: a focused review of paradigms and progress. *J Aging Res*. 2011;2011:261702.

[9] Fries JF. The theory and practice of active aging. *Curr Gerontol Geriatr Res*. 2012;2012;420637.p>