

Child Abuse or Rickets?

A Special Interview with Dr. David Ayoub

By Dr. Joseph Mercola

JM: Dr. Joseph Mercola

DA: Dr David Ayoub

JM: Child abuse is a terrible thing. But could it be evidence of something more foundational that isn't abuse at all? Hi, this is Dr. Mercola, helping you take control of your health. Today we're joined by Dr. David Ayoub, who is a practicing radiologist in Springfield, Illinois, working for one of the largest radiology groups in the country.

He's also a volunteer faculty member at the Southern Illinois University School of Medicine, where he instructs medical students and radiology residents. And actually, interestingly, this is not a common characteristic of... [He] may be the only person I've interviewed in the past. He was actually a patient of mine in the past. Welcome and thank you for joining us, Dr. Ayoub.

DA: Thank you, Dr. Mercola, for having me. It's a pleasure to see you again.

JM: We've been excited for this interview for a while now, because you've been compiling this evidence and have just had it recently published in a peer-reviewed journal. But why don't you give us a little background about how you started as a radiologist and started focusing on vaccines, vitamin D, child abuse, and rickets.

DA: Well, it's not a straight-line story. And actually, I have you to thank for my start. Because when I was looking, I was recovering from knee surgery probably about 10 years ago when I was in Chicago at Rush-Presbyterian [St. Luke's Medical Center]. I found your website. I was actually looking for a way to reverse my arthritis. I didn't want to have total knee replacements (TKR). I found your website and started reading some number of interesting things that I had not considered before in my career as a physician.

When I heard about the link between vaccines and autism, I was engaged and planning to get married and start a family. I really took a close look. I fell down the rabbit hole basically. I went to one of the conferences in which doctors were seeing children with vaccine injuries, treating them, and reversing some of their forms of autism and other neurodevelopmental disorders. I really got into vaccines and became an advocate for vaccine safety.

The last interview I did for you was really the link to my current work in infantile rickets. I was looking closely at aluminum adjuvants in vaccines and their association with diseases in early infancy. One of the classic diseases that aluminum is linked to is rickets. It's also plausible that aluminum is anticoagulant. In other words, it can induce bleeding conditions. What comes to mind is fractures and bleeding (what do you think of) in infants – shaken baby syndrome (SBS).

JM: Sure.

DA: The other link really was my good friend, Dr. Ed Yazbak, who's been on your program before. Ed is a pediatrician, a retired pediatrician. I think that he's practicing in Rhode Island at Brown University. He was testifying in court cases on shaken baby cases. We have worked together, writing a paper on the influence of vaccine in pregnancy.

Ed called me one day and asked me to look at some of his cases. He goes, "I know these kids weren't abused. They have bleeds in their brain. But some of them have fractures, and I can't figure it out. You're a radiologist. Would you look at these bones? Let me know what you see."

Initially, I had to call him and say, "I'd like to but I'm not a pediatric radiologist, and I don't know that much about bone disease in infants. Let me pass on that. But if I find somebody who's knowledgeable, I'll let you know." I thought about it for a while. The more I read about aluminum and vaccines, I thought, "You know what? I better reconsider that phone call." I grabbed our textbooks that we have at residency. I took all the pediatric radiology textbooks and copied the chapters on bone disease. I wanted to brush up. Before I looked at my first case, I wanted to brush up on bone disease in infants.

It really took me one night to read all of the material, all of the textbooks, and current pediatric radiology books. There was a stunningly thin amount of literature that was available. I wasn't satisfied. I started pulling references and citations. To make the long story short, I've read probably 2,000 to 3,000 items – literature going back to pre-1900, including the pathology literature. I've translated about seven languages, old books, chapters in books, and so forth. I did an extensive review and started looking at these cases. That's really how I got started.

JM: Terrific. That's quite an investigative journey that you've been on. Can you tell us some of the highlights of what you discovered in that journey?

DA: Well, yeah. Basically, the overview... It's a complicated story. Really, I didn't come to this conclusion overnight. When I started looking at cases... Let me tell you what my bias was. Having fallen down the rabbit hole with vaccines, I thought it was certainly likely that some cases of child abuse were unrecognized metabolic problems. It wasn't a stretch of imagination that they would miss in cases, that they would misdiagnose some cases. I thought I would see a case of child abuse, a case of child abuse, a case of child abuse, and then up metabolic bone disease. I didn't think I was going to see a huge number of misdiagnoses. I didn't really know frankly what to see.

The first case I saw was a preterm baby, a Florida case that Dr. Yazbak was working on. It was a case of terrible bones. They misdiagnosed it. We wrote a report. The case was dismissed. I'm like, "Wow, this is easy." The second case I got was a case of rickets and scurvy. It was a case in Southern States and a criminal case. We testified against a very seasoned elderly forensic radiologist, and we lost the case. It was a devastating loss because this kid has [inaudible 06:18] metabolic bone disease.

After that case, I really wanted to stop looking. I realized that there was a lot of politics. It's difficult to convince the judge and the jury that a general radiologist in Illinois is smarter than their backyard experts at their university hospitals.

JM: I'm just curious. What was the objection for the radiologist down there? I mean, why wasn't he convinced like you were? It just doesn't make sense.

DA: This was my wakeup call. Case number two: this case was so clear-cut. This child had plummeted off the growth curve. He had cataracts for God's sake – cataracts, you know. Unbelievable. Severe weight loss. He had classical changes of rickets and scurvy in the growth plates. I showed comparisons from the literature side by side, and of course, you're showing these to laymen. But even laymen... I mean, this is just showing a picture of a figure. You're looking at comparisons from established textbooks. All it took

for their expert to do – bow-tied, white-haired senior radiologist – is to shake his head, look at the jury, and say, “Scurvy is rare. Rickets is rare. This is not the disease,” and that’s it.

JM: But why wasn’t he convinced? I mean, he had the education and the experience to understand the evidence.

DA: That is a great question. I have reviewed over 400 cases. I’ve been in the legal process over a hundred times, whether it’s report writing or testifying. I have extensive experience. I’ve testified in other countries. I’ve testified in over half of the states in the United States. I’ve gone up against mostly university hospitals – big centers: Children’s Hospital in Boston, Philadelphia Children’s Hospital, Seattle Children’s Hospital, and so forth. I’ve really gone up against many of the top physicians.

I would say that I’m not going up against the radiologists that often. Most of the time, it’s the child abuse pediatrician who testifies in proxy for the radiologist for court. Many of the radiologists oddly enough don’t come to court.

That’s a political question, and one that goes in my head over and over again. But I will tell you this: what we’re seeing – this is probably one of the biggest explanations – is a rather big pill to swallow. For at least 25 years, we have seen rickets on X-rays in infants with fractures. From the very beginning (this is the research out of Boston’s Children’s Hospital), there has been a misdiagnosis or, let’s say, a diagnostic substitution that changes of healing rickets in infants were originally recognized in an autopsy study by Paul Kleinman, who’s a radiologist at Boston Children’s Hospital.

They looked at dead children, surveyed skeletal surveys, and looked under the microscope to correlate what they saw on X-ray with what they saw under the slide, under the microscope. They diagnosed unique fractures. They didn’t diagnose healing rickets. From that time on, the stamp of these changes on X-ray has been labeled child abuse, specific changes in child abuse. It’s a horrific thing to think about. The radiologists all over the country, prosecutors all over the country, and judges have seen these cases for decades. They said, “This is typical child abuse. These are corner fractures or bucket-handle fractures.”

JM: That makes perfect sense. They made this mistake early on. It just got perpetuated, and no one put on their critical thinking hat to analyze it more deeply like you did and took the time, effort, and energy to do an exhaustive literature review to come up with an objective conclusion.

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DA: There were a couple of other researchers before me who have really popped their head above water. They’ve made these claims. Colin Paterson is a bone specialist in the UK who was testifying in court and who was writing papers. He saw what I saw 20 years ago. He didn’t have the knowledge at that time of what healing rickets looks like.

I’ll tell you, in our textbooks, it’s gone. There’s over a 95 percent reduction in our textbooks from what was in the textbooks 50 years ago. Radiologists today and clinicians today do not know the full range of what you see in a radiograph in rickets.

The cases we’re seeing are 100 percent healing-phase rickets. I believe it’s the healing phase that makes these bones more susceptible to fractures. [It’s] not the active phase but the recovery that is the state of susceptibility.

When guys like Colin Paterson and the American researcher Marvin Miller, who’s sedating children, both almost about the same time started publishing papers on temporary brittle bone disease, a state of fragility and multiple fractures in early infants occurring in this two- to four-month window of age, they were

lambasted in the editorial pages. They were attacked by the child abuse community. Colin Paterson went through the General Medical Council (GMC) just like Andrew Wakefield did. He lost his license. There was a vicious attack of the credibility of their work for no reason. There was no science that refuted it; it was just the editorial processes used to say: how could it be? How can this condition exist?

The researchers at Stanford a few years ago, actually about the time I started reviewing cases, very well-known radiologists Kathy Keller and Patrick Barnes had published a four-case series, a very small series. It was published in the *Pediatric Radiology* journal. They described what I'm seeing. They said, "This is rickets." They didn't really put the details in their work that really concluded why this was rickets. They had some of the features of rickets that I knew of, but I didn't know what healing rickets looked like either. I thought their paper was interesting.

It was only once I read the pathology papers and the old radiology literature that I realized, "This is rickets. But it's not the rickets I was taught." It's not the rickets that you were taught about. There's no fraying. You probably remember in med school what rickets looked like. It's a classic they teach to medical students. The end of the bones looks like the end of a broom or a brush. It's a frayed or irregular border.

JM: Sure.

DA: We don't see that. We didn't see that in any of the cases, because rickets in infancy is much, much different from rickets in the classical age group, which is between one and two years of age.

JM: You've been involved with a hundred cases now.

DA: Legally, yes.

JM: Legally.

DA: I've reviewed well over that, though. I do consulting work. I review cases for other experts.

JM: What is the state of the situation at this point? Are most of the cases being won now, or are they still resistant and falling in the traditional methods of evaluating this?

DA: Well, there's a diverse answer to that question. I've testified in a number of courts, and I will tell you that... I know that I don't get involved in cases that are dropped. In other words, when one site... For example, one child abuse team, if they come to agree with us, we never get involved with the cases. That diagnosis may occur. They make the correct diagnosis, treat the child, evaluate for abuse, dismiss it based on absence, you know, social history, witnesses, and so forth. I don't know what the real impact is from our work.

But when we go to a center, when we testify at one site, our reports are disseminated to their team. We're basically educating them through the legal process, not the ideal way to educate anybody because the line is drawn in the sand. Once the legal process starts, nobody wants to lose. Prosecutors want to win at all cost. Child abuse pediatricians generally want to win at all cost. They've made their mind up. This isn't the investigating time; this is the battle. The courtroom is the battle.

JM: Okay.

DA: In some places, the decision process has been legitimate. The judges have really listened carefully. They have great decisions. In the other end of that spectrum, there's been what I believe to be dirty courts. The decisions have been abysmal. They've been appalling. The judges' reports that I have read have been as if they were in a different courtroom, hearing different things. In other words, the reports have been

buffed, the facts stretched. We've been in cases where data have been withheld. We've had experts absolutely state false things on the stand. Let me give you an example of [the] kind of false things that we hear from experts. This has changed over the years I've been testifying (I've been testifying for about six years).

The first argument was vitamin D deficiency is rare. Clearly that's not true. Then they state, "Well, vitamin D deficiency is common, but rickets is rare." No, we know from the scientific data, the current data, that that's not true. And then they said, "Rickets isn't common, but fractures and rickets is not rare." That's currently where we're at.

I had a case, a horrible case, in the state of Ohio in which a young baby [with] multiple fractures – the typical presentation – had a very, very low vitamin D, and one of the forms of vitamin D, which is 1,25-dihydroxyvitamin D, was remarkably elevated. If you know anything about these pathways, that form of vitamin D pulls calcium from bones. An elevated form of this vitamin D is bad for bone. It's a marker of calcium deficiency or vitamin D deficiency rickets.

The expert told the court that this [vitamin] D level is the active form. It is high, which means it's healthy for the baby, and the child can't have rickets based on this. Now, any good endocrinologist, pediatric endocrinologist, who heard the statement would realize that this was absolutely, completely, and utterly false. It means the exact opposite. You could look at any lab manual and realize that that form of vitamin D is detrimental to bone. That woman is in prison.

JM: That's sad. It takes such a long time for the truth to be spread. But thankfully, people like you are helping in this process. I'm wondering if you could expand a bit on your research on the evidence of metabolic bone disease in young infants and how they contribute to this ongoing fraud in child abuse radiology.

DA: It's such a complicated story. It's hard to know where to start. But let me start with really how it came to me, how we evaluated these cases, and came to discover what was going on. It was really about the 10th case or so that I've looked at that I started seeing the same things. It was a spectrum of changes in the bones. I started incorporating a more detailed history – not physical exam but [medical] history – from the mother.

Whenever rickets occurs in the first couple of months of life, it's not really an infantile issue per se; it originated in the mother. You have to look carefully at the mother's characteristics. When we started logging information on the mother, it was dramatic. It was really remarkable how many similarities we saw in conditions, risk factors, and signs in each mother.

Here's what we were seeing basically. The mothers, we had a predominance in northern latitude. Of course, the further north you go, the less effect of UVB light, lower population vitamin D levels. We saw a propensity of spring pregnancies. Of course, vitamin D levels are lowest in the March, April, and May timeframe in the northern latitudes. Again, not 100 percent of the mothers had all of these features. But it was clearly a pattern that we're seeing.

Mothers tended to be obese. Also, 80 percent of the mothers had a body mass index (BMI) greater than 30. What does that tell you? That's one risk factor for vitamin D deficiency.

Surprisingly, a high number of mothers actually smoked prior to or during pregnancy, about 30 percent. That's much higher than the background rates. We had an overrepresentation of twin pregnancies – that's a risk factor. The risk of rickets in a twin pregnancy is 25 times higher. Mothers had what we believe higher than background rates of gestational diabetes and difficult labor.

Half of the mothers had severe musculoskeletal symptoms that were undiagnosed by their clinicians. We had even a few mothers on narcotic drugs because of severe musculoskeletal pain. That's a feature of adult rickets. We use the term osteomalacia.

One of the things that really struck me is that reflux was very, very common in these pregnant women. I mean, not just a little bit of burping up; these women had debilitating reflux. About 75 percent of the mothers were taking antacids, and they were taking excessive amounts of antacids. The most commonly used medication was Tums, which is a calcium carbonate-based antacid. I was just surprised.

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And of course, that's not part of the medical record. This is an over-the-counter drug. You really have to ask the mother if she was taking antacids. I was surprised that the antacids were promoted by their obstetrician. I had a couple of people taking hundreds of Tums over a course of two or three days. It was way above the maximum recommended dose. That was one of the odd things. It was an outlier. I'll tell you why reflux is an important sign of vitamin D deficiency.

For one, when you think about the upper GI tract, from the pharynx to the lower third of the esophagus, that's skeletal muscle. Vitamin D, as you know, is important for muscle strength. When I reviewed the old medical literature in [vitamin] D deficiency, it was very commonly described in babies and in adults. Dyspepsia, bloating, constipation, and reflux symptoms in those patients because of esophageal motility and sphincter dysfunction made perfect sense. That could be a manifestation of vitamin D deficiency. Indeed, when we tested the mothers, the average [vitamin] D level in our mother population or maternal population was about 18 to 19 nanograms per milliliter, which is in the deficient range.

JM: Severely deficient range.

DA: That's significantly deficient for sure when you consider that the average vitamin D level in rickets in the United States in two modern studies is 21 nanograms per milliliter. The babies aren't born with mother's levels; the babies are born with about 60 to 70 percent of mothers' levels. A 19 in a mother is going to translate into a 13 or 14 in the baby. Clearly, we had a vitamin D-deficient population. The mothers... You know, we don't have a control group here, but we do have population values. The vitamin D levels in these moms are about 50 percent lower than what's been reported in general populations. That was, I think, the first epiphany that you have.

This is a unique population. We actually had more Caucasians than blacks, but the Caucasians were very light-skinned. Fitzpatrick Class I (which is a dermatology term), these are the pale women who avoid sun or use sunblock, which is, of course, a major risk factor for vitamin D deficiency.

The mothers also tended to have really poor diets: a lot of caffeine consumption, a very high prevalence of lactose intolerance, or just disliked dairy. Over 90 percent of the women avoided dairy products. [They] did not meet the minimum requirement of one cup of dairy a day.

You add calcium deficiency, you add vitamin D deficiency, and finally you add calcium carbonate in Tums in 75 percent of these mothers. Here's a shocker: this is obviously believed to be a safe medication [with] a generally recognized as safe (GRAS) classification by the FDA. Calcium carbonate is the active ingredient in the diets that they gave rats in the 1920s to 1950s in research studies to produce rickets. Tums's active ingredient is a rickets-causing chemical – calcium carbonate – due to its phosphate-binding properties.

JM: Okay. That's it. Because normally, you would think that it's a calcium supplement and it's a calcium source, it would decrease rickets. Can you expand on how the phosphate interferes with that?

DA: The mineralization of bone requires more than calcium. It's really the calcium-phosphorus ratio in the diet that optimizes mineralization. It has been known since the '20s that if you have high calcium and low phosphorous, high phosphorous and low calcium, or either of those imbalances from the normal ratio, which is about 2:1 calcium to phosphorous, when you go one way or the other too much, you don't mineralize. In other words, you can absorb the calcium; it's just not going into bone. There's not enough phosphorous to make the matrix, the mineralized matrix, to proceed to the mineralization, which is deficient in rickets.

JM: The Tums, the calcium carbonate, would precipitate out or bind the required phosphate necessary for bone mineralization?

DA: I believe it's a carbonate aspect actually that binds the phosphate. But calcium carbonate is actually used in dialysis patients as a phosphate binder. It's used more in Europe than it is in the United States. But it's known to do that.

JM: Interesting. I'm sorry I interrupted you. I just thought it was a good point to expand on.

DA: Well, you know, it stuns me. I guess nothing stuns me. Nothing shocks you [about] some of these everyday things that we use and we take for granted that they're safe. I actually called the drug company that makes Tums. I talked to one of their senior scientists. I asked him, "Where is the research that shows that this will not harm the fetus if it's taken by a mother particularly in large quantities? Will it impair bone mineralization?" I asked that question armed with the research studies from the 1920s, 1930s, and 1940s that show that actually it does that. They said, "We don't have research. It's generally believed to be safe by clinicians, therefore it is." I thought that was a pretty absurd answer.

But it is not just infant populations, you know.

JM: Sure.

DA: There are other studies in adults that show calcium carbonate supplements, including Tums, increase the risks of fractures of the humerus and other parts of the body in elderly people.

JM: I believe it's still widely promoted for osteoporosis prevention as a form of calcium supplements. It's acceptable and there's certainly no caution, widely adopted caution, warning against this.

DA: No. It's, you know. Again, mercury was put in vaccines. It was under the radar. People assumed it was safe. It is the same thing with calcium carbonate.

There are two other little oddities about calcium carbonate that are worth noting: (1) calcium carbonate is contaminated. Commercial products are contaminated with heavy metals. One of those metals is lead. Lead is a known cause of rickets. (2) It's contaminated with aluminum. Aluminum is a known cause of rickets. Calcium carbonate and Tums are contaminated with two metals, at least two metals, that are linked to bone disease as well.

JM: Yeah. With respect to aluminum, I remember in our previous interview, you took the position and provided us with some compelling evidence to support that aluminum as a preservative in vaccines is actually far more dangerous than the thimerosal or the mercury component.

DA: Yeah, I still believe that.

JM: It's a serious issue. I'm glad you're bringing that to the forefront. Why don't you continue with your journey?

DA: We started getting these profiles in the moms. As far as the infants now... This disease, really, it's just an absolutely perfect mimicker of child abuse. These children are born with lower levels of vitamin D from their mother. We believe there's pretty good evidence in the literature that in the first few weeks, vitamin D levels...

The half-life of vitamin D is about three weeks. Babies, if they don't adjust to the oral diet, if they have reflux, if the fact is their liver doesn't mature quick enough (that's pretty common in even full-term infants), if their liver isn't at full functional capacity, doesn't produce bile salts and bile acids to the optimal amount to allow for fat-soluble vitamin absorption, you will get a further drop in vitamin D. We know that babies lose weight a little bit normally. The mineralization of normal babies diminishes in the first few weeks of life.

You have this window if they're born in a compromised state with low vitamin D stores. If they dip a little lower, you're now in a window of significant problem.

Symptoms in the infants can be zero basically. You don't have any obvious signs until they fracture. But we did see some features in infants, which are reported commonly in the older rachitic literature, in the older rickets literature. It's hard to find this stuff in the new science and new review papers.

One of the odd features that these mothers stated over and over again is that the children, the babies, had head-sweating. They were irritable at night, they didn't sleep well, and they were covered with sweat. Why they're covered with sweat, I can't tell you for sure. But it's classically described in numerous sources in the old literature. I would be very concerned if a baby is perspiring heavily at night, especially around the face, head, and neck. They're described as soaking their pillows. They had to change the sheets, because they're so wet. That's one of the odd, lesser-known signs of infantile rickets.

Upper respiratory tract infections and sinus infections are very prevalent in this group of babies. Failure to thrive, which is a classic sign of rickets, was not predominant. We only saw that... Of course, the babies aren't very old. These kids, the average age of fracture was two to three months of age. We only had one case beyond eight months of age. If you look at the bell curve of our kids when they're presented with fractures, a two- to four-month window was the absolute peak. It was a smooth, skewed bell curve.

I think the earliest case I've ever seen was about (it is not in our series; it came in after we published our series) eight days of age. But typically, about five weeks is when they start fracturing from – for example, a diaper change, changing a shirt, putting an arm in the car seat, or, you know, a sibling playing with the kid and being a little rough, that sort of thing, and then they find healing rib fractures and so forth.

The age distribution was stunning. This isn't a random pattern of violence, okay? If this is a real child abuse... I've not actually ever seen a case over 12 months of age.

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Believe me, I have three kids. The anger of a parent is much greater in a two, three, four, or five-year-old, even older than in a two-month-old baby who's adorable, cuddly thing who sure may cry at nighttime. But it doesn't make any sense why these kids would present at the same period or window of time. There's no sense or whatsoever.

JM: Yeah, it certainly wouldn't seem rational. Before we go into some of the other associations you've uncovered, I'd like to expand on the importance of the recommendation for all pregnant women to make sure they have enough vitamin D. Because everything you're describing is virtually 100 percent preventable from the implementation of one simple measure, which is to normalize your vitamin D levels when you're pregnant.

Everyone should have normal vitamin D levels. But one of the most at-risk populations is a pregnant woman. I mean, to me, it's reprehensible medical malpractice not to perform that analysis and provide that device for a pregnant woman.

DA: I couldn't agree with you more. It's medical negligence not to monitor and replete women [of vitamin D]. We know so much about vitamin D in pregnancy right now. The prevalence rates are absolutely unbelievable.

I like to quote the Bodnar study, Dr. Lisa Bodnar. The research's in the University of Pittsburg. I think I sent you a copy of that paper, the abstract. In northern latitude Pittsburg, they looked at 200 white women and 200 black women. They found that really it's only about one in 10 black women and one in three white women who have normal vitamin D levels. Unbelievable. And 90 percent of that population is on recommended supplements. In spite of supplements (which, as you know, 400 units is a measly amount of vitamin D), there's still quite that deficiency.

JM: And still clinically insignificant.

DA: Yeah, that's right. I mean, that might bump an infant, might bump a one-week-old baby a little bit, but an adult...

JM: It's not going to touch it. They would need at least 10 to perhaps 20 times as much to make a dent. I thought it's an important tangent because the message is: anyone listening to this, if you're pregnant, of course, you need to do that. But if more than likely you're not pregnant but you have a friend, a relative, or a neighbor who is, this is a simple measure. They don't need a doctor to implement this therapy. They can just get vitamin D.

Now, obviously, it's very difficult to do in the winter. I strongly and firmly believe that the ideal way to get vitamin D is through exposing your skin in ultraviolet B radiation, and oral is a poor secondary supplement. Unfortunately, it really is the only option that's available to most in the winter. It's certainly better than the alternatives that you're describing as a complication as a result of not enough vitamin D sufficiency.

DA: I agree with you. I mean, I think the sun... I think UVB is the best way to get it. Unfortunately, there's even a prevalence rate of deficiency in people who are exposed to the sun. There have been studies that looked at that. There's a phenomena. This isn't in the medical science; this is in the environmental science. Have you heard of the term "global dimming"?

JM: Briefly, but I can't remember the specifics.

DA: Well, I was curious about it. Of course, this doesn't show up in our literature. But it appears that there is worsening of vitamin D in the population and something is going on. It isn't better awareness. There are a few serial studies that compared historical prevalence rates. It found that really, our population and populations in other countries are worsening with regards to vitamin D status.

When you think about it, the sun is the biggest source of vitamin D in the average adult. Global dimming is the phenomena of diminished UVB production by the sun, decreased contact on the earth's surface by sun. A number of environmental studies have shown decline in UVB production. These are monitors that were placed all over the world, and this has been going on for two to three decades.

JM: This is solar UVB production, or is it just actually a shielding within the atmosphere?

DA: They haven't solved that. I mean, what's hitting the earth's surface is not producing UVB. It's either sun production issue or it's environmental. It's hazing in the skies.

JM: Sure.

DA: I favored the latter. But obviously, that's a question that could be answered by a scientist. But that's a true phenomenon. That's contributing. I think there's something else that's contributing to the populations.

In my day to day practice, I normally read more adults than children. The use of antacids – proton-pump inhibitors, H2 blockers, Tums, and things like that – is epidemic. I mean, elderly people [have] very high prevalence rate of acid suppression. What happens when you suppress stomach acid? A lot of things: (1) you have impaired absorption of calcium. (2) You impair absorption of magnesium, which is important for impaired thyroid function. Your thyroid gland in the neck helps regulate calcium absorption. You have two hits on calcium: direct and indirect.

When you lower the body's state of calcium, you consume vitamin D. You convert the storage form 25-D to 1,25-D, and your levels drop. It is certainly... It has been shown experimentally. It has been shown clinically. When you suppress the calcium status of the human body, you deplete vitamin D. We're now talking about a secondary vitamin D deficiency due to the overuse, the widespread use, of acid suppressants.

JM: Yeah, another important vitamin or nutrient that's suppressed is the ability to absorb vitamin B12.

DA: B12.

JM: Which can make a difference in neuropsychological symptoms. The bottomline in this is for you or someone you know or love [who's] using some form of therapy for acid suppression to stop it immediately and treat the causes, which are relatively easy to treat. It's really a dietary issue and maybe an acid supplementation – not an acid suppression – issue. We've discussed that in many articles previously. I would strongly recommend pursuing those if that's your case or the case of one of your friends or loved ones.

But you've also found an association between leukemia, I believe, CML, which is a variant of leukemia, in some of your patients. Can you expand on that correlation among rickets, vitamin D, and this CML form of leukemia?

DA: I'm sorry, Dr. Mercola, I use that acronym. But in radiology circle, CML is classic metaphyseal lesion.

JM: Oh, I'm sorry.

DA: I should know better, but you know.

JM: Okay.

DA: In my area, that's what CML indicates.

JM: Well, what is CML? It certainly fooled me. I'm sure many other people don't know what it is.

DA: This is the basis of our paper that's just been published in January in the *American Journal of Roentgenology*. CML is the term that stands for classic metaphyseal lesion, a metaphysis at the end of the growing bone. This is the work out of Boston Children's Hospital, which we reviewed and we critiqued. These are the fractures at the end of bones in infants which are allegedly diagnostic, nearly pathognomonic of inflicted injury to the bone. What we have found... Our review basically began with a review of the literature.

CML is what basically every pediatric radiologist believes is diagnostic of child abuse. It looks like the bone, it looks like a fracture. They call it a bucket-handle fracture (where a piece of bone is elevated above the growth plate like this) or a corner fracture. If you rotate the leg, you can always see the corner. It's a projectional phenomenon with how an X-ray is taken. But it was interpreted as a fracture across the very end of the boney growth plate. Before you get into the cartilage, it is the bone itself.

It was published again by Dr. Kleinman in his research. Although we've looked at the entire world's literature, it turned out there is no world's literature. There is only one researcher who published nine papers on this. The biggest problem we found is that they haven't been replicated outside of Boston Children's Hospital. A very oddly known series of papers, which has now become the bible in child abuse radiology.

What the researchers had done is in dead children – sudden deaths. We don't know the nature. There were very little details in this publication on how the children died. They X-rayed the children, looked under the microscope at some of the material, and concluded that these bucket-handle fractures in bone were also seen in pathology. And therefore when you see this, you're seeing child abuse. You don't see this in the accidental literature. It's specific to grabbing a baby and shaking a baby, the limbs flailing, and they get these unique tears. That was the hypothesis.

We found in our paper – we outlined, I think, something like 17 issues – 17 major criteria flaws with the paper.

This is the controversy. When you have research that's this far off the mark, you have to wonder how they could make these diagnoses and how could they come up with a radical hypothesis that was never replicated by anybody else. There were a number of problems:

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(1) The first thing is that they had a gentleman who looked under the microscope. Of course, radiology is not a stand-alone science. Radiology is a shadow of real disease. Everything that I diagnose on an X-ray, somebody has done studies, generally multiple places, that establish when you see a mass on a lung, you know under pathology that's going to be a cancer. [There's] a good chance it's going to be a cancer and so forth. You need pathological correlation.

To get pathological correlation, you need a pathologist. A pathologist is a physician who's trained to look at diseased tissue – not just normal tissue, but diseased tissue. The individuals who serve as pathologist in this paper, in this series of papers, were a radiologist and a dentist. I love dentists; I just had dental work done. I have a lot of respect for dentists. But dentists are not pathologists. I don't think anybody at the time knew that this radiological-pathological study was performed by a non-pathologist. That's a big problem.

(2) They didn't screen these children for metabolic bone disease. There's no description of it. They assumed that metabolic bone disease was rare. You might ask, "These were dead infants that were screened. What's the prevalence of rickets in dead infants?" In the 1980s, 1990s, when they did this research, we didn't know. We had historical studies from the 1950s onwards. Still after that era of epidemic (the epidemic was in the '20s and '30s), the prevalence rates were up to 80 percent in infants incidental. This is subclinical rickets found at autopsies.

We have an answer now. Marta Cohen, who's at the National Health Service (NHS) Trust in London, pediatric pathologist, did an autopsy study. It was published last year. They looked at a relatively small number of infants who died. They found rickets in the first year of life: 87 percent; first four months of life: 100 percent; after one year of age: zero percent. Rickets is epidemic and histological. They're seeing this.

In other words, we go back to researchers at Boston. When they looked at dead children and took X-rays, these kids were the same age group that we're seeing. They were not beyond... I don't think... I think the average age was two to three months. The oldest child was 10 and a half months of age, the same type of a skewed bell curve. The prevalence of rickets in that population would have been 87 percent – very high. He didn't see it.

JM: And assumed it was zero or close to zero.

DA: That's right. A healing rickets looks like – guess what – a bucket handle. There's the bone. When you heal rickets, the mineralization front floats above the bone. It looks like a piece of bone that's torn off and then it fills in. You mimic a bucket handle when you have healing rickets. And bingo – that's what they saw. There are more details. There are a lot of nuances in our paper. [They're] quite technical.

But he described in great detail the histological feature of the cartilage cells called chondrocytes. Hypertrophic chondrocytes were in excess. He said that's because the blood supply was disrupted from fractures. Well – guess what – the hallmark pathology in rickets is an excess of hypertrophic chondrocytes. It's overproduction of cartilage when you fail to mineralize it.

There was a number of unfortunate diagnostic substitutions and misinterpretations of data.

Actually, I believe it's a miracle that our paper was published, because it is so controversial not from the fact that it's not validated [or] there's not a good basis for our issue with this science. But we understand that there are thousands of cases misdiagnosed a year. This has been going on for 25 years, 30 years, or longer. How many courts are going to want to accept the fact that any case they've had of multiple fractures in infants was probably a misdiagnosis?

JM: Yeah, it's pretty radical and trendsetting. I'm really happy and delighted that you're able to get it published. What journal was it published in?

DA: The *American Journal of Roentgenology*.

JM: Oh, that's a standard. That is the number one peer-reviewed journal in your profession or your discipline.

DA: Yeah, it's a very good journal. The editor did a great job of getting it peer-reviewed. She sent it to – I don't know who the individuals were. The marks we got were remarkable. They actually didn't ask [us] to do a single edit [in the] paper.

JM: Wow, congratulations. That's a quite a feat and accomplishment.

DA: I work with two other authors I should acknowledge. Dr. Chuck Hyman is a retired child abuse pediatrician from Loma Linda. He lives in Redlands, California. He's been involved in cases like this before I got started. Marvin Miller, who's a bone specialist, pediatric bone specialist, and geneticist at Dayton Children's Hospital, is also a collaborator on this.

JM: Terrific. Getting back to the risk factors, you had described the use of antacids – proton-pump inhibitors, calcium carbonate, and such – and low vitamin D in the pregnant mother. Are there any other risk factors for the mother that would lead her to have a child with rickets and potentially be falsely accused of child abuse syndrome?

DA: There is a genetic component that's hard to put your finger on. When you ask the parents, "Is there a history in the family of bone conditions – insufficiency fractures, spine compression fractures, hip

replacement surgery – and all these kinds of things linked to vitamin D deficiency,” it’s pretty commonly confirmed. I even had cases where they say, “Yeah, my uncle had rickets when he was a child.”

From what we know about mineralization, the genetic aspect of it’s amazing. There are polymorphisms in every possible pathway you can think of. Vitamin D is not so straightforward. Vitamin D works through a receptor. There are polymorphisms of these receptors. There are vitamin D binding proteins that are part of this equation. It is really, really a complex metabolic question.

But there is a genetic component. I believe that [it’s like], I think, vaccine adverse events with regards to autism. We know autoimmune disease, for example, occurs frequently in families that have autistic children. So, yeah, there is a genetic component. It’s hard to quantify. We just ask these questions. I don’t know what the background rates are. But I think that’s supported in the current literature.

JM: Okay. There’s also a potential implication with women who aren’t breastfeeding and using certain formulas that are high in palm oil.

DA: Yes.

JM: Can you just expand on that?

DA: Yeah. Post-natal issues, the two major post-natal issues we’re seeing: (1) something I really didn’t think about when I reviewed this older literature is the growth rates. The linear growth rate in babies is enormous. When you have severe nutritional deficiency where the baby doesn’t grow, you cannot get rickets. Rickets requires growth. The mineralization process is a two-step process: you lay down a cartilage precursor, and then you mineralize it. When you’re growing rapidly, and by rapidly... You know what growth charts look like, baby growth charts.

JM: Sure. I’m sure any parent has seen that.

DA: Right. The slope of that chart corresponds to the rate of growth. If you remember, from birth on, that vertical growth is almost linear. I mean, that’s vertical basically. That is the time of particular high demand for calcium salts. It stresses the mineralization process. That’s another big reason why we see rickets in this early life, the first few weeks of life. It’s because the demand for bone growth is so great. It’s like the stress test for coronary blockage, right? You’re stressing the bone, and this is when rickets manifest.

(2) Those moms that we don’t have actually... A typical population in childhood rickets is breastfeeding, prolonged breastfeeding – not just breastfeeding but prolonged breastfeeding. Most of our moms were actually on formula. Because of reflux in the babies, the babies are often on multiple formulas, alternative formulas. There was a high prevalence of soy formula and palm olein.

I started researching the content of these formulas. Palm olein is a synthetic triglyceride meant to mimic palmitic acid that’s in breast milk. There are others. A review article by Winston Koo (I believe he’s at the University of Cincinnati now), who looked at the studies of the effects on bone mineral of palm olein-containing formulas. Shockingly, virtually every paper that looks at the effects of bone showed osteopenia or diminished mineralization when babies are put on these formulas. Now, you have a formula that causes bone disease or that’s linked to bone disease.

JM: How common is this fatty acid in infant formulas?

DA: It’s in the majority of them. Next time you’re out shopping, go down the baby formula aisle, turn it over, and look and see how many have palm olein. It’s in most of the formulas now.

JM: That's just shocking, but not surprising. Anytime you get away from what the natural program was, which is obviously breastfeeding, you're going to run into complications. The further on we go and study this, the more we become aware of the side effects. This is a new one. I certainly haven't heard of it before.

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DA: I think breastfeeding gets a little bit of a bad knock with regards to vitamin D, because historically we've labelled it... Actually, the early papers link formula to rickets. Did you know that? They didn't implicate breastfeeding. They said that obviously these patients are artificially fed. This is in the 1920s, okay? Across the boards, they recognized that formula feeding was a risk factor. That's been flip-flop. Why? It's because mothers are vitamin D deficient, their milk is D deficient – nothing more than that.

My wife breast-fed our last and third baby. [For] two months, we tested his vitamin D levels. [He's] 100 percent breastfed. His vitamin D levels were 55 nanograms per milliliter on breast milk.

JM: No supplementation?

DA: Oh, absolutely. We supplemented that with the sun. We supplemented, we maintained, and we measured. We measured my wife's vitamin D levels. She managed her levels in the... I think she's right at about 74 nanograms per milliliter.

JM: Okay. But no supplementation in your child?

DA: Oh, no, just breastfeeding.

JM: That is surprising.

DA: That's right. Perfectly normal, healthy, optimal levels of vitamin D with nothing other than breast milk from a mother who's had her vitamin D levels optimized. Because traditionally... In fact, what I first put in my articles is that breast milk is not going to be a source. But that was because the average woman was overly deficient in vitamin D and couldn't possibly give it to her child.

JM: That's right. And we have a lot of women who just don't take calcium. They're lactose-intolerant. They use soy. They're not getting really good calcium nutrition involved. It's really a two-pronged issue with moms.

DA: Are there any other guidelines or recommendations you formulated as a result of your involvement with this for pregnant women?

JM: Well, the cases that I have reviewed largely were fracture cases. But a number of cases, let's say about a third of the cases I reviewed, had brain bleeds. Their allegations weren't just battered child but shaken baby brain injury. Obviously, the child abuse pediatricians just say, "There are fractures, but there are bleeds in the head. This is obviously head injury. You must be wrong that the bleeds indicating a violent attack to the whole body, including the head, can have anything to do with bone disease." That absolutely turns out to be untrue. There is a link between risks of bleeding in the brain and rickets. It's a little bit more than just bone health.

Again, it's not one thing. It's not so straightforward. But the old pathology literature, even pre-1900 in the French literature, [says] hemorrhage in the brain, over the convexities of the brain, the subdural compartment. They didn't use "subdural hematomas." They used "pachymeningitis interna hemorrhagica." It was the term from the old literature. It was very common in rickets. Why do you get bleeds in children with rickets? A number of factors:

(1) The cases that I've reviewed, where these children presented catastrophically, they basically, during feeding, choke or gag, their eyes roll up, they collapse, they get cardiopulmonary resuscitation (CPR), they die, and they have thin-film subdurals, a small amount of subdural bleeding in the head, and fractures in various states of healing.

It's been shown by researchers in United Kingdom – Marta Cohen, Waney Squier, Irene Scheimberg, a London pathologist, pediatric pathologist, neuropathologist – did autopsy studies on babies that die of natural causes in the first few months of life, and found about 20 percent of those babies (higher in subsets) have bleeds in the head – subclinical, undetected bleeds. They've linked this to hypoxic events. In other words, if you diminish perfusion of blood and oxygen delivery to the brain, you can predispose that brain to bleeding. That can occur very acutely.

What's happening, they believe, is that the venous outflow is becoming obstructed through vasomotor tone. The blood vessels have muscles in them. If you deprive those blood vessels, walls of the blood vessels of living tissue of oxygenation, they can clump down, venous pressures build up, and they can ooze blood around the brain.

Do children with rickets have hypoxic events? Absolutely. Seizures, okay? I've had a case, a legal case, in Texas that was in the news actually. The baby [had] seizures and died. The calcium was critically low – completely ignored. Even in the courtroom, a critically [low] calcium level was completely dismissed by the experts. Seizures, hypoxia, kid stops breathing, has a thin film subdural hematoma, and of course, the skeleton shows rickets and fractures in various states of healing. That's one association.

(2) Again, in the old literature, spasm of the upper airway, they can occur spontaneously. You've heard about carpopedal spasm. For example, people with hypocalcemia, they get hands [that] go into spasm, muscle cramps, or muscle spasms. In children in particular, they're susceptible to spontaneous closure of the airway. What happens [is that] particularly during coughing, feeding, because of the low calcium state, there's a trigger of airway spasm, and children choke to death. Spontaneously, they just can't take the air. They die. The hypoxia, diminished oxygenation of the brain, causes hemorrhage. You do have advanced with rickets through hypocalcaemia, seizures, and airway spasm.

(3) You also have vitamin C deficiencies. Ninety percent of children with rickets have vitamin C deficiency. This is from the Russian literature mostly. But in the old American literature, they didn't quantify what many researchers said: vitamin C deficiency is very, very common in rickets. Coexisting nutritional deficiencies.

(4) Vitamin K metabolism occurs through vitamin D pathways as well. Clotting also occurs more exuberantly in cases of vitamin D deficiency. We know that deep vein thrombosis (DVT), for example, thrombosis of leg veins and clots in the lungs, occurred more in winter and in more African Americans. That's linked to vitamin D deficiency. The veins in the head can clot in vitamin D deficiency. That causes back bleeding in the brain.

(5) There's a condition where water builds up in the brain. It's called external hydrocephalus. There was a paper in China published a few years ago that reported 73 cases of rickets with this condition of external hydrocephalus or water surrounding the frontal parts of the head. It causes enlargement of the head. About 10 to 20 percent of those children get subdural hemorrhages, just like what we see in shaken baby syndrome. There are a number of things that will link bleeds in the brain to rickets and fractures.

It really looks... For all practical purposes, you're right. You look on the surface, and you think, "This person was pummeled by a caregiver." Bleeds in the head, bleeds in the retina, or simply just venous hypertensions. Swelling of the brain causes the veins in the back of the retina to bleed and nothing more, not shaking of the retina.

JM: Yeah. There are many physiological reasons why vitamin D deficiency would cause this, as you just expanded on. The challenge that we have in contemporary society is that there's been an industry built up around protecting the child from child abuse that has sort of become entrenched in these older views. They're well-intentioned. Of course, they want to protect these children. But they're operating many times under insufficient information, inadequate information, outdated information, or a combination of all of them. This is the challenge that we have.

I'm wondering about two things: if you can comment on what can be done to change that system, and then what can parents do to protect themselves if they or someone they know or love is accused of child abuse when they know that's not the case, and it's more likely a result of vitamin D insufficiency.

DA: I think it's important. I think the legal system is relying heavily on the existing science, which is flawed. Physicians have to be responsible. We have to do research. And it is being done. Number one: we've got to get the papers out that establish what these diagnoses really are. We certainly have to be sensitive of the fact that abuse does occur. We have to be sensitive that when you have a broken bone, you can still abuse a child with rickets. There still needs to be a process in which these families are evaluated for mishandling and real child abuse.

But in my experience and the cases I've seen, I have not seen any high-risk family. I don't believe any case of fractures I've seen has been a result of real physical child abuse, that it's been metabolic.

Now, as you know, there's science that links vaccines with autism. Why isn't that science believed? Well, it's attacked. It's marginalized because there are competing papers, generally very flawed papers, which refute their claims. [They] design studies in order to give the answer that they want.

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That's going to happen when you have an industry this strong. The government is a big industry. Child Protection Services (CPS) is a behemoth, believe me. There's a lot of money that is generated from the job of protecting children from abuse.

JM: That's somewhat surprising for someone who's not involved in the industry. I mean, it's maybe not surprising, but I wouldn't have guessed that, that there's been such a large industry around that.

DA: That's dark politics. Now, this is very deep politics. There are people better and more adept than I am in talking about the courtroom and the CPS politics. But it is another rabbit hole. There are some major problems in the way we protect families.

JM: Okay. That's an issue. Obviously, it's not the time to go into specifics here. But just to be aware of that, you can pursue that on your own, because there's a lot of other people who have expanded on this.

Okay, so, what can the average parent do if they're accused of child abuse and they know that's not the case?

DA: These are my general recommendations when I get a phone call from a defense attorney or sometimes when I'm contacted directly by a caregiver or parent. My recommendations are:

(1) Go to your obstetrician. Get a vitamin D level [test] immediately, because the baby's vitamin D levels increase dramatically. By the time they get around testing infant's vitamin D, it's often two or three months after they present. The vitamin D level in a baby in the first 15 months of life can go up three, four, or five-fold. It rises dramatically. Even on breast milk, it rises dramatically.

That vitamin D level in the baby does not reflect what the baby was developing with. A mother's level stays. A mother's level that's depressed will stay depressed for at least nine months after pregnancy. Getting the mother's level afterwards will tell you what the baby was born with and what the baby was dealing with in the first few weeks of life. [That's] number one.

(2) They need to seek out a pediatric endocrinologist, because those specialists are the best specialists to assess the clinical circumstances – biochemical assessment, clinical assessment – of vitamin D deficiency metabolic bone disease. Those specialists are not getting involved in the courtroom very often. That is one useful specialist they can seek out.

Some parents have control of the babies or otherwise the baby is with the grandparent. The grandparent can take the baby to a doctor. If they're in custody, temporary custody, somewhere else and social services are involved, they may not have the ability to take that baby to a specialist. I suggest... I've sent several of the mothers to Dr. Michael Holick in Boston. As far as I know, virtually 100 percent of the time, he's diagnosed conditions in the mother. Those conditions are often osteomalacia, which is adult vitamin D deficiency.

Oddly enough, I didn't mention this earlier. A significant percentage of these cases, the mothers are diagnosed with Ehlers-Danlos syndrome (EDS). It's a clinical diagnosis. It's a connective tissue disease characterized by hypermobile skin and hypermobile joints. The genetic testing is not good for most of the forms. It's usually not a clinical basis. I frankly don't know whether they really have Ehlers-Danlos. Vitamin D deficiency can mimic Ehlers-Danlos because it's associated with joint hypermobility as well.

We know that collagen requires vitamin D as well. We didn't know that traditionally. But research really in the last 10 years has shown it's important for collagen pathways like vitamin C is. It may just be a manifestation of vitamin D deficiency. It may be a comorbidity. But it's useful for the mother to get worked up for metabolic bone disease as well.

JM: Perfect. I know you've mentioned the genetic testing wasn't that great, but that's probably going to change with DNA sequencing techniques. There is an organization called 23andMe that was providing that for 100 dollars and could easily assess that. Unfortunately, the FDA shut them down for claims that they were making. But they'll probably be back up at some point.

DA: I think you asked me about prevention. What would you do to assure that your baby doesn't have fragile bones? Well, it's what you've been telling your patients and what you've been telling people on your program: make sure your vitamin D is replete. Don't be afraid to ask for a test. Do your own self-test if you need to. Now, there are two labs. I think ZRT Labs. You can order from vitamin D to calcium.

JM: That's one.

DA: Doctor's Data now has a test kit.

JM: And Grassroots Health, too, is another organization that we work with. The cost is pretty similar, maybe even less than what you get at a typical lab, unless insurance covers it. They are doing ongoing studies to help advance the science of vitamin D.

DA: Right. I think there's also a confusion of what level of vitamin D constitutes adequate. My personal belief is that you really need to be well above 40 nanograms per milliliter. I prefer it to be in the 70s. You may have a different opinion but...

JM: No, I feel similar. I think from Bruce Holick?

DA: Bruce Hollis.

JM: Hollis, yeah. I mixed him up with Holick. Bruce Hollis did some studies that convinced me that vitamin D sufficiency was about 50. Anything below 50 is probably a little less than optimal. So, 40 and 50 is good. But I mean, if you can get it a little above 50, that's fine. Seventy is probably even better.

DA: But really, if you have poor calcium, the vitamin D is going to be limited, what it can do to bones. The mothers need to have an adequate source of calcium. Natural calcium is best. But those who can't, I don't know what the suggestion is, you know. [It's] certainly not calcium carbonate.

JM: Right. Dairy typically tends to be a relatively good one if you're tolerant of that. That's basically... Not butter, but the other forms of dairy.

DA: Right, yogurt and so forth.

JM: Yeah, yogurt, milk, and kefir. Well, that's great. I'm really grateful that you're able to have that paper published and really provide a new standard in this important area, because it's just really tragic when parents who absolutely love their children are accused of this tragic crime. Nothing could be further from the truth. I mean, the only crime that they committed is that they were ignorant of the importance of vitamin D. But that's really more a responsibility of the physician that they were under, who should have been more knowledgeable.

I do consider the vitamin D education of professionals one of the successes we've been able to catalyze through our sharing of information, that more and more physicians are aware of it. But I still think there's a significant number especially in the OB field who obviously don't get it, otherwise these cases would not be occurring.

DA: Well, the ACOG has not pushed forward the recommendations. Their recommendations are abysmal.

JM: ACOG, for those who are not familiar with it, is the American Congress of Obstetricians and Gynecologists.

DA: Right. They basically acknowledge that vitamin D deficiency is prevalent. They acknowledge that vitamin D deficiency is linked to a number of conditions in pregnancy, including gestational diabetes, preeclampsia, hypertension, preterm labor, difficult labors, and increased C-section rates.

But they turned around and stated that they needed more randomized studies before they change the recommendations, which is absurd. You don't need randomized study. This isn't a drug trial. Health is at stake. We need to replete women. We have to improve these abysmal vitamin D deficiency rates. Their policy makes absolutely no sense whatsoever.

The AAP is even worse. Not only are they not raising the vitamin D requirements for infants – the compliance rates are five to 20 percent in early infancy with regard to taking vitamin D on breast milk, which is the highest-risk patients in general – but they're [also] telling parents not to put their child in the sun. Wear a hat. Wear long sleeves. They're really anti-vitamin D policy. It's not even...

JM: They're certainly anti-sun. The AAP is the American Academy of Pediatrics.

DA: Right.

JM: They seem to have some influence from the dermatology community, because that's their long-held position.

It's a battle. It's a challenge. But the truth ultimately will come out. The real sad aspect of this is that there are many innocent people who will suffer, and innocent children and infants who suffered needlessly, because of lack of information.

My message to anyone listening to this is that you can significantly make a difference. You can really change someone's life with this information, because of the factors that Dr. Ayoub just mentioned. The professional organizations are reluctant to have this widely disseminated as the standard of care. Until that happens, we're going to need people who understand the truth to spread this message to save this needless tragic pain and suffering.

I applaud you for your efforts, Dr. Ayoub. I really appreciate what you're doing and for providing us with some really foundational studies that can help make this transition occur in a more rapid basis.

DA: Thank you, Dr. Mercola, for what you're doing and for giving a platform for researchers like myself and others to tell the general population what they know.

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JM: Yes. It's a real privilege to be able to do that. This is one of the exact types of information that we seek to spread – really helpful and basically almost free to implement. Essentially, vitamin D is one of those. If you take the oral one, it's one of the least expensive supplements you can possibly purchase. If you just get it from the sun, there's no cost. It's a simple process. It just needs to be implemented.

Again, thanks for all that you do and will continue to do. I mean, you've just been doing this for six years. You've got a long career ahead of you. I really appreciate that.

DA: Thank you. [It's] my pleasure.

[END]