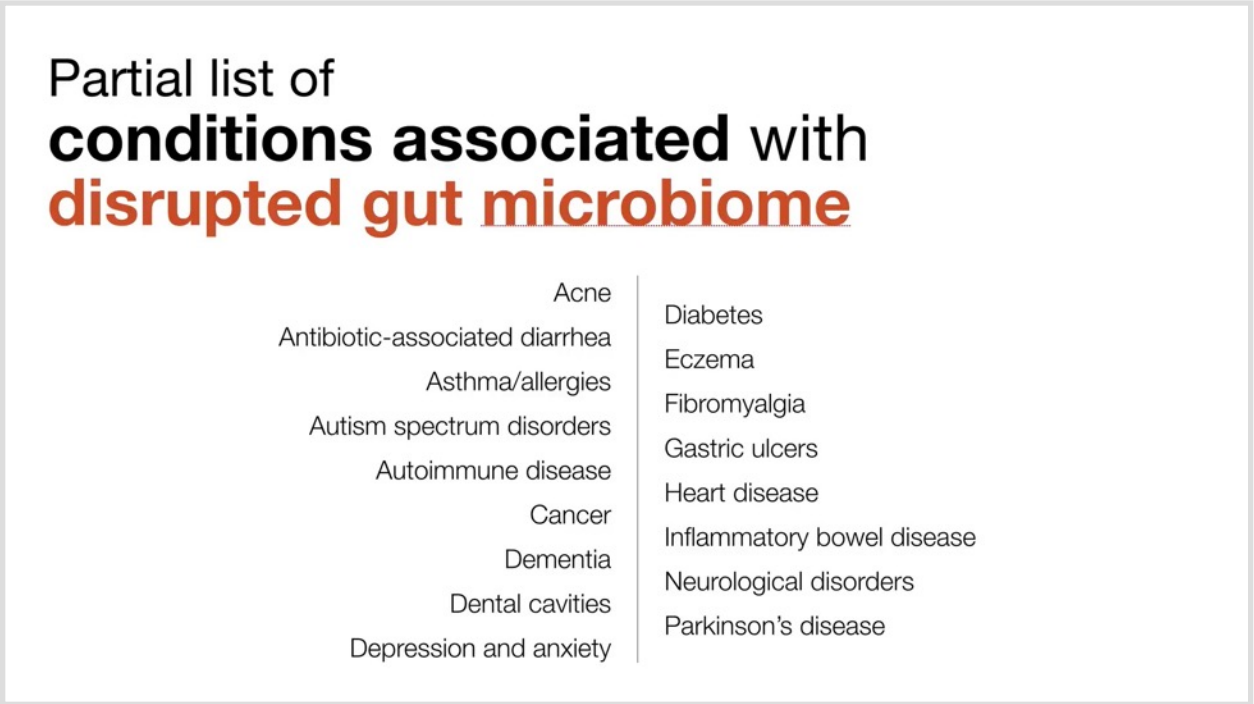
Microbiome & Intestinal Barrier - Part One

Hey, everyone, in this section we're going to talk about the microbiome and the intestinal barrier. I'm going to give you, again, a high-level review here. There's a ton written about this, several great books that are oriented towards both clinicians and the lay public, and I'll make some recommendations in the supplemental materials for this week. I write and speak about these topics of course regularly. It's a rapidly evolving area so please follow my blog and podcast for updates if you're not already, and feel free to ask questions during the Q&A. Again, the focus here is on what you need to know to be an effective practitioner, there's a ton of really interesting info out there, but a lot of it is not yet clinically actionable, and we're really just barely scratching the surface in terms of what we know about this, so whatever I say now is probably going to change within three to six months, not to mention a year or two from now, so we're going to keep it pretty focused here, and I will continue to provide relevant updates as they occur and as I think they're necessary.

The gut contains over a hundred trillion microorganisms from a thousand different species. That means we have ten times more microbes in the human body than human cells, and microbes have a hundred times more genes than we have in the human genome, so it's not inaccurate to say that at a cellular level we're more microbe than we are human. In fact, Justin Sonnenberg, a microbiologist at Stanford, has said humans can be regarded as elaborate vessels evolved to permit survival and propagation of microorganisms, so that certainly casts a different light on humans and what we're doing here.

Gut microbes are absolutely crucial to health; they promote normal GI function, protect against infection, regulate metabolism, and are home to a majority of the immune cells in our body, which explains why changes to the gut microbiota can result in diseases ranging from Hashimoto's hyperthyroidism to Parkinson's to type one diabetes.



As I'm sure you know, unless you've been living in a cave for the past several years, altered gut microbiome is now implicated in just about every chronic inflammatory disease. This is just a partial list here on the slide. New ones are added on an almost monthly basis, it seems. I have research alerts set up for microbiome and topics related to that, and I get emails several times a week with new studies published establishing these connections. Having said that, causation is not always clear, and is probably bidirectional in many cases, so for example, we know that there is a link between the gut and the brain, and we know that problems in the gut can, like inflammation, et cetera, can lead to a higher risk of problems associated with the brain, like dementia or autism spectrum disorders or neurological conditions, depression and anxiety, Alzheimer's, Parkinson's, et cetera, but we also know that problems in the brain, including all the ones that I just mentioned, can lead to dysfunction in the gut, because a large percentage of the output of the brain goes into the pontomedullary area, which in turn goes into the vagus nerve, which enervates the entire digestive tract, so there are bidirectional relationships in a lot of these cases, and causality is not always cut and dried.

It's really important for you to understand these connections and be able to communicate them with your patients, because they are the basis to some degree to a large degree for the gut testing that we do, and as you'll see, I advocate this gut testing even in patients without gut symptoms. So if a patient walks into your office and their main complaint is acne and high cholesterol, you need to have enough familiarity with these concepts to explain the potential connection between gut dysfunction and their skin problems and their cholesterol, in order for them to feel comfortable moving forward with ordering gut testing even though they didn't come to see you for gut complaints, and I've found that that's relatively easy. A lot of patients are already familiar with those connections and it doesn't require a lot to convince them that it's a good idea, but still, the more conversant you are with these connections, the easier it will be for you.

Until very recently, we believed—scientists believed, that is—that the gut was sterile. When a developing baby is in utero and colonization of the gut begins, as the baby starts the passage through the birth canal, but new research actually suggests that bacteria may cross the placenta and begin to colonize the fetal gut. Even if that's true, which it does look like it is, the majority of colonization still happens when the infant passes through the birth canal and swallows his or her mother's native bacteria, and this of course explains why the method of delivery strongly influences how microbiota initially develop in the infant gut. Vaginal birth, a birth through the birth canal, is the bacteria that a baby will be exposed to in that form of birth is incredibly different from the bacteria that the baby will be exposed to if it is born via Caesarian. In that case, the baby's first exposure is to bacteria in the hospital, whereas in a vaginal birth, as I said, the first exposure is swallowing the mother's native bacteria. Research has shown that the location of the initial exposure strongly affects the composition of the infant's gut, not only for the first few months of the infant's life, but even for several years after and perhaps permanently. And this explains why children that are born via C-section are at greater risk for asthma, obesity, type one diabetes, and a number of other conditions.

Of course, the infant's diet and then the younger child's diet, to some extent the mother's while she's breastfeeding, also affect the composition of the gut flora. Babies that are exclusively formula-fed are known to have significant differences in gut microbiota compared to fully or partially breastfed babies, and this is important because pioneer bacteria, which are the first bacteria to colonize the infant gut, have been shown to alter gene expression to create a more favorable environment for themselves and a less favorable environment for later bacteria, so if the baby doesn't receive these really important early pioneer species, not only is their initial imprint going to be different, but the way their gut is colonized later is different, because these pioneer species are not there to create that favorable environment.