Editor’s Note: On November 4, 2017 the Spondylitis Association of America hosted a free Spondylitis Educational Seminar in San Diego, CA, and livestreamed the presentations for all who couldn’t attend in person. Our speakers for the day were Rheumatologist John Reveille, MD and Physical Therapist Angelo Papachristos, MBA, BSc (PT.) We are including highlights here from the highly informative Q&A session with Dr. Reveille.

The recording of Dr. Reveille’s and Mr. Papachristos’ presentations are available on SAA’s website, at spondylitis.org/Seminars-and-Webinars.

Q: I’ve been through several TNF blockers, and they all seem to work initially, but over time they become less and less effective. Is that true of all of them? What’s your experience?

Dr. Reveille: The short answer is it does happen. It might be the fact that when you get on TNF blockers for a while you form antibodies to it.

We’re actually about to do a study looking at that. Specifically, a study out of The Netherlands is looking at people who are taking golimumab (Simponi) and looking for antibodies to see if there’s an associated lack of effectiveness. We know in rheumatoid arthritis there is, but the data in spinal arthritis is less clear on whether the presence of those antibodies affects how well the drug is working.

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Q: You have mentioned that everybody should get DEXA scanning (bone density scanning.) Could you speak more to that?

Dr. Reveille: Yes. They need to have you screened for osteoporosis. 40% of patients with AS have osteoporosis, and if you don’t treat it you have an even more brittle spine, and higher likelihood of fracture. Data from UCSF (University of San Francisco) has shown us that cervical spine fractures in a patient with AS have a very high operative mortality rate. I mean hospital mortality. It’s a big problem and we recommend screening.
Q: What is the difference between DISH and AS with fusion? The characteristics sound so similar.

Dr. Reveille: DISH has not been well studied genetically. DISH, that’s diffuse idiopathic skeletal hyperostosis, is a variant of osteoarthritis. People with DISH develop these big, fat osteophytes as opposed to syndesmophytes. It’s more common in people who are overweight, it’s associated with metabolic syndrome, and comes on in middle age. The difference is the SI joints are not affected (in DISH.) You may see a bone spur on the SI joints from the osteoarthritis, but you don’t see fusion.

You can have both (AS and DISH.) In some of our AS patients who are getting into their 50s and 60s - I see both. I have some very interesting patients who are B27 positive, have elevated CRP, fused SI joints, and their spine is all DISH. They have both diseases. DISH has not been well studied genetically. I wouldn’t be surprised if some of the bone formation genes like PTGER4 would be shared, but it’s not an inflammatory process as far as we can tell.

Q: What are your thoughts on long-term use of biologics, especially for younger patients?

Dr. Reveille: The data shows that the longer you’re on an anti-TNF agent, the less likely you are to have radiographic progression. I think you’re sort of stuck with it. Now, the new guidelines coming out from the European Union are suggesting for the first time that for a person who has been in sustained remission - now they didn’t find how long they sustained remission - but they recommended actually cutting the dose, if not discontinuing the drug. So I think as we get more information we’ll have better data in that regard. But as for right now, the standard recommendation is to continue the biologic; and as for long term use - as far as toxicity and the like - the long term data aren’t showing any more toxicity than acutely.

Q: Could you speak about bio-similar effectiveness, how they compare to some of the originals, and if you treated anybody with a bio-similar?

Dr. Reveille: Well, they haven’t gotten to Texas yet. When they come, I’ll use them. Now why is it called a bio-similar and not a generic? Because these are molecules made in biological systems. So they may chemically look like the drug they’re trying to simulate, but there could be differences in how the sugar residues and all that stuff are on them because they’re being made in a biological system. It’s like a fingerprint. It’s going to be a little different.
I’m not aware of any data that suggests they’re any worse. There are several adalimumab biosimilars out there. An infliximab one I think was just approved. They haven’t gotten to my patients yet because they’re in that immediate post-approval phase. So I have no personal experience with them, but I wouldn’t have problems with trying them. I think what’s going to end up happening is that the insurance companies demand that we use them because they’re cheaper. I think what you may see is a reduction in price of the ones out there. Certain drugs like golimumab, to an extent etanercept - believe it or not even though it’s the first one out it’s going to be one of the last to go off patent - and certolizumab have some time ahead still, but already adalimumab and infliximab are going bio-similar.

“Ankylosing spondylitis is not an autoimmune disease.”

Q: Do you see ankylosing spondylitis associated with vitiligo?

Dr. Reveille: Good question, and the answer is no - and I’ll tell you why. Vitiligo is an autoimmune disease. Ankylosing spondylitis is not an autoimmune disease. The genetic ... Remember I showed the slides with all those genes? If I were to put up lupus, and rheumatoid arthritis, and type-1 diabetes, and Sjogren’s syndrome, and autoimmune thyroid disease up there - we’d have the circles overlapping with each other, but it’d be completely different genes. It’s a different category.

What we think is going on here is basically the person who has ankylosing spondylitis, or psoriasis or psoriatic arthritis or inflammatory bowel disease, they have a hole in their immune system. That hole results from the interaction of HLA and ERAP; so when these bugs that are always coming across the intestinal wall, and trying to gain entry into your body hit up against the protective barrier - that barrier just doesn’t work very well in those people.... What we have then is a situation - it’s a little bit of an over simplification - but we start off with the body’s inability to handle certain immune challenges, and then add in genes that cause it to overreact to those challenges. Whereas in autoimmune disease you actually break tolerance to yourself, and your body is attacking your own tissues. That’s not psoriasis, that’s not AS, that’s not inflammatory bowel disease. These are different categories of disease.

There’s another group of diseases called autoinflammatory diseases. They start very early in life and there the genes just turn on and start causing inflammation. So there are three groups of disease: Autoinflammatory, autoimmune, and basically immune mediated – where the body reacts to something that you’re not dealing effectively with. I hope that was clear. Most people don’t know that.

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Q: You’d mentioned that spondylitis is not autoimmune. Would you expand a bit on that?

Dr. Reveille: Autoimmune diseases are characterized by associations with a different set of HLA molecules called HLADRRDQ – that’s number one. Number two - they’re characterized by the formation of antibodies to specific tissues in your body. Examples are - Rheumatoid factor, which is antibodies to immunoglobulin or CCP antibodies, citrullinated peptides or various components inside the cells. These are actually antibodies to components of your own tissues. We don’t see this in diseases like ulcerative colitis, Crohn’s disease, ankylosing spondylitis, psoriatic arthritis, reactive arthritis. You might see antibodies to the bugs, but we don’t see antibodies to one’s own tissues. There’s not any evidence that tolerance to yourself has been broken. That’s the cardinal thing. We don’t see auto-antibodies.

And the genetic networks, all the list of genes - if I were to have shown a slide, and I actually have another talk where I do show the slide, where we show the genes associated with rheumatoid arthritis, and lupus, and autoimmune thyroid disease.. It’s a different ... They’re different genetic networks. Okay, there are some overlap genes. Those have to do with immune responsiveness, but by and large they’re different genetic networks. We’re really coming at these diseases from a totally different point of view. SpA is not autoimmune. It is immune mediated. There’s a difference.