For my very first Spondylitis Plus column, I’d like to start with a heartfelt Thank You to each and every one of you who helped to make the Spondylitis Association of America the driving force behind advancements and innovation in the field of spondylitis.

In my new role as CEO, I have been continually awed by the success of the organization as a whole; the top level ratings from all of the Charity Watchdog groups; the dedication of our volunteer Board of Directors; the superior credentials of our Medical & Scientific Advisory Board; the passion and engagement of our brand ambassadors and advocates; and most of all, the unending and seemingly inexhaustible commitment of our members and other financial supporters like you.

You are the lifeblood of this organization and SAA’s achievements are your achievements.

I also want to thank the strong and determined leaders who blazed the trail I now find myself upon. Jane Bruckel, who, newly-diagnosed, found herself in a world in which no resources existed for those affected by spondylitis, and said, “No more.” Her remarkable vision changed the landscape of spondylitis in the U.S. and continues to offer hope and deliver better outcomes to millions of people.

And Laurie Savage, who elevated and expanded the programs and services that SAA provides and grew SAA’s reputation both nationally and internationally. Laurie will continue to be actively involved in the association’s research agenda and I look forward to working closely with her to build upon the important work already done.

It is truly a privilege to join such an esteemed group of motivated people and I’m geared up to both follow in their footsteps, and make some of my own.

2018 marks SAA’s 35th Anniversary and I believe that with the dedication and commitment of all of the amazing people in the spondylitis community, we can make it the best year ever.

35 Years of Research, Education, and Advocacy.

#SAA35

With much gratitude,

Cassie
Sharing good news - I had my appointment with a new primary care doctor today. I came prepared with several brochures from SAA just in case he was uninformed or misinformed about spondylitis. It turns out he is fresh out of medical school, and he knows that ankylosing spondylitis is not just a man’s disease, and that people who are HLA-B27 negative can have it! Hooray! Awareness is growing, thanks to SAA.”

~ Kalyn Gabriel
Deming, WA

I was diagnosed in 1986 when I was only 18 years old. The SAA was the only place I could turn to for information about this disease. Happy 35th Anniversary SAA! Thank you for your support, your educational materials, your research, and for your staff who have worked SO hard for SO many others! We all love you!”

~ Cristina Aukstkalnis Morgan
Via Facebook

“I can’t imagine having to be on this journey without the information and support that SAA has provided. Happy anniversary and thank you so much for fighting for us!

~ Ginga Cobb MacLaughlin
Via Facebook

“As a beneficiary of your education and patient forums, I am very grateful for your existence and efforts.”

~ Ruth Olson
Via Facebook

“I really hope you guys at SAA will consider selling those new spondylitis awareness t-shirts you were giving away on your Facebook page. I want one! I think they would sell very well.”

~ Sara Shaw
Pittsburgh, PA

Editor’s Note: Due to popular demand, we did indeed add the new “Spondy What?” awareness t-shirts to our online store. You can find them at spondylitis.org/Shop/Product/Official-SAA-T-Shirt.

Readers Forum Continued on page 4

LETTERS TO THE EDITOR
We want to hear from you! Send your thoughts, questions, opinions, and rebuttals.

Please send letters to:
Elin@spondylitis.org
Letters to the Editor/SAA
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Van Nuys, CA 91406

Please note that we reserve the right to edit for space and clarity.
“My name is Susan Lorenzana and I am a lifelong Ventura County resident. I’d been battling a chronic inflammatory illness for five years that was diagnosed as reactive arthritis at early onset. In June, I received an official diagnosis of ankylosing spondylitis (AS.)

While looking over the latest Spondylitis Plus magazine issue, I realized that I am not alone in this fight. I think it is about time that I share my story and begin the healing process.

In September of 2012, I fell desperately ill with widespread chronic inflammatory symptoms that shut my body down within a month of onset. These symptoms came out of nowhere and weren’t pretty. I experienced progressive chronic overall body pain, joint pain, along with tissue bruising around the joints/tendons, making it nearly impossible to move freely. And yet, test after test returned no answers. (Even though I am HLA-B27 positive.)

With years of uncertainty and battling depression, I retreated. In June of 2017, I finally received official diagnoses of ankylosing spondylitis, fibromyalgia, and peripheral neuropathy. I started a new biologic, Cosentyx, by monthly injection, which seems to be helping the pain, but it’s too early to tell if it is helping with inflammation. Holistic medicine has also been extremely beneficial over the years.

In March of 2016, Debbie Fox at Fox Fine Jewelry in Ventura had approached me about doing an art show the following year. I had no works to display at the time but I had established a goal and purpose to keep moving forward. In the years leading up to this wonderful opportunity, I could never have imagined this turn of events.

My art glass ended up being the best medicine as it has pulled me out of the dark into the light. I have now fully embraced and accepted my illness. My current work reflects this and speaks the language of healing on many levels. I have so much love and respect for all that this life has to offer, and will do everything in my power to inspire.

For many years I asked myself, “Why me?” Today, I can say with confidence, “Why not me?” My body may have severe limitations, but my mind is wide open for the first time in my life!

My father, John W. Durkin, was diagnosed with multiple sclerosis (MS) in 1976. He participated in the first ever clinical trial at UCLA Medical Center for MS. Dad passed in 1992, but I will never forget his strength, courage, and the example he passed onto others. I feel that I need to set an example as well, and do everything possible to help others by becoming an SAA member and sharing my story.

I would also love to be of assistance to the Spondylitis Association by donating custom art glass to raise money or donating a percentage of generated profits from my upcoming shows.

I will continue working on new pieces as my overall health allows in the coming months. I have been blessed with some fairly good days while finishing up my latest piece for my upcoming show named “Over The Moon,” which will showcase romance inspired pieces. It’s a must see!

This has been quite a journey in rediscovering myself, and I have much I am grateful for. My husband Ross and I just celebrated our 22nd wedding anniversary in July. Ross continues to be my rock! I thank God for my family and friends and for their continued support. I face the unknown with optimism, and that is a wonderful feeling.”

~ Susan Lorenzana
Ventura, CA

Editor’s Note: We connected with Susan and Debbie in November, and a successful fundraiser for SAA was created. You can see pictures of some of Susan’s beautiful art glass on page 26-27 of this issue. Big thanks to Susan, Debbie, and Amy Lynn!
Ankylosing spondylitis (AS) remains an enigmatic disease which many research groups such as my own are still trying to decipher. Current treatments for AS can be extremely expensive, have undesirable side effects, or work poorly in several patients. There is therefore a pressing need for more scientific research to work out the causes of AS and find new therapeutic strategies that could be used to develop better treatments, or ideally a cure, for this debilitating disease.

"The microbiome refers to the trillions of bacteria which normally colonize the human body and mainly reside in our intestines. In fact there are just as many microbial cells in our body as human cells, so in some respects we are only 50% human!"

Our studies focus on a novel area of AS research known as the microbiome. The microbiome refers to the trillions of bacteria which normally colonize the human body and mainly reside in our intestines. In fact there are just as many...
microbial cells in our body as human cells, so in some respects we are only 50% human!
This previously understudied part of our bodies remarkably has been linked to a huge number of inflammatory diseases such as rheumatoid arthritis, inflammatory bowel disease (IBD), multiple sclerosis (MS), cardiovascular disease, several cancers and even diseases such as autism and depression.

The reason the microbiome has been linked to so many diseases is partly due to a paradigm shift in our understanding of our immune system, the critical part of human physiology that helps to fight foreign infection or destroy harmful human cells in our body such as cancer cells. This is also what we try to boost when we get ourselves or our loved ones lifesaving vaccinations. Looking at text books published only five years ago, the main components of our immune system were considered to be organs such as the thymus or the spleen, our lymph nodes, or our bone marrow. While these are certainly key components of our immune system, arguably the most important part of our immune system is our intestine.

This raises the question, why are we now teaching medical students that the gut is such an important part of immunity?? Firstly, the intestine is home to more white blood cells (the major immune cells in our body) than any tissue in our body. Secondly, our gut is exposed to more foreign material than any other site in the body, for instance the bacteria we are exposed to all around us, such as those in our food, living on our pets, or residing within soil. This also includes food itself, which can at times be recognized by the immune system as ‘foreign’ (the major job of the immune system.) Hence our enormous intestinal immune system is constantly faced with the difficult task of either fighting off infection or switching itself off so as to not launch inflammatory responses to those harmless bacteria or food we ingest on a daily basis. Thirdly, it is now thought that immune cells in the gut are rather like salmon, geese or buffalo. That is to say they are highly migratory. This means immune cells that develop or are activated in the gut can travel to other tissues in our body, which could include the spine or the joints of spondyloarthritis patients. It is therefore possible that they may contribute to inflammation at these sites.

It is important to stress that the overwhelming majority of microbes that normally colonize the body are harmless and even play several roles critical to our health and well-being. These include digestion of many dietary components, provision of vitamins, and helping our immune system develop normally. It is thought however that in AS patients this healthy population of bacteria becomes imbalanced with the loss of benign or harmless bacteria and an expansion of pathogenic or disease-causing bacteria. This perturbed state in the microbiome is known in scientific jargon as ‘dysbiosis’. Further to this dysbiotic state, it is also known that the majority of AS patients exhibit some form of bowel inflammation. Therefore many roads lead to the gut in AS.

“ It is now thought that immune cells in the gut are rather like salmon, geese or buffalo. That is to say they are highly migratory.”
A current problem is that we do not fully understand which bacteria are most beneficial to our health, and which bacteria might be the culprits in triggering the inflammatory responses that drive AS. Nor do we yet understand why dysbiosis occurs in AS patients in the first place. These are all questions that are novel and are currently under investigation in my laboratory, with support from the SAA. To do this we are currently collecting stool swabs and/or blood samples from volunteers with AS, as well as their healthy relatives. In fact, we have recently placed an advertisement recruiting for this study on the SAA website (another example of how SAA supports our research!) Findings from this study will enable us to identify which bacteria in the gut are the most ‘immunogenic’ - or in other words inflammatory - and may be driving disease in AS patients. They will also allow us to determine whether there are more migratory immune cells in AS patients by looking for their presence in blood. Another possibility we are examining is that bacteria (or bacterial components, such as their cell wall) may be getting into the blood of AS patients from the gut and potentially contributing to AS symptoms. This is currently a very busy time for the lab, and we are still welcoming new recruits to our studies (including yourself!)

Editor’s Note: See the “Participate in Research” box for more on this study.

One particularly exciting prospect is that unlike our own cells, our microbiome can be altered by several lifestyle changes such as diet, prebiotics, or probiotics. We may therefore be able to restore the imbalanced microbiome of AS patients and prevent the out-of-control inflammatory responses that are caused by their immune system. These approaches also include microbiome transplantation in which healthy donor feces are transplanted to a sick recipient to try to resolve the disease. All in all, together with many dedicated collaborators, colleagues, and volunteers, we are identifying a huge number of new potential treatments based on our microbiome, for which we are incredibly indebted for the support of the Spondylitis Association of America.

www.stopas.org
Spondylitis Plus: You came on board in September of last year. What has it been like so far? What have been some highlights for you?

Cassie: It’s been an exciting and busy first five months! Even before my first day on the job, I was invited to attend the Spondyloarthritis Unmet Needs Conference. It was a fantastic two-day conference held on the grounds of the National Institutes of Health (NIH) in Bethesda, MD, and the third conference of this type sponsored by SAA. (You can read more about it starting on page 13 of this issue.) The conference brought together thought-leaders from around the world, in a variety of specialties, to set the agenda for the next decade’s direction in spondyloarthritis research. It was a baptism by fire, but a wonderful window on the big work to come!

Other highlights include attending my first American College of Rheumatology (ACR) conference in November, and the patient seminar we put on that same week where I had the pleasure of meeting some of our members. Getting to know some of the attendees and hearing their personal stories was very motivating.

Another exciting opportunity - we were approached by ACR to partner on the 2015 AxSpA Treatment Guidelines update, and I am thrilled to have us play a major role in this project. Two members of our spondylitis community were selected and asked to participate as voting members on the panel - representing the patient’s voice and perspective. This is a great example of how SAA uses your generous contributions to further our shared mission. Through this collaboration with ACR, our collective voices can be heard and are even more powerful.

Spondylitis Plus: Our readers may be interested to know a bit about your background. We’ve heard you jokingly say in the office, “I’ve worked my way around the body.”

Cassie: Yes, my career has been devoted to nonprofit management for almost 30 years. I actually started in the late ‘80s as a volunteer on the “Dance For Heart” committee for the American Heart Association. I enjoyed it so much I joined their staff and was there for almost 18 years. It was really a great experience. I was hired as a Field Director in the San Francisco Bay Area, and gained invaluable experience in community organizing, fundraising, programs and mission awareness, marketing/communications, and advocacy - all from a grassroots level. I left Heart as the Chief Operating Officer of the Western States Affiliate. From there I accepted an executive role with the American Lung Association of CA for a couple of years, until my husband, after retiring from the U.S. Navy, was offered a job on the east coast. So away we went to Maryland. Rounding out my resume, I found myself as the President and CEO of the National Kidney Foundation of Maryland - another fantastic experience. After almost five years, my husband was transferred back to California and I joined the American Diabetes Association, first as the Executive Director for the Los Angeles office. After about a year and a half I was asked to take on a new role as Regional Vice President. One of the things SAA has in common with ADA and NKF is the passion and commitment the communities have for the cause. That’s one of the main reasons I was so drawn to SAA; learning how engaged the spondylitis community is, and how closely they (you) are involved in SAA’s mission, made me feel connected to the organization before I even started.
Most people don’t know that I lived in Japan for almost four years. My husband was stationed there while in the Navy and I worked as a government contractor, running the Navy’s Level I Health and Fitness program.

So yes, I really have been working my way through the body starting with the heart (and brain), then the lungs, kidneys, pancreas, and now the spine and joints.

*Spondylitis Plus: What might people be surprised to learn about you?*

**Cassie:** Most people don’t know that I lived in Japan for almost four years. My husband was stationed there while in the Navy and I worked as a government contractor, running the Navy’s Level I Health and Fitness program. While there I climbed Mt. Fuji twice, and played on a Japanese women’s volleyball team. It was so much fun! To this day I miss it.

I love gardening, although I don’t get to do it as much as I’d like with my chronic back and neck pain. I enjoy hiking with my husband (as long as there are no snakes, I’m a total ophidiophobic.) I also love swimming, cycling, traveling, and gourmet cooking. And - this usually makes people’s eyes pop - I used to be a body builder and actually competed in several shows. Yes, really! I have the pictures and a couple of trophies to prove it (and it was 25 years ago.)

*Spondylitis Plus: What is your vision for the next five years for SAA? Where do you hope to see the organization go?*

**Cassie:** Of course research will remain a key priority, and I’d like to see us widen the field of researchers focusing on SpA. We will continue pushing for earlier diagnosis to shorten the diagnostic delay.

I’d like to see us heighten our focus on advocacy - take up initiatives on affordable prescription medications, access to quality and affordable healthcare, and advocating on the hill for increased funding of arthritis research.

We have plans for enhancing some of our existing programs for our membership and I look forward to rolling those out.

Lastly, SAA has earned quite a national and international footprint already, and I would like to continue that legacy by expanding that footprint even further. It’s crucial that we remain the go-to source for spondyloarthritis information, for community support; that we remain easily accessible, and that we continue to increase our reach and awareness of this disease and find new and more effective treatments. If all of this happens to the level I know is possible - and it will only happen with the ongoing commitment and support that our members and donors have demonstrated over the past 35 years - perhaps at our 40th or 50th anniversary the general public won’t say “Spondy What?” but will know what spondylitis is. Now there’s a goal!
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.

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.
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 is the difference between DISH and AS with fusion? The characteristics sound so similar.

Dr. Reveille: The data shows that the longer you’re on an anti-TNF agent, the less likely you are to have radiographic progression.

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.

I hope that was clear. Most people don’t know that.

“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.

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.

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.
Over the past decade, the field of spondyloarthritis (SpA) has seen significant progress—from a greater understanding of the genetic and biologic underpinnings of these diseases to novel treatments that improve the lives of patients. Despite this progress, gaps in our knowledge about them remain and serve as a continued focus of research efforts across the United States and throughout the world.

For two days in September of 2017, the Spondylitis Association of America brought together an international group of leading researchers to the National Institutes of Health (NIH) in Bethesda, Maryland, to identify pressing gaps, share insights, discuss scientific and treatment issues, and provide direction for the next decade of spondyloarthritis research. The Spondylitis Association of America and the National Institute of Arthritis, Musculoskeletal and Skin Diseases (NIAMS) “Spondyloarthritis 2017: Unmet Needs Conference III” generated interdisciplinary collaborations among researchers and clinicians, and opened new areas of investigation to expand the breadth and depth of spondyloarthritis research. The 2017 gathering builds on SAA’s inaugural Unmet Needs conference held in 1998, and Unmet Needs II in 2006.

“The success of previous scientific exchanges, together with substantial developments in science and technology, suggested it was time for Unmet Needs III,” said Planning Committee Chair Robert Colbert, MD, PhD, chief of the NIAMS Intramural Research Program’s Pediatric Translational Research Branch, member of SAA’s Medical and Scientific Advisory Board, and a pediatric rheumatologist. “This conference lays the groundwork for the next several years of research and investigation into spondyloarthritis.”

A conference committee—comprised of former SAA Executive Director Laurie Savage; Ellen Gravallese, MD, of the University of Massachusetts Medical School; Maripat Corr, MD, of the University of California, San Diego School of Medicine; Michael Ward, MD, of the NIAMS Clinical Trials and Outcomes Branch; Michael Weisman, MD, of Cedars-Sinai Medical Center; and Dr. Colbert—planned a two-day scientific conference designed to generate new research ideas and fertilize new collaborations.

“Our goal,” said Savage, who is now an SAA research consultant, “was to gather leading experts in their fields to not only discuss the progress we’ve made to date but to examine the road ahead in terms of research on and treatment of these conditions.”

The conference brought together a stellar group of presenters to discuss a wide range of topics—from disease genetics and pain management to metabolism, immunology, and imaging.
More than 100 scientists, physicians, and thought-leaders in SpA and related fields attended Unmet Needs III, sharing innovative approaches and cutting edge research. A main goal of the conference was to expand horizons and learn as much as possible from research conducted in fields complementary to spondyloarthritis, which, in some cases, is ahead of SpA research.

**Day One**

After a brief introduction by Drs. Colbert and Gravallese, the first group of panelists examined the genetics, epigenetics, and functional genomics of rheumatic and inflammatory diseases. They covered a range of topics, including the genetics of human inflammatory diseases and the links many of these diseases have to SpA; the genotype (genetic) and phenotype (physical characteristics) of inflammatory bowel disease, a significant complication of SpA; and the epigenetics—the environmental factors that change the way genes are switched on or off without altering the DNA sequence—of rheumatic diseases.

The panelists included Daniel Kastner, MD, PhD, of the NIH’s National Human Genome Research Institute; Dermot McGovern, MBBS, DPhil, of Cedars-Sinai Medical Center; Soumya Raychaudhuri, MD, PhD, of Brigham and Women’s Hospital; and James Lee, MD, PhD, of Harvard University and the University of Cambridge.

Among the unmet needs identified by the group were defining new therapies for IBD based on genetic variants in the disease, identifying targetable genetic mutations that drive gene regulation and disease susceptibility, and characterizing cell states in which disease alleles (one of two or more forms of a gene that arise by mutation and are found at the same place on a chromosome) affect gene regulation.

A second group of panelists delved into a range of subjects concerning the generation, perception, and management of pain during the afternoon session. These scientists and clinicians discussed the mechanisms of pain, including inflammation at joint sites and pain governed by the spinal cord and brain; the pathogenic (caused by disease) and clinical features of pain; the use of neuroimaging to regulate pain; and epigenetic factors that influence chronic pain.

In this session, panelists included Stephen McMahon, FMedSci, FSB, PhD, of King’s College, London; Yvonne Lee, MD, of Brigham and Women’s Hospital; Daniel Clauw, MD, of the University of Michigan School of Medicine; and Kasey Hemington, PhD, of the University of Toronto.

The panelists cited a number of unmet immediate needs, including identifying mechanisms that link inflammation and pain, both centrally and peripherally; understanding the effects of TNF-alpha (a molecule released by the body that plays a critical role in controlling inflammatory and immune responses) on the brain; and treating chronic pain as a disease state in and of itself, rather than as a symptom of other diseases.

**Day Two**

The second day of the conference began with a morning session on microbes, metabolism, and host immunity, followed by an afternoon session that focused on various aspects of imaging as it relates to spondyloarthritis.

In the first session, the presenters discussed how metabolism regulates bone maintenance and resorption and how the immune system is affected by interactions between the host (a human) and various microbes. A portion of the session focused on the microbiome—the micro-organisms in our body, especially our gut—and its relationship to diseases such as spondyloarthritis.
as well as a new treatment that employs fecal implants to target the microbiome in disease.

Panelists here included Lionel Ivashkiv, MD, of the Hospital for Special Surgery; Yasmine Belkaid, PhD, of the National Institute of Allergy and Infectious Diseases; Ramnik Xavier, MD, PhD, of Massachusetts General Hospital; Dirk Elewaaut, MD, PhD, of Ghent University Hospital; and Colleen Kelly, MD, of Brown University’s Alpert School of Medicine.

The group identified a number of unmet needs, including manipulating the gut microbiota as an effective treatment for spondyloarthritis, understanding how inflammation impacts metabolic pathways, and determining the link between gut and joint inflammation in the pathogenesis of SpA.

During the final session, the panelists examined a wide range of issues regarding imaging of spondyloarthritis and related diseases. These talks included an overview of SpA imaging, quantitative imaging of the spine, molecular imaging of musculoskeletal pain and inflammation, and the use of imaging to quantify syndesmophyte (a bony growth originating inside a ligament and a characteristic component of spine pathology in SpA) growth and development.

The presenters in the final session were Walter Maksymowych, MD, of the University of Alberta; Sharmila Majumdar, PhD, of the University of California, San Francisco School of Medicine; Sandip Biswal, MD, of Stanford University Medical Center; and Michael Ward, MD, of NIAMS.

These panelists identified standardized imaging evaluation, validation of biomarker technologies using multiplexing, or combination, imaging platforms, and using anatomy to inform pathogenesis as unmet needs in spondyloarthritis imaging.

“Going forward,” said Dr. Colbert, “our hope is to build on the insights shared by our distinguished panelists to begin to address the unmet needs in spondyloarthritis imaging.

“Editor’s Note: SAA will be submitting a manuscript outlining the highlights of the Unmet Needs III Conference for publication in a peer reviewed medical journal. We will keep you updated in these pages.

Photos by Barbara Alper Photography
35 years ago, a small group of AS patients changed the course of spondyloarthritis, and jump-started its research in the U.S. This is SAA’s “Your Story,” which begins with a personal need, and a woman named Jane Bruckel.

“You might think that as a Registered Nurse with access to doctors and medical information, I would have a quick path to diagnosis,” tells Jane Bruckel, BSN, RN to Spondylitis Plus. “You’d be wrong.”

Out of this sense of frustration and isolation, I finally asked my doctor if he would invite his AS patients to a support meeting. Simultaneously, another AS patient asked his doctor to do the same. And so, about 12 of us met for the first time in January of 1983, creating the country’s first-ever AS support group. We were all excited to finally meet. And women, too! We found that we’d all experienced this sense of frustration, anger, and isolation. We decided to meet monthly. As word got out, our numbers rapidly grew and with that growth the need to create an organization focusing on AS became clear. We selected a board of directors, called ourselves ‘The Ankylosing Spondylitis Association,’ and went into fast gear.”

What was going on in the Ankylosing Spondylitis landscape back in 1983?

“The short answer is nothing was going on. No publications for patients, no way to connect with other AS patients (no internet, no support groups, no organization), and the number of researchers and amount of funding going to AS research was quite small. There was very little interest among rheumatologists, and absolutely no public awareness.

Everything we did from there on was a first in the U.S. Six months after our first gathering, we launched a quarterly newsletter. It had to be put together the old way. Articles were typed, then cut and pasted into a storyboard, then sent off to the printer to finish. We held monthly meetings with guest speakers, such as rheumatologists, physical therapists, experts in medical and social security disability insurance, etc.

Then, a whole new world suddenly opened up to us. A physical therapist just back from a two-week AS course in Great Britain told us that AS organizations existed in countries throughout
And so, about 12 of us met for the first time in January of 1983, creating the country’s first-ever AS support group.

Europe! She introduced us to the British organization, NASS, which shared their patient publications with us, helping us create our own. Soon after, we were among the founding organizations of the Ankylosing Spondylitis International Federation, a worldwide organization that, among other things, is charged with helping AS organizations. Imagine, from not knowing anyone else, to having a worldwide AS connection!

As ASA grew, and took more and more of Jane’s time, Jane realized she needed to leave her nursing job to devote herself fully to the young organization’s mission. The Board authorized a salary, enabling Jane to become its first official employee. She learned to write grant proposals and began fundraising in earnest. She tells Spondylitis Plus about the first large grant she wrote - applying to the Dallas based Harold Simmons Foundation for $50,000 to go towards an AS awareness campaign. It was rejected. Not one to give up, Jane flew to Dallas to meet the foundation’s representatives and personally deliver the revised draft of the grant. It was accepted, and went a long way in helping raise the organization’s profile.

Shortly after, the first AS public service announcement was created - which featured Ed Asner, perhaps best known as the newsman, Lou Grant, from The Mary Tyler Moore Show. Various publications printed pieces about AS and the association, and the ASA was finally on the public radar. In fact, the publicity resulted in so many calls and letters that the young association had to hire its second official employee – an administrative assistance to handle the inquiries. The U.S. Spondylitis Community was forming, and ASA was at its epicenter.

The 80s also saw the publication of the first comprehensive book for AS patients “Straight Talk On Ankylosing Spondylitis,” the start of ASA’s Medical and Scientific Advisory Board, the first (and second) patient and physician scientific symposiums on AS, and ASA’s first steps into the research arena - which would shape the next big chapter for the organization.

The 90s and beyond – Research is King

While we can’t possibly fit into this piece the numerous research milestones accomplished, we will briefly touch on a few early items that were key to shaping SAA’s future.

In 1992, The Ankylosing Spondylitis Association, (ASA)
In 2003, the FDA was considering approving Enbrel as the first biologic drug indicated for AS. As an advocate for SAA, I was invited to testify. I brought with me numerous testimonials from our members who, like me, were already taking the drug off-label.

changed its name to the Spondylitis Association of America (SAA) to reflect a broadened mission encompassing the totality of spondyloarthritis - the family of related diseases.

In 1995, SAA’s Board of Directors changed the organization’s major focus to spearheading and funding research efforts into uncovering the causes of and cure for spondyloarthritis.

That same year, a leading gift of $30,000 from the Jean and E. Floyd Kvamme Foundation, and Damon Kvamme kicked off SAA’s fundraising for spondyloarthritis research. With mission and now funds aimed at research, Jane connected with the National Institutes of Health (NIH), meeting with NIAMS Director, Dr. Stephen Katz to discuss future collaborations and SAA’s commitment to research. A fruitful relationship was formed that has served SAA and the spondylitis community for decades since.

Just three years later, in 1998, SAA would fund Drs. Reveille and Jin to start the first major, nationwide AS genetic study. The year after that, the NIH would recognize the importance of this work, providing a $6.5 million grant for the study, as well as designating SAA as a clinical coordinating center. The AS Family Genetic Project had taken form, which would morph into the international Triple A Australo-Anglo-American Spondyloarthritis Consortium (TASC), and go on to eventually identify two additional genes in 2008 - ERAP 1 and IL23R - that play a role in susceptibility to spondyloarthritis. A huge breakthrough for spondyloarthritis research.

We end our story as we have begun it - with Jane, who shares one last memory.

“In 2003, the FDA was considering approving Enbrel as the first biologic drug indicated for AS. As an advocate for SAA, I was invited to testify. I brought with me numerous testimonials from our members who, like me, were already taking the drug off-label. They told heartfelt stories of how the drug gave them their lives back and many told of the hardship since their insurance wouldn’t pay for it. I came prepared to tell some of these stories and to tell my own story. Since I had never disclosed which biologic I was taking, I planned not to specify the drug I was on. The panel of experts listened to testimony from researchers and a young man with juvenile AS who had also been invited to speak. Between each speaker, I listened to the panelists discuss some of their thoughts. At one point, I found it hard to believe hearing them debate among themselves whether Enbrel should only be approved for early disease and only for men. I could hardly contain myself. So, when I got up to testify, I read from the letters as planned. But I went on to disclose to the panel that I also take Enbrel and that it has changed my life. I told how, after years of walking very gingerly, I could now run down the street, run up and down stairs (not recommended), and how I was so elated at the change that I even showed our friends at New Year’s Eve that I could jump rope! (Also not recommended). I then emphasized to the panel that “I’m a woman, and I’m NOT newly diagnosed!”

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Enbrel would soon be approved for all adults with active AS, and Jane would be quoted in the press release announcing the very thing she helped make possible. Though retired, Jane is never far from SAA (or its staff) and remains an invaluable resource. “Since my retirement 12 years ago, I am overwhelmed by the achievements and incredible impact this organization has continued to make under the leadership of Laurie Savage (who herself has recently retired). Way too many to mention! Laurie kept us moving forward at a fast pace. I believe this forward momentum has been possible because she and the Board of Directors stayed very focused on our mission. I am confident that our new CEO, Cassie Shafer, has the ‘know-how’ and enthusiasm to continue this fast-paced march toward finding a cure, and empowering those affected to live life to the fullest. I look forward to seeing all they accomplish in the next decade to bring us closer to our ultimate goal of ending this disease forever. And in the meantime, continue advocating on our behalf, and improving the quality of our lives.”
Last year when I was 52, the rheumatologist who had seen me since I was 22 retired. I was sad to lose someone who had been in my life that long, but off he went to enjoy retirement, and off I went to find a new doctor. That wasn’t easy, as mine was considered by many as the best in the DC metro area. I didn’t click with the first doctor I tried, but I liked the second. He was younger and had a practice by himself. I liked that he didn’t dwell on the past and instead wanted to know, rather flatly, why I was sitting in front of him. “I have ankylosing spondylitis,” I said.

“No, you don’t,” he said.

“Yes, I do,” I responded, “since I was 21.”

“You don’t look like someone who as AS,” he responded.

There was at least one more round of “no you don’t, yes I do.” And at last he said, “Fine. Get the X-rays and we’ll see what you have when you come back.”

I was irritated as I left, because by this point I am an expert in what I have and this guy had just met me. But by the time I got to my car I decided that it was a compliment—a testament to how hard I worked over the years. Three weeks later, I was sitting in the room waiting for him. He walked in and laughed. “You have ankylosing spondylitis.”

“I know,” I rolled my eyes, and laughed back. “I work hard.”

That day I decided to write this article—not for myself, and not even for others like me. I wanted to write it for the children and young adults whose parents are reading this magazine. I’ll explain why.

First, let me share some history. One day when I was 21, I was making my bed—something I still do every day so that if I accomplish nothing else, I at least did that. But by the time I got to my car I decided that it was a compliment—a testament to how hard I worked over the years. Three weeks later, I was sitting in the room waiting for him. He walked in and laughed. “You have ankylosing spondylitis.”

“I know,” I rolled my eyes, and laughed back. “I work hard.”
One day when I was 21, I was making my bed—something I still do every day so that if I accomplish nothing else, I at least did that. But this day there was a stab when I bent over. “Uh oh,” I thought. “Pulled muscle.”

scan showed something was wrong. One doctor thought I might have a bone infection and did a bone biopsy of my right sacroiliac (SI) joint. That was bad. After waiting months to be seen at Johns Hopkins in Baltimore, a doctor there looked at my X-rays, talked to me for five minutes, and told me I had rheumatoid arthritis. That was bad too. “Go home to Northern Virginia and find a good rheumatologist,” he had said as we sat there in the exam room. I had never heard of a rheumatologist before. It was an orthopedist who had done the bone biopsy.

Within an hour of meeting the rheumatologist who would be my doctor for so many years, and after he pored through X-rays, reports, and bloodwork, he knew what I had. It wasn’t rheumatoid arthritis, and he was horrified that someone had told me that and that someone did a bone biopsy. In some ways, I was classic. I’d had a urinary tract infection before the back pain started. I had had iritis a few times afterward. Mornings could be rough, but then I normally loosened up. I was HLA-B27 negative and I was female. Those last two weren’t quite as classic. As I left his office that day, I had hope that I would be one of those people who would see the disease go away. But that wasn’t to be.

I went through my 20s and 30s in a lot of pain—probably half the time. I’ve always thought it strange that people with extensive AS can have little pain, and those with mild AS could have terrible pain. Mine was typically considered moderate, and it could be terrible when it decided to act up. I would hop from NSAID to NSAID, as they would stop working, with a few other drugs mixed in. I could be perfectly normal on some days, and feel perfectly miserable on others. In many cases, it would strike around 1:00 in the morning. A stab would wake me, and even thinking about moving would send a searing hot knife into one of my SI joints.

I could be perfectly normal on some days, and feel perfectly miserable on others. In many cases, it would strike around 1:00 in the morning. A stab would wake me, and even thinking about moving would send a searing hot knife into one of my SI joints.

When I was around 41, I decided to use a biologic. The doctor had told me about them previously, but I hesitated because it seemed like a big step. I wish I hadn’t waited so long to start. First it was Enbrel; later it was Humira; and these days it’s Cimzia. Along the way I developed ulcerative colitis, a related disease, and the Cimzia treats that too. The day I took the first injection of Enbrel was the...
last day I ever had a serious AS flare. Twice a year I get a steroid injection into my left SI joint when it starts to act up, and that calms things down.

"Around the age of 25, I joined a gym. I was in a busy job (human resources) and frequently worked long hours. Between the hours and the AS flares, my workouts were hit or miss. But I did my best."

That's the history. But that’s not why I wrote this article, because everything I said so far is probably pretty common. The reason I wrote this article is the thread that runs through my story. It's the thread that parents and young people need to know about.

Around the age of 25, I joined a gym. I was in a busy job (human resources) and frequently worked long hours. Between the hours and the AS flares, my workouts were hit or miss. But I did my best. If I had minor pain, I went. If I had major pain, I stayed home and sat on a heating pad. At 27, my dad died and life spiraled out of control for a time. But we (my mom, my brother, and his kids) rolled on. I pushed on at the gym over the years. When I was around 43, my manager, an avid gym guy, commented on my erratic workout schedule and suggested I invest in a fitness trainer to help me get consistent. Now remember, I had started the biologics around 41 and was managing the pain fairly well. I decided it was a good investment. We started at twice a week, then I worked my way down to no trainer after about three months. It worked—from then on, I was consistent. And I was hooked. For me that just meant three hard workouts a week. By then I had a dog and was also taking two good walks a day with her.

I'm making that all sound so easy, but it wasn’t. It took work. Evenings were best back then. My mom was diagnosed with Alzheimer’s, and moved from California to Virginia to live near me. Life got grueling. There was independent living for her, and I was balancing work and caregiving. I worked out. We later moved my mom to an assisted living facility, which had me zipping back and forth between work, home, and her facility. I worked out. We had a thief there, and caught her on the nanny cam, which led to court dates. I kept working out. Soon after, my brother died suddenly of a heart attack at 48 and my world again spiraled downward. I ended up moving Mom back to her condo with the caregiving handled by a caregiving company, and we found some steadiness. I worked out. A month before my mom died, after over eight years of battling the disease, her caregiver snapped and assaulted her. Thankfully, she wasn’t seriously injured, but once again, it was on the nanny cam. After Mom’s death, I entered a four-year period of court proceedings, both county and federal, as I had inadvertently stumbled on more than an assault – it involved immigration and other kinds of fraud. I WORKED OUT. I would see different trainers here and there. Now on my own, without being a caregiver, I work out more consistently. And very recently, I walked away from the normal work world and started my own company, putting my schedule in my own hands. Now I start almost every day except Sundays at the gym. And I work out hard.

My point is that despite the pain and despite all that life could throw at me, I pushed myself to get to the gym.

Over all these years, I saw multiple doctors and multiple physical therapists who would tell me to do gentle exercises. I would smile and commit, and then go to the gym and work way harder than they said to. It was the fitness trainers at the gym who really kicked my butt and pushed me. Sometimes, they would have me do something that ended up hurting me and I’d pay for it in pain for a couple of weeks. But generally, I learned on my own what I could and could not do.

"My mom was diagnosed with Alzheimer’s, and moved from California to Virginia to live near me. Life got grueling. There was independent living for her, and I was balancing work and caregiving. I worked out."

In the end, I created a workout cobbled together from everything the trainers showed me that I could tolerate most of the time. I do cardio, weight lifting, and floor work. Last year I figured out one of the exercises I had done for a long time (an overhead seated press where you push weights straight up) was making my pain worse. Sitting in the machine one day, I thought about how it had
to be compressing my spine. I stopped, and my pain decreased soon after. I had been making it worse. But in general, I am much better overall for working out hard. I’m trim and have “mostly good” posture. Over those years I went from exercising grudgingly, to exercising because I knew I needed to, to exercising because I enjoyed it. And now I really do enjoy it. The camaraderie is nice, too.

I don’t know what the future holds. I have multiple autoimmune issues. The AS moved to my cervical spine (neck) over the years, and my head doesn’t turn quite like it used to. I participate in an AS study at the National Institutes of Health (NIH), and see a knowledgeable doctor there who gives me an added layer of advice. I’m great in the mornings, which is when I go to the gym. Most there have no idea I have a problem. But by the end of the day, it’s there. Twinges that remind me I can’t stand too long or walk too far. I don’t take chances on the ice, and I wear those shoe spikes from L.L. Bean if there’s any sign of it, because a fall could spell disaster anywhere along my spine. Last year, I ended up in the hospital in agony because of terrible pain where my upper ribs joined my spine. It wasn’t until I was home four days after I went in that I figured out by myself what was wrong. I popped ibuprofen pills that I wasn’t supposed to take anymore, and the pain went away immediately. Since my head wasn’t turning far enough, I was turning at my shoulders and twisting my spine, doing a number on my rib joints. I learned to turn at my hips instead.

That is the story I want to share with young people who might be able to change the progression of AS. I could worry about a lot of things, but I try not to. And, I work out.

"I’m great in the mornings, which is when I go to the gym. Most there have no idea I have a problem. But by the end of the day, it’s there. Twinges that remind me I can’t stand too long or walk too far."

Julie Nielsen is the managing principal of Oyster Organizational Development, LLC, based outside of Washington, DC. She wrote a book called Finding Grace (search “Finding Grace Nielsen” on Amazon) to share what she learned about caring for the elderly and especially those with Alzheimer’s. In her free time, she coaches people on how to thrive at work and she walks her dog Sadie. And, she works out...
This issue of Spondylitis Plus is made possible through the generous support of...

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Editor's Note: We kicked off our “Q&A With A Medical Professional” column in the Spring 2017 issue, with Dr. Gensler answering your rheumatology questions. We follow up here with our second installment, as there were many great questions we were unable to cover. Our thanks to Dr. Gensler for so generously giving of her time, and to our readers for the excellent questions.

Part 2 Of Our Q&A With A Rheumatologist, With Dr. Lianne Gensler

Have there been any clinical studies or anecdotal comments regarding the withdrawal effect (tiredness, susceptibility to illness, etc.) from eliminating a TNF inhibitor after long-term usage?

Few studies have looked at withdrawing a TNFi in a systematic manner. There is no known withdrawal effect and no dependency on the drug as we see in other medications (prednisone, pain medications, etc.), however, withdrawal of a TNFi can result in disease flare. Recently, the ‘ABILITY 3’ study examined stopping of adalimumab (Humira) in patients reaching remission after 28 weeks of treatment. In this study, stopping adalimumab was not associated with greater susceptibility to illness (though this was not a primary endpoint). Flare of disease was seen (as we might expect) and in the setting of flare, fatigue is quite common.

I have osteoporosis (spine BMD is -3.0), AS, with a completely fused spine, and I broke a vertebra and two arm bones when I fell this summer. My concerned rheumatologist wants me to switch from Boniva to Forteo, which purports to re-build bone, for the next two years (maximum allowed). He is hopeful that it will strengthen my spine. I am on a TNFi and have been for over a decade.

Here’s my question- When I researched Forteo, I discovered a medical article in which the authors (two MDs) state that because of the way Forteo builds bone, they will not prescribe it for their AS patients with osteoporosis. I can find no other literature that touches on this subject. My doctor wants me to begin this treatment NOW. Can you help me, via anecdotal information or research, to weigh the risks versus benefits in my situation?

I understand your rheumatologist’s concern when you sustained fractures and were already on a bisphosphonate (Boniva). It partly depends how traumatic the fall was mediating the fracture. A minor fall while walking on a flat surface for example would be more concerning than a fall down the stairs or off a ladder. Bisphophonates may not help prevent fracture with high impact trauma. In addition, we should consider what the other risk factors for fracture are in AS. Unlike the general population, where low bone density is the major player, in AS there is also risk because of the ankylosis of the spine making it more rigid and brittle. There is risk because the fall risk is greater and there is probably risk because the bone quality is worse. We have no evidence that an anabolic agent like teraparatide (forteo) will address these in the setting of a fused spine. There is no evidence that teraparatide will decrease your fracture risk (in the setting of AS) and I agree with the referenced authors’ concern, that theoretically this could make the AS worse by making your spine more rigid with further ankylosis. This in fact could theoretically further increase spinal fracture risk. Therefore in a patient with advanced AS and a fused or partially fused spine, my expert opinion only (without strong evidence) would be to avoid an anabolic agent.
Is it a common occurrence for a patient with AS to be plagued with tendonitis in the elbows and hips? Steroid injections help, but what can be done as a preventative measure?

AS patients can develop enthesitis, which is related to tendonitis but not exactly the same. Enthesitis is inflammation where the tendons and ligaments insert on bone. The primary location of the inflammation is the bone, not the tendon. The classic location is the heel bone with Achilles tendonitis. It can also affect the elbows and side of the hips at the bony insertion. In general, steroid injections around tendons are not recommended, as there is a risk of tendon rupture with this procedure. Treating the enthesitis would be similar to the treatment of AS. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are first line and then the biologics can be used and also work. Non-pharmacologic interventions like regular exercise, stretching, strengthening and maintaining a healthy weight may be important too.

Are there established standards for properly assessing disease progression? For example BASDI frequency, specific spine/mobility evaluations and their suggested frequency, imaging, blood work frequency?

There are a few ways to gauge whether the disease is under control. One way is to measure the disease activity. This is in fact recommended by the American College of Rheumatology/ Spondylitis Association of America /Spondyloarthritis Research and Treatment Network guidelines and can be performed by measuring the inflammation in the blood, typically by the C Reactive Protein, though the Erythrocyte Sedimentation Rate is also sometimes helpful(1). In addition, the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) is a composite patient questionnaire asking about pain, fatigue, and morning stiffness and is important to determine the patient-reported disease activity(1). Finally, the above measures can be combined to derive the Ankylosing Spondylitis Disease Activity Score (ASDAS). How often to measure these is dependent on the individual patient. If the patient has active disease, they should be measured as frequently as the patient is seen to try to achieve a lower state of disease activity. If the patient is stable and well controlled, a once a year measure may be adequate. I would not do it less frequently than that. An international taskforce published a recommendation last year on what the target should be(2). Disease progression can also be measured by performing the metrology measures in the clinical visit with the rheumatologist (and comparing it to prior results). Examples of these are the modified Schober test, the thoracic expansion score, and the occiput to wall measurement. Finally, imaging helps determine if there has been progression. X-rays can help to determine if there is damage, especially in the spine over time. They should not be performed more than every two years and should only look at the side views (lateral views) in follow up. MRI is also sometimes helpful to look for inflammation and structural changes not seen well on the X-rays(3).

References:
“Life’s most persistent and urgent question is, what are you doing for others?” — Dr. Martin Luther King, Jr.

It was my wise mother who told me when I turned 13 that it was my duty to volunteer and give back to my community. It’s also possible she simply wanted me out of the house! In either case, I inherited a tradition of volunteerism that shapes my life to this day. I immediately threw myself into an endless list of car washes, pancake breakfasts, senior citizen dances, floor hockey events, stair climbs, craft events, 5k runs, and magazine sales. I stuffed envelopes until my fingers went numb. I read to children dressed as the Easter bunny through a costume I couldn’t see out of. I suddenly found a sense of purpose that filled me with pride. I fell in love with being a volunteer. I learned that fundraising can change lives and every penny counts.

In my role at the Spondylitis Association of America as a Development & Annual Giving Manager, I never cease to be amazed by the passion, dedication and creativity of our volunteers. Did you know that SAA is a national organization with volunteer fundraisers that happen across the country? SAA has a robust volunteer program that has a rich history of innovation. Past volunteer events include ice fishing, internet radio programming, beard growing and/or shaving, art raffles and live bands. By creating your own personal fundraising event, you can help SAA with spondylitis awareness on a local level and bring new participants into our community. Which is incredibly important! Volunteer fundraising spreads awareness to communities far beyond the reach of our national office and major U.S. cities. I have heard many times from potential volunteers that they don’t know where to begin or aren’t sure if their event will be a success. People decide to give because they are moved by their relationship with you. They are touched by your story. They believe in you. I believe if a volunteer fundraiser touches one person and raises one dollar for your cause, that’s progress!

One of the many hats I wear is “volunteer cheerleader.” It may seem like it takes a perfect storm to make your volunteer fundraiser click. However, getting a few events under your belt to see what works and what doesn’t is the best way to make sure that “perfect storm” happens the next time you plan. A great way to begin to fundraise is directing donations in lieu of gifts to SAA. We would love to be the beneficiary for wedding favors, birthdays, graduations or any event where the desire of the honoree is to give back to the community. We want to be your charity of choice!

One of the many things I love about being a volunteer is the chance to meet people I never would know otherwise. What am I doing for others? What skills and talents do I have that cost me nothing, but to a nonprofit would mean the world? I remember a time when I was performing radio shows for seniors at a center in the Midwest. After the performance, the cast spent time with the audience in the theater. The first person I spoke to told me she was in her 90s. She remembered hearing the
People decide to give because they are moved by their relationship with you. They are touched by your story. They believe in you. I believe if a volunteer fundraiser touches one person and raises one dollar for your cause, that’s progress!

This is where you come in. This year SAA celebrates 35 years of supporting research, creating programs, and pursuing a cure for spondylitis. You have been there every step of the way. Your stories have given a face to spondylitis when there wasn’t one. You are part of an incredible culture of volunteerism and giving that is deeply engrained in the spondylitis community. I believe we can continue this tradition and create some amazing fundraisers this year. I want to hear your stories and make sure they are shared through volunteer fundraising events in a way that is meaningful to you. It is an incredible feeling to know your event helped provide funding for research, a speaker for a seminar, or resources for a support group. It feels good to do for others!

We need your creativity, energy and passion more than ever before. SAA receives no government funding. We rely on the financial support of the individuals we serve. With your generous support from volunteer fundraisers, we will continue to advance research into the causes and the cure, as well as provide the programs and services so many have come to rely upon. I am here to help you tell your story so your fundraiser will be a success. A volunteer fundraiser is a chance to have the conversation about spondylitis. It’s a chance to share with others how spondylitis has impacted the lives of friends and loved ones with a call to action. Help us in the quest to find a cure by holding a volunteer fundraiser in 2018. In the process, you may just discover something about yourself you didn’t know. Perhaps, like I did, you may find that in helping others you discover your own passion for service.

Please reach out to me for information or guidance on creating your volunteer fundraiser. You can reach me by email at sean.ewert@spondylitis.org or by phone at (818) 855-2106. I look forward to hearing from you!

“Tini Time” by Susan Lorenzana. Image used with permission from susanlorenzana.com.
The Spondylitis Monthly Automatic Rewards Team

S.M.A.R.T. is a safe, secure and convenient way to put more of your money to work advancing the spondylitis community's shared mission. Just specify a monthly amount and SAA will automatically deduct the contribution from your credit card. At the end of the year, we'll send you a summary of your giving and a tax receipt. Your dependable monthly gift of $100, $50, $25, $15 or even $10 will boost the impact of your SAA membership gift many times over.

To sign up for the S.M.A.R.T. Givers Program, go to www.StopAS.org/smart or contact Helene Hart at (818) 855-2109 or at hhart@spondylitis.org