I wish to extend my thanks to the ORA for providing me with this educational grant that has allowed me to attend the 2016 ACR. The conference, as you know, is massive with unlimited educational opportunities. It is impossible to attend every single session. It is vital to identify and attend a number of key talks.

Highlights of the 2016 ACR meeting in Washington included areas of advocacy, research, models of care and practice efficiency. Many of the topics incorporated more than ORA pillar. The opening lecture by Dr. Kvedar entitled "Harnessing the Internet" discussed the use of telehealth as an evolving part of healthcare. It allows patient's in distant areas to be treated with minimal disruption and still provide access to excellent care. He also emphasized the importance that the internet will play in the future of medicine and the need to embrace it into practice. The ARHP key note address focused on Health centered care rather than patient centered care. It provided an interesting perspective on how we do business.

I am very interested in learning about how health care practitioners can work together to provide an ideal model of care. The circle of care was a joint session presented by a Rheumatologist Dr. Bruce Hoffmann, the physiotherapist Carol Oatis and an Orthopaedic surgeon Dr. Ryan Hoffman. It discussed a patient's journey with unremitting rheumatoid arthritis and eventual total knee arthroplasty. It provided a good discussion about each health care professional's role on the patient's journey and the importance of team collaboration to maximize outcomes. The discussion revolved around improving patient access to the health care provider and how to maximize each team member's role in order to make enhance practice efficiency. It emphasized that the circle of care can improve efficiencies in practice as it enhances patient care and improves patient advocacy.

I found that the primary focus of ACR 2016 was on announcing the latest information/ research that is available to clinicians. The information was provided at both the basic science level which helps understand where rheumatology is heading as well as the clinical picture which helps in day to day care. Research is one of the four key pillars of the ORA.

This year's ACR saw a significant amount of the research geared towards basic science. In the area of research I felt that attendance at the year in review session helped to establish the highlights in 2016. Dr. Ingrid Lundberg discussed the year in respect to clinical rheumatology. She discussed the shift in research focus from the treatment of established disease to predicting disease. The year saw the increased use of ultrasound as a modality to detect inflammation

[Highlights of ACR 2016]

before there is evidence of erosion. Disease predictors including genetic predictors, environment triggers as well as the systemic symptoms.

Dr. Lundberg discussed:

- The use of ultrasound and MRI in individuals who were at risk for developing RA.
- She stated that ultrasound in anti-CCP positive individuals without clinical synovitis helped predict progression to arthritis.
- Subclinical MRI inflammation in individuals with clinical arthralgia helps predict disease development.
- She examined the role of autoantibodies and the induction of pain.
- ACPA induced osteoclastogenesis. The ACPA bind osteoclast precursor cells and directly promote the differentiation into bone-resorbing osteoclasts.
- IL8 blocking in ACPA induced mice may act as a mechanism to blockade bone destruction and decrease pain.
- She discussed the association of smoking and age with inflammatory joint signs among unaffected first degree relatives.
- Research in AS patients demonstrated that 33% of healthy first degree relatives had clinical and/or imaging signs of AS.
- The use of Tocilizumab for induction and maintenance of remittance in giant cell arteritis
- The use of Tocilizamab in PMR
- Gout: increased risk of cardiovascular disease
- Effect of smoking on AS axial spondylitis:
 - Decreased adherence to medications
 - Higher disease activity at baseline
 - Decreased response to medication

The second part of the year in review was the basic science review which was completed by Dr. Bruce Cronstein who discussed:

- The Mitochondria and ATP
- OMICS roles and reactions of activities of various molecules.
- In aging and OA there is Impaired chondrocyte with decreased ATP
- chondrocyte ATP depletion decrease mitochondrial reserve results in chondrocalcinosis and OA
- Dead chondrocytes versus angry chondrocytes

[Highlights of ACR 2016]

In another lecture I attended based on scientific research was the lecture series on OA novel treatment targets. There were three different scientists who discussed current research in OA.

Dr. Anne-Marie Malfait discussed:

- Pathways to pain, the most rapidly rising condition associated with disability
- Autophagy
- Wnt B pathways, growth factor, anabolic, innate immune systems, mediators
- Cytokines upregulated in the synovial fluid proalge
- Post traumatic OA the role of Anakinera
- Chemokine's CCR2 activators TLR activation TLR4 ligands
- Nerve growth factor release neuropeptin which inhibits pain but resulted in more rapid OA
- osteonecrosis antiNGF and NSAID
- The use of NGF inhibition and Tanezumeb targets nerve growth factor going into phase III trails

Dr. Louise Reynard discussed the epigenetic regulation of zinc transport and inflammation in OA. She discussed OA as a heterogeneous disease where multiple different pathways lead to the same outcome:

- Age, obesity, gender, genetics which is different in different joints
- She felt that OA of the hip could be divided into two distinct groups that did not behave the same.
- Cellular zinc homeostasis
- intracellular zinc
- IL1αinduces upregulation of ZIP8in chondrocytes from OA hip group 1 but not OA hip group 2

Dr. Frank Beier discussed PPAR mediated pathways as targets in OA:

- Adipoteines, cytokines, lipids, sugars and systemic metabolism
- ROS autophagy, cholesterol mitochondrial, and intracellular metabolism
- Metabolic syndrome and OA
- HTN
- Obesity common mediator in OA hyperglycemia
- Autophagy and OA
- Ample paths PPAR4
- Nuclear receptors ligand activated transcription factors DNA binding

[Highlights of ACR 2016]

The more clinical research talks that I was able to attend included Dr. Felson's evidence based review of current nonsurgical treatment of Osteoarthritis:

- He stated that IA steroids at low dose can improve symptoms from 1 to 4 weeks
- He stated that a higher dose (80 mg depomedrol) works better and lasts longer, based on the work by Neill et al 2015.
- He discussed the synovial tissue volume being the treatment target in knee OA.

Update on Ankylosing Spondylitis 2016 by Dr. Lianne Genser:

- She discussed the association of anterior uveitis and AS
- That disease activity predicts progression
- She highlighted the ACR guidelines and the new EULAR guidelines
- She discussed the role of NSAIDS as well as DMARD therapy
- The roles of Monoclonal antibody IL17 Secukinumab (Cosentyx) how it is a breakthrough for skin but does it make IBD worse.

Those are my highlights from the ACR 2016 scientific programme. Attending this conference provided me with an opportunity to expand my knowledge base and assist in the delivery of patient centered care. The less scientific opportunities at ACR include the chance to network with a number of healthcare providers from around the world. The daily poster sessions are overwhelming in the content and number of submissions. It is truly mind boggling.

I sincerely appreciate the opportunity to attend ACR 2016.

Thank you for supporting me in this manner.

Caroline Jones