The fourth meeting of the 2017/ 2018 session was held in Lecture Theatre 2 of GDH on Tuesday 16th January 2018 commencing at 7pm.

There were 44 members and guests in attendance. Apologies were received from 10 members. The minutes of the previous meeting, published online were approved.

The President, Professor Jeremy Bagg, welcomed the members and guests to the meeting.

The President introduced the speaker Dr Shauna Culshaw and invited her to give her address to the society entitled *“Periodontal disease, inflammation and general health”.*

Dr Culshaw started her lecture by thanking the President for asking her stating it was a privilege and slightly intimidating. She declared that she had no conflicts of interest. She then mentioned the National Institute for Health Research and invited the audience to complete the Oral and Dental PSP survey and give their opinion ([www.bit.ly/DentalResearch](http://www.bit.ly/DentalResearch))

Dr Culshaw then explained that she was going to talk about periodontal disease, inflammation and health. She described what happens in health and compared that to what happens in periodontal disease. She posed the question ‘Why look at Inflammation?’ Non-surgical treatment is effective in the management of periodontal disease. It works in some patients and in some sites. There may be 1-2mm recession but healthy and stable periodontal tissue. Dental plaque is necessary but not sufficient for periodontal destruction. She illustrated this by describing a study carried out by Loe et al in 1986 where they travelled to Sri Lanka over 30 years. The population they studied was isolated with very little access to dental care. They looked attachment loss over time and found there were three groups:-the first with hardly any attachment loss, the next with some attachment loss and the third with rapidly progressing attachment loss. Plaque and calculus didn’t change over time or between groups. 8% population were highly susceptible, 11% were strongly resistant and the remainder had moderate progression.

She postulated that the response is due to a deregulated immune response which targets bacteria but also targets the host, possibly an autoimmune component in serum and gingival tissue. She then described the murine model of periodontal disease in which mice are given antibiotics and then infected with Porphyromonas gingivalis. The bone loss around the mice’s teeth was then measured clinically and with micro CT. Infected mice had reduced bone levels and increased Antibody (Ab) levels. Antibodies (Abs) are associated with a self destructive immune response. If the same experiment is carried out in Ab deficient mice there are no Abs produced with some bone loss which doesn’t progress. From this it has been concluded that if the Abs are removed, bone destruction is removed. Dr Culshaw then went on to discuss other cell types involved in inflammation. Mast cells are present in the periodontium and activated in periodontal disease. Mast cell deficient mice have no bone loss; mice with normal mast cells have bone loss. She then discussed cytokines / soluble proteins. Interleukin 33(IL-33) is a cytokine which is released by mast cells following cell damage. It exacerbates alveolar bone loss. In the presence of infection the bone loss is greater. It doesn’t directly affect bone. Cytokines work in networks / pairs. Using the cytokine map it was suggested that RANKL (Receptor activator of nuclear factor kappa-Β ligand, a membrane protein which is a member of the tumour necrosis family), RANK (its receptor) and OPG (RANKL binds to [osteoprotegerin](https://en.wikipedia.org/wiki/Osteoprotegerin), a protein secreted mainly by osteoblasts which inhibits osteoclast formation by preventing binding of RANKL to RANK) might be involved. RANKL and OPG are balanced in health. IL33 up regulates RANKL and causes bone destruction. OPG stops this by reversibly binding to RANKL and preventing bone resorption. The drug denosumab mimics this interaction.

Dr Culshaw then discussed what happens in patients taking these drugs and their oral health. Inflammation is the key to periodontal destruction and it can be modified. When looking at periodontal disease and general health the evidence is confusing and some links are possible but not proved. She started by looking at periodontitis and rheumatoid arthritis (RA). RA is a chronic, inflammatory and systemic disease characterised by extensive synovitis resulting in bone destruction and loss of function so there are obvious similarities. It affects approximately 1% of the population (a relatively large number of young people in addition to old people). It is associated with an increased risk of infection, cardiovascular, GI, respiratory and malignancy problems. There is a 3-12 year reduction in lifespan and it costs the UK approximately £5Billion. With the current gold standard treatment approximately 1/3 patient achieve disease remission, approximately 1/3 patients achieve partial remission and just under ½ patients have no response. Dr Culshaw then examined the evidence of a link between periodontitis and RA. She first discussed whether or not it was biologically plausible. She concluded that it was – bacteria from ulcerated epithelium could lead to bacterial DNA in joints. Areas of inflammation are ‘sticky’ and those modified proteins could trigger an autoimmune response. C-reactive protein levels decrease after periodontal treatment. Anti – CCP (anti cyclic citrullinated peptide) autoantibody are elevated in patients with RA and samples taken before and after periodontal treatment show a reduction in the levels of anti CCP autoantibody. Its levels increase before arthritis clinically. Patients with periodontal disease have autoantibodies at low levels. These levels reduce if the periodontal disease is treated. If the level in smokers and non-smokers are examined patients who are non-smokers get a much more marked reduction.

The suggestion of systemic autoimmunity is present but slight. Dr Culshaw discussed whether or not patients with RA are more likely to have periodontal disease. The evidence suggests that if you have RA then there is more attachment loss around teeth. Plaque scores in the general population and in people with RA are similar so plaque scores don’t fully account for it. Eriksson et al looked at a possible link between RA and periodontal disease. They found that smoking and ageing are risk factors for periodontitis, both in RA and controls but they found no evidence of an increased prevalence of periodontitis in patients with established RA compared to healthy controls. She therefore concluded that there is not enough evidence to suggest that patients with RA are more likely to have periodontal disease.

Dr Culshaw then examined the evidence that patients with periodontal disease are more likely to have RA. In 2015 Chou et al looked at the Taiwanese National Health Insurance Research Database to investigate whether there was an association between periodontal disease and the risk of developing RA. They compared three groups- the first did not have periodontal disease and were used as controls, the second group were those that only received routine scale and polishing and the third group had periodontal treatment including surgery. They looked at how many of each group remained free of RA over time. The group with periodontal disease had a higher incidence of RA than the other two groups.

Dr Culshaw then discussed the evidence that RA is worse in RA patients with periodontitis. CRP levels are higher in patients with RA and periodontal disease than in patients with either RA or periodontal disease. She then asked ‘Is treating periodontal disease helpful to RA?’ A small number of studies have looked at this over the last 10 years. They looked at the effect of periodontal treatment on DAS 28 (Disease Activity Score which is a an arthritis scoring system which measures disease activity in RA in 28 joints, it looks at CRP / biological mediators, clinical signs in the 28 joints examined, patients pain scores and how patients think their RA is). When periodontal disease is treated the DAS 28 reduces in almost all studies. In one study there was an improvement at 3/12 which was not sustained at 6/12.

Dr Culshaw concluded that inflammation is important in periodontal disease. There are links with inflammation and systemic disease. Periodontal disease involves inflammation and its treatment allows patients to keep their teeth. Periodontal treatment decreases inflammation and might make some patients feel better too. She suggested that the reason there is not definitive data to show that treating periodontal disease is good for general health is because periodontal treatment doesn’t always work, it takes a long time, costs a lot and relapse is common. She then recounted an analogy of chronic disease e.g. lupus and ‘spoons (packet of energy) theory’ – there are a limited number of ‘spoons’ to expend in a day and she related how many ‘spoons’ it costs the patient to have periodontal treatment. She suggested that if we can find a way of treating patients without expending their energy it may help with other inflammatory diseases.

She acknowledged the people and companies involved in her work. She finished by asking the audience to complete the Oral and Dental PSP survey.

Dr Culshaw was happy to answer questions.

The Vote of Thanks was proposed by Dr Alun Scott who started by thanking the audience for turning out on an arctic evening. He then thanked the speaker for making something very complex sound very straightforward, she was very obviously in control of her subject. He then asked the audience to thank the speaker in the usual manner. The speaker was presented with an Odontological Society paperweight after the meeting.

Under AOCB the President asked Mr Iain Ogilvy, the winner of the Odontological Society Undergraduate Prize to come forward and receive his prize. Mr Ogilvy was unable to attend the meeting in December as he was on placement. His prize was £200 and was given for the best case presentation in fourth year. He had an opportunity to show the members his winning case presentation in GDEC at registration prior to the meeting proper.

He then asked that any member wishing to propose another member for Honorary Membership status to contact the Secretary with their nominee.

He informed the audience that booking tickets for the Dinner is now open and your ticket can be booked on the website. It is in the RCPS Glasgow on **Saturday 24/02/18.** The cost of a ticket is £65. Dress code: Black Tie. There is a reduced rate for overnight accommodation that evening and a proposal for a post Dinner Reception in the Dakota Hotel. Details are available on the website.

The next meeting is on **Tuesday 20th February 2018** in the **Lecture Theatre 2, GDH.** This is the ***Presidential Address*** and Professor Bagg will give his lecture entitled *“Murky meanderings in microbiology”.*