

**[P54] CHANGE IN FAT PAD COMPOSITION IN THE NEUROPATHIC DIABETIC FOOT AND IT'S ASSOCIATION WITH DYNAMIC PLANTAR FOOT PRESSURE**

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**Aim:** Diabetic neuropathy is associated with physiological and biomechanical abnormalities in the foot that increase risk for ulceration. These include micro-haemorrhage, neuropathy-induced atrophy, and non-enzymatic glycosylation of proteins in the cushioning fat-pads of the foot. The objective was to assess if the composition of fat pad tissue changes in the neuropathic diabetic foot and is associated with increased repetitive stress on the foot during walking.

**Method:** Fourteen patients with diabetic neuropathy (mean age 57.9 years) and five age-matched healthy controls underwent T1-weighted sagittal plane spin-echo MRI of the rearfoot at 1.5 Tesla. Two-point Dixon Chemical Shift Imaging was used to create fat-only and water-only images from which the fat signal fraction in a defined ROI of the sub-calcaneal fat pad was calculated. The barefoot plantar pressure distribution during walking was measured using an Emed pressure platform and peak pressure in the heel region was calculated.

**Results/Discussion:** Mean  $\pm$  SD fat signal fraction was significantly lower in patients ( $0.55 \pm 0.11$ , range 0.34-0.67) than in healthy controls ( $0.72 \pm 0.03$ , range 0.70-0.76,  $p < 0.005$ ), and was explained more by a lowering in fat signal ( $R^2$  0.87) than an increase in water signal ( $R^2$  0.32). Mean  $\pm$  SD peak dynamic pressure at the heel was  $391 \pm 119$  kPa for patients and  $325 \pm 53$  kPa for healthy controls (non-significant). Fat signal fraction and peak pressure were significantly inversely correlated ( $r = -0.59$ ,  $p < 0.01$ ).

**Conclusion:** Dixon MRI shows changes in sub-calcaneal fat pad composition in diabetic neuropathy, through a decrease in fat rather than an increase in water content. Both neuropathic and non-neuropathic factors may be attributed to this outcome, which include increased amounts of collagen as a result of non-enzymatic glycosylation or neuropathy-induced fat pad atrophy. As a result, fat pad function is compromised, as indicated by increased repetitive stress on the foot. These findings improve our understanding of the relationship between foot structure and function, and with that of the factors that underlie foot ulceration in diabetic neuropathy. Although plantar heel ulcers are not common, the associations found may act as a model for pathological changes that occur in the forefoot, where ulcers mostly occur.