**Focal adhesion components are altered in childhood nephrotic syndrome**

**Background-** Nephrotic syndrome (NS) is a common paediatric glomerular disease. It is characterised by podocyte effacement and can be categorised according to response to glucocorticoid (GC) therapy, where steroid resistant NS is associated with a worse prognosis. Mutations in an increasing number of genes expressed by podocytes have been shown to cause NS: including genes that encode focal adhesion (FA) proteins such as integrin receptors. To date there has not been a comprehensive investigation of FA components in human NS. We hypothesised that podocyte FA components would be differentially expressed in NS subtypes and this could form the basis of a biopsy biomarker panel. **Aims-** We investigated FA proteins in glomeruli in healthy human renal cortical tissue and compared with biopsy samples from patients with GC-sensitive, GC-dependent and GC-resistant NS. **Methods-** We performed immunofluorescence using previously characterised antibodies recognising active integrin isoforms, total integrin levels and downstream FA signalling molecules. The biopsy samples were formalin fixed and paraffin embedded. Visualisation was conducted using fluorescent microscopy and antigen expression levels were quantified using Fiji (Image J). We also performed laser dissection of normal human glomeruli followed by analysis with mass spectrometry-based proteomics. **Results-** The successful optimisation of 18 antibodies was achieved, and 4 were chosen for staining human NS biopsies. All patients with NS had decreased expression of total integrin β1. When grouped by response GC, the GC-sensitive and GC-dependent individuals had decreased total integrin β3 and increased vinculin expression. In contrast talin expression did not differ between the diseased and control samples. The proteomic analysis identified 10 adhesion complex components and 18 extracellular matrix components. **Conclusion**- In this investigation we highlight the potential role of FA components as biopsy biomarkers in human NS and furthermore we demonstrate an association between the integrin β1-talin-vinculin axis as and NS disease progression. Finally laser capture coupled with mass spectrometry-based proteomics gave good coverage of FA and matrix components and this approach could be further developed for biomarker discovery in human NS.