**Introduction** The pathogenesis of Inflammatory Bowel Disease (IBD) is unclear which hinders effective targeted drug development. IBD and murine models of colitis are associated with the abnormal accumulation of activated dendritic cells (DCs) in the colonic epithelium. DCs play a critical role in promoting inflammatory responses and blockade of their activation prevents colitis development in mouse models. We now propose to address the mechanisms underlying the aberrant accumulation of DCs in the gut by focusing on microbial danger stimuli that drive activation and migration of DCs. **Methods** We analysed the expression of migration-associated markers on DCs from normal and colitic mice. Bone marrow derived DCs (BMDCs) from WT or Beta-2 integrin^−/− (ITGB2^−/−) mice were cultured in vitro and their migration and activation analysed in response to control (Phosphate buffered saline - PBS), bacterial lipopolysaccharide (LPS), live Escherichia coli (EC), and live Bacteroides fragilis (BF) in the presence and absence of the lamina propria extracellular matrix component fibronectin. **Results** During colitis there was a marked increase in a population of CD103^+ (α integrin) DCs. We were able to mirror these populations in vitro. DCs moved via random motion and their velocity after stimulation with LPS and EC, in the absence of fibronectin, was significantly decreased. In comparison, stimulation with BF significantly increased DC velocity (p < 0.001 for all). In the presence of fibronectin, there was no change in DC velocity. The track displacement length (the distance between the start and finishing point of a given cell migration track) was significantly decreased after EC stimulation and significantly increased after BF stimulation (p < 0.001 for both). Unstimulated ITGB2^− BMDC velocity and track displacement length were significantly increased in comparison to that of unstimulated WT BMDC (p < 0.001 for both). This was more marked in the absence of fibronectin. **Conclusion** We have shown that in vitro WT DC cultures contain DCs with similar integrin-defined phenotypes to those found in colonic DCs in colitis. Differential bacterial stimulation causes opposing fibronectin-dependent effects on BMDC migratory behaviour whilst absence of ITGB2 significantly alters the migratory behaviour of BMDC. Our data implicates a complex relationship between specific components of gut microbiota, extracellular matrix, and migration and activation of DCs that could potentiate the aberrant accumulation of DCs in the colitic gut. If this complex relationship is further elucidated, it may be possible in the future to develop therapies that reduce colitis by controlling DC migration. **Disclosure of Interest** None Declared.