A new mechanism for bacterial uptake in mucosal tissues is proposed that is mediated by dendritic cells endowed with the ability to make intercellular adhesive links directly with epithelial cells thereby preserving the integrity of the gut barrier.
localised at the most apicolateral parts of the cells, someway from the basal parts of the cells.

Extrajunctional E-cadherin and desmosomal cadherins are also known to be expressed in the lateral membranes of epithelial cells,14 and thus may provide molecular handholds for initial points of interaction between the two cell types. In addition, formation and stabilisation of the tight junction is intimately related to the assembly of adherens junctions.15

DCs have been localised in vivo within the epithelium of normal rat intestine so they may be routinely anchored there below the tight junctions by other epithelial type cell-cell contacts awaiting a signal to interact with tight junctions, perhaps facilitated by upregulation and redistribution of occludin and claudin. In this context, it may be significant that in these experiments DCs could not interact from the apical side of the epithelium where they would not have access to other epithelial junctional proteins in cells with mature contacts.

Neutrophils can also migrate across epithelia, and occludin influences this process.16 When neutrophils interact with epithelium or endothelium they are capable of traversing from the apical side paracellularly as well as from the basolateral side,17 suggesting that they may interact with or influence tight junctions via different mechanisms.

Another notable aspect of this study is the observation that transepithelial resistance (TER) and the structure of tight junctions are not altered during interactions between DCs and epithelial cells. This absence of tight junction alteration and TER change has also been confirmed in studies of neutrophil migration.18 Interestingly, neutrophils have been shown to preferentially migrate across endothelial monolayers at tricellular points of cell contact where tight junction discontinuities are normally observed and this has been proposed as a reason why tight junctions need not be disrupted in this system.19 It is unknown whether this phenomenon exists in intestinal epithelia.

This paper therefore provides evidence for a novel and elegant method of bacterial sampling that may represent a very important component of mucosal surveillance, not least because the contribution of the non-Peyer’s patch gut epithelium greatly increases the surface available for detection and response to microorganisms. It will be of interest to know whether similar mechanisms exist in other mucosal sites such as the lung where DCs of similar type have been localised in vivo within the epithelium.18 It is unknown whether similar mechanisms exist in other mucosal sites such as the lung where DCs of similar type have been described and, in addition, whether DCs can be used as a mode of entry and spread by pathogenic microorganisms.

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