The tooth is the only structure in the body that breaches its integrity at the level of the oral mucosa. To palliate to this weak link in the body, the oral mucosa has adapted by developing a specialized structure called the junctional epithelium (JE) which forms an adhesive ‘collar’ around the tooth that essentially seals off periodontal tissues from the oral environment. Integrity of the JE is thus essential for maintaining a healthy periodontium and preventing the spread of bacterial infection. Disease sets in when the structure of the JE starts to fail. The JE is considered as an incompletely differentiated epithelium that produces components for tooth attachment instead of progressing along a keratinization pathway. It is a stratified squamous non-keratinizing epithelium whose cells derive from basal cells situated away from the tooth surface. The basal cells rest on a typical basal lamina (BL), which interfaces with dermal connective tissue. Suprabasal cells all have similar appearance, and quite remarkably maintain the ability to undergo cell division. The JE turns over quite rapidly, at least in some species. The most superficial cell layer of the JE provides the actual attachment of the gingiva to the tooth surface by means of a structural complex called the epithelial attachment. This complex consists of an atypical BL formed and maintained by the flattened superficial cells. This BL adheres to the mineralized tooth surface rather than connective tissue in a yet unknown manner and the JE cells attach to it by hemidesmosomes. Characterization of the structural components of the atypical BL of the JE has been particularly challenging. However, it is now widely accepted that it contains laminin-332 but not γ1 chain laminins, type IV and VII collagens, setting it apart functionally and compositionally from typical BLs binding to connective tissues. In this invited presentation, I will review the structure of the JE, present novel constituents of the BL that may mediate its adhesion to mineral, and discuss regeneration of the JE. In particular, I will focus on a protein called odontogenic ameloblast-associated (ODAM). This unique protein with no known homology is normally expressed only by the JE but is highly upregulated during neoplastic transformation of epithelial cells. We have thus hypothesized that ODAM is a matricellular protein that participates both in structuring of the atypical BL and in modulating the cell status of JE keratinocytes and transformed epithelial cells.

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Fig. 1: (A) Scanning electron micrograph of a section showing the various components of the junctional epithelium (JE). (B) Electron micrograph of a region similar to the boxed area. Immunoperoxidase (C) and immunogold (D) labeling illustrating the expression of ODAM by the JE and its localization among cells of the JE and in the basal lamina.