Developmental Biology ■ (■■■■) ■■■–■■■

Contents lists available at ScienceDirect

Developmental Biology

journal homepage: www.elsevier.com/locate/developmentalbiology

Cranial placodes: Models for exploring the multi-facets of cell adhesion in epithelial rearrangement, collective migration and neuronal movements

Marie Anne Breau [a,b,c,*], Sylvie Schneider-Maunoury [a,b,c]

[a] Sorbonne Universités, UPMC Univ Paris 06, IBPS-UMR7622, F-75005 Paris, France
[b] CNRS, UMR7622, Institut de Biologie Paris-Seine (IBPS) - Laboratoire de Biologie du Développement, F-75005 Paris, France
[c] INSERM, U1156, F-75005 Paris, France

ARTICLE INFO

Article history:
Received 2 September 2014
Received in revised form
8 December 2014
Accepted 9 December 2014

Keywords:
Placode
Morphogenesis
Adhesion
Collective migration
Sprouting
Neuronal migration
Epithelial morphogenesis

ABSTRACT

Key to morphogenesis is the orchestration of cell movements in the embryo, which requires fine-tuned adhesive interactions between cells and their close environment. The neural crest paradigm has provided important insights into how adhesion dynamics control epithelium-to-mesenchyme transition and mesenchymal cell migration. Much less is known about cranial placodes, patches of ectodermal cells that generate essential parts of vertebrate sensory organs and ganglia. In this review, we summarise the known functions of adhesion molecules in cranial placode morphogenesis, and discuss potential novel implications of adhesive interactions in this crucial developmental process. The great repertoire of placodal cell behaviours offers new avenues for exploring the multiple roles of adhesion complexes in epithelial remodelling, collective migration and neuronal movements.

Introduction

Adhesion molecules: Dynamic anchoring sites and signalling platforms

Cell adhesion is a major driving force in multicellular morphogenesis, and its misregulation leads to developmental defects and tumor invasion. Adhesion molecules are transmembrane glycoproteins which couple the microenvironment of cells with their internal mechanics and biology. Their extracellular domain binds to adhesive receptors of neighbouring cells (intercellular adhesion) or to components of the extracellular matrix (ECM) (cell/matrix adhesion), while cytoplasmic tails interact with cytoskeleton filaments and signalling proteins. Cadherins and integrins are the best characterised players in intercellular and cell/matrix adhesion, respectively. Cadherins engage in homophilic interactions at the level of specialised adhesive platforms, the adherens junctions. αβ integrin heterodimers bind to ECM components which form 3D meshworks with various geometries and physical properties in the cell environment.

Adhesion proteins exhibit a dual function. Their first recognised role is structural: they initiate and maintain tissue cohesion, and provide anchoring sites for pushing and pulling forces required for cell shape changes and morphogenetic movements. However, it is now clear that adhesion molecules play more diverse roles, which do not only depend on their sticky properties. This can be attributed to their ability to sense and transduce mechanical cues and crosstalk with signalling pathways that regulate cytoskeleton dynamics, proliferation, survival and differentiation (Hynes, 2002; Stepniak et al., 2009; Schwartz and DeSimone, 2008). Adhesive complexes can therefore be seen as dynamic anchoring sites and signalling platforms.

The unique morphogenetic properties of cranial placodes

Neural crest (NC) cells have long been used as an experimental paradigm to study the function of adhesion molecules in cell migration and morphogenesis (McKeown et al., 2013). Here, we focus on another embryonic cell population with unique morphogenetic properties, the placodal cells. Cranial placodes are discrete patches of ectodermal cells which give rise to crucial parts of sensory organs and ganglia in the vertebrate head, including the olfactory epithelium, the lens, the entire inner ear and cranial sensory ganglia, as well as mechanosensory lateral lines in aquatic vertebrates (Fig. 1A) (Streit, 2008; Schlosser, 2010).

* Corresponding author at: Group "Vertebrate Brain Morphogenesis", INSERM ERL U1156, IBPS-Developmental Biology Laboratory (LBD), CNRS UMR7622, Université Pierre et Marie Curie, 9 Quai Saint-Bernard, Bât C 7ème étage, case 24, 75252 Paris Cedex 05, France.
E-mail address: marie.breau@upmc.fr (M.A. Breau).

http://dx.doi.org/10.1016/j.ydbio.2014.12.012
0012-1606/

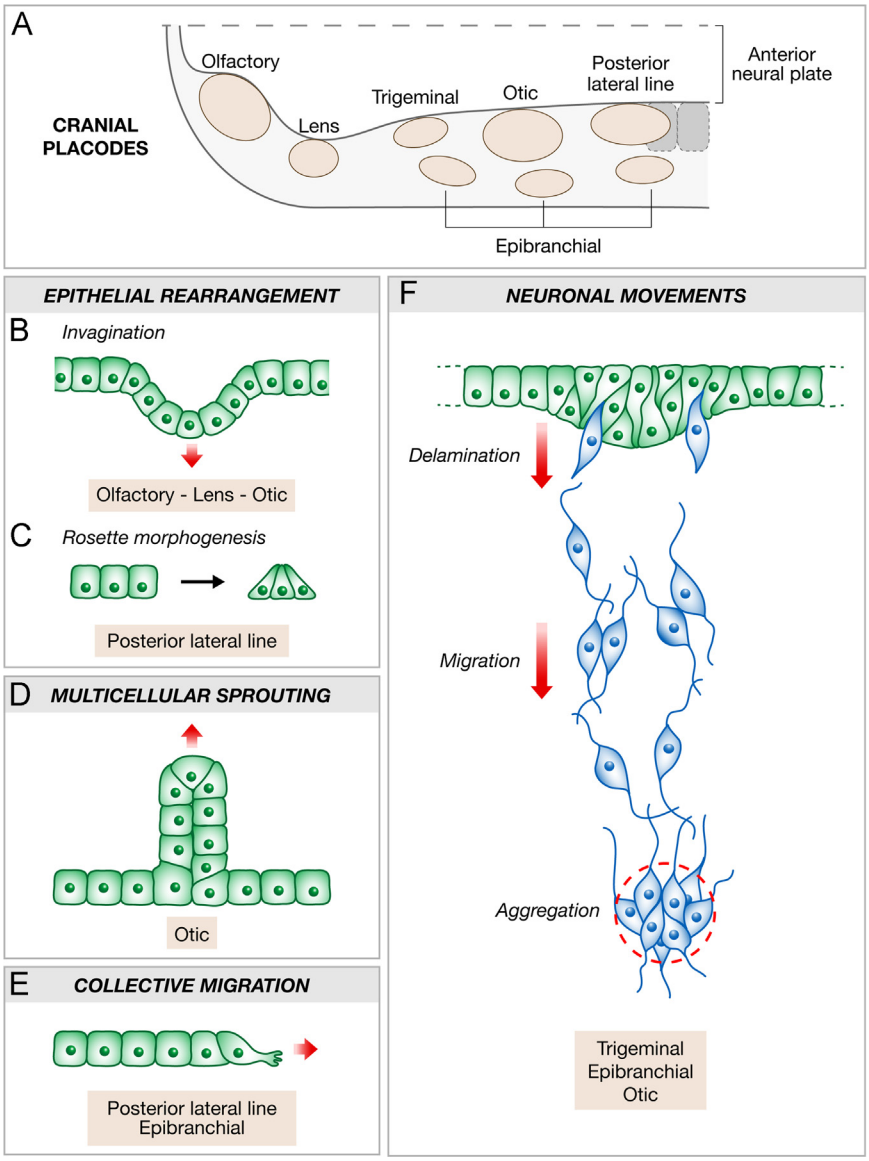

Fig. 1. The repertoire of placodal cell behaviours. (A) Dorsal view of half of a theoretical vertebrate embryo, anterior to the left (adapted from Breau and Schneider-Maunoury, 2014). Placodes whose morphogenesis are not discussed in the text are not represented. After the segregation of placodal precursors and their coalescence, cranial placodes appear as discrete patches of ectodermal cells that occupy specific positions along the anteroposterior axis in the head region of the embryo, next to the brain. From this stage, these placodal tissues can undergo epithelial rearrangement such as invagination (B) and formation of rosette-like structures (C), sprouting of multicellular strands (D) or migration as cohesive groups (D). Differentiating neurons produced by neurogenic placodes delaminate from the pseudostratified epithelium of the surface ectoderm, and migrate as streams of bipolar cells towards the site of sensory ganglia aggregation (F).

Progenitors of cranial placodes are initially partially intermixed within the crescent-shaped pan-placodal domain surrounding the anterior neural plate (Streit, 2008; Schlosser, 2010). As development proceeds, three steps of placode morphogenesis can be distinguished. First, intermingled placode progenitors segregate into adjacent placodal cell domains. Second, these apposed cell populations coalesce into compact and individualised placodes occupying specific positions along the anteroposterior axis, next to the brain (Fig. 1A) (Breau and Schneider-Maunoury, 2014). Third, subsequent morphogenetic processes take place, such as invagination, delamination and diverse types of migration, giving rise to the final pattern of placodal derivatives (Schlosser, 2010). In this review, we focus on the two last steps of cranial placode morphogenesis. The mechanisms driving the first step of placodal precursor segregation, including the hypothesis of an active sorting-out mediated by differential adhesion, have been recently discussed (Breau and Schneider-Maunoury, 2014) and will not be covered here.

Placodal cells share common features with NC cells: their origin at the neural plate border, and their capacity to leave the ectoderm and move through embryonic tissues to generate a variety of internal structures. However, there are also clear differences between NC cell and placode morphogenesis. After their detachment from the neural tube through epithelium-to-mesenchyme transition (EMT), NC cells migrate as isolated or loose groups of mesenchymal cells, and differentiate into neurons or other cell types only when they have reached their final destination (Blentic et al., 2011; Strobl-Mazzulla and Bronner, 2012; Theveneau and Mayor, 2012). In contrast, placodal cells can rearrange as epithelial cells (Fig. 1B and C), move or sprout as cohesive clusters (Fig. 1D and E) and migrate as streams of differentiating neurons (Fig. 1F). These are three unique morphogenetic properties of placodal cells compared to NC cells. Such behaviours must rely on dynamic adhesive interactions with surrounding cells and ECM components. Functional studies of adhesion proteins in placode ontogeny are still relatively scarce and often lack mechanistic

Please cite this article as: Breau, M.A., Schneider-Maunoury, S., Cranial placodes: Models for exploring the multi-facets of cell adhesion in epithelial rearrangement, collective migration and neuronal.... Dev. Biol. (2015), http://dx.doi.org/10.1016/j.ydbio.2014.12.012