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Specification, plasticity and evolutionary origin of peripheral glial cells

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Peripheral glia includes predominantly myelinating and non-myelinating Schwann cells in addition to satellite, terminal and enteric glia as well as other unresolved subtypes with localized functions. Of these subtypes, all of them originate from neural crest-derived embryonic Schwann cell precursors (SCPs). Specific gene regulatory networks control neural crest specification and downstream events, including SCP differentiation and myelination. Embryonic SCPs are multipotent and generate neuroendocrine cells, parasympathetic and enteric neurons, melanocytes and other cell types. The evolutionary origin of peripheral Schwann cell lineage is not widely discussed in the literature, despite numerous similarities between central and peripheral glia. Here, we review the major features of the Schwann cell lineage and proceed to an evolutionary discussion around possible relations between central and peripheral glial cells.

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Introduction

Peripheral glial cells dwell in different and specific locations of our body and perform a plethora of functions linked to myelination, neuronal support, regulation of synaptic connectivity and sensory function.

During embryonic development, peripheral glial progenitors originate from neural crest cells (NCCs) that assimilate on the surfaces of developing nerves and differentiate into Schwann cell precursors (SCPs). SCPs live in association with embryonic nerves and exhibit striking similarities to their maternal population, the multipotent neural crest (NC), since they generate melanocytes, autonomic neurons, peripheral glia and other cell types [1–3] (for an in-depth discussion please see [4]). During development, SCPs will transit into immature Schwann cells (SCs) that will later generate adult peripheral glial cell types (Figure 1).

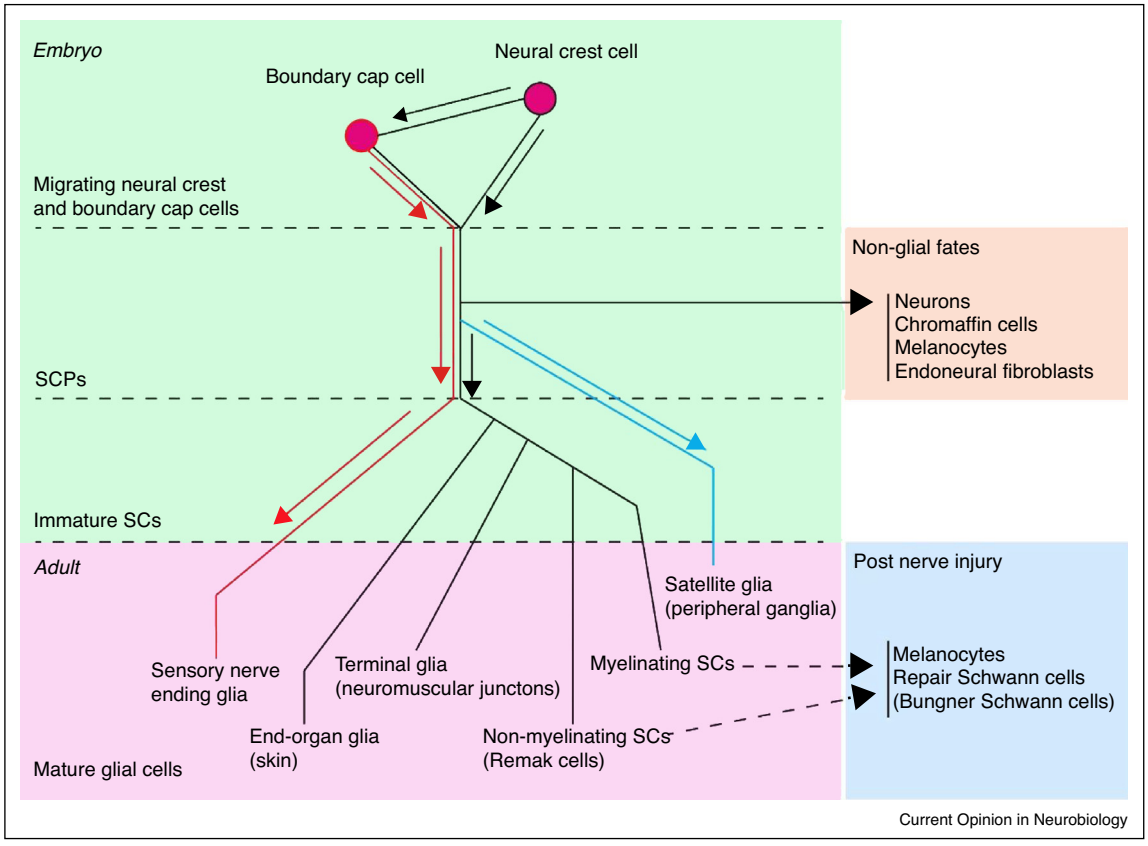
Adult myelinating and non-myelinating SCs surround the nerves and constitute the majority of peripheral glial cells. Satellite glia occupies peripheral ganglia and intermingles between the neurons (reviewed in [5]), while terminal glial cells shape, both functionally and structurally, the neuromuscular junctions (NMJs) [6]. Another type of specialized glial cell guides fine sensory fibres into the epidermal skin layer. These cells originate from the boundary cap (BC) population, migrate along the developing sensory nerves and inhabit the skin [7•]. Other specialized glial subtypes include glia found in the cutaneous sensory end-organs (Ruffini endings, Krause end bulbs, Meissner's and Pacinian corpuscles) [8,9] (Figure 1).

Despite extensive knowledge of the transcriptional mechanisms driving peripheral glial specification and myelination (for instance, role of *Egr2/Krox20* and *Zeb2*, see [10••,11]), the precise mechanisms dictating fate acquisition and the relative proportions of rare non-myelinating glial subtypes are not understood (Figure 2). Additionally, it remains unclear whether the emergence of different peripheral glia subtypes is a consequence of distinct molecular signatures or of the location within the developing body.

Development and signalling code of the Schwann cell lineage

SCPs are specified from the FOXD3+/SOX10+ migratory NC upon the formation of spinal roots, cranial nerves, sensory and autonomic neurons. FOXD3, a transcription factor (TF) found in migrating NCCs, promotes glial fate while biasing against the alternative neuronal and melanocytic fates [12–14]. SOX10, another key NC-specific TF, is necessary for the specification and later maturation of SCPs towards myelinating SCs [15,16]. SOX10 precedes neuronal differentiation and is downregulated during neurogenesis, while, in contrast to FOXD3, it is necessary for melanocytic differentiation and survival. Apart from these cell-intrinsic factors, the neuronal membrane-anchored growth factor NEUREGULIN 1 (NRG1) maintains the SCP fate and survival via extrinsic activation of ErbB2/3 receptors expressed by SCPs [17,18]. Interestingly, satellite glia cells in dorsal root ganglia are able to develop in Erbb2/3-mutants,

Figure 1



Overview of peripheral glia subtypes. SCPs: Schwann cell precursors; SCs: Schwann cells.

suggesting that they are specified independently of NRG1-signalling [19,20].

During the subsequent developmental phase, committed SCPs give rise to immature (or promyelinating) SCs (for the involved mechanisms see [21]). Among the molecules controlling the myelinating acquisition, KROX20 (EGR2) is a key TF controlling the maturation of SCs, as well as the initiation and maintenance of myelination [11,22]. At the same time, NOTCH signalling plays an important role in these processes [23]. The NRG1-ERBB signalling axis also controls the myelinating versus non-myelinating fate switch of SCPs and myelin sheath thickness [24,25]. Finally, immature SCs acquire myelinating capacity around birth and establish a 1:1 ratio with large calibre axons that will be myelinated, while sparing the small calibre axons — a process known as a radial axonal sorting (reviewed by [21]).

Another population of embryonic glia will associate with axons, remaining throughout adulthood as non-myelinating SCs (or Remak bundles). Typically, non-myelinating cells are found along nociceptive fibres and nerves extending from autonomic ganglia. These small calibre axons express much lower levels of NRG1, and, thus, SCPs associated with them do not receive the signals that would drive radial sorting and subsequent myelination [17,25]. However, the fate selection mechanisms of satellite glia, terminal glia of NMJs or sensory-nerve endings are currently unknown and require further investigation.

Similarity of PNS glial subtypes to CNS glia fuels evolutionary discussions

Oligodendrocytes (OLs) are the myelinating cells of the central nervous system (CNS) and in that sense they are very similar to Schwann cells — the myelinating cells of the peripheral nervous system (PNS) — including common features in their myelination gene expression program (i.e. Sox10, Egr2/Krox20, see Figure 2). Notably, the overall morphology of the myelin produced by OLs and SCs is similar, even though differences are observed on the structural level. For instance, both CNS and PNS myelin contain the proteins PLP, MBP and MAG, but are distinguishable by the presence of a unique set of proteins, such as CNP, MOG and O4 in the CNS and PMP22, and P0 in the PNS (Figure 2).

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