Stem Cell Reviews and Reports (2023) 19:358–367
https://doi.org/10.1007/s12015-022-10460-7

New Insights on the Role of Satellite Glial Cells

Junhou Lu[2,5] · Dongyan Wang[1] · Jianwei Xu[4] · Huanxiang Zhang[5] · Wenfeng Yu[3]

Accepted: 30 September 2022 / Published online: 15 October 2022

Abstract

Satellite glial cells (SGCs) that surround sensory neurons in the peripheral nervous system ganglia originate from neural crest cells. Although several studies have focused on SGCs, the origin and characteristics of SGCs are unknown, and their lineage remains unidentified. Traditionally, it has been considered that SGCs regulate the environment around neurons under pathological conditions, and perform functions of supporting, nourishing, and protecting neurons. However, recent studies demonstrated that SGCs may have the characteristics of stem cells. After nerve injury, SGCs up-regulate the expression of stem cell markers and can differentiate into functional sensory neurons. Moreover, SGCs express several markers of Schwann cell precursors and Schwann cells, such as CDH19, MPZ, PLP1, SOX10, ERBB3, and FABP7. Schwann cell precursors have also been proposed as a potential source of neurons in the peripheral nervous system. The similarity in function and markers suggests that SGCs may represent a subgroup of Schwann cell precursors. Herein, we discuss the roles and functions of SGCs, and the lineage relationship between SGCs and Schwann cell precursors. We also describe a new perspective on the roles and functions of SGCs.

Keywords Satellite glial cells · Dorsal root ganglia · Schwann cell precursors · Schwann cells · Stem cells

Introduction

Dorsal root ganglia (DRG) sensory neurons can transmit information about the location, type, and intensity of noxious stimuli to the cerebral cortex via the spinal cord, and play an important role in the pain pathway [1–3]. Sensory neurons are surrounded by a layer of satellite glial cells (SGCs) that support, nourish, and protect these neurons [4]. It has been considered that under pathological conditions, SGCs regulate the environment around neurons through gap junctions or by releasing neuronal activity modulating factors, such as adenosine 5'-triphosphate (ATP), nitric oxide, and tumor necrosis factor-alpha (TNF-α) [3, 5–7]. Recent studies have demonstrated that following nerve injury, SGCs around the DRG sensory neurons upregulate nestin and sex-determining region Y-box 2 (Sox2) expression, and differentiate into neurons [3, 8]. Interestingly, several markers specific for Schwann cell (SC) precursors or SCs were also found to be expressed in SGCs [9, 10]. This review aims to provide an overview of changes that occur in SGCs following nerve injury and the lineage relationship among SGCs, SC precursors, and SCs. Specifically, our review will help researchers focusing on the roles and functions of SGCs.

Junhou Lu and Dongyan Wang contributed equally.

✉ Huanxiang Zhang
hzhang@suda.edu.cn

✉ Wenfeng Yu
wenfengyu2013@126.com

1 Department of Physiology, School of Basic Medical Sciences, Guizhou Medical University, Guiyang 550025, China

2 Translational Medicine Research Center, Guizhou Medical University, Guiyang 550025, China

3 Key Laboratory of Endemic and Ethnic Diseases, Ministry of Education and Key Laboratory of Medical Molecular Biology of Guizhou Province, School of Basic Medical Science, Guizhou Medical University, Guiyang 550025, China

4 National Guizhou Joint Engineering Laboratory for Cell Engineering and Biomedicine Technique, Center for Tissue Engineering and Stem Cell Research, Guizhou Province Key Laboratory of Regenerative Medicine, Guizhou Medical University, Guiyang 550004, China

5 Department of Cell Biology, Medical College of Soochow University, Suzhou 215123, China

Identification of SGCs

SGCs are located in the ganglia of the peripheral nervous system, such as DRG and trigeminal ganglion (TG), and wrap completely around the sensory neurons. Each SGC is separated from its parent neuron by a gap of approximately 20 nm [11, 12]. In general, several SGCs can form an SGC sheath enveloping each sensory neuron, which can slow down the diffusion of most substances, particularly large molecules [11]. Studies have shown that SGCs are connected to each other through gap junctions and express a variety of ion channels, such as inwardly rectifying K^+ channels (Kir), voltage-dependent outward K^+ channels, and small-conductance Ca^{2+}-activated SK3 channels [13]. These properties of SGCs are thought to give these cells the ability to control over the neuronal environment.

Till date, SGCs have been identified mostly based on their location, morphology, and the expression of a combination of specific proteins, such as glutamine synthetase (GS), which is widely used to distinguish SGCs from other cell types in the DRG and TG both in vitro and in vivo [8, 11, 14–17]. Recently, using single-cell sequencing, Avraham et al. showed that GS mRNA is expressed in many cell types within the DRG [9]. However, GS protein is specifically expressed in SGCs [8, 9, 11, 18–22]. Moreover, several in vivo studies have also shown that only SGCs, but not SCs, express GS protein [11, 14, 18, 22, 23]. Accordingly, DRG and TG derived cells that express GS protein were identified as SGCs [20, 24–28].

The reason why SGCs express GS protein may be that they can convert glutamate into glutamine and participate in the rapid removal of glutamate from the perineuronal space by SGCs [13]. Studies have shown that SGCs express glutamate transporters, such as glutamate-aspartate transporter (GLAST) and glial glutamate transporter (GLT-1), which are responsible for the uptake of glutamate released by neurons into the synaptic cleft [11, 12, 29]. Under the action of GS, glutamate is converted into glutamine and released to the synaptic cleft. Then glutamine is returned to presynaptic terminals of neurons through glutamine transporters and converted back to glutamate to be reused [13]. Glutamate-glutamine cycle formed between neurons and SGCs can effectively prevent the cytotoxicity caused by a large accumulation of glutamate and realize the repeated recycling of glutamate.

S100 proteins are also expressed in SGCs; however, they are not considered as the ideal markers for SGCs because SCs also express these proteins [11]. Fatty acid binding protein 7 (Fabp7, also known as BLBP and BFABP) is expressed in astrocytes, radial glial cells and neuronal progenitors in the central nervous system [30]. It has been reported that Fabp7 can participate in neurogenesis as a positive regulator of proliferation in neural stem progenitor cells and is important for dendritic growth and neuronal synapse formation as well as for astrocyte proliferation during reactive gliosis [31–33]. In the peripheral nervous system, Fabp7 is usually expressed in SC precursors and immature SCs [34–36] and participate in regulating Schwann cell-axon interactions [37]. However, recent in vivo studies have shown that in adult mice, Fabp7 is a specific marker for SGCs and does not label SCs surrounding axons in the DRG or in the sciatic nerve [9]. Thus, in adult mice, Fabp7 can be used to identify SGCs. Considering that SGCs might represent a subpopulation of SC precursors (For details, see 4. The close relationship between SGCs, SC precursors, and SCs) and that central nervous system injury most likely increases the immature state of cells in the adult, additional markers or morphology or location information are also needed to identify SGCs under pathological conditions.

Following nerve injury, glial fibrillary acidic protein (GFAP) protein expression is upregulated in SGCs, therefore, GFAP protein can also be used as a marker of SGCs under pathophysiological conditions [38–40]. In addition, Kir4.1, SK3, connexin 43, and endothelial PAS domain protein 1 (Epas1) can also be used as markers of SGCs [41].

Through Kir4.1 channel, SGCs can limit the extracellular levels of K^+ in the sensory ganglia to maintain the neuronal resting membrane potential and neuronal excitability [42]. Studies has shown that injury suppresses Kir4.1 function in SGCs, which may contribute to pain [43]. Reduced Kir4.1 permeability likely depolarizes SGCs, inducing them to release excitatory mediators such as ATP that can activate the neurons. In the rat TG, specific silencing of Kir4.1 using RNA interference leads to spontaneous and evoked facial pain-like behavior in freely moving rats [44]. These findings suggested that a decrease in Kir4.1 expression after injury reduces K^+ buffering capacity of SGCs, raises extracellular K^+ concentration in the vicinity of neurons and thus results in depolarization and hyperexcitability of neuronal somata to give rise to chronic pain [13]. The results of immunohistochemistry showed that SGCs express Kir4.1, whereas the neurons, blood vessel endothelial cells and SCs do not [44]. Thus, Kir4.1 can be used for SGC recognition under physiological and pathophysiological conditions [44–48].

SK3 immunoreactivity is described in peripheral neurons, which leads to the controversy of SK3 as a specific marker of SGC in peripheral ganglia [49, 50]. However, according to the results of some studies, SK3 is a specific immunomarker of SGCs in vivo [14, 51]. Like Kir4.1 protein, SK3 is involved in either directly or indirectly, in potassium ion (K^+) buffering and can influence the level of neuronal excitability [51]. A recent study that induced trigeminal neuropathic pain in rats through a partial transection of the infraorbital nerve reported a decreased pain threshold, together with a lower expression of SK3 in the TG of the animals that underwent the infraorbital ligation when

compared to control group. In addition, administration of SK3 channel agonist (CyPPA) could significantly improve the pain threshold, and the pain threshold decreased after administration of SK3 channel antagonist (Apamin) [52, 53]. These findings suggest that SK3 play a pivotal role in neuropathic pain and may be one of the potential targets for the treatment of neuropathic pain. In all, SK3 can be used to mark SGCs under physiological and pathophysiological conditions [9, 14, 54].

Connexin 43, a gap junction protein expressed in SGCs has been shown to play an important role in several pain models [51, 55, 56]. Accumulating evidence demonstrates that after peripheral nerve injury, the expression of Connexin 43 increased in SGCs [55, 57, 58], and blockade of the gap junctions formed by Connexin 43 resulted in alleviated pain response of the mice [46, 59]. At present, Connexin 43 is commonly used to indicate activated SGCs after nerve injury.

Epas1, also referred to as hypoxiainducible factor (HIF)2α, plays an important role in tumours [60]. Recently, Epas1 was found to be expressed in SGCs by using single-cell RNA sequencing. validation by RNAscope confirmed unique expression of Epas1 in DRG SGCs, but not in sciatic nerve [41]. This result indicates that Epas1, as a new marker of SGCs, can be used to identify SGCs. In conclusion, with the increasing research on SGCs, the specific markers for SGCs recognition will be constantly discovered, andthese specific markers enable us to better identify SGCs and investigate their functions.

Dynamics of Molecular Signaling in SGCs Following Peripheral Nerve Injury

Along with changes in neurons following injury to the proximal or distal part of the peripheral nerve, SGCs also undergo characteristic changes [12]. One noticeable change in SGCs following injury is an increase in GFAP expression. Under resting conditions, GFAP protein is expressed at low levels in SGCs, but the expression of this protein increases significantly following axonal damage or inflammation [38–40, 61]. It has been reported that GFAP protein expression in SGCs peaks between 3 and 6 h following carrageenin injection and returns to the basal level at 24 h, which coincides with the normalization of the mechanical nociceptive threshold [62]. GFAP can anchor the glutamate-aspartate transporter (Glast) to the plasma membrane of glial cells. Glast is responsible for the uptake of glutamate released by neurons into synaptic clefts so that glutamate can be converted into glutamine by the enzyme GS. Therefore, increased expression of GFAP in SGCs could be related to the rapid removal of the accumulated glutamate from the perineuronal space to prevent its cytotoxic effects [12, 13].

Using 5-bromo-2′-deoxyuridine (BrdU) labeling combined with immunohistochemistry for SGC-specific proteins, researchers have demonstrated that SGCs proliferate following nerve injury [54]. In accordance with this, studies showed that following oxaliplatin injection, the number of activated SGCs in the DRG significantly increases [63], and in the monoarthritis or collagenase arthritis model, the arthritic rats exhibit SGC activation and proliferation [64, 65]. Studies also showed that activated SGCs express high levels of brain derived neurotrophic factor (BDNF), p75, and fibroblast growth factor 2 (FGF2) [4, 66]. It has been proposed that increased proliferation along with increased expression of growth factors in SGCs following injury contribute to recovery and regeneration.

The mitogen-activated protein kinase (MAPK) pathway plays an important role in the initiation and maintenance of nociceptive responses in glia and neurons of the sensory ganglion. MAPK pathway includes three major proteins: extracellular signal-regulated kinase 1/2 (ERK1/2), P38, and c-Jun N-terminal kinase (JNK) [42]. Accumulating evidence demonstrates that nerve or spinal cord injury leads to the profound activation of MAPK signaling in SGCs [67]. In CFA-treated TGs, the expression levels of p-ERK, p-P38, and p-JNK were readily observed in SGCs. Almost no p-MAPKs were expressed on SGCs in the vehicle group, but the expression levels of p-MAPKs were significantly increased in the CFA group. Similarly, upregulation of p-ERK has been reported in SGCs in animal models of local inflammation of the dura mater, temporomandibular joint inflammation, and migraine [68–70]. MAPKs phosphorylation is associated with SGCs activation and increased production of interleukin-1β (IL-1β) and TNF-α [16, 25]. It has been proposed that SGCs exhibit the features of inflammatory cells. Similar to macrophages, SGCs can be activated by monocyte chemoattractant protein-1 (MCP-1) through the CCR2 receptor to produce high levels of TNF-α and IL-1β [12]. These inflammatory cytokines released from the activated SGCs can enhance the excitability of neurons in the trigeminal ganglion, which in turn may promote the development and maintenance of orofacial allodynia/hyperalgesia [71–73].

In addition to the above-mentioned changes, there is a large significant increase in the expression of gap junction, decrease in Kir expression, enhancement in ATP release, and increase in P2XR and P2YR expression in SGCs following nerve injury [13, 42]. These characteristic changes affect the ability of SGCs to regulate the environment around neurons and give rise to chronic pain conditions.

SGCs have the Characteristics of Stem Cells

Recent evidence has added a new aspect to the functions of SGCs by showing that these cells have differentiation potential. In DRG explant culture, it has been observed

that a subpopulation of cells expressing nestin and p75 protein migrate from these explants and have the ability to differentiate into glial cells, neurons, and smooth muscle cells in vitro. Further, BrdU pulse-chase analysis showed that these progenitors likely originate from SGCs [74]. In vivo studies also demonstrated that following peripheral nerve injury, the protein expression of nestin and Sox2 increases in SGCs [75–77], and proliferating SGCs can be transformed into doublecortin-positive cells in the DRGs [78]. Similarly, it has been observed that chronic pain induces proliferation and upregulation of progenitor markers in the Sox2- and platelet-derived growth factor receptor alpha (PDGFRα)-positive SGCs [8]. Moreover, data of BrdU-labeling and genetic fate mappings demonstrated a chronic pain-induced nociceptive neurogenesis in DRG from Sox2-positive SGCs [8]. In line with this, our previous study showed that DRG-derived SGCs highly express neural crest cell markers including nestin, Sox2, Sox10, and p75, and could differentiate into nociceptive sensory neurons in the presence of VPA, CHIR99021, RO4929097, and SU5402 [3]. Similar results were also found in TG derived SGCs (our unpublished results). These findings confirmed that SGCs indeed have differentiation potential.

Recently, using single-cell RNA sequencing and RNA scope, Segal et al. found that DRG-derived SGCs can be distinguished on the basis of high levels of Epas1, as this protein is not expressed in diverse SC types. Furthermore, Epas1 has also been defined as a top marker of DRG glial precursors. In fact, several genes that are highly expressed in DRG-derived SGC clusters are also highly expressed in DRG glial precursors. These results suggest that DRG-derived SGCs retain the gene signature of DRG glial precursors [41].

As early as 2004, researchers proposed that SGCs have stem cell characteristics. The authors found a cell type of embryonic DRG of the PNS - the satellite cell - can develop into oligodendrocytes, SCs and astrocytes and proposed satellite cells of DRG are multi potential glial precursors [79]. Subsequently, evidence that SGCs express stem cell markers and can differentiate into neurons is also constantly being discovered. These evidences promote our further understanding of the roles and functions of SGCs, showing that SGCs not only have the functional properties of glia, but they could also play the role of stem cells under pathological conditions. However, we still know little about the mechanism of SGCs turning into neurons or even specific types of neurons. Strengthening research in this field will help us understand the mechanism of pathological pain and treat sensory neuron related diseases (Fig. 1).

The Close Relationship Between SGCs, SC Precursors, and SCs

Besides SGCs, SCs are also a much-touted cell type with stem cell properties in the peripheral nervous system. Increasing evidence suggests that SC precursors and even adult SCs can give rise to several cell types including those of parasympathetic ganglia during neurodevelopment as well as after injury, highlighting the broad developmental potential of these cells [80–86]. Studies showed that SC precursors, unlike SCs, die when separated from axons in vitro. It is traditionally believed that SC precursors, which are a transient population and dependent on axonal contact for survival, are specified during embryonic day 14 (E14) and E15 (mouse E12 and 13), and differentiate into immature SCs during E15-17 and mature SCs during perinatal stages [87–91]. At present, there is little evidence regarding the persistence of SC precursors after E17/18 in rats [92].

Studies suggest a close relationship between SGCs, SC precursors, and SCs. CDH19, MPZ, PLP1, SOX10, ErbB3, and FABP7 are specific markers for SC precursors and have been used to identify these cells [80, 88, 89, 92–95]. Based on the PLP1 or SOX10 promoter, many researchers have investigated the progeny of SC precursors in transgenic mice [80, 83, 96–100]. However, recent studies showed that these genes are also expressed in SGCs (Table 1) [8, 9, 101, 102]. Egr2 (also known as Krox20) is expressed in boundary cap cells, immature SCs, and myelinating SCs, and is not expressed in SGCs in vivo [10, 103, 104]. Interestingly, Egr2 is expressed in cultured SGCs [10, 102]. George et al. [18] showed that SGCs are transcriptionally, morphologically, and functionally identical to SCs. Considering that SGCs are multipotent, these studies suggest that SGCs might represent a subpopulation of SC precursors; however, a recent single-cell RNA-sequencing study indicated that SGCs are molecularly distinct from SCs [9]. Studies also suggest that SGCs adopt the morphology of SCs when cultured for extended period of time [18, 28] and SCs exhibit, to some extent, plasticity upon nerve injury [84, 92] raising an interesting possibility concerning the lineage of SGCs with respect to SC precursors or SCs.

The Origin of SGCs

It is well known that SGCs, sensory neurons, and SCs originate from neural crest cells (NCCs), a transient population in the embryo that can give rise to multiple cell types, including most neuronal and all glial components of the peripheral nervous system (Fig. 1) [104, 105]. During sensory neuron development, multiple signaling pathways such as Wnt, FGF, and Notch, induce the expression of sensory neuron-specific transcription factors including Ngn1, Ngn2, Brn3a, Isl1, Runx1, Shox2 and Runx3 in NCCs leading to their differentiation into nociceptive sensory neurons, mechanical sensory neurons, and proprioceptive neurons [3, 106, 107]. Moreover, during SCs development, NCCs differentiate into SC precursors and immature SCs before finally differentiating into myelinating and non-myelinating SCs [34, 89, 108].

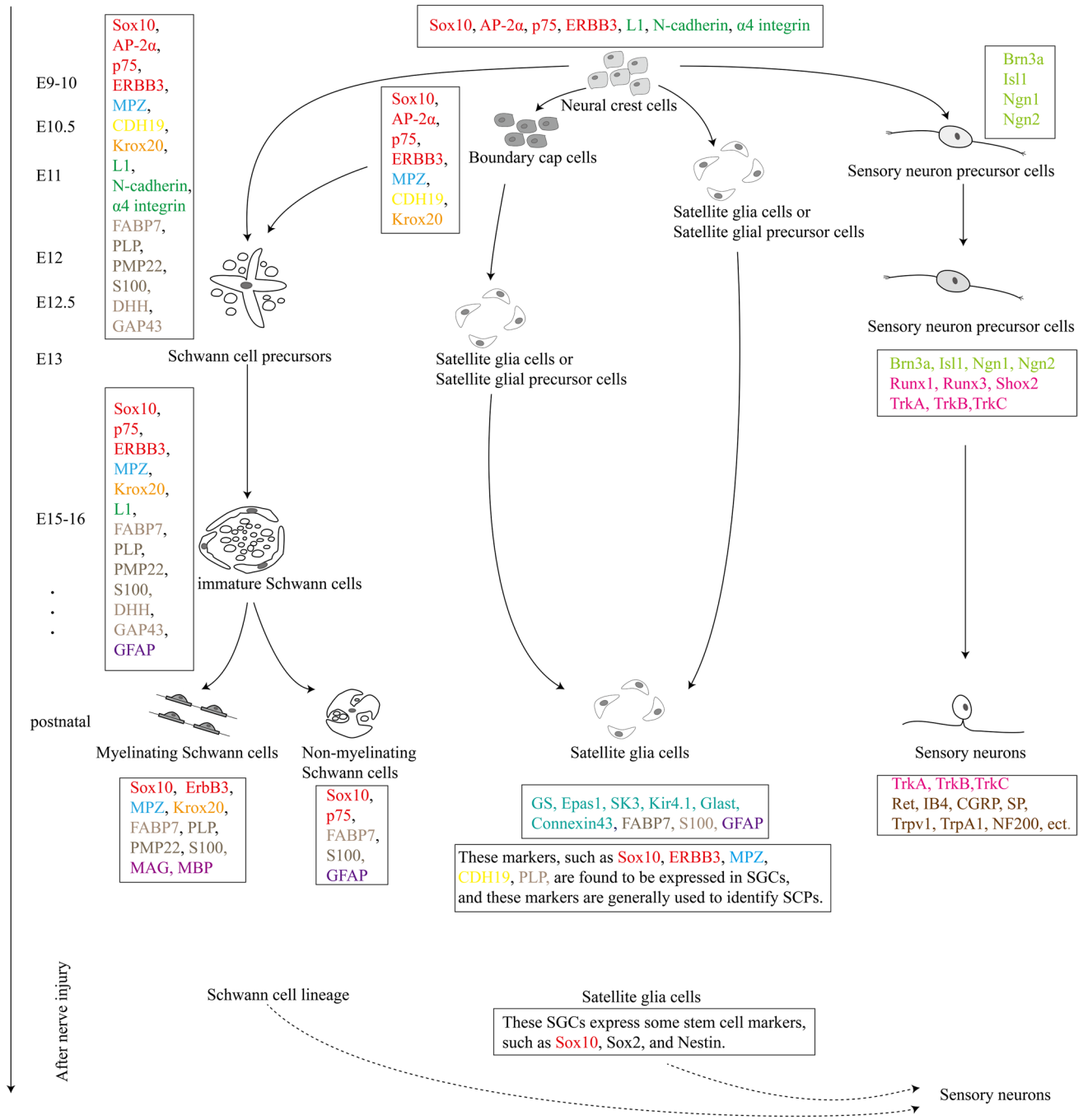

Fig. 1 The development of satellite glial cells, Schwann cells and sensory neurons and molecular expression at the main development stages. Font colors are used for easier identification of markers that are expressed in several lineages. The embryonic stage (E) corresponds to mouse development time

However, unlike the detailed understanding of the development of sensory neurons and SCs, little is known about the development of SGCs.

Studies suggest there are two waves of peripheral gliogenesis during embryonic development. For SGCs development, the first wave is at E11 in mice. At this time, a fraction of SGCs is specified directly from the migratory NCCs [102, 109]. Another source of SGCs is the boundary cap cells, which are also derived from NCCs. Boundary cap cells are found at E10.5 at the dorsal sensory entry point and the ventral motor exit point, and give rise to the remaining fraction of SGCs at E12.5 [102, 104].

Evidence suggests that histone deacetylases1 and 2 (HDAC1/2) are essential for differentiation of NCCs to

Table 1 Comparison of satellite glial cells and Schwann cell precursors

Cell type	Satellite glial cells	Schwann cell precursors
Common identification markers	GS [8, 11, 21, 22, 48]; FABP7 [9]; Epas1[41]; SK3 [14, 51]; S100 [46, 47, 110]; GFAP [46, 70, 111]; Kir4.1[44–48]; Glast [112, 113]; Connexin 43 [19].	SOX10; CDH19; ERBB3; MPZ; PLP1; FABP7; DHH; GAP43; PMP22; AP-2α; L1; Krox20; Ncad; α4 integrin [34, 88, 89, 102].
	According to the literature, the specific markers for SGCs are GS, Epas1, SK3, Kir4.1, and Connexin 43. Markers specific for SC precursors, such as CDH19, MPZ (mRNA), PLP1 (mRNA), SOX10, ERBB3 and FABP7, are also expressed in SGCs [8, 9, 18, 101, 102].	
Origin	Neural crest cells (at E11) [109, 114] Boundary cap cells (at E11.25) [104]	Neural crest cells (at E11) [109, 114] Boundary cap cells (at E12.5) [104]
progeny	1. Satellite glial cells 2. Neurons [3, 8, 74]	1. Schwann cell lineage 2. Parasympathetic neurons [80, 115] 3. Some mesenchymal stem cell [116]

SGC lineage. In mice, NCC-specific deletion of *HDAC1/2* leads to a strong reduction in Sox10 and Pax3 expression in NCCs along with the loss of SGCs and SC precursors in the DRG and peripheral nerves [109]. In NCCs, HDAC1/2 bind to the Pax3 promoter and induce the expression of this transcription factor, which was required to maintain high Sox10 levels and trigger the expression of Fabp7. In addition, HDAC1/2 also activate P0 transcription by binding to the P0 promoter. Therefore, HDAC1/2 direct the specification of NCCs to SC precursors and SGCs by controlling the expression of Pax3 and the concerted action of Pax3 and Sox10 on their target genes [102, 109].

The time of development of SGCs is slightly later than that of sensory neurons, but similar to that of SC precursors. Although several studies have described the process and mechanism of neural crest specification into peripheral glia, the focus of description has always been SC lineage, and very few studies have paid close attention to the development of SGCs. Considering the increasing evidence that SGCs have stem cell characteristics and are similar to SC precursors, it is necessary to investigate the SGC lineage in the future (Table 1).

Conclusion

SGCs are the most important type of glial cells in the sensory ganglion. Following nerve injury, SGCs undergo a series of changes, leading to their proliferation, high expression of neurotrophins and cytokines, and activation of MAPK kinases. Activated SGCs regulate the environment around neurons, and participate in the repair process. It has been suggested that SGCs may have the ability to replenish damaged neurons, which expands the role of these cells. With the application of single-cell sequencing technology to SGCs, researchers have explored the characteristics of SGCs and the differences between SGCs and SCs. These studies have undoubtedly advanced our understanding of SGCs. However, based on present literature, it is still impossible to distinguish SGCs from SC precursors. Although several studies have focused on SGCs, the origin and nature of these cells remain unclear, and their lineage remains uncharacterized. Further investigation is required to establish a criteria for distinguishing SGCs from SC precursors and SCs, and to determine the lineage of SGCs.

Acknowledgements We thank Jigang Pan and Lu Wang for their help in writing this paper.

Author Contributions All authors contributed to the study conception and design. The first draft of the manuscript was written by Junhou Lu, Dongyan Wang, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Funding This work was supported by the Young Scientific Talents Growth Project of Guizhou Provincial Education Department (KY [2022]228), Doctoral Foundation of Guizhou Medical University [2021]020, and the Science and Technology Fund Project of Guizhou Health Commission (gzwkj2022-511, gzwkj2022-257).

Data Availability Not applicable.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Competing Interests The authors declare that they have no competing interests.

References

1. Basbaum, A. I., Bautista, D. M., Scherrer, G., & Julius, D. (2009). Cellular and molecular mechanisms of pain. *Cell, 139*(2), 267–284.
2. Meyer, K., & Kaspar, B. K. (2014). Making sense of pain: are pluripotent stem cell-derived sensory neurons a new tool for studying pain mechanisms? *Molecular Therapy, 22*(8), 1403–1405.

3. Wang, D., Lu, J., Xu, X., Yuan, Y., Zhang, Y., et al. (2021). Satellite glial cells give rise to nociceptive sensory neurons. *Stem Cell Reviews and Reports, 17*(3), 999–1013.
4. Lin, G., Bella, A. J., Lue, T. F., & Lin, C. S. (2006). Brain-derived neurotrophic factor (BDNF) acts primarily via the JAK/STAT pathway to promote neurite growth in the major pelvic ganglion of the rat: part 2. *The Journal of Sexual Medicine, 3*(5), 821–829.
5. Spray, D. C., Iglesias, R., Shraer, N., Suadicani, S. O., Belzer, V., Hanstein, R., & Hanani, M. (2019). Gap junction mediated signaling between satellite glia and neurons in trigeminal ganglia. *Glia, 67*(5), 791–801.
6. Belzer, V., & Hanani, M. (2019). Nitric oxide as a messenger between neurons and satellite glial cells in dorsal root ganglia. *Glia, 67*(7), 1296–1307.
7. Zhang, X., Chen, Y., Wang, C., & Huang, L. Y. (2007). Neuronal somatic ATP release triggers neuron-satellite glial cell communication in dorsal root ganglia. *Proceedings of the National Academy of Sciences of the United States of America, 104*(23), 9864–9869.
8. Zhang, L., Xie, R., Yang, J., Zhao, Y., Qi, C., et al. (2019). Chronic pain induces nociceptive neurogenesis in dorsal root ganglia from Sox2-positive satellite cells. *Glia, 67*(6), 1062–1075.
9. Avraham, O., Deng, P. Y., Jones, S., Kuruvilla, R., Semenkovich, C. F., Klyachko, V. A., & Cavalli, V. (2020). Satellite glial cells promote regenerative growth in sensory neurons. *Nature Communications, 11*(1), 4891.
10. Murphy, P., Topilko, P., Schneider-Maunoury, S., Seitanidou, T., Baron-Van Evercooren, A., & Charnay, P. (1996). The regulation of Krox-20 expression reveals important steps in the control of peripheral glial cell development. *Development, 122*(9), 2847–2857.
11. Hanani, M. (2005). Satellite glial cells in sensory ganglia: from form to function. *Brain Research. Brain Research Reviews, 48*(3), 457–476.
12. Ohara, P. T., Vit, J. P., Bhargava, A., Romero, M., Sundberg, C., Charles, A. C., & Jasmin, L. (2009). Gliopathic pain: when satellite glial cells go bad. *The Neuroscientist : A Review Journal Bringing Neurobiology, Neurology and Psychiatry, 15*(5), 450–463.
13. Huang, L. Y., Gu, Y., & Chen, Y. (2013). Communication between neuronal somata and satellite glial cells in sensory ganglia. *Glia, 61*(10), 1571–1581.
14. Jager, S. B., Pallesen, L. T., & Vaegter, C. B. (2018). Isolation of satellite glial cells for high-quality RNA purification. *Journal of Neuroscience Methods, 297*, 1–8.
15. Capuano, A., De Corato, A., Lisi, L., Tringali, G., Navarra, P., & Russo, D. (2009). Proinflammatory-activated trigeminal satellite cells promote neuronal sensitization: relevance for migraine pathology. *Molecular Pain, 5*, 43.
16. Zhang, Y., Song, N., Liu, F., Lin, J., Liu, M., et al. (2019). Activation of mitogen-activated protein kinases in satellite glial cells of the trigeminal ganglion contributes to substance P-mediated inflammatory pain. *International Journal of Oral Science, 11*(3), 24.
17. Silva, J. R., Lopes, A. H., Talbot, J., Cecilio, N. T., Rossato, M. F., et al. (2017). Neuroimmune-glia interactions in the sensory ganglia account for the development of acute herpetic neuralgia. *Journal of Neuroscience, 37*(27), 6408–6422.
18. George, D., Ahrens, P., & Lambert, S. (2018). Satellite glial cells represent a population of developmentally arrested Schwann cells. *Glia, 66*(7), 1496–1506.
19. Procacci, P., Magnaghi, V., & Pannese, E. (2008). Perineuronal satellite cells in mouse spinal ganglia express the gap junction protein connexin43 throughout life with decline in old age. *Brain Research Bulletin, 75*(5), 562–569.
20. Lin, J., Liu, F., Zhang, Y. Y., Song, N., Liu, M. K., et al. (2019). P2Y14 receptor is functionally expressed in satellite glial cells and mediates interleukin-1beta and chemokine CCL2 secretion. *Journal of Cellular Physiology, 234*(11), 21199–21210.
21. Magni, G., Merli, D., Verderio, C., Abbracchio, M. P., & Ceruti, S. (2015). P2Y2 receptor antagonists as anti-allodynic agents in acute and sub-chronic trigeminal sensitization: role of satellite glial cells. *Glia, 63*(7), 1256–1269.
22. Jager, S. E., Pallesen, L. T., Richner, M., Harley, P., Hore, Z., McMahon, S., Denk, F., & Vaegter, C. B. (2020). Changes in the transcriptional fingerprint of satellite glial cells following peripheral nerve injury. *Glia, 68*(7), 1375–1395.
23. Castillo, C., Norcini, M., Hernandez, M., Correa, L. A., Blanck, G., & Recio-Pinto, E. (2013). Satellite glia cells in dorsal root ganglia express functional NMDA receptors. *Neuroscience, 240*, 135–146.
24. Zhang, Q., Zhao, J., Shen, J., Zhang, X., Ren, R., et al. (2019). The ATP-P2×7 signaling pathway participates in the regulation of slit1 expression in satellite glial cells. *Frontiers in Cellular Neuroscience, 13*, 420.
25. Liu, F., Zhang, Y. Y., Song, N., Lin, J., Liu, M. K., et al. (2019). GABAB receptor activation attenuates inflammatory orofacial pain by modulating interleukin-1beta in satellite glial cells: Role of NF-kappaB and MAPK signaling pathways. *Brain Research Bulletin, 149*, 240–250.
26. Vinterhoj, H. S. H., Stensballe, A., Duroux, M., & Gazerani, P. (2019). Characterization of rat primary trigeminal satellite glial cells and associated extracellular vesicles under normal and inflammatory conditions. *Journal of Proteomics, 190*, 27–34.
27. Laursen, J. C., Cairns, B. E., Dong, X. D., Kumar, U., Somvanshi, R. K., Arendt-Nielsen, L., & Gazerani, P. (2014). Glutamate dysregulation in the trigeminal ganglion: a novel mechanism for peripheral sensitization of the craniofacial region. *Neuroscience, 256*, 23–35.
28. Poulsen, J. N., Larsen, F., Duroux, M., & Gazerani, P. (2014). Primary culture of trigeminal satellite glial cells: a cell-based platform to study morphology and function of peripheral glia. *International Journal of Physiology, Pathophysiology and Pharmacology, 6*(1), 1–12.
29. Berger, U. V., & Hediger, M. A. (2000). Distribution of the glutamate transporters GLAST and GLT-1 in rat circumventricular organs, meninges, and dorsal root ganglia. *The Journal of Comparative Neurology, 421*(3), 385–399.
30. Killoy, K. M., Harlan, B. A., Pehar, M., & Vargas, M. R. (2020). FABP7 upregulation induces a neurotoxic phenotype in astrocytes. *Glia, 68*(12), 2693–2704.
31. Matsumata, M., Sakayori, N., Maekawa, M., Owada, Y., Yoshikawa, T., & Osumi, N. (2012). The effects of Fabp7 and Fabp5 on postnatal hippocampal neurogenesis in the mouse. *Stem Cells, 30*(7), 1532–1543.
32. Ebrahimi, M., Yamamoto, Y., Sharifi, K., Kida, H., Kagawa, Y., et al. (2016). Astrocyte-expressed FABP7 regulates dendritic morphology and excitatory synaptic function of cortical neurons. *Glia, 64*(1), 48–62.
33. Sharifi, K., Morihiro, Y., Maekawa, M., Yasumoto, Y., Hoshi, H., et al. (2011). FABP7 expression in normal and stab-injured brain cortex and its role in astrocyte proliferation. *Histochemistry and Cell Biology, 136*(5), 501–513.
34. Jessen, K. R., & Mirsky, R. (2019). Schwann cell precursors; multipotent glial cells in embryonic nerves. *Frontiers in Molecular Neuroscience, 12*, 69.
35. Kastriti, M. E., & Adameyko, I. (2017). Specification, plasticity and evolutionary origin of peripheral glial cells. *Current Opinion in Neurobiology, 47*, 196–202.
36. Jessen, K. R., & Mirsky, R. (2005). The origin and development of glial cells in peripheral nerves. *Nature Reviews Neuroscience, 6*(9), 671–682.

37. Miller, S. J., Li, H., Rizvi, T. A., Huang, Y., Johansson, G., et al. (2003). Brain lipid binding protein in axon-Schwann cell interactions and peripheral nerve tumorigenesis. *Molecular and Cellular Biology, 23*(6), 2213–2224.
38. Siemionow, K., Klimczak, A., Brzezicki, G., Siemionow, M., & McLain, R. F. (2009). The effects of inflammation on glial fibrillary acidic protein expression in satellite cells of the dorsal root ganglion. *Spine (Phila Pa 1976), 34*(16), 1631–1637.
39. Blum, E., Procacci, P., Conte, V., Sartori, P., & Hanani, M. (2017). Long term effects of lipopolysaccharide on satellite glial cells in mouse dorsal root ganglia. *Experimental Cell Research, 350*(1), 236–241.
40. Liu, H., Zhao, L., Gu, W., Liu, Q., Gao, Z., et al. (2018). Activation of satellite glial cells in trigeminal ganglion following dental injury and inflammation. *Journal of Molecular Histology, 49*(3), 257–263.
41. Tasdemir-Yilmaz, O. E., Druckenbrod, N. R., Olukoya, O. O., Dong, W., Yung, A. R., et al. (2021). Diversity of developing peripheral glia revealed by single-cell RNA sequencing. *Developmental Cell, 56*(17), 2516-2535e2518.
42. Fan, W., Zhu, X., He, Y., Zhu, M., Wu, Z., Huang, F., & He, H. (2019). The role of satellite glial cells in orofacial pain. *Journal of Neuroscience Research, 97*(4), 393–401.
43. Hanani, M., & Spray, D. C. (2020). Emerging importance of satellite glia in nervous system function and dysfunction. *Nature Reviews Neuroscience, 21*(9), 485–498.
44. Vit, J. P., Ohara, P. T., Bhargava, A., Kelley, K., & Jasmin, L. (2008). Silencing the Kir4.1 potassium channel subunit in satellite glial cells of the rat trigeminal ganglion results in pain-like behavior in the absence of nerve injury. *Journal of Neuroscience, 28*(16), 4161–4171.
45. Huang, B., Zdora, I., de Buhr, N., Lehmbecker, A., Baumgartner, W., & Leitzen, E. (2021). Phenotypical peculiarities and species-specific differences of canine and murine satellite glial cells of spinal ganglia. *Journal of Cellular and Molecular Medicine, 25*(14), 6909–6924.
46. Schmitt, L. I., Leo, M., Kutritz, A., Kleinschnitz, C., & Hagenacker, T. (2020). Activation and functional modulation of satellite glial cells by oxaliplatin lead to hyperexcitability of sensory neurons in vitro. *Molecular and Cellular Neuroscience, 105*, 103499.
47. Xiang, H., Xu, H., Fan, F., Shin, S. M., Hogan, Q. H., & Yu, H. (2018). Glial fibrillary acidic protein promoter determines transgene expression in satellite glial cells following intraganglionic adeno-associated virus delivery in adult rats. *Journal of Neuroscience Research, 96*(3), 436–448.
48. Rajasekhar, P., Poole, D. P., Liedtke, W., Bunnett, N. W., & Veldhuis, N. A. (2015). P2Y1 receptor activation of the TRPV4 ion channel enhances purinergic signaling in satellite glial cells. *Journal of Biological Chemistry, 290*(48), 29051–29062.
49. Bahia, P. K., Suzuki, R., Benton, D. C., Jowett, A. J., Chen, M. X., Trezise, D. J., Dickenson, A. H., & Moss, G. W. (2005). A functional role for small-conductance calcium-activated potassium channels in sensory pathways including nociceptive processes. *Journal of Neuroscience, 25*(14), 3489–3498.
50. Mongan, L. C., Hill, M. J., Chen, M. X., Tate, S. N., Collins, S. D., Buckby, L., & Grubb, B. D. (2005). The distribution of small and intermediate conductance calcium-activated potassium channels in the rat sensory nervous system. *Neuroscience, 131*(1), 161–175.
51. Vit, J. P., Jasmin, L., Bhargava, A., & Ohara, P. T. (2006). Satellite glial cells in the trigeminal ganglion as a determinant of orofacial neuropathic pain. *Neuron Glia Biology, 2*(4), 247–257.
52. Verissimo, C. P., Acosta Filha, L. G., Moreira da Silva, F. J., Westgarth, H., Coelho Aguiar, J. M., Pontes, B., Moura-Neto, V., Gazerani, P., & DosSantos, M. F. (2022). Short-term functional and morphological changes in the primary cultures of trigeminal ganglion cells. *Current Issues in Molecular Biology, 44*(3), 1257–1272.
53. Zhao, J., Zhang, Y., Liu, X., Rao, Y., Fu, J., Hua, L., & Ou, C. (2021). Activation of SK3 channel plays a pivotal role in modulation of trigeminal neuralgia. *Neurological Research, 43*(12), 1005–1012.
54. Donegan, M., Kernisant, M., Cua, C., Jasmin, L., & Ohara, P. T. (2013). Satellite glial cell proliferation in the trigeminal ganglia after chronic constriction injury of the infraorbital nerve. *Glia, 61*(12), 2000–2008.
55. Jin, Y. Z., Zhang, P., Hao, T., Wang, L. M., Guo, M. D., & Gan, Y. H. (2019). Connexin 43 contributes to temporomandibular joint inflammation induced-hypernociception via sodium channel 1.7 in trigeminal ganglion. *Neuroscience Letters, 707*, 134301.
56. Kaji, K., Shinoda, M., Honda, K., Unno, S., Shimizu, N., & Iwata, K. (2016). Connexin 43 contributes to ectopic orofacial pain following inferior alveolar nerve injury. *Molecular Pain, 12:*1–12.
57. Komiya, H., Shimizu, K., Ishii, K., Kudo, H., Okamura, T., Kanno, K., Shinoda, M., Ogiso, B., & Iwata, K. (2018). Connexin 43 expression in satellite glial cells contributes to ectopic tooth-pulp pain. *Journal of Oral Science, 60*(4), 493–499.
58. Ohara, P. T., Vit, J. P., Bhargava, A., & Jasmin, L. (2008). Evidence for a role of connexin 43 in trigeminal pain using RNA interference in vivo. *Journal of Neurophysiology, 100*(6), 3064–3073.
59. Li, Q., Wang, Y. Q., & Chu, Y. X. (2020). The role of connexins and pannexins in orofacial pain. *Life Sciences, 258*, 118198.
60. Zhao, J., Du, F., Luo, Y., Shen, G., Zheng, F., & Xu, B. (2015). The emerging role of hypoxia-inducible factor-2 involved in chemo/radioresistance in solid tumors. *Cancer Treatment Reviews, 41*(7), 623–633.
61. Warwick, R. A., & Hanani, M. (2013). The contribution of satellite glial cells to chemotherapy-induced neuropathic pain. *European Journal of Pain, 17*(4), 571–580.
62. Souza, G. R., Talbot, J., Lotufo, C. M., Cunha, F. Q., Cunha, T. M., & Ferreira, S. H. (2013). Fractalkine mediates inflammatory pain through activation of satellite glial cells. *Proceedings of the National Academy of Sciences of the United States of America, 110*(27), 11193–11198.
63. Di Mannelli, C., Pacini, L., Bonaccini, A., Zanardelli, L., Mello, M., & Ghelardini, C. (2013). Morphologic features and glial activation in rat oxaliplatin-dependent neuropathic pain. *The Journal of Pain: official Journal of the American Pain Society, 14*(12), 1585–1600.
64. Nascimento, D. S., Castro-Lopes, J. M., & Moreira Neto, F. L. (2014). Satellite glial cells surrounding primary afferent neurons are activated and proliferate during monoarthritis in rats: is there a role for ATF3? *PLoS One, 9*(9), e108152.
65. Adaes, S., Almeida, L., Potes, C. S., Ferreira, A. R., Castro-Lopes, J. M., Ferreira-Gomes, J., & Neto, F. L. (2017). Glial activation in the collagenase model of nociception associated with osteoarthritis. *Molecular Pain, 13*, 1744806916688219.
66. Verkhratsky, A., Parpura, V., & Rodriguez, J. J. (2011). Where the thoughts dwell: the physiology of neuronal-glial "diffuse neural net" *Brain Research Reviews, 66*(1–2), 133–151.
67. Zhang, Y. Y., Song, N., Liu, F., Lin, J., Liu, M. K., et al. (2020). Activation of the RAS/B-RAF-MEK-ERK pathway in satellite glial cells contributes to substance p-mediated orofacial pain. *European Journal of Neuroscience, 51*(11), 2205–2218.
68. Lukacs, M., Haanes, K. A., Majlath, Z., Tajti, J., Vecsei, L., Warfvinge, K., & Edvinsson, L. (2015). Dural administration of inflammatory soup or Complete Freund's Adjuvant induces activation and inflammatory response in the rat trigeminal ganglion. *The Journal of Headache and Pain, 16*, 564.

69. Bi, R. Y., Kou, X. X., Meng, Z., Wang, X. D., Ding, Y., & Gan, Y. H. (2013). Involvement of trigeminal ganglionic Nav 1.7 in hyperalgesia of inflamed temporomandibular joint is dependent on ERK1/2 phosphorylation of glial cells in rats. *European Journal of Pain, 17*(7), 983–994.
70. Qin, G., Gui, B., Xie, J., Chen, L., Chen, L., Cui, Z., Zhou, J., & Tan, G. (2018). Tetrandrine alleviates nociception in a rat model of migraine via suppressing S100B and p-ERK activation in satellite glial cells of the trigeminal ganglia. *Journal of Molecular Neuroscience, 64*(1), 29–38.
71. Takeda, M., Tanimoto, T., Kadoi, J., Nasu, M., Takahashi, M., Kitagawa, J., & Matsumoto, S. (2007). Enhanced excitability of nociceptive trigeminal ganglion neurons by satellite glial cytokine following peripheral inflammation. *Pain, 129*(1–2), 155–166.
72. Takeda, M., Kitagawa, J., Takahashi, M., & Matsumoto, S. (2008). Activation of interleukin-1beta receptor suppresses the voltage-gated potassium currents in the small-diameter trigeminal ganglion neurons following peripheral inflammation. *Pain, 139*(3), 594–602.
73. Takeda, M., Takahashi, M., & Matsumoto, S. (2008). Contribution of activated interleukin receptors in trigeminal ganglion neurons to hyperalgesia via satellite glial interleukin-1beta paracrine mechanism. *Brain, Behavior, and Immunity, 22*(7), 1016–1023.
74. Li, H. Y., Say, E. H., & Zhou, X. F. (2007). Isolation and characterization of neural crest progenitors from adult dorsal root ganglia. *Stem Cells, 25*(8), 2053–2065.
75. Koike, T., Wakabayashi, T., Mori, T., Takamori, Y., Hirahara, Y., & Yamada, H. (2014). Sox2 in the adult rat sensory nervous system. *Histochemistry and Cell Biology, 141*(3), 301–309.
76. Krishnan, A., Bhavanam, S., & Zochodne, D. (2018). An intimate role for adult dorsal root ganglia resident cycling cells in the generation of local macrophages and satellite glial cells. *Journal of Neuropathology and Experimental Neurology, 77*(10), 929–941.
77. Gallaher, Z. R., Johnston, S. T., & Czaja, K. (2014). Neural proliferation in the dorsal root ganglia of the adult rat following capsaicin-induced neuronal death. *The Journal of Comparative Neurology, 522*(14), 3295–3307.
78. Ma, W., Yang, J. W., Wang, X. B., Luo, T., Zhou, L., et al. (2021). Negative regulation by proBDNF signaling of peripheral neurogenesis in the sensory ganglia of adult rats. *Biomedicine & Pharmacotherapy, 144*, 112273.
79. Fex Svenningsen, A., Colman, D. R., & Pedraza, L. (2004). Satellite cells of dorsal root ganglia are multipotential glial precursors. *Neuron Glia Biology, 1*(1), 85–93.
80. Espinosa-Medina, I., Outin, E., Picard, C. A., Chettouh, Z., Dymecki, S., Consalez, G. G., Coppola, E., & Brunet, J. F. (2014). Neurodevelopment. Parasympathetic ganglia derive from Schwann cell precursors. *Science, 345*(6192), 87–90.
81. Espinosa-Medina, I., Jevans, B., Boismoreau, F., Chettouh, Z., Enomoto, H., Muller, T., Birchmeier, C., Burns, A. J., & Brunet, J. F. (2017). Dual origin of enteric neurons in vagal Schwann cell precursors and the sympathetic neural crest. *Proceedings of the National Academy of Sciences of the United States of America, 114*(45), 11980–11985.
82. Adameyko, I., Lallemend, F., Aquino, J. B., Pereira, J. A., Topilko, P., et al. (2009). Schwann cell precursors from nerve innervation are a cellular origin of melanocytes in skin. *Cell, 139*(2), 366–379.
83. Xie, M., Kamenev, D., Kaucka, M., Kastriti, M. E., Zhou, B., et al. (2019). Schwann cell precursors contribute to skeletal formation during embryonic development in mice and zebrafish. *Proceedings of the National Academy of Sciences of the United States of America, 116*(30), 15068–15073.
84. Milichko, V., & Dyachuk, V. (2020). Novel glial cell functions: extensive potency, stem cell-like properties, and participation in regeneration and transdifferentiation. *Frontiers in Cell and Developmental Biology, 8*, 809.
85. Merrell, A. J., & Stanger, B. Z. (2016). Adult cell plasticity in vivo: de-differentiation and transdifferentiation are back in style. *Nature Reviews Molecular Cell Biology, 17*(7), 413–425.
86. Kameneva, P., Kastriti, M. E., & Adameyko, I. (2021). Neuronal lineages derived from the nerve-associated Schwann cell precursors. *Cellular and Molecular Life Sciences, 78*(2), 513–529.
87. Jessen, K. R., & Mirsky, R. (1997). Embryonic Schwann cell development: the biology of Schwann cell precursors and early Schwann cells. *Journal of Anatomy, 191*(Pt 4), 501–505.
88. Mirsky, R., Woodhoo, A., Parkinson, D. B., Arthur-Farraj, P., Bhaskaran, A., & Jessen, K. R. (2008). Novel signals controlling embryonic Schwann cell development, myelination and dedifferentiation. *Journal of the Peripheral Nervous System: Jpns, 13*(2), 122–135.
89. Woodhoo, A., & Sommer, L. (2008). Development of the Schwann cell lineage: from the neural crest to the myelinated nerve. *Glia, 56*(14), 1481–1490.
90. Jessen, K. R., & Mirsky, R. (1999). Schwann cells and their precursors emerge as major regulators of nerve development. *Trends in Neurosciences, 22*(9), 402–410.
91. Mirsky, R., Jessen, K. R., Brennan, A., Parkinson, D., Dong, Z., Meier, C., Parmantier, E., & Lawson, D. (2002). Schwann cells as regulators of nerve development. *Journal of Physiology, Paris, 96*(1–2), 17–24.
92. Jessen, K. R., Mirsky, R., & Lloyd, A. C. (2015). Schwann cells: development and role in nerve repair. *Cold Spring Harbor Perspectives in Biology, 7*(7), a020487.
93. Kim, H. S., Lee, J., Lee, D. Y., Kim, Y. D., Kim, J. Y., Lim, H. J., Lim, S., & Cho, Y. S. (2017). Schwann cell precursors from human pluripotent stem cells as a potential therapeutic target for myelin repair. *Stem Cell Reports, 8*(6), 1714–1726.
94. Shi, H., Gong, Y., Qiang, L., Li, X., Zhang, S., et al. (2016). Derivation of Schwann cell precursors from neural crest cells resident in bone marrow for cell therapy to improve peripheral nerve regeneration. *Biomaterials, 89*, 25–37.
95. Aquino, J. B., & Sierra, R. (2018). Schwann cell precursors in health and disease. *Glia, 66*(3), 465–476.
96. Kameneva, P., Kastriti, M. E., & Adameyko, I. (2020). Neuronal lineages derived from the nerve-associated Schwann cell precursors. *Cellular and Molecular Life Sciences,78*(2), 513–529.
97. El-Nachef, W. N., & Bronner, M. E. (2020). De novo enteric neurogenesis in post-embryonic zebrafish from Schwann cell precursors rather than resident cell types. *Development, 147*(13), dev186619.
98. Kastriti, M. E., Kameneva, P., Kamenev, D., Dyachuk, V., Furlan, A., et al. (2019). Schwann cell precursors generate the majority of chromaffin cells in zuckerkandl organ and some sympathetic neurons in paraganglia. *Frontiers in Molecular Neuroscience, 12*, 6.
99. Furlan, A., Dyachuk, V., Kastriti, M. E., Calvo-Enrique, L., Abdo, H., et al. (2017). Multipotent peripheral glial cells generate neuroendocrine cells of the adrenal medulla. *Science, 357*(6346), eaal3753.
100. Kim, H. S., Kim, J. Y., Song, C. L., Jeong, J. E., & Cho, Y. S. (2020). Directly induced human Schwann cell precursors as a valuable source of Schwann cells. *Stem Cell Research & Therapy, 11*(1), 257.
101. Zhang, R., Chen, S., Wang, X., Gu, X., & Yi, S. (2021). Cell populations in neonatal rat peripheral nerves identified by single-cell transcriptomics. *Glia,69*(3), 765–778.
102. Jacob, C. (2015). Transcriptional control of neural crest specification into peripheral glia. *Glia, 63*(11), 1883–1896.
103. Topilko, P., Schneider-Maunoury, S., Levi, G., Baron-Van Evercooren, A., Chennoufi, A. B., Seitanidou, T., Babinet, C., &

Charnay, P. (1994). Krox-20 controls myelination in the peripheral nervous system. *Nature, 371*(6500), 796–799.
104. Maro, G. S., Vermeren, M., Voiculescu, O., Melton, L., Cohen, J., Charnay, P., & Topilko, P. (2004). Neural crest boundary cap cells constitute a source of neuronal and glial cells of the PNS. *Nature Neuroscience, 7*(9), 930–938.
105. Le Douarin, N., Dulac, C., Dupin, E., & Cameron-Curry, P. (1991). Glial cell lineages in the neural crest. *Glia, 4*(2), 175–184.
106. Pavan, W. J., & Raible, D. W. (2012). Specification of neural crest into sensory neuron and melanocyte lineages. *Development Biology, 366*(1), 55–63.
107. Marmigere, F., & Ernfors, P. (2007). Specification and connectivity of neuronal subtypes in the sensory lineage. *Nature Reviews Neuroscience, 8*(2), 114–127.
108. Monk, K. R., Feltri, M. L., & Taveggia, C. (2015). New insights on Schwann cell development. *Glia, 63*(8), 1376–1393.
109. Jacob, C., Lotscher, P., Engler, S., Baggiolini, A., Varum Tavares, S., et al. (2014). HDAC1 and HDAC2 control the specification of neural crest cells into peripheral glia. *Journal of Neuroscience, 34*(17), 6112–6122.
110. Leo, M., Schmitt, L. I., Kutritz, A., Kleinschnitz, C., & Hagenacker, T. (2021). Cisplatin-induced activation and functional modulation of satellite glial cells lead to cytokine-mediated modulation of sensory neuron excitability. *Experimental Neurology, 341*, 113695.
111. Vargas-Parada, A., Loeza-Alcocer, E., Gonzalez-Ramirez, R., Rodriguez-Sanchez, M., Raya-Tafolla, G., Floran, B., Felix, R., & Delgado-Lezama, R. (2021). gamma-Aminobutyric acid (GABA) from satellite glial cells tonically depresses the excitability of primary afferent fibers. *Neuroscience Research, 170*, 50–58.
112. Rabah, Y., Rubino, B., Moukarzel, E., & Agulhon, C. (2020). Characterization of transgenic mouse lines for selectively targeting satellite glial cells and macrophages in dorsal root ganglia. *PLoS One, 15*(9), e0229475.
113. Wang, F., Xiang, H., Fischer, G., Liu, Z., Dupont, M. J., Hogan, Q. H., & Yu, H. (2016). HMG-CoA synthase isoenzymes 1 and 2 localize to satellite glial cells in dorsal root ganglia and are differentially regulated by peripheral nerve injury. *Brain Research, 1652*, 62–70.
114. Morrison, S. J. (2001). Neuronal potential and lineage determination by neural stem cells. *Current Opinion in Cell Biology, 13*(6), 666–672.
115. Dyachuk, V., Furlan, A., Shahidi, M. K., Giovenco, M., Kaukua, N., et al. (2014). Neurodevelopment. Parasympathetic neurons originate from nerve-associated peripheral glial progenitors. *Science, 345*(6192), 82–87.
116. Kaukua, N., Shahidi, M. K., Konstantinidou, C., Dyachuk, V., Kaucka, M., et al. (2014). Glial origin of mesenchymal stem cells in a tooth model system. *Nature, 513*(7519), 551–554.