paper	Anatomy	Cellular	Molecular	Genetics	Time	Note
(Lin, Pan et al. 2012)	Around the Central canal NMLF	Motor neurons	Contactin-2(TAG-1)		Acute& Chronic(6 &11)	
Chen 2016	Around the central canal. NMLF	Overlap between L1.2 marker and Islet-1(motor neuron) HuC/D(immature neurons) GFAP(astrocytes)	L1.2		Chronic(6 &11)	
(Liu, Yu et al. 2014)	Caudal to the lesion site Around the central canal & in white matter	Motor neurons around central canal& immature neurons	Ptena		Acute(12h) & Chronic (6d) and back to basal level at 11d	inhibit regrowth of axons and reduce number of brain stem neurons (NMLF) with regenerated axons
(Ma, Yu et al. 2012)	NMLF IMRF SRF Neurons regenerating axons	Neurons	Cysteine and glycine-rich protein1a	Csrp1a gene	Chronic(3, 11,21)	Successful spinal cord regeneration requires at least two critical factors: the ability of an injured neuron to regrow its axon and a supportive extracellular environment for axon regeneration (Bulsara et al., 2002). In addition, rearrangements of the Intra-spinal connections of interneurons involving pre-synaptic and Post-synaptic structures need to be considered (Guo et al., 2011).
(Ma, Shen et al. 2014)	NMLF IMRF In caudal spinal cord		Legumain		Chronic(3 &11)	
(Ogai, Hisano et al. 2012)	NMLF IMRF	Upper motor neurons	Anti-apoptotic factors: Bcl-2 p-Akt		Chronic(1-6) Rapid activation	The regrowing axons from upper motor neurons reached the lesion site at 10–15 days and then crossed at 4–6 weeks after SCI

(Ogai, Nakatani et al. 2014)		Ependymal cells	Sox-2		Chronic(1-20)	Initiate Proliferation in ependymal cells
(Pan, Lin et al. 2013)	Around the central canal(gray matter) And spinal cord parenchyma	Ependymal cells Motor neurons and noradrenergic and dopaminergic neurons.	MVP(major vault protein)		Chronic(6 &11)	MVP and nestin are co-expressed in ependymal cells lining the central canal and in some cells distributed in the spinal cord parenchyma.
(Reimer, Sorensen et al. 2008)		Ependymoradial glial cells	Olig-2		Chronic(14)	Olig2 expressing ependymoradial glial cells proliferate and switch to motor neuron production after a lesion.
(Reimer, Kuscha et al. 2009)	Ependymal zone in ventrolateral position. immediate vicinity of the lesion site	Ependymoradial glial cells (proliferate to motor neuron progenitors and express these molecule)	Olig-2 Shha hh signaling (by measuring hh receptor *: patched1* Fgf signaling. retinoic acid signaling	 *spry4* crapb2a, cyp26a	Chronic(2 wpl-6)	Regrowth of axon from brain stem mRNA expression of *fgf3* and the downstream gene *sprouty4 (spry4*) was robustly increased in the lesioned spinal cord. robust increase in mRNA levels of retinoic acid receptor subunits (rarab, rxrga, rxrgb) and downstream genes (crapb2a, cyp26a)
(Schweitzer, Becker et al. 2003)	Spinal cord caudal to the lesion site (where descending axons from the brainstem regenerate)		P0(protein zero) mRNA		Chronic(14)	

(Schweitzer, Gimnopoulos et al. 2007)	NMLF IMRF Peripheral white matter	periventricular cell layer and in the peripheral white matter	Contactin1a		Chronic(6 &14)	
(Vajn, Suler et al. 2014)						Only functional tests.
(Yu, Gibbs et al. 2011)	NMLF		miR-133b		Acute(6 h) Chronic(1 &7d)	
(Yu, Cristofanilli et al. 2011)	NMLF Axonal regeneration Around the ventricle	Neurons & ependymal cells	Tenascin-C		Chronic(11)	
(Yu and Schachner 2013)	located in and around the central canal and in white matter	neuron- and glia-like cells	Syntenin-a		Chronic(6 & 11)	Syntenin-a expression was associated with synapse formation
(Goldshmit, Matteo et al. 2012)	along the midline and central canal of the spinal cord	large neuronal-like cells and glia-like cells	LPA(Lysophosphatidic Acid)		Acute and chronic(not exact time	Inhibit the normal regenerative response after SCI. LPA mediates neuronal death, microglial activation, astroglial proliferation, and GFAP up-regulation and, over the long term, inhibits neurite sprouting
(Becker, Bernhardt et al. 1998)	nucleus of the medial longitudinal fascicle(NMLF), the intermediate reticular formation, and the magnocellular octaval nucleus,	L1.2: glial cells(most likely astrocytes or Oligodendrocytes) All three molecules: motor neurons In the spinal cord caudal to the lesion site, L1.2 but not L1.1 or NCAM mRNA expression was increased in putative glial cells.	L1.1, L1.2, and neural cell adhesion molecule (NCAM)		Chronic(3 &7 and 14dpl) Chronic(7 and 14dpl) Chronic(7 and 14dpl)	Acts on axonal regrowth Mauthner cells: Mauthner cells are a bilateral pair of cerebrospinal projection neurons that could be individually identified in all preparations. These cells showed L1.1 upregulation after distal lesion.

	nucleus ruber, the nucleus of the lateral lemniscus, and the tangential nucleus					
(Barreiro-Iglesias, Mysiak et al. 2015)	Dopaminergic axons derived from diencephalon Serotonergic axons derived from descending axons of brain stem	Dopaminergic axons derived from diencephalon Serotonergic axons derived from descending axons of brain stem	serotonin and dopamine			Serotonin affects PMN-like ERG cells. They both act on regeneration, and promote motor neuron generation. PMN-like ERG cells and oligodendrocytes express serotonin receptors.
(Becker, Lieberoth et al. 2004)	NMLF and gray matter	neurons	L1.1		Chronic(1-6wpl)	Axonal regrowth
(Becker and Becker 2001)					Chronic	Pathway of descending axons switches from the white matter to the gray matter. A macrophage/microglia response was observed caudal to the transection at 2dpi.
(Borman, Roth et al. 1999)		Regenerating neurons neuron-associated glial cells	zfNLRR			Regulate the adhesive strength during neuronal growth in response to the extracellular environment.
(Briona, Poulain et al. 2015)	Blastema	Radial Glia cells	Wnt/ß-catenin signaling		Chronic(1-7dpi)	is required for progenitors to differentiate into neurons. Affects axonal regrowth and neurogenesis

(Fang, Pan et al. 2014)	NMLF HMGB1 was strongly upregulated in blood vessels at 6 days and decreased at 11 and 21 days after SCI compared to the sham injury group.	endothelial cells and motoneurons	HMGB1		Acute 12h and chronic(6 &11)	Acute effects: inflammation, HMGB1 translocates From nuclei into the cytoplasm of spinal motoneurons at 4 and 12 h (acute stage) following SCI, then accumulates in the nuclei of motoneurons during the ensuing chronic stage Chronic effects: HMGB1 promotes recovery from SCI not only through enhancing neuroregeneration, but also by increasing angiogenesis. HMGB1 released from microglia and/or degenerating neurons produces multiple inflammatory and neurotoxic factors. In contrast to the neurogenic properties of HMGB1, HMGB1 is highly inflammatory upon release from cells. HMGB1 is initially released to promote neurogenesis and cell survival, then downregulated to minimize inflammation during recovery. HMGB1 is involved in axonal regrowth. This molecule has dual effects.
(Hui, Nag et al. 2015)	Sox2 in grey matter, particularly in the ventricular zone around the central canal and in subependyma. A few cells near pial membrane are also showing colocalization of Sox2 and BrdU	Ependymal: sox2 injury epicenter, ependymal cells, neuron like cells, glia cells: pou5f1 gray matter cells: msx-b glia like cells and mesenchymal cells near to the ependymal bulb: vimentin	Sox2 pou5f1 vimentin msx-b		Chronic	Highest level of sox2 at day3. Sox2 is a neural progenitor marker. Highest proliferation at day7. Existence of progenitor cells both in early stage like 3, 7 dpi cord and in late stage like 10 dpi cord. Many newly formed Schwann cells in 10 dpi. Vimentin: 7dpi Msx-b: 3dpi Pou5f1: 7dpi

(Dias, Yang et al. 2012)	Ventromedial spinal cord(inhibits motor neuron generation at this place) Around the central canal.	***Her9*** dorsal half of the ventricular zone in the lesioned spinal cord. *Her4.5 in scattered cells.* *Motor neuron progenitors* ***Her4.5***was upregulated from undetectable levels in scattered cells ***Her9*** was expressed in dorsal midline cells in the unlesioned spinal cord and this expression domain expanded to comprise the dorsal half of the ventricular zone in the lesioned spinal cord	Notch signaling	*hairy-related* (*her*) genes	Chronic	14dpi: presence of newly generated motor neurons Notch signaling inhibits motor neuron generation and progenitor proliferation after injury. DAPT treatment increases the number of newly generated motor neurons and proliferating progenitor cells in the lesioned spinal cord by inhibiting notch signaling. Additional signals from the lesion are needed to make spinal progenitor cells competent to react to manipulations of Notch signaling. Receptors *notch1a* and *notch1b* were upregulated from undetectable levels around the central canal. The notch ligand *jagged1b* was upregulated from undetectable levels in ventricular cells of the dorsal midline and the ligand *deltaC* was upregulated in cells in the vicinity of the ventricular zone, predominantly in the ventral half of the spinal cord.
(Goldshmit, Sztal et al. 2012)		Glia cells, neurons	Fgf-signaling	*Fgf3 main downstream effector of fgf signaling(p-MAPK)*	3wpl 1dpl	zebrafish glia form an elongated morphology that joins the resected regions of the spinal cord. Glia bridge formation in 2-3wpl.
(Guo, Ma et al. 2011)		Ependymal cells, Newly generated neurons	Sox11b Nestin Ascl1 oct4(pou family)		Chronic and acute 12h& 3&11dpl	Sox11b & oct4 regulates nestin and Ascl1 expression.
(Kuscha, Barreiro-Iglesias et al. 2012)	Raphe nucleous: serotonergic Diencephalon:	tyrosine hydroxylase-positive (TH1β; mainly dopaminergic), and serotonergic (5-HTβ)	molecules: dopamine and serotonin		Chronic(evaluated just in some specific	TH1β and 5-HTβ innervations are massively altered in the successfully regenerated spinal cord of adult zebrafish. Major sources of dopamine in the spinal cord are TH1β axons coming from the brain and that TH1β terminals are mostly dopaminergic.

	dopaminergic				time points not at all times)	
(Hui, Dutta et al. 2010)		Apoptotic cell death at 6h and 1and 3dpl			Acute and chronic	3dpi: damaged axons are shown about to be engulfed by macrophages.
(Kuscha, Frazer et al. 2012)		interneurons: V2 interneurons(differentiated from the V2 interneuron progenitor (p2) domain)	Pax6 and Nkx6.1		chronic:2 and 6 wpl	In the ventricle, their place of origin.
(Briona and Dorsky 2014)		Neurogenic spinal radial Radial glia progenitors (dbx1a:GFP+)			Chronic (after 5dpl)	These cells are progenitors of neurons and also interneurons after injury.

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