Journal of Chemical Neuroanatomy 71 (2016) 1–5

Contents lists available at ScienceDirect

Journal of Chemical Neuroanatomy

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

BCL11B/CTIP2 is highly expressed in GABAergic interneurons of the mouse somatosensory cortex

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ARTICLE INFO

Article history:
Received 1 September 2015
Received in revised form 22 November 2015
Accepted 11 December 2015
Available online 14 December 2015

Keywords:
B-cell lymphoma/leukemia 11B
CTIP2
Mouse
Somatosensory cortex
GABAergic interneurons

ABSTRACT

In the nervous system, BCL11B is crucial for the development of deep layer corticospinal projection neurons and striatal medium spiny neurons and is often used as a marker for the aforementioned cell types. However, the expression of BCL11B in subtypes of non-excitatory neurons in the primary somatosensory cortex (S1) has not been reported in the mouse. In this study we show that BCL11B is extensively expressed in S1 GABAergic interneurons, throughout the three main subgroups (somatostatin-, parvalbumin- and 5HT3a-expresssing). Almost all BCL11B positive cells in the upper S1 layers were GABAergic interneurons and surprisingly, almost 40% of the BCL11B positive neurons in layer V were GABAergic interneurons. Single cell mRNA sequencing data revealed higher *Bcl11b* expression in S1 interneurons compared to deep layer pyramidal neurons. The highest levels of *Bcl11b* expression were found within the 5HT3a population, specifically in putative neurogliaform interneuron subclasses (5HT3a-positive but not expressing vasoactive intestinal peptide). In the light of our findings we suggest caution using BCL11B as a single marker to identify neurons.

1. Introduction

To elucidate the function of the brain despite its complexity we need to understand the diversity of the cellular components, the neurons, and how they interact with each other in networks. To investigate the outcome of a manipulation to the system, known molecular markers are often used as a proxy for cell identity and therefore also function. B-cell leukemia/lymphoma 11B (BCL11B) also called Chicken ovalbumin upstream promoter transcription factor interacting protein 2 (CTIP2) or radiation-induced tumor suppressor gene 1 (Rit1), is a zinc finger transcription protein important for development of the central nervous system, skin, hair and lymphocytes (Kominami, 2012). Mutant mice lacking both alleles with *Bcl11b* die shortly after birth with defects in the immune system, central nervous system, skin and hair (Kominami, 2012). The action of BCL11B is known to be necessary for the development of corticospinal projection neurons mainly found in cortical layer V (Arlotta et al., 2005; Chen et al., 2008; Leyva-Diaz and Lopez-Bendito, 2013). In the absence of BCL11B these neurons fail to project axons into the spinal cord (Arlotta et al., 2005). BCL11B is also crucial for the development of medium spiny neurons (MSNs) in the striatum (Arlotta et al., 2008). Thus BCL11B is often used as a marker for deep layer subcerebral projection neurons (Leyva-Diaz and Lopez-Bendito, 2013; Alcamo et al., 2008; Leone et al., 2015; Molyneaux et al., 2005; Britanova et al., 2008) as well as striatal MSNs (Delli Carri et al., 2013). In the striatum, in addition to the striatal MSNs, BCL11B was recently shown to be partly expressed by at least one group of GABAergic interneurons (Muñoz-Manchado et al., 2016), but the expression in subtypes of non-excitatory neurons in the neocortex has not been characterized.

Cortical inhibitory neurons is a very heterogeneous group of neurons. This diversity has been studied using features such as morphology, electrophysiology, membrane properties, molecular markers, cellular location, connectivity and/or recently single cell RNA-sequencing (Markram et al., 2004; Fishell and Rudy, 2011; Rudy et al., 2011; Ascoli et al., 2008; Zeisel et al., 2015; DeFelipe et al., 2013). Although complex, GABAergic interneurons can be divided based on three non-overlapping molecular markers (Rudy et al., 2011). These are: parvalbumin (PV), a calcium-binding protein which is expressed by 40% of the neocortical GABAergic interneuron, and the neuropeptide somatostatin (SST), and 5-hydroxytryptamine (serotonin) receptor 3A (5HT3a), each marking two groups of 30% of the interneurons. The group expressing 5HT3a can be further subdivided with regards to the presence or absence of vasoactive intestinal peptide (VIP) which constitute 40% and 60% of 5HT3a expressing interneurons respectively (Lee et al., 2010). These two 5HT3a-populations still contain more than one

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http://dx.doi.org/10.1016/j.jchemneu.2015.12.004
0891-0618/

subtype of interneurons based on morphology and firing patterns (Prönneke et al., 2015; Miyoshi et al., 2010). In this study we show that BCL11B is extensively expressed in neocortical GABAergic interneurons of all three major subgroups and thus its expression is not restricted to subcerebral projections cells.

2. Material and methods

2.1. Mouse lines

In this study we used outbred wild type CD1 mice (6 adult animals of both sexes) and the transgenic mouse line 5HT3a^{EGFP} (GENSAT project at Rockefeller University) on a CD1 background (5 animals, age P21-P22, females). All animal handlings were according to local ethical regulations under the permit N567/11 from *Stockholms norra djurförsöksetiska nämnd*, Sweden.

2.2. Tissue preparation

Mice were transcardially perfused with 4 °C phosphate buffered saline (PBS) solution followed by ice cold 4% paraformaldehyde (PFA) in PBS. The brains were dissected and post-fixated for 1 h in 4% PFA in PBS at 4 °C. These were then rinsed in PBS and cryoprotected in 30% sucrose in PBS overnight at 4 °C. Cryoprotected brains were embedded in optimal cutting temperature (OCT) compound, frozen and kept at −80 °C. Subsequently 40 μm thick coronal sections were obtained in a Leica cryostat and kept free floating in cryoprotect solution (30% Ethylene Glycol, 30% Glycerol, 10% 1× PBS in H_2O) at −20 °C until immunostaining.

2.3. Immunohistochemistry

The brain sections were washed in PBS, 0.1% Tween20 in PBS, 0.3% Tween20 in PBS and PBS again at room temperature and subsequently blocked in blocking buffer (5% normal goat serum (NGS), 0.5 M NaCl, 2.5% bovine serum albumin (BSA) and 0.3% Tween20 in 1x PBS) for one hour at room temperature, followed by incubation with the primary antibodies overnight at 4 °C in blocking buffer without NGS. Wild type mice were double stained with rat anti-BCL11B (1:500; Abcam; monoclonal [25B6]) (Arlotta et al., 2005; Leone et al., 2015; Molyneaux et al., 2005) and either rabbit anti-VIP (1:500; Immunostar; polyclonal), mouse anti-PV (1:1000; Sigma; monoclonal [PARV-19]) or rabbit anti-SST (1:500; Diasorin; polyclonal). 5HT3a^{EGFP} mice were stained with rat anti-BCL11B (as above) and the EGFP signal was detected by using chicken anti-GFP (1:2000; Abcam; polyclonal). After being washed in 0.1% Tween20 in PBS at room temperature the sections were then incubated in appropriate secondary antibodies (raised in goat) conjugated with Alexa Fluor dyes 488, 555 or 647 (Invitrogen) overnight at 4 °C. After washes with 0.1% Tween20 in PBS nuclear counterstaining was performed on all sections with 4,6-diamidino-2-phenylindole (1:1000 in H_2O; Molecular Probes) for 10 min at room temperature. The free floating sections were then mounted with Fluoromount-G (Southernbiotech) on microscope slides.

2.4. Image acquisition and data collection

The images were acquired by Carl Zeiss LSM700 confocal microscope with Plan Apochromat 10X/0.45 DIC II M27 (resolution: 0.74 μm) objective. We used automatic stitching made possible by the motorized stage to acquire a single image spanning all 6 layers of cortex (average area of cortex in each image was $0.77 \pm 0.12\ mm^2$). Two images from S1 per animal were acquired. S1 was determined according to The Mouse Brain in Stereotaxic Coordinates (Franklin and Paxinos, 2008). The cells were then counted using the ImageJ software (U.S. National Institutes of Health). The cortical area was manually delineated and measured by ImageJ's area measurement function. All data is presented as means ± standard error of the mean (SEM).

2.5. Single cell mRNA-sequencing

The single cell mRNA data from somatosensory cortex was obtained from Zeisel et al. (2015). The *Sst* neurons were defined as interneurons with more than 100 *Sst* mRNA molecules. The *Vip* neurons were defined as interneurons with more than 50 *Vip* mRNA molecules. All the FACS sorted EGFP$^+$ interneurons from the 5HT3a^{EGFP} mice excluding any cell without any *Htr3a* (5HT3a gene) mRNA molecules were defined as *Htr3a* interneurons. The different pyramidal neurons were defined as the classification in Zeisel et al. (2015). Since the expression data is not normally distributed we

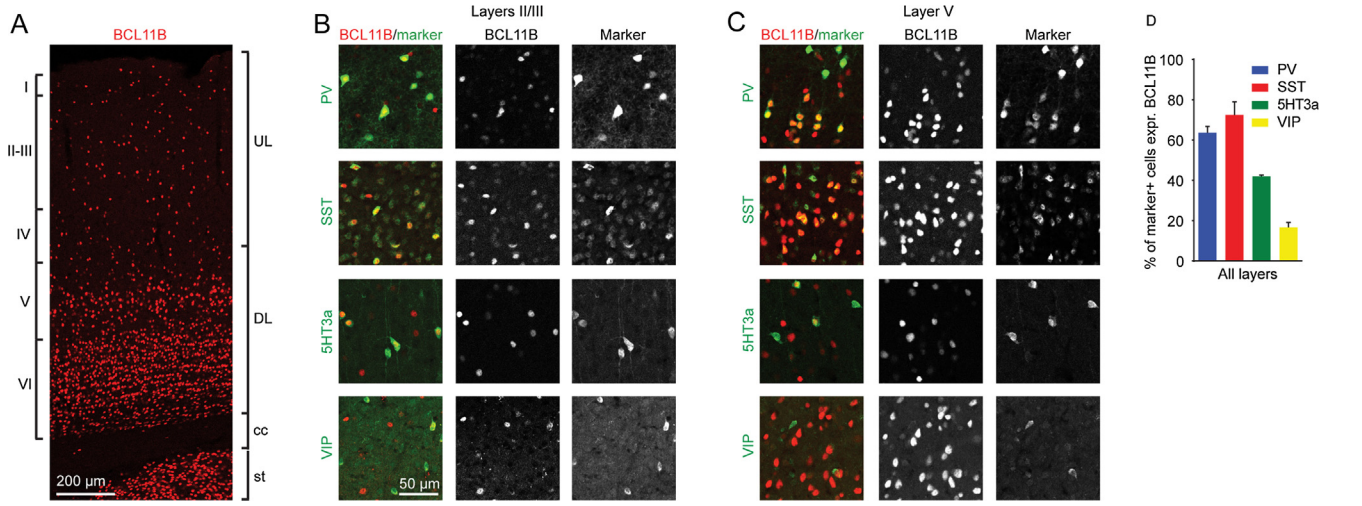

Fig. 1. Immunohistochemistry analysis reveals BCL11B was expressed by GABAergic interneurons. (A) Representative image of a coronal section immunostained for BCL11B showing the entire depth of S1 and parts of corpus callosum and striatum. (B) Representative images of co-expression of BCL11B and the different GABAergic interneuron subgroup markers in the layers II/III of S1. (C) Representative images of co-expression of BCL11B and the different GABAergic interneuron subgroup markers in the layer V of S1. Same scale as B. (D) Proportion of the different GABAergic interneuron subgroups which express BCL11B in all layers. (BCL11B + marker)/marker. Error bars represent SEM. Abbreviations: 5HT3a = 5-hydroxytryptamine receptor 3A, BCL11B = B-cell leukemia/lymphoma 11B, cc = corpus callosum, DL = deep layers, PV = parvalbumin, S1 = primary somatosensory cortex, SST = somatostatin, st = striatum, UL = upper layers, VIP = vasoactive intestinal peptide.

performed Mann Whitney *U*-test to compare the difference in *Bcl11b* mRNA expression between interneurons and deep layer pyramidal neurons (defined as S1PyrL5, S1PyrL5a, S1PyrL6, S1PyrL6b and S1PyrDL neurons as classified in Zeisel et al. (2015)). Data is presented with median and the interquartile range if not stated otherwise.

3. Results

3.1. BCL11B was expressed by all three major GABAergic interneuron subgroups

We used immunohistochemistry to determine the expression of BCL11B in the mouse S1. Although BCL11B is mainly expressed in the deep layers of the neocortex we could see a substantial number of neurons in the upper layers (layers I–IV) expressing BCL11B (Fig. 1A) . Immunohistochemical staining of the GABAergic interneuron subgroup markers PV ($n=5$), SST ($n=3$), and 5HT3aEGFP ($n=5$) in S1 revealed that on average there were 175.5 ± 17.1 PV-expressing neurons, 150.3 ± 11.7 SST-expressing neurons, and 147.6 ± 10.4 5HT3aEGFP positive neurons per square millimeter. The 5HT3aEGFP population can be further subdivided by the expression or absence of VIP and we found 38.2 ± 2.7 ($n=3$) VIP-positive neurons/mm^2. We then investigated the expression of BCL11B within the populations of GABAergic interneurons in a semi-quantitative manner. Our results showed that cells from all the major GABAergic subgroups expressed BCL11B to a large extent in both upper layers II/III and deep layer V (Fig. 1B and C). Counting across all layers, $64 \pm 3\%$ of PV-expressing neurons ($n=5$ animals; in total 1302 cells), $72 \pm 7\%$ of SST-expressing neurons ($n=3$ animals; in total 650 cells) and $42 \pm 1\%$ of 5HT3aEGFP positive neurons ($n=5$ animals; in total 1172 cells) expressed BCL11B (Fig. 1D). Within the 5HT3a-positive VIP-expressing population, $17 \pm 2\%$ of VIP-expressing neurons co-expressed BCL11B ($n=3$ animals, in total 180 cells) (Fig. 1D). We further analyzed the layer specific co-expression of BCL11B and the four interneuron markers across all six layers delineated using nuclear staining (Fig. 2A) finding that there was a slightly higher co-expression of BCL11B and each interneuron marker in the upper layers (Fig. 2A). We also analyzed the proportion of all BCL11B-expressing neurons within each layer that came from each major non-overlapping GABAergic subgroup (Fig. 2B and C) showing that the majority of cells expressing BCL11B in layers I–IV were GABAergic interneurons. Surprisingly, almost 40% of all the BCL11B expressing cells in layer V were GABAergic interneurons, mostly PV expressing (Fig. 2B). Of all the BCL11B expressing cells in the upper layers $33 \pm 3\%$ expressed PV, $30 \pm 3\%$ expressed SST and $20 \pm 2\%$ were 5HT3aEGFP neurons (Fig. 2C).

3.2. Bcl11b mRNA expression levels in interneurons and excitatory cells

Analyzing single cell mRNA-sequencing data from Zeisel et al. (2015) allowed us to analyze the *Bcl11b* mRNA expression levels between different S1 GABAergic interneurons and deep layer pyramidal neurons. One caveat with the used data set is that it contains few cells expressing *Pvalb* (parvalbumin gene) and therefore we cannot confirm the expression in this cell type. The *Htr3a* interneurons had the highest *Bcl11b* mRNA expression (3.0, interquartile range 0.0–9.0) among the GABAergic interneurons (Fig. 3A). The relatively low *Bcl11b* mRNA expression among the *Vip* interneurons (0.0, interquartile range 0.0–1.0) demonstrate that most of the *Bcl11b* mRNA in the *Htr3a* interneurons were expressed in the non-*Vip* expressing *Htr3a* interneurons. These non-*Vip* expressing neurons are mainly Reelin-positive multipolar cells including the neurogliaform cells (Rudy et al., 2011). A closer analysis of specific molecular subgroups expressing *Bcl11b* as delineated in Zeisel et al. (2015) showed that while we could observe transcripts scattered throughout most subgroups as suggested by immunohistochemistry, the putative neurogliaform subclasses Int11, Int12, Int14 and Int16 all uniformly expressed *Bcl11b* at higher levels (Fig. 3B). Layer V and Va pyramidal neurons showed a low *Bcl11b* mRNA expression level in the somatosensory cortex (1.0, interquartile range 0.0–2.8 and 0.0, interquartile range 0.0–0.0, respectively) (Fig. 3A). Interestingly, the *Bcl11b* mRNA expression in all *Bcl11b*$^+$ interneurons (6.0, interquartile range 2.0–10.0) was significantly higher than all the *Bcl11b*$^+$ deep layer pyramidal neurons (3.0, interquartile range 1.0–7.0) (Fig. 3C).

4. Discussion

In this study we have shown that BCL11B is highly expressed in several different subgroups of GABAergic interneurons and that they in fact constitute a majority of the cells expressing BCL11B in the upper layers of S1. As supported by previous studies (Arlotta et al., 2005; Chen et al., 2008; Leyva-Diaz and Lopez-Bendito, 2013; Alcamo et al., 2008; Leone et al., 2015; Molyneaux et al., 2005; Britanova et al., 2008), BCL11B was dominantly expressed in the deep layers of S1. Interestingly, almost 40% of the BCL11B positive neurons in layer V were also positive for GABAergic interneuron

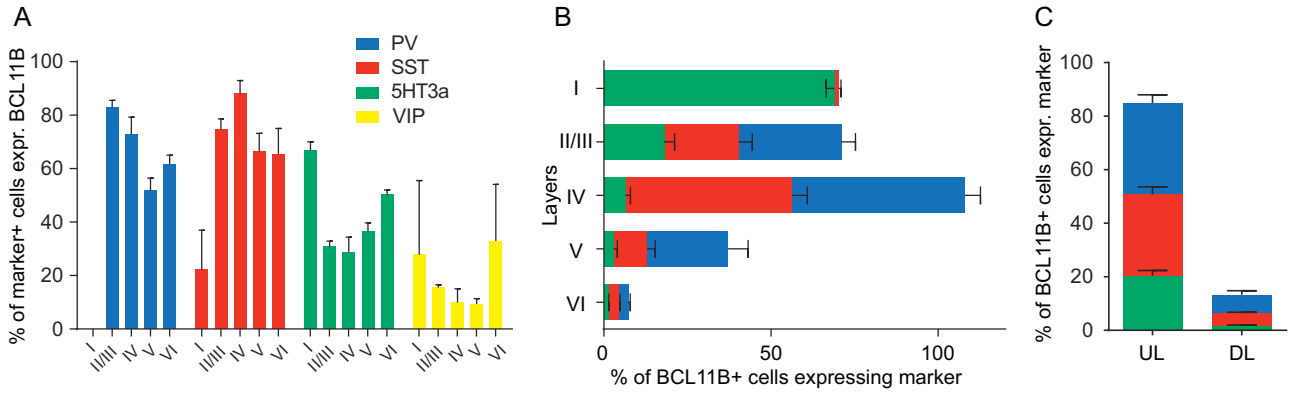

Fig. 2. BCL11B expression in GABAergic interneurons in S1. (A) Layer specific expression of BCL11B by GABAergic interneuron subgroup marker positive cells. (BCL11B + marker)/marker. (B) Layer specific proportion of BCL11B + cells expressing GABAergic interneuron subgroup marker. (BCL11B + marker)/BCL11B. (C) Proportion of BCL11B + cells expressing GABAergic interneuron subgroup marker divided in upper layers (layers I–IV) and deep layers (layers V–VI). (BCL11B + marker)/BCL11B. Error bars represent SEM. Abbreviations: 5HT3a = 5-hydroxytryptamine receptor 3A, BCL11B = B-cell leukemia/lymphoma 11B, DL = deep layers, PV = parvalbumin, S1 = primary somatosensory cortex, SST = somatostatin, UL = upper layers, VIP = vasoactive intestinal peptide.

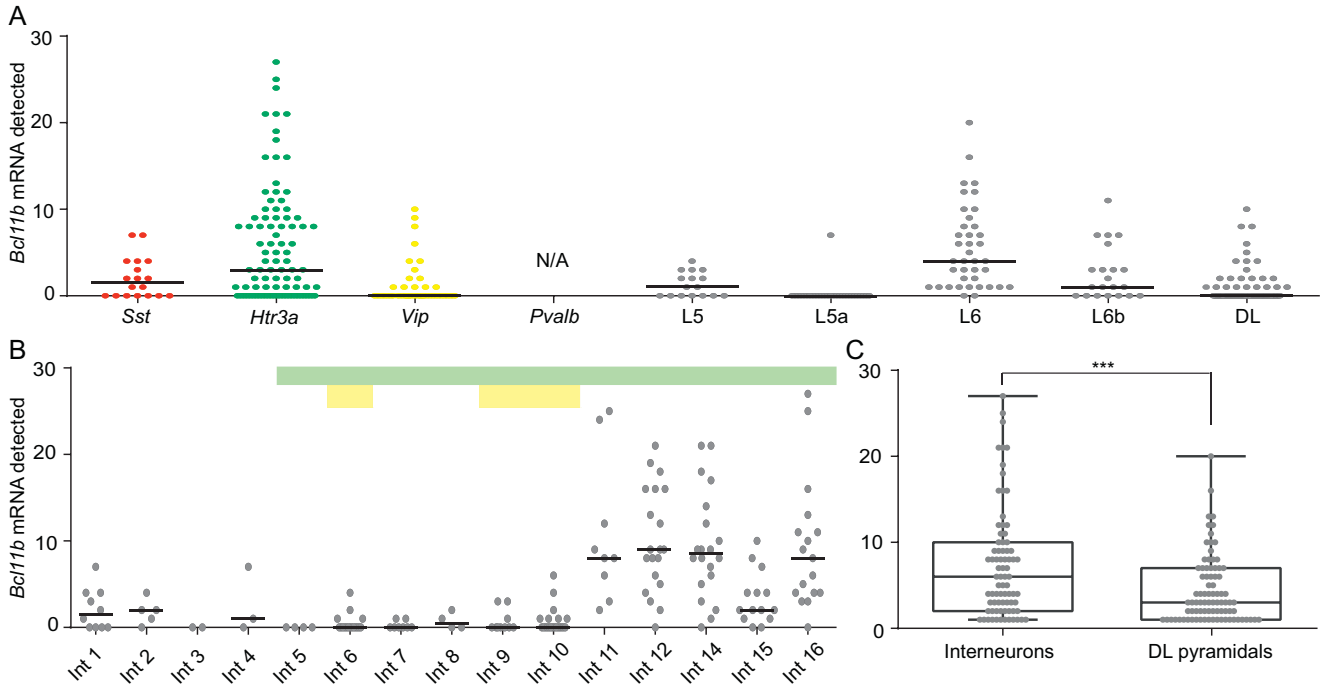

Fig. 3. *Bcl11b* mRNA expression in S1 by single cell mRNA-sequencing. (A) Dot plot showing *Bcl11b* mRNA molecules detected in different cell types. The horizontal lines show the median number of *Bcl11b* mRNA molecules in each cell type. (B) Illustrating the *Bcl11b* mRNA molecules detected in different interneuron subclasses (Int 1–Int 16) as defined in Zeisel et al. (2015). Color code above graphs denotes *Htr3a* expressing (green) and *Vip* expressing (yellow) subgroups. Horizontal lines represent the median values. (C) *Bcl11b* mRNA molecules detected in all interneurons ($n = 78$) and all deep layer pyramidal neurons ($n = 85$) with at least one *Bcl11b* mRNA molecule in S1. Boxplots represent minimum to maximum value. Box boundaries range from 25th to 75th percentile. Line inside box indicates the median. *** = $p = 0.0010$. All data in this Fig. is modified from Zeisel et al. (2015) with each dot representing one cell. Abbreviations: *Ht3ra* = 5-hydroxytryptamine receptor 3A, *Bcl11b* = B-cell leukemia/lymphoma 11B, DL = deep layers, Int = interneuron, L5 = layer V, L5a = layer Va, L6 = Layer VI, L6b = layer VIb, *Pvalb* = parvalbumin, S1 = primary somatosensory cortex, *Sst* = somatostatin, *Vip* = vasoactive intestinal peptide.

subgroup markers. This complements earlier studies that have suggested BCL11B mainly as a marker specifically expressed by subcerebral projection neurons in neocortical layer V (Arlotta et al., 2005; Alcamo et al., 2008; Leone et al., 2015; Molyneaux et al., 2005; Britanova et al., 2008). Although a powerful marker for subcerebral projection neurons, our study suggests that one has to be careful using single channel immunohistochemistry of BCL11B to assign neuronal identity.

In this study we did not perform staining of a pan-interneuronal marker such as GAD1. But previous work has shown that the interneuron markers used in this study are restricted to the GABAergic population and that by using the three main markers one can account for most, if not all, of the interneurons in S1 (Lee et al., 2010). Although the data presented in this study is from the primary somatosensory cortex of the mouse many molecular markers of interneuron subtypes are conserved across cortical areas and species (Ma et al., 2013; Gonchar et al., 2008). Indeed, a previous study using transgenic rats in which GABAergic cells are genetically labeled reported a considerable overlap with BCL11B in superficial layers of primary and secondary motor cortex (Ueta et al., 2014).

BCL11B is known to be important for the development of axonal projections of projection neurons in cortex. Is there a similar function in cortical interneurons where perhaps it influences local axonal projections? Why do not all neocortical interneurons express BCL11B? It is increasingly clear that the division into parvalbumin, somatostatin, VIP or 5HT3a groups does not catch the entire complexity of interneuron morphologies. Could there be general differences in features predicted by if the interneurons express BCL11B or not? If our hypothesis that Int11–16 mainly are neurogliaform cells is true, then the cells expressing *Bcl11b* at high levels, as measured by single cell sequencing, indeed do have a very different axonal arrangement than the non *Bcl11b*-expressing ones. Future studies of neurogliaform cells in the *Bcl11b* conditional knock-out mice are warranted.

5. Conclusions

BCL11B is expressed not only by corticospinal excitatory cells but also by inhibitory interneurons. BCL11B expression is seen in all three major subgroups (SST, PV and 5HT3a^{EGFP}) of interneurons in S1.

Author contributions

K.N. and J.H-L. designed the study. K.N., A.M-M. and J.H-L. wrote the paper. K.N. and A.M-M. performed experiments.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

We thank Tibor Harkany, Florent Ginhoux and Low Kian Soon Donovan for help with reagents and the “Click” facility at the Karolinska Institutet for providing imaging equipment. The study was funded by Swedish Research Council, StratNeuro and the Jeansson Foundation.

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