Transcriptional regulation of MGE progenitor proliferation by PRDM16 controls cortical GABAergic interneuron production

Miguel Turrero Garcia, José-Manuel Baizabal, Diana N. Tran, Rui Peixoto, Wengang Wang, Yajun Xie, Manal A. Adam, Lauren A. English, Christopher M. Reid, Salvador I. Brito, Matthew A. Booker, Michael Y. Tolstorukov and Corey C. Harwell
DOI: 10.1242/dev.187526

Editor: François Guillemot

Review timeline
Original submission: 17 December 2019
Editorial decision: 27 January 2020
First revision received: 1 October 2020
Accepted: 5 October 2020

Original submission

First decision letter

MS ID#: DEVELOP/2019/187526

MS TITLE: Transcriptional regulation of MGE progenitor proliferation by PRDM16 controls cortical GABAergic interneuron production

AUTHORS: Corey Harwell, Miguel Turrero Garcia, Jose-Manuel Baizabal, Diana Tran, Rui Peixoto, Wengang Wang, Yajun Xie, Manal Adam, Salvador Brito, Matthew Booker and Michael Tolstorukov

I have now received the reports three referees on your manuscript and I have reached a decision. The reports are appended below and you can access them online: please go to BenchPress and click on the 'Manuscripts with Decisions' queue in the Author Area.

As you will see, all the referees are enthusiastic about your work, but they also have significant criticisms and recommend a substantial revision of your manuscript before we can consider publication. If you are able to revise the manuscript along the lines suggested, which may involve further experiments, I will be happy to receive a revised version of the manuscript. Your revised paper will be re-reviewed by one or more of the original referees, and acceptance of your manuscript will depend on your addressing satisfactorily the reviewers' major concerns. Please also note that Development will normally permit only one round of major revision.

Please attend to all of the reviewers' comments and ensure that you clearly highlight all changes made in the revised manuscript. Please avoid using 'Tracked changes' in Word files as these are lost in PDF conversion. I should be grateful if you would also provide a point-by-point response detailing how you have dealt with the points raised by the reviewers in the 'Response to Reviewers' box. If you do not agree with any of their criticisms or suggestions please explain clearly why this is so.

Reviewer 1

Advance summary and potential significance to field

In their manuscript, Turrero Garcia and colleagues address important questions in cortical development. They follow upon an earlier study of the Harwell lab and investigate the role of PRDM16 in the regulation of cortical interneuron development. They use conditional mouse genetics approaches and delete PRDM16 specifically in Nkx2.1+ interneuron progenitors and their descendent postmitotic GABAergic interneurons. The authors analyzed the phenotype of PRDM16 loss of function at mature (P30) stages and found a significant reduction of Nkx2.1 lineage derived interneurons in most cortical layers. This phenotype is likely caused by a defect in MGE progenitor proliferation rather than loss by cell death of aberrant migration of nascent interneurons. The authors then analyzed how PRDM16 associates with cis-regulatory elements and compared their new data with their own previously published data set (E15 cortex) and with the data set from Sandberg et al 2016 (E13 MGE histone modifications). Intriguingly but perhaps not surprisingly they find similar but also convergent binding sites when they compare excitatory versus inhibitory neurons. These data very nicely extend the findings in Baizabal et al 2018. Finally, the authors analyzed expression levels of two genes from their data set and involved in neuronal differentiation (Pdzm3 and Gad2) and found increased expression levels upon Prdm16 deletion.
Overall the study by Turrero Garcia et al., is carried out very carefully and mostly well presented. The findings quite nicely extend the findings and concepts that the group reported before for excitatory projection neurons. A few points should however still be addressed in order to be entirely conclusive.

Comments for the author

Major point:
1. A major missing point is that the authors did not carry out gene expression analysis and compare control to conditions with Prdm16 deleted in Nkx2.1 lineage. Already the title of the manuscript raises the expectation for gene expression data. In my opinion such data is quite important for this paper. While PRDM16 binding sites may indicate repression activities it would be actually important to see the consequent gene expression pattern in control and upon loss of PRDM16 function in cKO. Based on the data presented in Figure 5, RNAseq data would significantly add to the study in an essential way.

Minor points:
2. Throughout the paper the authors label the images with KO. I think it would be more appropriate to at least indicate cKO when the authors show panels where Prdm16 was conditionally deleted by using Nkx2.1-Cre.

3. Figure 1B, it would be more informative to also have a high resolution image so that individual cells could be observed.

4. Figure 2A, the images appear to show a ‘stripe’ pattern. If this is due to image processing please revise or alternatively use different representative images.

5. Figure S3B, the images show an overview of interneuron migration. However, given the role of Prdm16 in excitatory neurons as shown in Baizabal et al., 2018, it would be informative and important to quantify the migration of interneurons in control versus cKO.

6. The authors generate retrovirus-labeled clones to analyze proliferation dynamics in MGE progenitors. While the data is in principle convincing, the authors should probably mention in the discussion that the overall clone size (and thus also the effect of Prdm16 deletion) is likely an underestimate due to potential silencing. Such phenomenon has been observed and/or discussed previously in a number of papers (e.g. Brown et al., 2011, Science or Ciceri et al., 2013, Nat Neuro) and in fact in an earlier paper by Harwell et al., 2015 and the companion paper by Mayer et al., 2015, both in Neuron.

7. In the discussion the authors briefly mention an interesting point: Genes that are regulated by PRDM16 in both cortical and MGE progenitors such as Pdzm3 could have a conserved molecular but

different overall cellular function. This is an important aspect of the entire study and deserves more in depth elaboration and discussion.

8. Their earlier study is mostly cited as Baizabal et al., 2018 but sometimes as Baizabal et al., 2017 (e.g. in the paragraph 'PRDM16 downregulates neuronal differentiation genes'). If this is not a typo, the Baizabal et al., 2017 reference is missing.

Reviewer 2

Advance summary and potential significance to field

In this manuscript, the authors have investigated the role of the transcriptional regulator PRDM16 in progenitors of the mouse MGE. Their previous work has shown that this factor controls proliferation and neurogenesis in cortical progenitors, in part, by regulating the epigenetic status of certain regulatory regions controlling neuronal development genes. The interneurons of the cortex derive largely from the MGE and CGE. In this paper, the requirement of PRDM16 in MGE progenitors is examined using a conditional allele together with Nkx2.1-cre mice. Using this approach, the authors show that loss of PRDM16 alters proliferative capacity in MGE progenitors and leads to significant reductions in the MGE-derived parvalbumin (PV) and somatostatin (SST) interneurons. Accordingly, this results in reduced inhibitory inputs to the cortical pyramidal neurons in the PRDM16 mutants, despite an apparent increase in CGE-derived interneurons. Their ChIP-seq studies show that PRDM16 associates with regulatory regions of genes involved in neuronal development/function which overlap with those previously identified in cortical progenitors (e.g. Pdzrn3) and others that are unique to the MGE lineage (e.g. Gad2). These genes are upregulated in the PRDM16 mutants suggesting precocious neurogenesis in conjunction with the reduced proliferative capacity of the MGE progenitors.

Comments for the author

This study is a well done and it adds to the literature in a very meaningful way. I believe it is deserving of publication in Development. However, I have a few questions/suggestions for the authors to consider before publication.

Are all neuronal populations derived from the MGE (e.g. striatal interneurons and globus pallius neurons) reduced in the PRDM16 mutants similar to the PV and SST cortical interneurons? I know this paper is focused on cortex but some mention of this would be good.

It seems clear that MGE proliferation is altered in the PRDM16 mutants, however, it would be interesting if the authors could examine the impact on each of the progenitor subtypes. It almost seems in Fig. 4A that there are more apical progenitors (APs) in the VZ of the mutant.
Are the subapical progenitors (SAPs) in the VZ (Pilz et al, 2013) reduced even more? The authors could examine this by co-staining PH3 with Ascl1. Perhaps a reduction in SAPs accounts for both the reduced PH3 in the VZ as well as the SVZ (i.e. basal progenitors).

The peaks in Pdzrn3 between the MGE and cortex are quite clear. However, the boxed peaks in Gad2 (Fig. 6B) seem rather different between the 2 MGE replicates. In fact, there may be another peak in both replicates to the left (i.e. over the "d" in Gad2) which is completely missing in the cortex data. Have the authors done ChIP-PCR to confirm any of these peaks?
Moreover, the authors mention that Ascl1 binding sites are enriched in many of the PRDM16 peaks. Do the called PRDM16 peaks in Gad2 overlap with called peaks in Ascl1 ChIP studies (Castro et al 2011)? If they could overlay the called Ascl1 peaks with their own, it would be quite useful.

This is a minor point but why did the authors only look at inhibitory inputs on L2/3 cortical neurons? The interneurons are also reduced in L5/6 in the PRDM16 mutants – do those neurons show similarly reduced inhibitory inputs?

Reviewer 3

Advance summary and potential significance to field

Rev of Harwell 2020

This group recently published an outstanding paper on the role of PRDM16 in cortical development, and here are focusing on conditional loss of this gene in the ventral telencephalon.
The studies are solid, if pretty similar overall to the dorsal phenotype. A few comments below.

Comments for the author

Fig 3 legend. All values are displayed, with mean ± S.E.M. indicated in black. Mann-Whitney tests (panels I, J)

There are no panels I, J in the figure. More importantly since 3 mice of each genotype were compared, the N of the experiment cannot be more than 3. Mann-Whitney tests require a minimum of N=4 for one group and 5 for the other. It appears that the statistical N may have been the pooled number of neurons recorded, which would not be appropriate since within the same brain data from multiple neurons are not independent measures.

"Different subtypes of transit-amplifying MGE progenitors are biased towards the generation of PV+ and SST+ cortical interneurons (Petros et al., 2015),"
Actually, that paper used in vivo fate mapping of VZ versus SVZ neurogenesis, and in vivo enhancement or repression of this neurogenesis, to suggest that PV interneurons originate mainly from SVZ divisions whereas SST interneurons mainly arise from VZ divisions.

This paper does not address differential influences of outputs of VZ versus SVZ neurogenic events--the results are consistent with a reduction in VZ neurogenesis as well as, and consistent with VZ seeding of SVZ, SVZ neurogenesis.

Also, the comparison of NexCre results from previous paper to the Nkx2.1Cre results of the current paper is oversimplified. The discussion suggests greater effects on cortical SVZ neurogenesis than that in the current paper's MGE focus is suggestive of distinct roles of PRDM16 in the system. That may be accurate, but there are at least two important sources of variability that complicate this interpretation. First, it is not clear exactly when PRDM16 protein is eliminated in the two transgenics. From the current paper it appears some protein is present in the MGE as late at E13.5, and it is not clear when most of the decrease is first present. Second, MGE neurogenesis, while not studied in the detail of the neocortex, seems to precede that of neocortical neurogenesis, especially regarding the transition of most neuronogenic divisions shifting from the VZ to the SVZ (not MGE, but Sheth and P Bhide showed in LGE that shift occurs by 11.5 whereas various labs showed it occurs around a day later in neocortex). Thus the timing of loss of protein in the two models, in relation to the timing of the transition to the SVZ/transit amplifying cells being the main source of neuronogenesis, will complicate the comparisons between the models. Using a non-conditional if it lives long enough (to e13), or a nestin-cre that would hit both dorsal and ventral simultaneously, would help.

Regarding Fig. 6, the chip seq data suggests that PRDM16 represses genes expressed in differentiating neurons, and the data suggest ectopic expression of GAD2. It would be helpful to know whether that expression is occurring in mitotic cells. If so, that argues more forcibly for a role of PRDM16 at repressing neural genes. If not, PRDM16 function may be more related to maintaining cell cycle, as any gene whose loss result in cell cycle exit will result in upregulation of genes expressed by postmitotic cells.

First revision

Author response to reviewers' comments

We thank the reviewers for their helpful comments that have helped us to substantially improve our manuscript. After carrying out important additional experiments and further analyses we have been able to address most of the reviewers' concerns in our revised manuscript. Most notably, we have included RNA-Seq data to examine differential gene expression between the MGE of WT and *Prdm16* cKO embryos.

Below is a detailed answer to each point raised by the reviewers. We reference in our replies the new sections of text and figures and address the reviewers' specific criticisms. We have uploaded a color coded copy of our reply that includes a figure for addressing a comment from reviewer #2.

Reviewer 1
Major point:
A major missing point is that the authors did not carry out gene expression analysis and compare control to conditions with Prdm16 deleted in Nkx2.1 lineage. Already the title of the manuscript raises the expectation for gene expression data. In my opinion such data is quite important for this paper. While PRDM16 binding sites may indicate repression activities it would be actually important to see the consequent gene expression pattern in control and upon loss of PRDM16 function in cKO. Based on the data presented in Figure 5, RNAseq data would significantly add to the study in an essential way

We completely agree with the reviewer's suggestion that RNA-Seq analysis of *Prdm16* cKO would significantly add to our study. We have performed bulk RNA-Seq of MGE samples collected from control WT and cKO embryos at E14. This experiment gave us a deeper understanding of the molecular mechanisms by which *Prdm16* deletion causes the phenotypes we observed, which we have incorporated into the manuscript in the shape of a new Figure 5 and a corresponding section in the Results.

Minor points:
2. Throughout the paper the authors label the images with KO. I think it would be more appropriate to at least indicate cKO when the authors show panels where Prdm16 was conditionally deleted by using Nkx2.1-Cre.

We have changed the labeling to cKO throughout the entire manuscript to more clearly indicate the conditional deletion of *Prdm16* with our Nkx2.1-Cre mouse line.

3. Figure 1B, it would be more informative to also have a high-resolution image so that individual cells could be observed.

We have now included new high-resolution insets into panel 1B, corresponding to WT and cKO. This allowed us to highlight how PRDM16 is still present in the dorsal MGE, a region with no *Nkx2.1* expression.

4. Figure 2A, the images appear to show a 'stripe' pattern. If this is due to image processing please revise or alternatively use different representative images.

Unfortunately, these striations are a sectioning artifact produced by the particular machine that we used to obtain this set of brain sections. We have attempted to choose images that have less distracting striping patterns, but could not eliminate them completely.

5. Figure S3B, the images show an overview of interneuron migration. However, given the role of Prdm16 in excitatory neurons as shown in Baizabal et al., 2018, it would be informative and important to quantify the migration of interneurons in control versus cKO.

This is a very important point, which illustrates how the role of *Prdm16* might differ between cortical/pyramidal and MGE-derived interneurons. We have now quantified the extent of interneuron migration (see Figures S3B and C) and found no differences between WT and cKO

samples; we have incorporated this into the text accordingly.

6. The authors generate retrovirus-labeled clones to analyze proliferation dynamics in MGE progenitors. While the data is in principle convincing, the authors should probably mention in the discussion that the overall clone size (and thus also the effect of Prdm16 deletion) is likely an underestimate due to potential silencing. Such phenomenon has been observed and/or discussed previously in a number of papers (e.g. Brown et al., 2011, Science or Ciceri et al., 2013, Nat Neuro) and in fact in an earlier paper by Harwell et al., 2015 and the companion paper by Mayer et al., 2015, both in Neuron.

This is an excellent point, and we now make certain to account for this caveat in our discussion of this data.

7. In the discussion the authors briefly mention an interesting point: Genes that are regulated by PRDM16 in both cortical and MGE progenitors such as Pdzm3 could have a conserved molecular but different overall cellular function. This is an important aspect of the entire study and deserves more in-depth elaboration and discussion.

We have expanded upon this point using Pdzrn3 as a useful example. Pdzrn3 appears to be repressed by Prdm16 in both cortex and MGE. Pdzrn3 is critical for radial migration in the cortex, however migration is unperturbed in MGE derived neurons of Prdm16cKOs

8. Their earlier study is mostly cited as Baizabal et al., 2018 but sometimes as Baizabal et al., 2017 (e.g. in the paragraph 'PRDM16 downregulates neuronal differentiation genes'). If this is not a typo, the Baizabal et al., 2017 reference is missing.

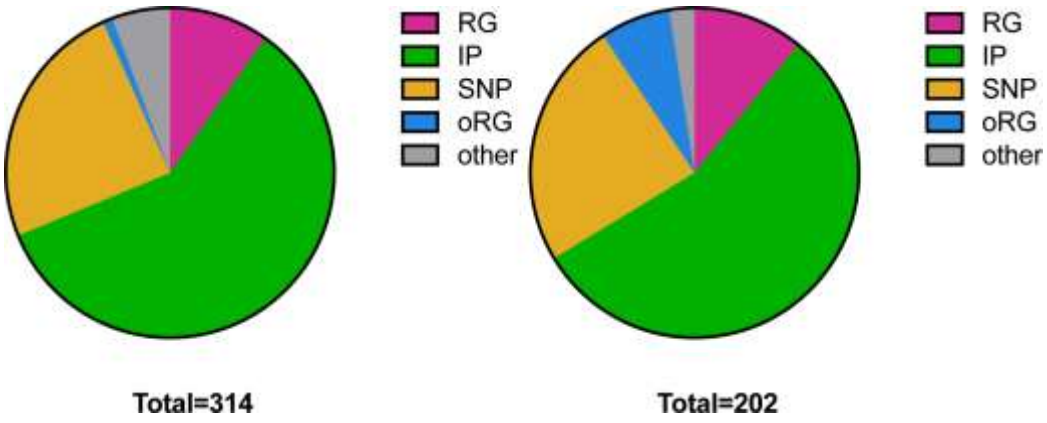
We have now corrected this error.

Reviewer 2

Are all neuronal populations derived from the MGE (e.g. striatal interneurons and globus pallius neurons) reduced in the PRDM16 mutants similar to the PV and SST cortical interneurons? I know this paper is focused on cortex but some mention of this would be good.

We thank the reviewer for bringing attention to other forebrain structures that are populated by MGE-derived cells. In addition to our extensive counting of interneurons in the hippocampus (Figure S1A-H), we have also included counts showing reduction of tdT+ cells in both the striatum (Figure S1I) and the nucleus accumbens (Figure S1J).

It seems clear that MGE proliferation is altered in the PRDM16 mutants, however, it would be interesting if the authors could examine the impact on each of the progenitor subtypes. It almost seems in Fig. 4A that there are more apical progenitors (APs) in the VZ of the mutant. Are the subapical progenitors (SAPs) in the VZ (Pilz et al, 2013) reduced even more? The authors could examine this by co-staining PH3 with Ascl1. Perhaps a reduction in SAPs accounts for both the reduced PH3 in the VZ as well as the SVZ (i.e. basal progenitors).



When we quantified pH3+ cell density in eight WT and seven cKO samples for each experimental group we found a significant reduction in both VZ and SVZ/MZ. Although not included in our primary data we attempted to classify progenitor types based upon morphology of our GFP retrovirus labeled clones as well as pVim staining. We did not detect a significant difference in the ratio of apical versus subapical progenitors (see graphs above - WT is on the left, cKO on the right). This is consistent with our hypothesis that loss of *Prdm16* leads to reduced self-renewing RG cell divisions and a shift towards more direct neurogenic divisions. This would lead to the reduction of both apical and basal progenitor divisions as we observe.

The peaks in Pdzrn3 between the MGE and cortex are quite clear. However, the boxed peaks in Gad2 (Fig. 6B) seem rather different between the 2 MGE replicates. In fact, there may be another peak in both replicates to the left (i.e. over the "d" in Gad2) which is completely missing in the cortex data. Have the authors done ChIP-PCR to confirm any of these peaks?

We agree with the reviewer, even though *Gad2* locus peaks met our statistical enrichment threshold for being defined as a reproducible peak they were not very visually compelling. We have replaced *Gad2* with *Lmo1*, an MGE-specific PRDM16 target gene that was also found to be differentially expressed in our *Prdm16* cKO RNA-seq. *Lmo1* displays a clear peak near its TSS, as shown in Figure 7B. This change allowed us to elaborate on the lineage-specific genetic programs that are regulated by *Prdm16*.

Moreover, the authors mention that Ascl1 binding sites are enriched in many of the PRDM16 peaks. Do the called PRDM16 peaks in Gad2 overlap with called peaks in Ascl1 ChIP studies (Castro et al 2011)? If they could overlay the called Ascl1 peaks with their own, it would be quite useful.

We agree with the reviewer's helpful suggestions and have now included an overlay of the genomic loci and the nearest associated genes of overlapping ASCL1 and PRDM16 peaks (see Table S7). One major caveat of this analysis is that the genome-wide data from Castro *et al.* is derived from a neural stem cell line (NS5), while ours was obtained from mouse tissue. Despite this, the data provides support for the hypothesis that PRDM16 may derive some part of its specificity in genomic associations through interactions with bHLH transcription factors.

This is a minor point but why did the authors only look at inhibitory inputs on L2/3 cortical neurons? The interneurons are also reduced in L5/6 in the PRDM16 mutants – do those neurons show similarly reduced inhibitory inputs?

We decided to record from L2/3 pyramidal neurons due to both practical reasons (they are easily identifiable layers with an abundance of recordings in the literature for comparison) and due to the data presented in Figure 2, where we described a potentially compensatory increase of CGE-derived reelin+ interneurons specific to upper layers (layers 1 and 2/3); recording from L2/3 proved that this increase is not sufficient to maintain the inhibitory input onto cKO excitatory cells at WT levels.

Fig 3 legend. All values are displayed, with mean ± S.E.M. indicated in black. Mann- Whitney tests (panels I, J). There are no panels I, J in the figure.

This was an unfortunate mistake carried over from a previous version of the figure. We have corrected it.

More importantly since 3 mice of each genotype were compared, the N of the experiment cannot be more than 3. Mann-Whitney tests require a minimum of N=4 for one group and 5 for the other. It appears that the statistical N may have been the pooled number of neurons recorded, which would not be appropriate since within the same brain data from multiple neurons are not independent measures.

In the mISPC recordings shown in Figure 3, sample size (n) refers to individual neurons and not mice; this is a valid and standard approach in the field as these recordings compare properties of similar neural populations (L2/3 pyramidal cells) across genotypes, not across animals. An exception to this would be if the experimental manipulation itself could induce variability across

animals of the same group (for example when using viral strategies for induction of transgenes), but this is not a concern when comparing transgenic animals with tissue-specific deletions.

Different subtypes of transit-amplifying MGE progenitors are biased towards the generation of PV+ and SST+ cortical interneurons (Petros et al., 2015)," Actually, that paper used in vivo fate mapping of VZ versus SVZ neurogenesis, and in vivo enhancement or repression of this neurogenesis, to suggest that PV interneurons originate mainly from SVZ divisions whereas SST interneurons mainly arise from VZ divisions.

This paper does not address differential influences of outputs of VZ versus SVZ neurogenic events--the results are consistent with a reduction in VZ neurogenesis as well as, and consistent with VZ seeding of SVZ, SVZ neurogenesis

We have revised this section of the discussion to more accurately reflect the findings of Petros et al. We agree that our findings are indeed consistent with a role for *Prdm16* in VZ proliferation that leads to a reduction in seeding and subsequent SVZ neurogenesis.

Also, the comparison of NexCre results from previous paper to the Nkx2.1Cre results of the current paper is oversimplified. The discussion suggests greater effects on cortical SVZ neurogenesis than that in the current paper's MGE focus is suggestive of distinct roles of PRDM16 in the system. That may be accurate, but there are at least two important sources of variability that complicate this interpretation. First, it is not clear exactly when PRDM16 protein is eliminated in the two transgenics. From the current paper it appears some protein is present in the MGE as late at E13.5, and it is not clear when most of the decrease is first present. Second, MGE neurogenesis, while not studied in the detail of the neocortex, seems to precede that of neocortical neurogenesis, especially regarding the transition of most neuronogenic divisions shifting from the VZ to the SVZ (not MGE, but Sheth and P Bhide showed in LGE that shift occurs by 11.5 whereas various labs showed it occurs around a day later in neocortex). Thus the timing of loss of protein in the two models, in relation to the timing of the transition to the SVZ/transit amplifying cells being the main source of neuronogenesis, will complicate the comparisons between the models. Using a non- conditional if it lives long enough (to e13), or a nestin-cre that would hit both dorsal and ventral simultaneously, would help.

Nkx2.1-Cre expression in this line is reported to be detected as early as E10.5 (https://www.jax.org/strain/008661; Xu et al 2008). Emx1-Cre in the pallium (Gorski et al., 2004) is detected at a similar E10.5 timepoint. *Prdm16* is specifically expressed in radial glia and not neuroepithelial cells, which transition into neurogenic radial glia around E10.5. Cre recombination at E10.5 should largely coincide with or precede the onset of *Prdm16* expression in neurogenic radial glia in both the cortex and MGE. We hope that our higher magnification immunohistochemistry images shown in Figure 1 clearly demonstrate that PRDM16 in the MGE ventricular zone is absent from *Prdm16* cKO, with the exception of the dorsal MGE, which does not express Nkx2.1-Cre.

We very much agree with the reviewer that both the timing and the magnitude of the contribution of the SVZ (i.e., the number of proliferative divisions) between the cortex and MGE are likely quite different, however the general organization of progenitors (RG make up the bulk of apical progenitors and give rise to more differentiated progenitors in the SVZ) is largely conserved. However, loss of *Prdm16* from the pallium with Emx1-Cre and subpallium with Nkx2.1-Cre should occur at the start of neurogenesis, allowing us to observe the impact of *Prdm16* loss on the entire period of neurogenesis.

Regarding Fig. 6, the chip seq data suggests that PRDM16 represses genes expressed in differentiating neurons, and the data suggest ectopic expression of GAD2. It would be helpful to know whether that expression is occurring in mitotic cells. If so, that argues more forcibly for a role of PRDM16 at repressing neural genes. If not, PRDM16 function may be more related to maintaining cell cycle, as any gene whose loss result in cell cycle exit will result in upregulation of genes expressed by postmitotic cells.

Upregulated gene expression is likely occurring in both progenitors and neurons, as we showed in Baizabal et al. There are a number of PRDM16 target genes that are misexpressed at every stage

of the lineage. In particular we previously showed that *Pdzrn3* was misexpressed in Pax6+ cortical progenitors in the VZ and in neurons in the intermediate zone of the cortex. The upregulation that we show in the VZ with smFISH is likely derived from progenitors as well since we know that the overall structure of the MGE VZ in *Prdm16* cKO is still intact.

Second decision letter

MS ID#: DEVELOP/2019/187526

MS TITLE: Transcriptional regulation of MGE progenitor proliferation by PRDM16 controls cortical GABAergic interneuron production

AUTHORS: Corey Harwell, Miguel Turrero Garcia, Jose-Manuel Baizabal, Diana N Tran, Rui Peixoto, Wengang Wang, Yajun Xie, Manal A Adam, Lauren A English, Christopher M Reid, Salvador I Brito, Matthew A Booker, and Michael Y Tolstorukov
ARTICLE TYPE: Research Article

I am delighted to tell you that your manuscript has been accepted for publication in Development, pending our standard ethics checks.