

Xiang et al. showed that loss of RP58 (ZNF238, ZBTB18), a BTB/POZ and [zinc finger](#) containing [transcription factor](#), in the [mouse](#) brain lead to [microcephaly](#), [agenesis of the corpus callosum](#), [cerebellar hypoplasia](#) and that it is required for normal [neuronal differentiation](#). The transcriptional programs regulated by RP58 during this process are not known. They reported for the first time that in embryonic mouse neocortical neurons a complex set of genes normally expressed in other cell types, such as those from [mesoderm](#) derivatives, must be actively repressed in vivo and that RP58 is a critical regulator of these repressed transcriptional programs. Importantly, the GSEA analyses of these transcriptional programs indicate that repressed genes include distinct sets of genes significantly associated with glioma progression and/or pluripotency. They also demonstrated that reintroducing RP58 in [glioma stem cells](#) not only leads to aspects of neuronal differentiation but also to loss of [stem cell](#) characteristics, including loss of stem cell markers and decrease in stem cell self-renewal capacities. Thus, RP58 acts as an [in vivo](#) master guardian of the neuronal identity [transcriptome](#) and its function may be required to prevent brain disease development including glioma progression ¹⁾.

¹⁾

Xiang C, Fietze KK, Bi Y, Li Y, Dal Pozzo V, Pal S, Alexander N, Baubet V, D'Acunto V, Mason CE, Davuluri RV, Dahmane N. RP58 represses transcriptional programs linked to non-neuronal cell identity and glioblastoma subtypes in developing neurons. Mol Cell Biol. 2021 Apr 26;MCB.00526-20. doi: 10.1128/MCB.00526-20. Epub ahead of print. PMID: 33903225.

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