p27 Kip1 down-regulation as achieved by two clinically feasible means did not induce proliferation of supporting cells in the rat neonatal cochlea in vivo

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 In mammals, the cochlear sensory epithelium becomes quiescent early during development. After the first postnatal week, there is no cell replacement or proliferation, and severe damage leads to permanent deafness. Supporting cells? trans-differentiation has been suggested as a way to regenerate cochlear hair cells after damage. However, they are also needed for proper functionality. Cdkn1b (p27 Kip1) participates in the cochlear terminal mitosis state achieved during development. Its expression is maintained in adult supporting cells and its postnatal deletion has induced cochlear proliferation in vitro and in vivo. Therefore, its manipulation has been proposed as a feasible way to induce proliferation of supporting cells after birth. Nevertheless, the literature is scarce regarding feasible methods to directly decrease p27 Kip1 in the clinical domain. The effects of p27 Kip1 knockdown using viral vectors are not completely elucidated and no pharmacological approa