Supplementary Figure 1. *In situ* hybridization of *PK2*, *Ngn1* and *Mash1* in adult and P0 WT OB with S35-labeled probes. Scale bar = 1mm.
Supplementary Figure 2. Immunostaining showing Ngn1 expression (red) in post-mitotic neurons (shown in green by antibody against Tuj1) in the GCL, MCL and PGL of the OB at P2. Cell nuclei were stained with Hoechst.
Supplementary Figure 3. Size reduction and structural alteration in the OB of P0 Ngn1-/- and PK2-/- mice. (a). Macroscopic view of the brain of E16 and E18 Pk2-/- and WT mice. The OB of Pk2-/- embryos were smaller than wild-type control. (b). Diagram of OB in sagittal plane. The vertical line illustrates the position of cross section shown in d and e and used for quantification. (c). Quantification of the OB size in cross sections. *, p<0.01, unpaired t-test, n=4; **, p<0.0001, unpaired t-test, n=6. (d-e). Cresyl violet staining of cross section of P0 OB. (d). Low magnification, scale bar = 1mm. (e). High magnification, scale bar = 200 μm. GCL, granular cell layer; MCL, mitral cell layer; GL, glomerular layer.
Supplementary Figure 4. A model for PK2 gene as a common functional target gene for different bHLH transcription factors in the regulation of their respective functions. Our present data show that PK2 is a target gene of proneural bHLH factors Ngn1/MASH1 in regulating OB neurogenesis. We previously showed that another pair of bHLH factors CLOCK1/BMAL1 regulated PK2 in the SCN, the central circadian rhythm pacemaker (Cheng, M.Y. et al. *Nature* **417**, 405-10, 2002). It has also been suggested that PK2 might be controlled by HIF-1α, a key mediator for angiogenesis under hypoxia condition (Lecouter J, et al, *Proc Natl Acad Sci U S A* **100**, 2685-90, 2003).