

Correction: Variants in *NR6A1* as a cause for congenital renal, vertebral and uterine anomalies

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Correction: Human Genetics (2026) 145:25
<https://doi.org/10.1007/s00439-025-02806-1>

Following publication of the original article, the author identified an error in Figure legend that has been incorrectly tagged, it should be tagged correctly to the respective figures.

For completeness and transparency, new correct versions are display below with the tagging of the correct figure legends.

The original article can be found online at <https://doi.org/10.1007/s00439-025-02806-1>.

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The Corrected Version of Fig. 1.

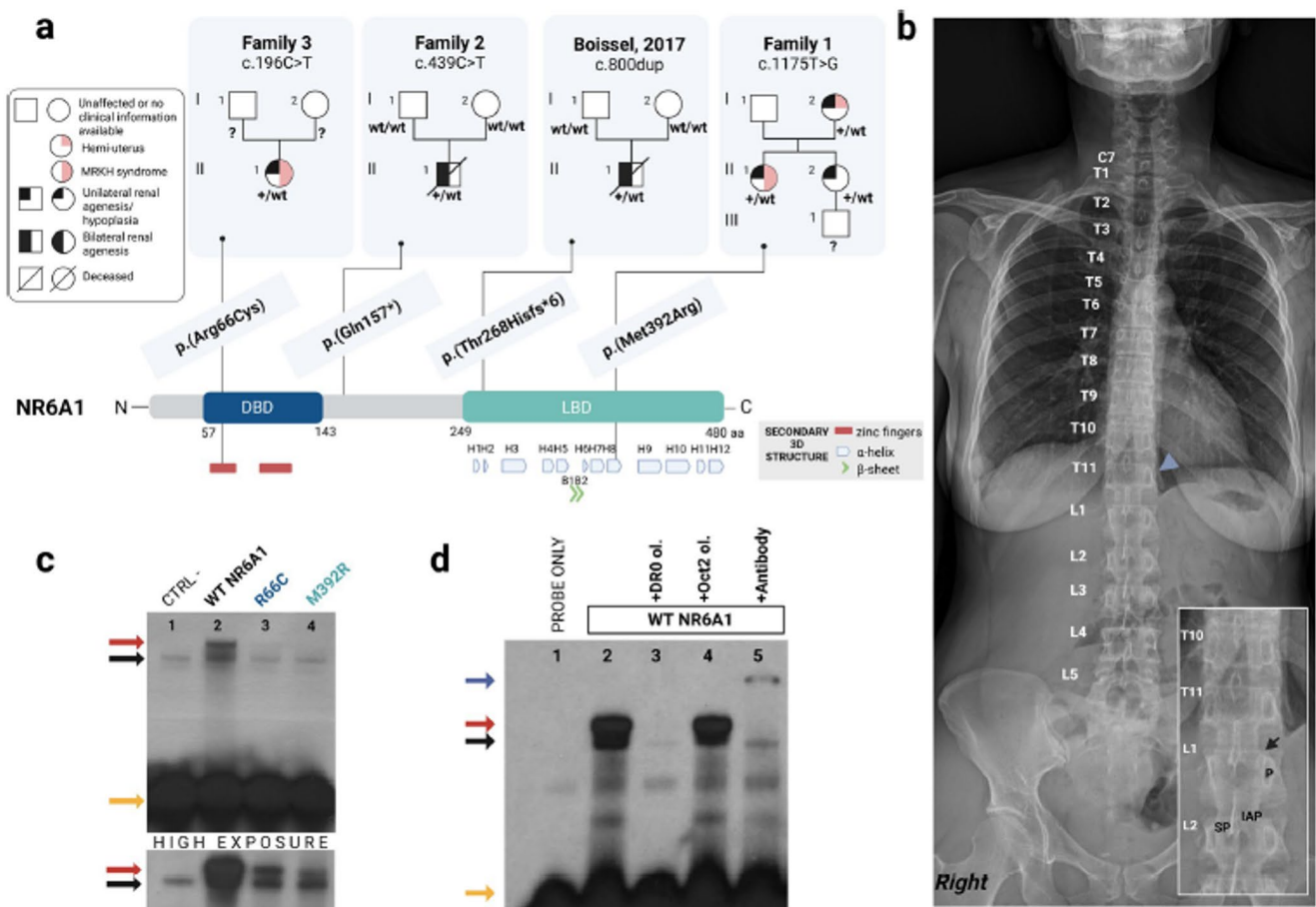


Fig. 1 Heterozygous variants in *NR6A1* in individuals with renal agenesis/hypoplasia with/without uterine and skeletal anomalies. **a** Pedigree and localization of the truncating and missense variants along the NR6A1 protein (NM_033334.4). The genotype of relatives is mentioned as “?” if not tested. NR6A1 is depicted with its main functional domains and 3D structure. The DNA-binding domain (DBD in blue) include two zinc fingers (red rectangles). The 3D structure of the ligand binding domain (LBD in turquoise) is composed of twelve alpha-helices (H1-H12) and two beta-turns (B1-B2). **b** X-ray of the spinal column (Family 1; individuals II:1) showing 11 thoracic vertebrae (T), 10 ribs on the right and a 11th hypoplastic rib on the left (blue arrowhead), and a normal number of five lumbar vertebrae (L). The black arrow points to the interspace of the facet joint visible between the last thoracic vertebrae and the first lumbar vertebrae given its orientation. IAP=inferior articular process, P=pedicle, SP=spinous process. **c** Electrophoretic mobility shift assay with ³²P-radiolabeled

DR0 consensus sequence, showing lower complex formation for the mutated hsNR6A1 proteins compared to controls (red arrow). An additional band (black arrow) is detected in the four samples corresponding to non-specific interaction of DR0 probe with proteins of reticulocyte lysate. The yellow arrow points to the unbound labeled DR0 oligonucleotides. The negative control (CTRL-) corresponds to incubation of probe with reticulocyte lysate without any mRNA. WT NR6A1, R66C and M392R correspond to wildtype and mutant NR6A1 proteins synthesized in reticulocyte lysates. **d** Competition analyses and supershift assay. The red arrow points to the band formed by the DR0-NR6A1 complex, the black arrow points to non-specific protein-DNA complexes from the reticulocyte lysate, the yellow arrow points to the unbound labeled DR0 oligonucleotides, and the blue arrow points to the shifted complex in presence of anti-Myc antibody. Created in BioRender. Humaine, G. (2025) <https://BioRender.com/sxmzsth>.

The Corrected Version of Fig. 2.

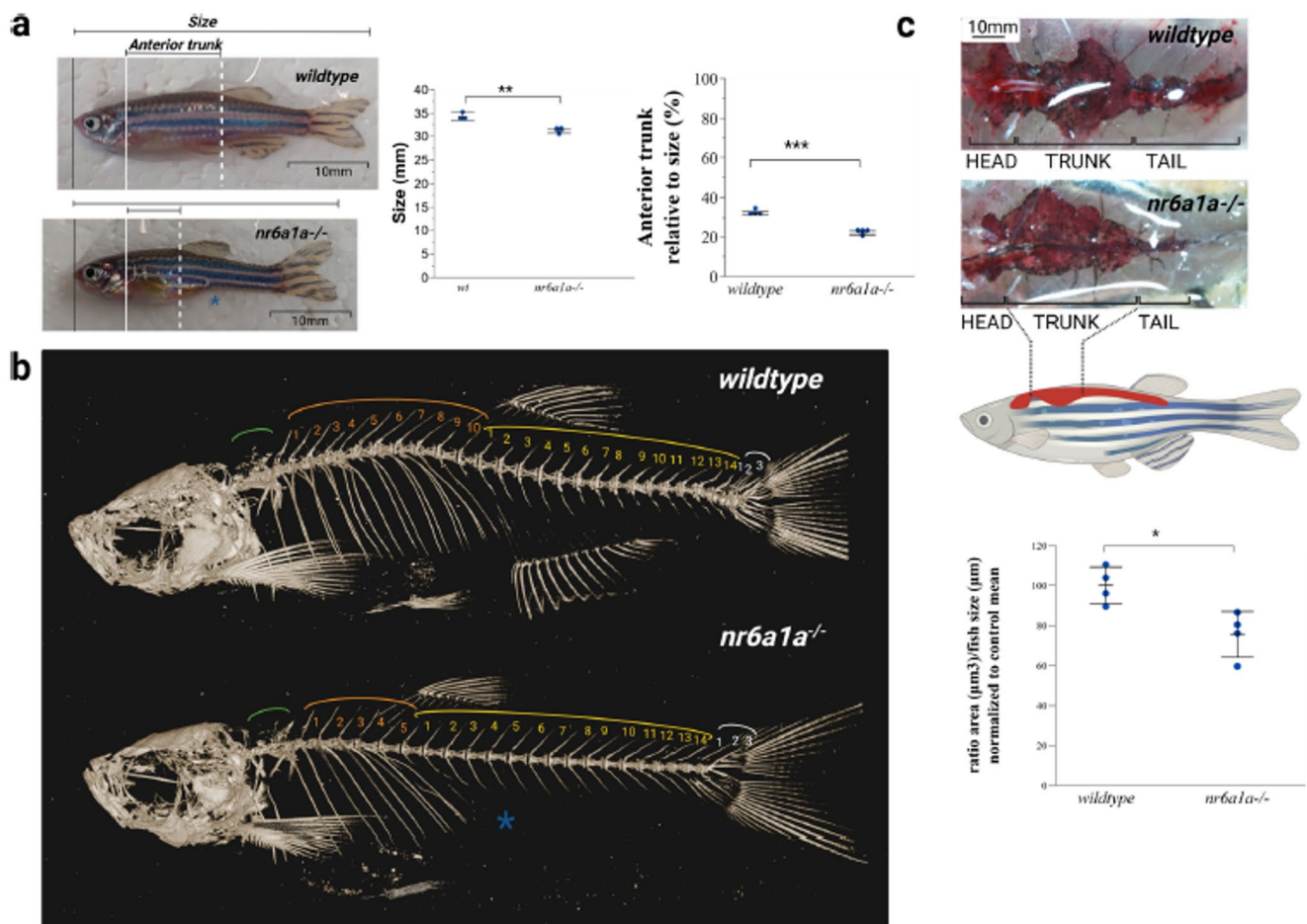


Fig. 2 Smaller size, decreased number of rib-bearing vertebrae and abnormal size and morphology of the kidney in $nr6a1a^{-/-}$ adult zebrafish. **a** Comparison of the size of wildtype ($n=3$) and mutant ($n=3$) siblings at 5-month $\frac{1}{2}$ (mean size wildtype: 34.295 mm; mean size mutants: 31.125 mm; difference: 3.17 mm (CI 95% 1.47–4.87) (p value=0.0095)). Comparison of the length of the anterior part of the trunk relative to the fish size between wildtype ($n=4$) and mutants ($n=4$) (mean ratio wildtype: 32.048%; mutants: 22.036%; difference: 10.01% (CI 95% 8.43–11.59) (p value<0.0001)). **b** X-ray microtomography of 3-month-old wildtype and mutant. The number of rib-bearing precaudal vertebrae (orange) is decreased in mutants (#5 instead of 10 in wildtype). The rib-bearing precaudal vertebrae are recognizable by the presence of ribs, and the absence of hemal arches

and spines, that are them specific of the caudal vertebrae (Bird and Mabee 2003). The number of caudal vertebrae (yellow, and light blue for the caudal fin vertebrae) is normal in mutants (#17). The anal fin (beginning at the 1st caudal vertebrae in wildtype) is absent in mutant (*). The Weberian vertebrae (vertebrae 1 to 4) are highlighted in green and seems normal although not investigated in details. **c** Mesonephros morphology of 15-month-old wildtype and mutants. The mutants present an abnormal shape of the mesonephros, dissected after injection of Rhodamine-dextran for visualization. Compared to wildtype ($n=4$), the surface of the mesonephros relative to the fish size (after normalization by the control mean), is decreased by 24.35% (CI 95% 6.15–42.54) (p value=0.017) in $nr6a1a^{-/-}$ mutants ($n=4$). Created in BioRender. Humaine, G. (2025) <https://BioRender.com/r29gpwg>.

The Corrected Version of Fig. 3.

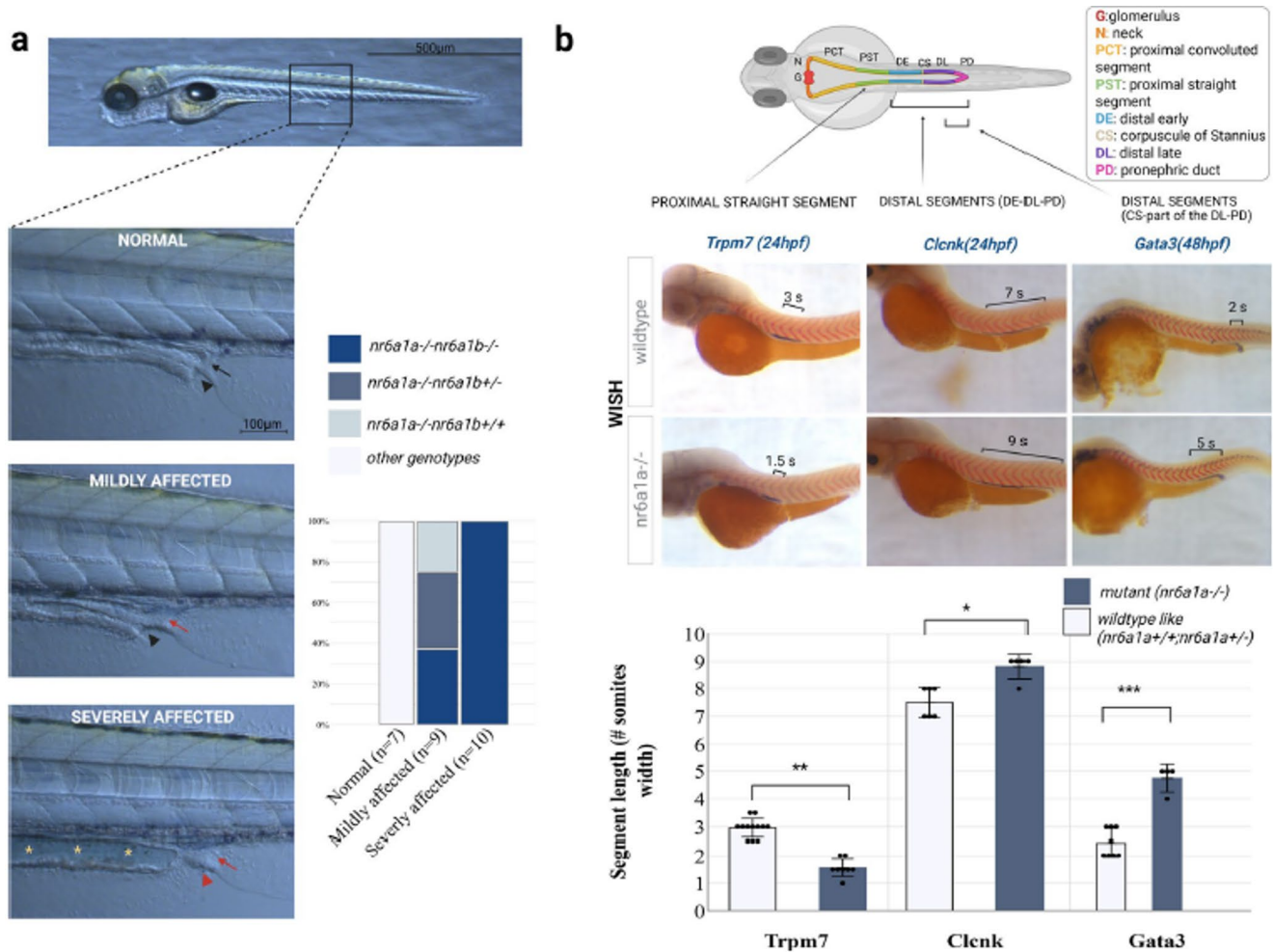


Fig. 3 Defects in cloacal morphogenesis and pronephros patterning in *nr6a1a*^{-/-} and *nr6a1a*^{-/-}*nr6a1b*^{-/-} mutants. **a** Brightfield images of the cloaca at 120hpf in embryos of *nr6a1a*^{+/+}*nr6a1b*^{+/+} incross. Classification of the phenotype as normal if the cloaca presents a typical inverted U-shaped with a visible pronephric duct in its terminal part (black arrow), and a visible opening of the gut (black arrowhead); mildly affected if the lumen of the gut reaches the cloaca (black arrowhead) whereas the terminal part of the pronephric duct is not visible (red arrow); severely affected if both ends are not visible (red arrow and red arrowhead). Some severely affected embryos present dilation of the gastrointestinal tract (yellow stars), suggesting imperforation. 26 embryos were selected for genotyping (normal (n=7); mildly affected (n=9); severely affected (n=10)). Embryos showing normal morphology are wildtype or heterozygous for *nr6a1a* and show

variable *nr6a1b* genotypes. Affected embryos are all *nr6a1a*^{-/-} with severely affected embryos being homozygous for both *nr6a1a* and *nr6a1b*. **b** Double whole-mount in situ hybridization with the somite boundary marker *xirp2a* and the pronephros segment markers *trpm7*, *clcnk* and *gata3* in embryos from *nr6a1a*^{+/+} incross. The width of one somite is used as reference to measure the length of the expression domain. Expression in *nr6a1a*^{-/-} mutants compared to wildtype/heterozygotes is increased with a difference of +2.3 somites ($p < 0.0001$) for *gata3* (n=4 for *nr6a1a*^{-/-} and n=8 for *nr6a1a*^{+/-or +/+}), +1.3 somites ($p = 0.0022$) for *clcnk* (n=5 for *nr6a1a*^{-/-} and n=6 for *nr6a1a*^{+/-or +/+}), and -1.4 somites ($p < 0.0001$) for *trpm7* (n=8 for *nr6a1a*^{-/-} and n=12 for *nr6a1a*^{+/-or +/+}). Created in BioRender. Humaine, G. (2025) <https://BioRender.com/dauy9pq>.

The Corrected Version of Fig. 4.

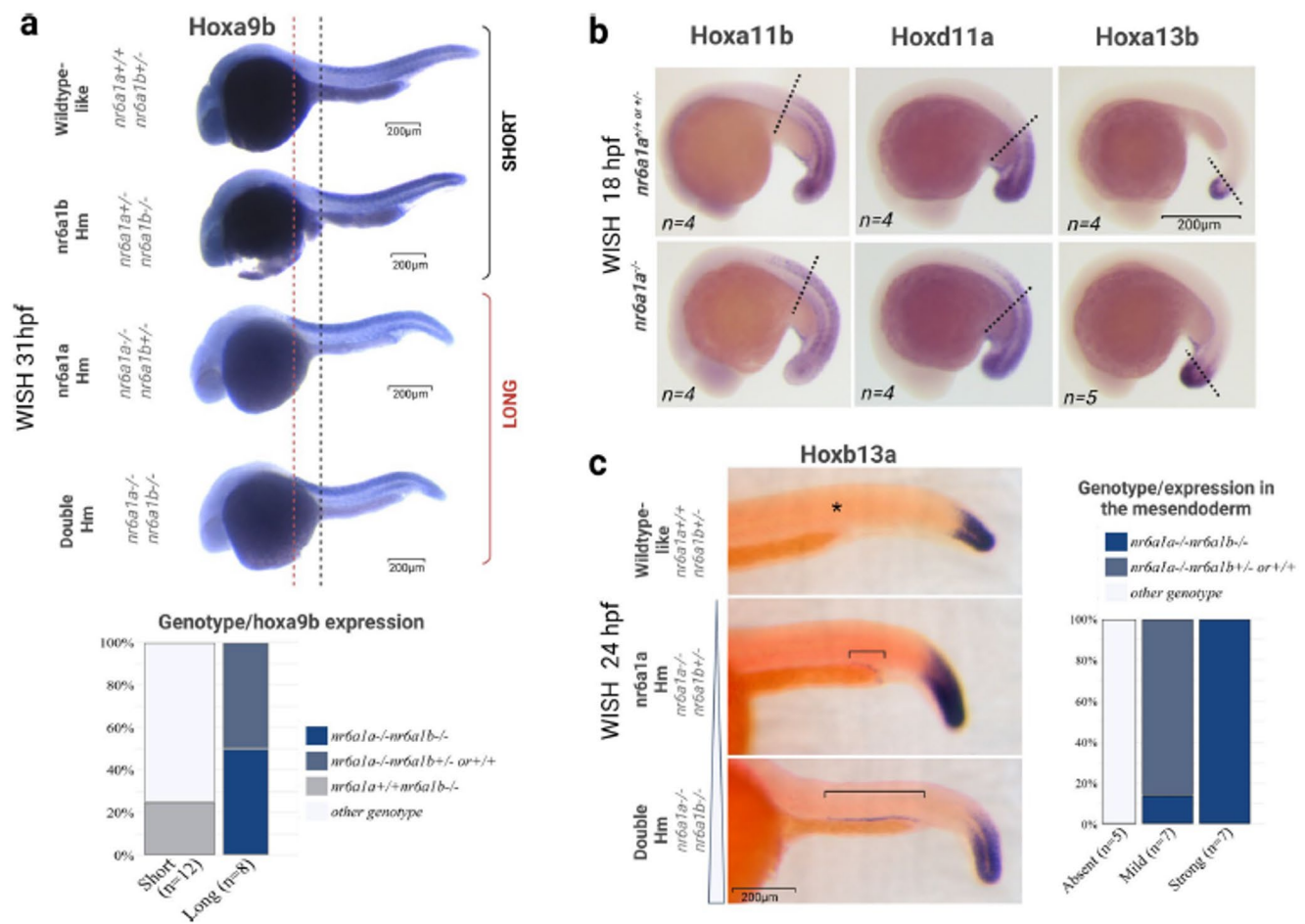


Fig. 4 Abnormal expression of posterior Hox in *nr6a1a*^{-/-} and *nr6a1a*^{-/-}*nr6a1b*^{-/-} mutants. **a** Whole-mount in situ hybridization with *hoxa9b* at 31hpf in embryos from an incross between *nr6a1a*^{+/+}*nr6a1b*^{+/+} and *nr6a1a*^{-/-}*nr6a1b*^{-/-} fish. A longer anterior axial staining (neural tube) was observed in 1/4 of the stained embryo (18/68). Genotyping was performed for 20 embryos (short phenotype: n=12; long phenotype: n=8). **b** Whole-mount in situ hybridization with *hoxa11b*, *hoxd11a*, *hoxa13b* at 18hpf in embryos from an incross between *nr6a1a*^{+/+}*nr6a1b*^{-/-} showing an extended anterior staining in *nr6a1a*^{-/-} mutants. Genotyping was performed for 8

embryos for *hoxa11b* (staining as expected: n=4; extended anterior staining: n=4), 8 embryos for *hoxd11a* (staining as expected: n=4; extended anterior staining: n=4), 9 embryos for *hoxa13b* (staining as expected: n=4; extended anterior staining: n=5). **c** Whole-mount in situ hybridization with *hoxb13a* at 24hpf in embryos from an incross between *nr6a1a*^{+/+}*nr6a1b*^{+/+} double heterozygotes. Classification of the embryos following the extend of the staining in the posterior mesendoderm (from absent to severely extended). Genotyping was performed for 19 embryos. Created in BioRender. Humaine, G. (2025) <https://BioRender.com/u7d75jt>.

The original article has been corrected

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