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**Missense variants in ANKRD11 cause KBG syndrome by impairment of stability or transcriptional activity of the encoded protein (vol 24, pg 2051, 2022)**

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## CORRECTION

# Missense variants in *ANKRD11* cause KBG syndrome by impairment of stability or transcriptional activity of the encoded protein



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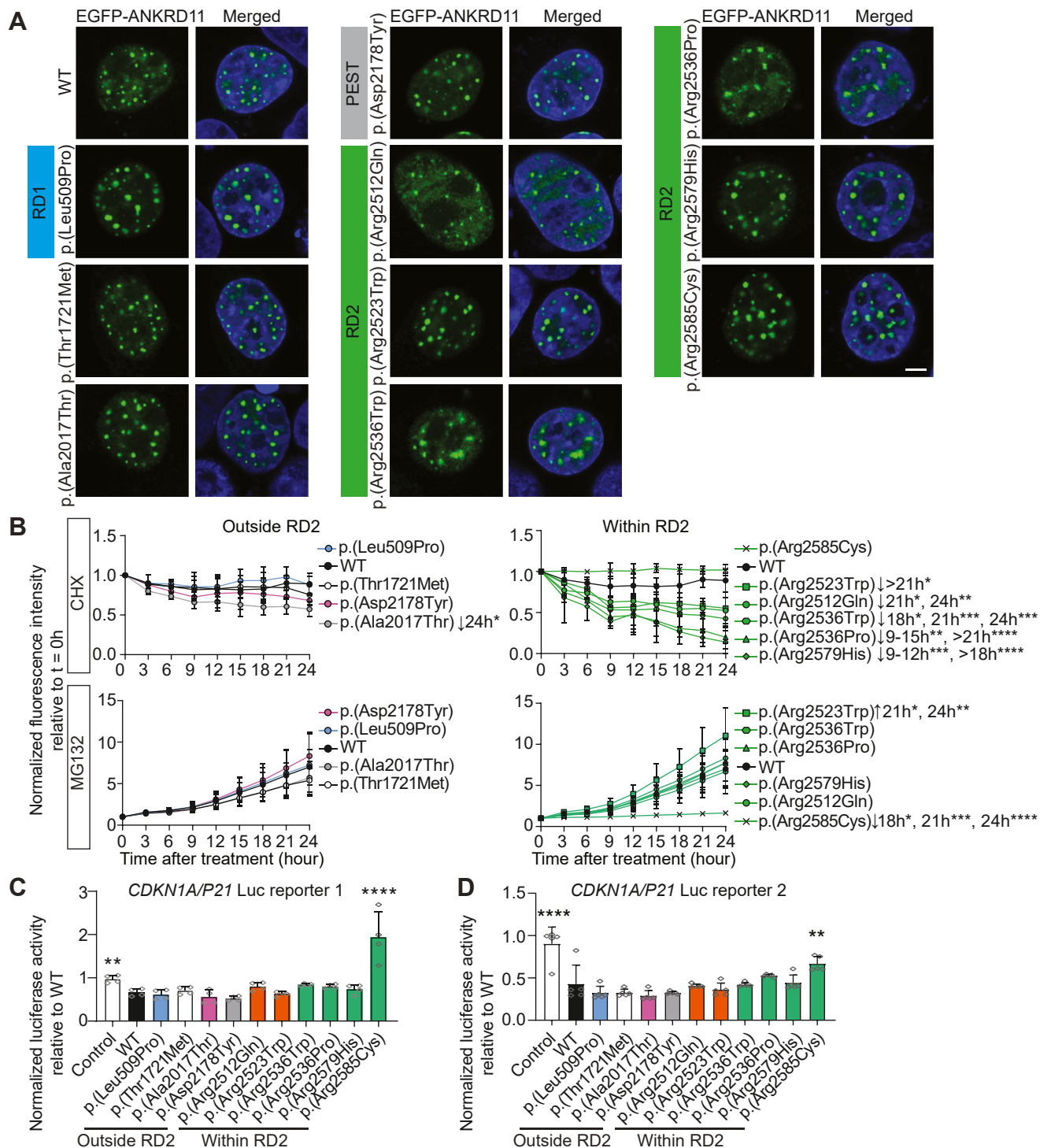
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In the article “Missense variants in *ANKRD11* cause KBG syndrome by impairment of stability or transcriptional activity of the encoded protein” (*Genet Med* 2022;24:2051–2064), the following update was made. On page 2060, [Figure 3](#) had an error in the artwork (the EGFP and Merged fluorescence imaging of ANKRD11 p.Leu509Pro and p.Arg2512Gln are identical). The revised [Figure 3](#) is shown below. The authors would like to apologize for any inconvenience this may have caused. The article has been corrected online and can be accessed at <https://doi.org/10.1016/j.gim.2022.06.007>.



**Figure 3 ANKRD11 variants in RD2 result in reduced protein stability and impaired proteasome degradation or loss of *CDKN1A/P21* transcriptional repression.** A. Direct fluorescence imaging of cells expressing EGFP-tagged variants of the ANKRD11 protein using confocal microscopy. Wild type and all variants showed a speckle-like pattern in the nucleus. Nuclei are stained with Hoechst 33342 (blue). Protein domains in which variants are located are indicated. Results are representative of 3 independent experiments. Scale bar = 5  $\mu$ m. B. Relative fluorescence intensity of EGFP-tagged ANKRD11 variants overexpressed in HEK293T/17 cells treated with translation inhibitor cycloheximide (CHX; 50  $\mu$ g/mL) shown in upper panels and with proteasome inhibitor MG132 (5  $\mu$ g/mL) in the lower panels. Equal volume of dimethyl sulfoxide was used as a vehicle control. Fluorescence intensity was measured for 24 hours with 3-hour intervals. Values are expressed relative to  $t = 0$  hour and represent the mean  $\pm$  SD of 3 independent experiments, each performed in triplicate (\* $P < .05$ , \*\* $P < .01$ , \*\*\* $P < .001$ , \*\*\*\* $P < .0001$ ; 2-way analysis of variance and a post hoc Dunnett's test). C-D. Results of luciferase assay with constructs containing WT and ANKRD11 variants and 2 firefly luciferase reporter constructs with a *CDKN1A/P21* promoter. Values are expressed relative to the control condition that used an EGFP-C2 construct without ANKRD11 and represent the mean  $\pm$  SD of 4 (C) or 5 (D) independent experiments, each performed in triplicate (\* $P < .05$ , \*\* $P < .01$ , \*\*\*\* $P < .0001$  vs WT; 1-way analysis of variance and a post hoc Dunnett's test). RD, repressor domain; WT, wild type.