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CORRESPONDENCE

CTNNB1 mutations in papillary thyroid carcinoma with prominent myofibroblastic stromal component

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To the Editor:

We read with great interest the article by Suster et al. titled "Papillary thyroid carcinoma with prominent myofibroblastic stromal component: clinicopathologic, immunohistochemical, and next-generation sequencing study of seven cases", in which they report *BRAF* mutations in the follicular cell component and *CTNNB1* mutations in the stromal component [1].

The desmoid-type fibromatosis variant of papillary thyroid carcinoma (DTF-PTC) is a very rare variant of PTC.

Fig. 1 Phenotypic and genotypic descriptions of the seven cases of analyzed by Suster et al. [1]. Compare the *CTNNB1* genotype of cases 1 and 6 [c.121A>G (p.Thr41Ala)] to that of case 3 [c.124A>G (p.Thr41Ala)]. From Suster et al. [1].

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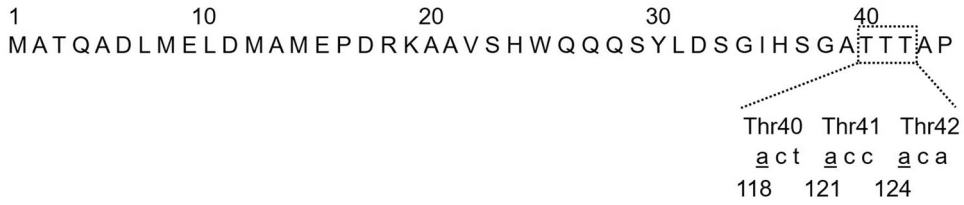

Fig. 2 Partial cDNA and protein sequence of CTNNB1. Note that nucleotides 121 and 124 belong to different codons (41 and 42, respectively), which code for consecutive threonines. The nucleotide sequences of the codons corresponding to the threonines at positions 40–42 are shown; the numbering corresponds to the underlined adenines. Sequences from National Center for Biotechnology Information Reference Sequence NM_001904.4 (https://www.ncbi.nlm.nih.gov/nuccore/NM_001904.4).

It is essentially a dual tumor with a component of classical PTC with malignant epithelial proliferation and another component of mesenchymal (stromal) proliferation. In two studies on non-thyroidal DTF cancers, accumulation of β-catenin due to an activating mutation in *CTNNB1* was found in 89 and 92% of the total cases [2, 3]. *CTNNB1* encodes β-catenin, a downstream effector of the Wnt signaling pathway that is generally responsible for regulation of cell growth and survival [4, 5]. Several studies have also detected *CTNNB1* mutations in the desmoid-type fibromatosis tissue in DTF-PTC [4, 6, 7].

In the text of the article by Suster et al. it is mentioned that "three cases showed a *CTNNB1* c.121A>G (p. Thr41Ala) mutation" [1]. However, Fig. 4 of their article actually indicates two cases with a CTNNB1 c.121A>G (p. Thr41Ala) mutation and a third case with a CTNNB1 c.124A>G (p.Thr41Ala) mutation [1] (Fig. 1). Because nucleotides 121 and 124 belong to different codons, they cannot both affect the same amino acid residue (Thr41). Unfortunately, because threonines are present at both positions 41 and 42 of the CTNNB1 protein (Fig. 2), it cannot be concluded from the information available in the article by Suster et al. [1] where the error lies, and which of the two contradictory affirmations is accurate. We suggest that the authors examine this issue and correct their article accordingly.

Compliance with ethical standards

Conflict of interest The authors declare no competing interests.

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