

CORRESPONDENCE



Response and correction to correspondence on "CTNBB1 mutations in papillary thyroid carcinoma with prominent myofibroblastic stromal component"

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TO THE EDITOR:

We thank the authors of "CTNBB1 mutations in papillary thyroid carcinoma with prominent myofibroblastic stromal component" for their short correspondence in which they identify an error in Fig. 4 of

the original paper [1, 2]. The original sequencing report was reviewed and shows a *CTNNB1* c.124A>G (p.Thr41Ala) mutation that was reported incorrectly in the original report. This was a typographical error in the original report. Review of the sequencing data for case 3 shows this should have indeed been a *CTNNB1* c.121A>G (p.Thr41Ala) mutation. This mistake was unfortunately transcribed and propagated into Table 3 and Fig. 4 and not caught during review of the Table and Figure. Table 3 and Fig. 4 have been revised (see Table 1 and Fig. 1). The body of the manuscript requires no revisions. The overall study findings remain unchanged.

Table 1. Revised Table 3: Immunohistochemical and molecular genetic findings in seven patients with PTC-DTF and PTC-NFS.

Case	Myofibroblastic Stroma IHC	PTC IHC	Molecular
1	Beta Catenin+ (nuclear) Keratin AE1/AE3- MIB1 <5% PAX8- P53- SMA+	Beta Catenin+ (membranous) Keratin AE1/AE3+ MIB1 <5% PAX8+ P53- SMA-	CTNNB1 c.121A>G, (p.Thr41Ala)
2	Beta Catenin+ (nuclear) Keratin AE1/AE3- MIB1 <5% PAX8- P53- SMA+	Beta Catenin+ (membranous) Keratin AE1/AE3+ MIB1 <5% PAX8+ P53- SMA-	CTNNB1 c.133T>C, (p.Ser45Pro) Epithelial component not available for testing, however previously tested ¹³ and positive for BRAF c.1799T>A (p.Val600Glu)
3	Beta Catenin+ (nuclear) Keratin AE1/AE3- MIB1 <5% PAX8- P53- SMA+	Beta Catenin+ (membranous) Keratin AE1/AE3+ MIB1 <5% PAX8+ P53- SMA-	CTNNB1 c.121A>G, (p.Thr41Ala)
4	Beta Catenin+ (nuclear) Keratin AE1/AE3- MIB1 <5% PAX8- P53- SMA+	Beta Catenin+ (membranous) Keratin AE1/AE3+ MIB1 <5% PAX8+ P53- SMA-	Failed repeat sequencing/QNS, However, epithelial and mesenchymal component were previously tested and reported positive ¹³ for: CTNNB1 c.133T>C, (p.Ser45Pro) BRAF c.1799T>A, (p.Val600Glu)
5	Beta Catenin— (nuclear) Keratin AE1/AE3— MIB1 ~5–10% PAX8— P53— SMA+	Beta Catenin+ (membranous) Keratin AE1/AE3+ MIB1 <5% PAX8+ P53- SMA-	BRAF c.1799T>A, (p.Val600Glu) USP6 rearrangement negative by NGS
6	Tissue exhausted	Tissue exhausted	CTNNB1 c.121A>G, (p.Thr41Ala) NRAS c.175G>A, (p.Ala59Thr)
7	Beta Catenin+ (nuclear) Keratin AE1/AE3- MIB1 <5% PAX8- P53- SMA+	Beta Catenin+ (membranous) Keratin AE1/AE3+ MIB1 <5% PAX8+ P53- SMA-	CTNNB1 c.134C>T, (p.Ser45Phe) BRAF c.1799T>A, (p.Val600Glu)

NGS next generation sequencing, IHC immunohistochemistry, QNS quantity not sufficient.

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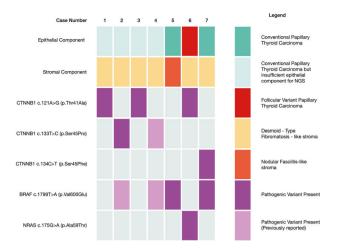


Fig. 1 Revised Figure 4—Sequencing results demonstrate 3 cases with a *CTNNB1* c.121A>G (p.Thr41Ala) mutation.

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REFERENCES

- 1. Roukain A, Sykiotis GP. CTNNB1 mutations in papillary thyroid carcinoma with prominent myofibroblastic stromal component. Mod Pathol. 2021. In press.
- Suster D, Michal M, Nishino M, Piana S, Bongiovanni M, Blatnik O, et al. Papillary thyroid carcinoma with prominent myofibroblastic stromal component: clinicopathologic, immunohistochemical and next-generation sequencing study of seven cases. Mod Pathol. 2020;33:1702–11.

CONFLICT OF INTEREST

The authors declare no competing interests.

ADDITIONAL INFORMATION

Correspondence and requests for materials should be addressed to S.S.

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