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Letter

DETECTION OF VIM3 IN ORAL SQUAMOUS CELL CARCINOMAS: METHODOLOGICAL LIMITATIONS

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We thank Von Brandenstein and colleagues for their interest in our study entitled "*Vimentin 3 as a New Potential Biomarker in Oral Squamous Cell Carcinomas*"[1], and for their valuable comments[2].

We acknowledge that the VIM-3B4 antibody used in our study recognizes an epitope located within the rod domain of vimentin, a region shared by both full-length vimentin and truncated isoforms, particularly Vim3[3]. Therefore, we agree that this antibody cannot definitively distinguish Vim3 from full-length vimentin. This represents a methodological limitation and is an important consideration in the interpretation of our findings.

The aim of our study was to investigate the association between vimentin expression (as detected by the VIM-3B4 antibody) and clinicopathological parameters in oral squamous cell carcinomas[1]. Variations in staining extent and intensity observed in tumor tissues may provide preliminary insights into potential biological relevance, even in the absence of isoform specificity.

We concur that further validation using isoform-specific antibodies or complementary techniques, such as Western blotting and mass spectrometry, is necessary for the definitive identification of Vim3[4]. This constitutes a limitation of our study and represents an important direction for future research.

In both clinical and research practice, particularly in resource-limited settings, the use of commercially available and practical antibodies is often essential. In our study, the VIM-3B4 antibody was selected based on its prior use in the literature, availability, and suitability for our experimental conditions.

Importantly, the primary objective of our study was not to establish a definitive molecular distinction of the Vim3 isoform, but rather to explore the potential role of immunoreactivity detected by the VIM-3B4 antibody. In this context, our findings provide preliminary evidence that vimentin-associated alterations, including those potentially involving truncated isoforms, may have biological and diagnostic relevance in these tumors.

In conclusion, while we acknowledge that findings obtained using the VIM-3B4 antibody should be interpreted with caution and not attributed absolute specificity to Vim3, we believe that our results remain valid within the framework of immunohistochemical evaluation and contribute to the existing literature on vimentin isoforms in tumor biology.

References

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