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TRIM26 Accelerate Oxidative Stress Injury of Vascular Endothelial Cell in Model of AS by NTH1 Function

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ABSTRACT AS (AS) is an important pathological process of the most common cardiovascular diseases. This study investigated the role and mechanism of TRIM26 in patients with AS. TRIM26 mRNA expression was upregulated in patients and mice with AS. In the mice model, TRIM26 was found to accelerate oxidative stress and AS. Overexpression of TRIM26 induced TH1 expression to promote oxidative stress in the in vitro model through inhibiting NTH1 expression. Conversely, si-TRIM26 induced NTH1 expression to reduce oxidative stress in the in vitro model. TRIM26 protein was found to interact with the NTH1 protein, and TRIM26 increased the ubiquitination of the NTH1 protein. In summary, TRIM26 was found to contribute to oxidative stress injury in AS, particularly under oxidative stress conditions. Therefore, TRIM26 may act as a critical factor in the induction of the AS model.