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LETTER TO THE EDITOR

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ID1 and IGFBP3: roles in cellular senescence, cardiac development, angiogenesis and cancer diagnosis

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To the editor,

The protein known as Inhibitor of DNA-binding 1 (ID1) is a component of the helix–loop–helix (HLH) group of proteins [1]. Insulin-like growth factor binding protein-3 (IGFBP3) exerts its influence by managing the bioavailability of the insulin-like growth factor (IGF) [2]. After a complete search for infinite definite words of "ID1 and IGFBP3" in PubMed, only 12 references were found from 2003 to 2023 years (Fig. 1) and we summarized the relative literatures on the relationship between ID1 and IGFBP3 (Fig. 2).

ID1 and IGFBP3 in cellular senescence

TGF-β1 is postulated to be a primary factor in prostate aging, facilitating premature senescence and promoting myofibroblast differentiation [3]. Both cellular senescence and TGF-β-induced growth arrest and differentiation of prostate basal cells lead to the downregulation of ID1. Furthermore, IGFBP3 mRNA levels are reduced after a 24-h exposure to TGF-β1, 2 and 3 [3].

ID1 and IGFBP3 in prostate cancer

Research indicates that normal prostate epithelial cells display low or virtually non-existent ID1 expression levels, contrasting with the heightened levels observed in prostate cancer. Therefore, the integration of ID1 into normal prostate epithelial cells might present a method for examining the early stages involved in the onset of prostate cancer. IGFBP3 has been discovered to exhibit both inhibitory and stimulatory effects on cells, with these effects being mediated via specific IGFBP3 binding proteins/receptors [1].

ID1 and IGFBP3 in cardiac development

Early research reported that ID1 and ID3 double knockout (dKO) mouse embryos suffer mid-gestation demise due to multiple cardiac defects [4]. Changes in the expression patterns of vascular, fibrotic, and hypertrophic markers were observed in the ID dKO hearts, but IGFBP3 introduction restored vascular and fibrotic gene expression patterns [4]. This implies that deletion of ID genes in the vasculature results in distinct postnatal cardiac phenotypes and highlight IGFBP3 as a possible link between ID and its vascular effectors [4]. It is plausible that ID1 suppresses IGFBP3 within the endothelium, given that both proteins have been found there.

ID1 and IGFBP3 in angiogenesis in endothelial cell

Bone morphogenetic protein 2 (BMP2) is crucial for endometrial decidualization and the invasion of trophoblast cells [2, 5]. Interestingly, IGFBP3 promotes cell migration and angiogenesis in endothelial cells, while ID1 has a regulatory impact on IGFBP3 expression in rat prostate epithelial cells [2]. The researchers verified that ID1 and IGFBP3 promote human trophoblast invasion and the formation of endothelial-like tubes. Additionally,

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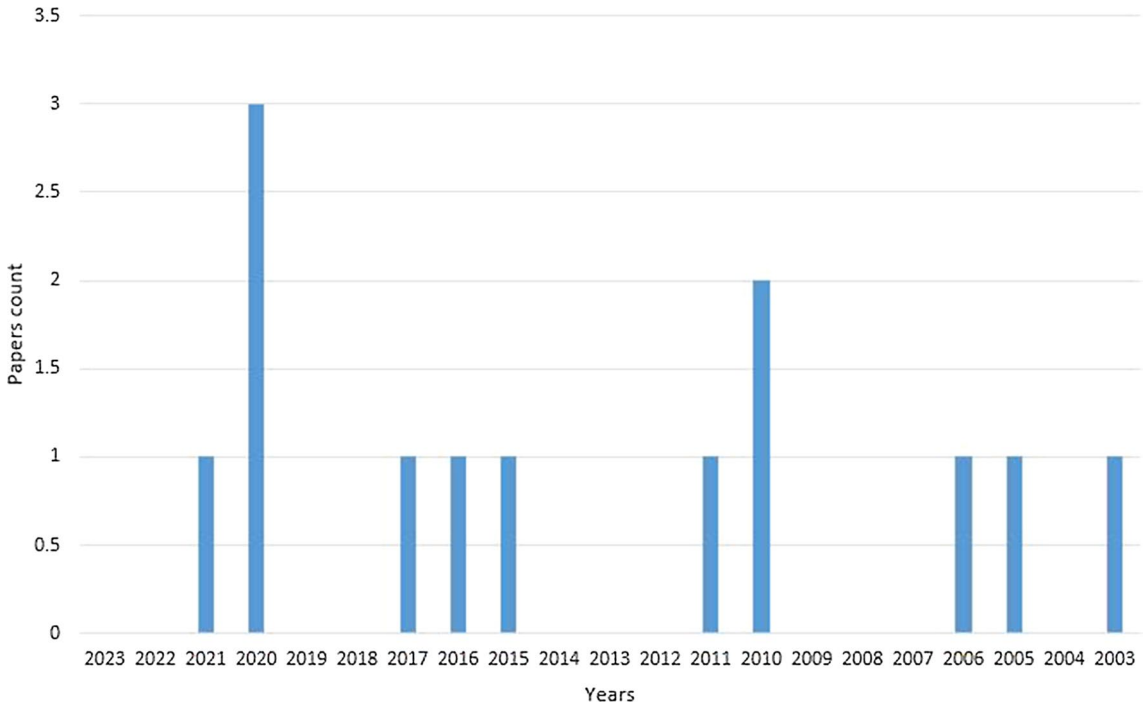

Fig. 1 Number of publications of ID1 and IGFBP3 in PubMed

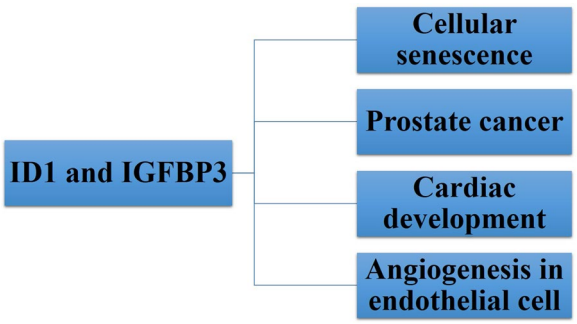

Fig. 2 Relative contents on the role of ID1 and IGFBP3

they showed that ID1 plays a role in mediating the BMP2-induced increase in IGFBP3 expression [5].

In conclusion, ID1 and IGFBP3 each have their own unique biological properties, and there are currently few research results on the relationship between them. Hence, exploring the mechanisms that underlie this specificity would be a compelling area for future research.

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Author contributions
CS was involved in drafting the manuscript. CS was involved in acquisition of data. CS designed and revised the manuscript. Author has read and approved the final manuscript.

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Availability of data and materials
All data for this study are publicly available and are ready for the public from database of hospital.

Declarations

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Consent for publication
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The author has no conflicts of interest to declare.

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