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# Infantile hemangioma is originated from placental trophoblast, fact or fiction?

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**Summary** Infantile hemangiomas are common, benign tumors, distinctive for their perinatal presentation, rapid growth and subsequent involution. Hemangiomas can pose serious concerns to the cosmetic and psychosocial development of the afflicted child, but none of the current therapeutic modalities is ideal to date, partly because the origin of the pathogenic ECs in infantile hemangioma is unknown.

Many clues and evidences suggest a link between infantile hemangiomas and the maternal placental trophoblasts. Shared expression of distinct endothelial markers in hemangioma and placental tissues raises a possibility that infantile hemangioma is originated from placental trophoblast. Moreover, the findings of a very high similarity between the transcriptomes of placenta and hemangioma provide strong support for this theory. Furthermore, epidemiologic and clinical evidences accumulated in recent years also suggest the placental trophoblast as the cell of origin for infantile hemangioma. These findings imply a unique relationship between hemangioma and the placental trophoblast and suggest a hypothesis that infantile hemangioma is originated from placental trophoblast.

The hypothesis could provide new understanding of these vascular tumors of childhood and may become the most promising research fields for the etiology of infantile hemangiomas. Further study of the precise mechanisms for the

*Abbreviations*: Glut-1, type 1 glucose transporter; PCNA, proliferating cell nuclear antigen; VEGF, vascular endothelial growth factor; MMP, metalloproteinase; bFGF, basic fibroblastic growth factor; TIMP, tissue inhibitor metalloproteinase; vWF, von willebrand factor; ECs, endothelial cells; PFO, patent foramen ovale; CVS, chorionic villus sampling; ''='', equal expression; ''>'', stronger expression; ''>>'', much stronger expression.

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placental trophoblast originated hemangiomas will produce new preventive strategies and therapeutic avenues, possibly immunologic treatment, to the very difficult problem.
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## Introduction

Infantile hemangioma, the most common benign tumors of infancy, is characterized by rapid growth during the first year of life (proliferative phase) followed by a decline in growth (involutional phase) over the next 5—6 years, with complete regression of the lesion by age 9 (the involuted phase). 90% of these tumors spontaneously regress, 5% cause serious tissue damage and approximately 1—2% are life threatening [1,2]. Although rarely life threatening, hemangiomas can pose serious concerns to the cosmetic and psychosocial development of the afflicted child.

Several theories can rationally elucidate the pathogenesis of proliferation hemangiomas formation, but the biologic origin of these lesions continues to elude us. Up to now, none of the current therapeutic modalities is ideal, partly because the origin of the pathogenic ECs in infantile hemangioma is unknown [3].

Many people could not believe there has some correlation between infantile hemangiomas and placental trophoblasts. If saying that infantile hemangioma is originated from placental trophoblast, you would only ignore it just as a fiction. But, in fact, it may be a fact.

Many clues reveal that there may be a link between infantile hemangiomas and the maternal placental trophoblasts. Also, epidemiologic and clinical evidences accumulated in recent years suggest the placental trophoblast as the cell of origin for infantile hemangioma. Further more, coexpression of some distinct cell markers in placental trophoblast and infantile hemangioma which was found recently support this placental trophoblastic origin. As outlined above, it is conceivable that infantile hemangioma is originated from placental trophoblast. Although speculative at present, the theory seems to explain many epidemiologic and clinical features.

## The hypothesis

We hypothesize that infantile hemangioma is originated from placental trophoblast, to the specific, placental ECs could embolize and become dislodged into the fetal circulation to receptive tissues during gestation, which can form infantile hemangioma after birth.

## Clues for the placental trophoblastic origin

It has been reported that infantile hemangiomas have been described to occur more frequently in association with chorangiomas (a type of hemangioma), which is the most frequently occurring benign tumor of the maternal placental trophoblasts [4]. It reveals that there might be some correlation between infantile hemangioma and placental trophoblast. Further more, several recent studies have suggested an increased incidence of hemangiomas in infants whose mother had undergone CVS, which is a means of rapid prenatal diagnosis in early pregnancy. Burton et al. [5] describes CVS at 9—12 weeks of gestation has been associated with a 21% increased incidence of infantile hemangiomas. In addition, intentional placental trauma, produced during embryoscopy prior to elective pregnancy termination, results in rapid development of fetal ecchymotic lesions. Although the intrinsic reason has not been studied specifically, we postulate that CVS and other placental trauma can cause placental injury and intravascular shedding of placental ECs and then direct emboli of the cells reach to receptive fetal tissues during gestation.

Besides, as we know a variety of growth factors play essential roles during hemangioma proliferation and involution, of interest was that many of these factors also play a role in placental physiology (see Fig. 1).

Moreover, many people found that the perinatal presentation of these tumors has their typical pattern of rapid proliferation and subsequent involution, and it could just mirror the programmed limited life span of human placental endothelium [6]. Though this phenomenon supports our hypothesis, the underlying pathophysiologic mechanism of this relationship is not well understood.

These clues, although indirect, make the placental trophoblastic origin of infantile hemangioma more or less conceivable.

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