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Editorial: Wnt signaling in endocrine and metabolic disorders

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Editorial on the Research Topic

Wnt signaling in endocrine and metabolic disorders

The present Research Topic, entitled “*Wnt signaling in endocrine and metabolic disorders*” aims to emphasize the functional role of the Wnt signaling pathway in human endocrinology, focusing on metabolic disease. Endocrine and metabolic disorders encompass a wide range of conditions affecting various organ systems and physiological processes. The Wnt signaling pathway, originally recognized for its role in embryonic development and tissue homeostasis (1, 2), has emerged as a crucial player in the pathogenesis of several human disorders, including cancer (3, 4), and greatly contributes to disease progression and potential therapeutic implications (5–7).

The first study in this Research Topic clarified that one of the mechanisms by which the “*Modified Qing' E Formula*” (MQEF), used for more than 1,300 years in China as a treatment for lumbodysnia, may exert its therapeutic effect on steroid-induced ischemic necrosis of the femoral head, is through targeting exosomal microRNAs (miRNAs) to regulate multiple signaling pathways, including Wnt, PI3K-Akt, and MAPK (Zhu et al.). In another original report investigating miRNAs and Wnt signaling, Tripathi et al. demonstrate that miR-539-3p overexpression in osteoblasts downregulates several components of the Wnt signaling pathway and deteriorates trabecular microarchitecture, leading to decreased bone formation in ovariectomized mice. In the third original article in our Research Topic, a group of investigators led by Xiaolin Tu found that the small molecule C91 (CHIR99021) promotes osteogenic differentiation of bone marrow stromal cells via the activation of Wnt signaling (Wang et al.).

Vilaseca et al. provide an interesting overview of the functional roles of estrogen deficiency in the processes involved in the development of Alzheimer's disease, including Wnt signaling and glucose transport in the brain, amyloid precursor protein processing to form senile plaques, and Tau phosphorylation forming neurofibrillary tangles.

A very comprehensive review concludes our Research Topic: Franco et al. elegantly explain the main differences between the physiological roles of canonical Wnt signaling

(essential for cell growth, tissue remodeling, and organ formation) and its pathological involvement in the development of several human diseases, including cancer. Correctly interpreting the molecular bases of Wnt signaling and metabolism, ideally in a cell-type and tissue-specific manner (Franco et al.; 8–10), may provide formidable knowledge to biomedical scientists and clinicians, holding the promise of producing novel therapies.

In conclusion, understanding the intricate interplay between Wnt signaling and endocrine/metabolic disorders holds great promise for the development of targeted therapies and improved patient outcomes.

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FC: Writing – review & editing. GS: Writing – original draft

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Conflict of interest

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References

- Li D, Sun J, Zhong TP. Wnt signaling in heart development and regeneration. *Curr Cardiol Rep* (2022) 24(10):1425–38.
- Steinhart Z, Angers S. Wnt signaling in development and tissue homeostasis. *Development* (2018) 145(11):dev146589.
- Zhou Y, Xu J, Luo H, Meng X, Chen M, Zhu D. Wnt signaling pathway in cancer immunotherapy. *Cancer Lett* (2022) 525:84–96.
- Bugter JM, Fenderico N, Maurice MM. Mutations and mechanisms of WNT pathway tumour suppressors in cancer. *Nat Rev Cancer* (2021) 21(1):5–21.
- Rim EY, Clevers H, Nusse R. The Wnt pathway: from signaling mechanisms to synthetic modulators. *Annu Rev Biochem* (2022) 91:571–98.
- Albrecht LV, Tejeda-Munoz N, De Robertis EM. Cell biology of canonical Wnt signaling. *Annu Rev Cell Dev Biol* (2021) 37:369–89.
- Akoumianakis I, Polkinghorne M, Antoniadou C. Non-canonical WNT signalling in cardiovascular disease: mechanisms and therapeutic implications. *Nat Rev Cardiol* (2022) 19(12):783–97.
- Clevers H, Nusse R. Wnt/beta-catenin signaling and disease. *Cell* (2012) 149(6):1192–205.
- Chen X, Tan H, Xu J, Tian Y, Yuan Q, Zuo Y, et al. Klotho-derived peptide 6 ameliorates diabetic kidney disease by targeting Wnt/beta-catenin signaling. *Kidney Int* (2022) 102(3):506–20.
- Aamir K, Khan HU, Sethi G, Hossain MA, Arya A. Wnt signaling mediates TLR pathway and promote unrestrained adipogenesis and metaflammation: Therapeutic targets for obesity and type 2 diabetes. *Pharmacol Res* (2020) 152:104602.