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1992	전남대학교 의과대학 학사
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1999	일본 교토대학교 제2내과 방문교수 (mentor: Prof. Itoh Hiroshi / Prof. Kazuwa Nakao)
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[관심분야]

심혈관 및 골격근육 리모델링의 후생성 조절

[논문]

1. Circular RNA circSMAD4 regulates cardiac fibrosis by targeting miR-671-5p and FGFR2 in cardiac fibroblasts Mol Therapy - Nucleic Acids, 34: 102071, 2023
2. Circular RNA circSmac1-2 regulates vascular calcification by acting as miR-874-3p spongy in vascular smooth muscle clells. Mol Therapy - Nucleic Acids, 27: 645, 2022
3. Regulation of MDM2 E3 ligase-dependent vascular calcification by MSX1/2. Experimental and Molecular Medicine 53: 1181, 2021
4. The roles of non-coding RNAs in vascular calcification and opportunities as therapeutic targets. Pharmacology & Therpeutics 218: 107675, 2021
5. S-nitrosylation of histone deacetylase 2 by neuronal nitric oxide synthase as a mechanism of diastolic dysfunction. Circulation 143: 1912, 2021

RXX regulates balance of skeletal muscles and adipose tissues

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The RXX, also identified as TRIM-XX, plays multifunctional roles, notably as an oncoprotein and a regulator in various biological processes. Our laboratory has been at the forefront of exploring RXX's involvement in skeletal muscle diseases and its broader biological implications. RXX functions as a pro-tumorigenic gene, integrating into tumor suppressor genes, and displays dual functionality as both a transcription regulator and an E3 ligase. A significant breakthrough came when we identified RXX's interaction with the enhancer of polycomb 1 (EPC1), a transcription activator. This interaction modifies the transcriptional activity of the EPC1-containing protein complex, shedding light on the intricacies of gene expression regulation. Further investigations revealed RXX's high expression in the satellite cells of undifferentiated skeletal muscles and during early muscle development stages, indicating a pivotal role in muscle differentiation. RXX obstructs skeletal muscle differentiation by extracting EPC1 from the serum response factor (SRF), essential for initiating muscle differentiation. Moreover, RXX instigates the degradation of MyoD, a key muscle differentiation protein, in an E3 ligase-dependent manner, underscoring its intricate involvement in muscle physiology. Our research also extended to RXX's impact on adipose tissue, revealing that RXX knockout mice subjected to a high-fat diet exhibited less weight gain and improved metabolic health compared to wild-type counterparts. This was evident in the reduced weight increase in both inguinal and epididymal white adipose tissues, alongside normalized blood glucose levels and improved glucose and insulin tolerance. The generation of adipose-tissue-specific knockout mice further corroborated these findings, with inhibited adipocyte differentiation observed in isolated stromal vascular cells from RXX knockout mice. These studies unveiled RXX's interference with PPAR-γ function, suggesting its role in promoting body fat accumulation by enhancing adipogenic differentiation. This multifaceted research highlights RXX's significant implications in muscle differentiation, adipogenesis, and potential therapeutic targets in related diseases.