제 119회
ORGAN ON A CHIP
기술교류회

2025.06.05 목 오후 4시 30분

한림대학교 의료·바이오융합연구원 포스터홀

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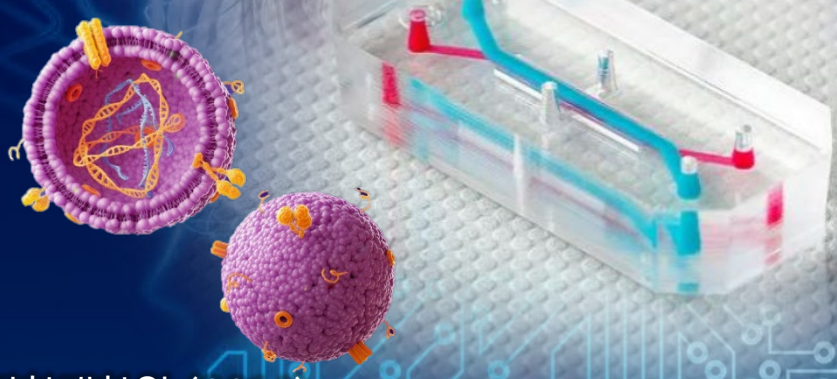

이찬희 교수

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1. Education

박사: 한국과학기술원 의과학대학원 (2016)
학사: 연세대학교 생화학과 (2011)

2. Experience

2021 ~ 현재	한림대학교 의약신소재전공, 조교수
2020 ~ 2021	UT Southwestern Medical Center, Post-doc
2019 ~ 2020	울산대학교 의과대학, 연구조교수
2016 ~ 2019	서울아산병원 생명과학연구원, Post-doc

시상하부 자율신경계 신호가 암-악액질에 미치는 영향

Hypothalamic sympathetic signaling mediates the development of cancer-associated cachexia

Background: Cancer-associated cachexia (CAC) is a debilitating syndrome characterized by adipose tissue and skeletal muscle wasting. The hypothalamus controls whole-body energy metabolism by regulating catabolic programs through the sympathetic nervous system (SNS). However, the detailed mechanism by which the hypothalamus contributes the adipose tissue and skeletal muscle wasting remains unclear. This study investigated the role of interleukin-4 (IL-4) in promoting CAC, particularly through SNS activation.

Methods: This study centrally administered IL-4 and examined its effects on feeding behavior, as well as adipose tissue and muscle wasting. To investigate the mechanism by which central IL-4 signaling impacts adipose and muscle wasting, sympathetic denervation was performed, and mifepristone was administered. To determine the role of the hypothalamic microglia IL-4R in CAC development, we injected IL-4R shRNA-expressing AAV into the hypothalamic arcuate nucleus (ARH) of tumor-bearing CX3CR1-creERT2 mice. Additionally, we chemogenetically suppressed the activity of hypothalamic POMC neurons to assess their contribution to CAC development.

Results: Central IL-4 administration reproduced key features of CAC, including weight loss, anorexia, and adipose and muscle wasting. The mechanisms underlying adipose and muscle wasting differed, involving the hypothalamic-pituitary-adrenal (HPA) axis and autonomic stimulation, respectively. IL-4R is expressed in hypothalamic microglia but not in astrocytes and POMC neurons. Deletion of IL-4R in hypothalamic microglia ameliorated B16F10-induced CAC. Additionally, chemogenetic inactivation of POMC neurons prevented B16F10-induced adipose and muscle wasting.

Conclusions: Our findings suggest that targeting hypothalamic microglial IL-4 signaling and POMC neurons could represent a pivotal therapeutic strategy for CAC intervention.

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