



Vienna Cachexia Network

Upcoming Vi-CaN Meeting

23rd February 2026, 4:00pm

CeMM, 8th floor, Large Seminar Room

FGF21 protects from adipose tissue wasting and systemic inflammation in cancer cachexia

Besides muscle atrophy, loss of adipose tissue depots is frequently observed in cancer cachexia (CAC). Adipose tissue atrophy in turn increases systemic inflammation and insulin resistance, thereby accelerating whole body catabolism. Using syngeneic murine tumor models of CAC, we identified fibroblast growth factor 21 (FGF21) as a thermoneutrality-associated factor that is upregulated in cachectic mice as well as in human cancer patients with cachexia. Genetic ablation and pharmacological administration of FGF21 revealed a protective role for this hepatokine, as FGF21 attenuated adipose tissue wasting, mitigated systemic inflammation, and improved muscle glucose handling. Our findings suggest that FGF21 functions as an endogenous, compensatory endocrine signal with therapeutic potential to alleviate CAC.



Raimund Oberle performed his PhD at the Max Perutz Laboratories and did Postdoctoral training at the UKE Hamburg in translational oncology. Currently he is working at the Center for Pathobiochemistry and Genetics at the Medical University of Vienna. A major research focus of his laboratory is to elucidate alterations in organ homeostasis, metabolic regulation and inflammatory processes in cancer cachexia.

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