Inflammation and myosteatosis in pancreatic cancer cachexia

Propositions

Accompanying the dissertation

INFLAMMATION AND MYOSTEATOSIS IN PANCREATIC CANCER CACHEXIA

1. The positive correlation between plasma C1q and C3a/TCC concentrations in patients with pancreatic cancer suggests that complement activation in these patients could be the result of activation of the classical complement pathway. (this thesis)

2. Elevated circulating lipocalin-2 levels are a result of neutrophil activation in patients with pancreatic cancer cachexia. (this thesis)

3. The correlation between circulating complement factors and neutrophil activation markers in patients with pancreatic cancer calls for more research into their potential causal relationship and its role in the pathogenesis of cancer cachexia. (this thesis)

4. The combination approach of LC-MS/MS-based lipidomics and MALDI-MSI enables the quantification and spatial mapping of intramyocellular lipid alterations. (this thesis)

5. Intramyocellular lipid content increases in pancreatic cancer patients and is associated with their inflammatory and cachectic status. (this thesis)

6. To treat pancreatic cancer more effectively, we should prioritize anti-cachexia treatment over targeting the tumor.

7. Since the majority of patients with pancreatic cancer is diagnosed with metastases, most research on this deadly disease should be focused on methods to improve early detection.

8. Since cancer cachexia is a multiorgan failure disease, novel multiorgan-on-a-chip approaches will help to advance our understanding of inter-organ communication in cancer chachexia.

9. A better understanding of the mechanisms underlying cancer cachexia as provided in this thesis will help pharmaceutical companies to develop new drugs against cancer-associated cachexia and benefit cachectic patients as well as reduce health care costs.

10. "Somewhere, something incredible is waiting to be known." - Carl Sagan

11. "The more we know, the more we realize how much we don't know." - Aristotle