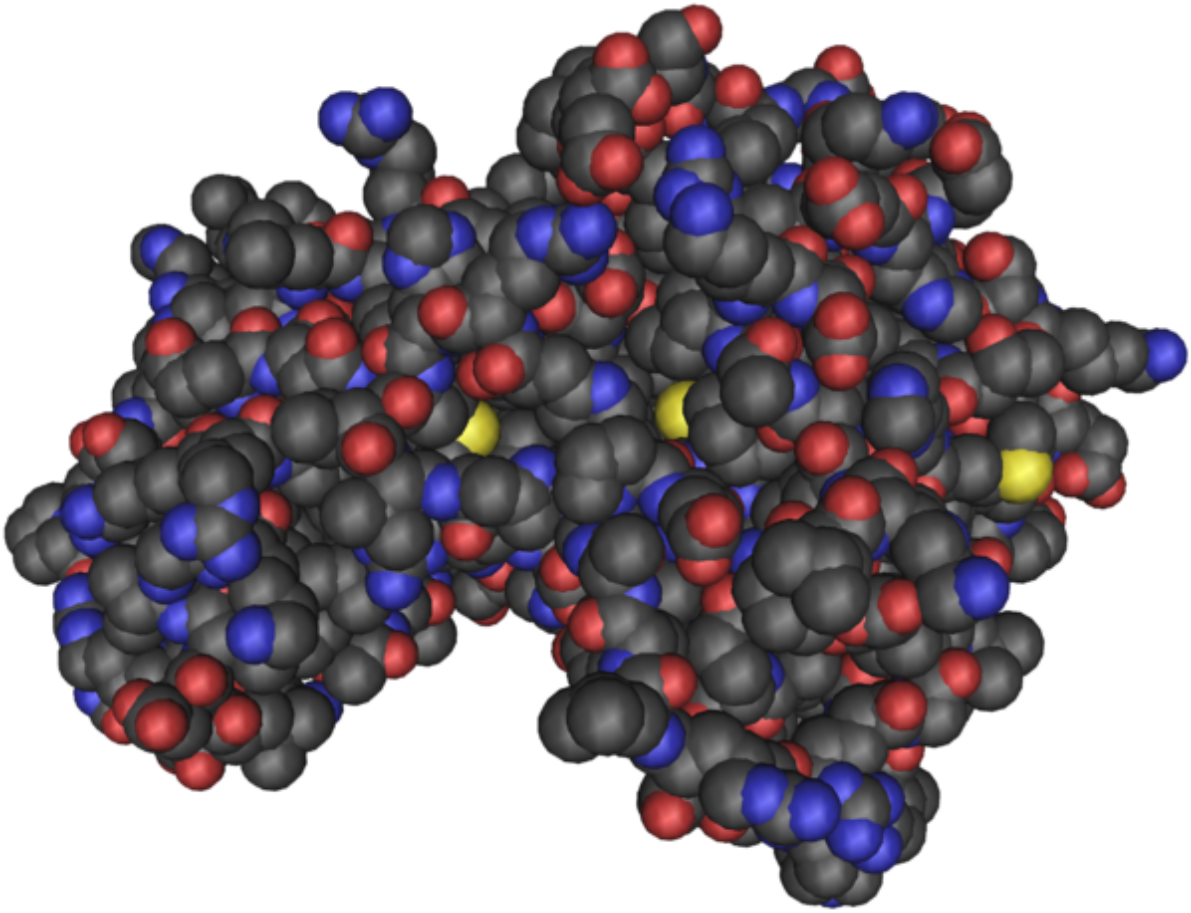
Molecularly shutting down cancer cachexia

August 30 2016



Molecule model of AMP-activated protein kinase. Credit: Nevit Dilmen / Wikipedia / Licence CC BY SA 3.0

Healthy fat tissue is essential for extended survival in the event of tumor-

induced wasting syndrome (cachexia). In *Nature Medicine*, researchers at Helmholtz Zentrum München show that selective manipulation of an enzyme can stop unwanted metabolic processes.

Cancer often results in weight loss due to unwanted metabolic complications. This so-called cancer cachexia is accompanied by a poor prognosis with regard to disease progression, quality of life, and mortality. After sepsis, cachexia is the most frequent cause of death in cancer patients. It is not entirely clear which biochemical mechanisms play a role. To date there have also not been any pharmacological possibilities for selectively influencing tumor-associated wasting syndrome.

Stopping energy wasting molecularly

Researchers at the Institute for Diabetes and Cancer (IDC) at Helmholtz Zentrum München have identified the AMP-activated protein kinase (AMPK) as the central enzyme in cancer cachexia. AMPK is normally responsible for protecting cells from energy deficiency. In the case of cancer cachexia, however, AMPK activity is inhibited due to the illness, resulting in a pointless waste of the body's own energy store.

Selective AMPK reactivation was successfully carried out in tumor models. The therapeutic manipulation took place through a specific peptide which prevents the interaction between AMPK and the lipid droplet-associated protein Cidea, and which consequently can stop the increased fat breakdown (lipolysis) found in tumor diseases.

"Our data suggest that the preservation of "healthy" adipose tissue can promote not only the quality of life, but also the response to treatment and the survival of cancer patients," says Prof. Stephan Herzig, IDC Director. "The interaction between AMPK and Cidea can be taken as a starting point for developing new lipolysis inhibitors which could then

prevent the breakdown of energy stores in the fat of tumor patients." He furthermore sees possibilities for transferring the acquired insights to other wasting disorders, such as with sepsis or burn injuries.

More information: Maria Rohm et al. An AMP-activated protein kinase–stabilizing peptide ameliorates adipose tissue wasting in cancer cachexia in mice, *Nature Medicine* (2016). DOI: 10.1038/nm.4171

Provided by Helmholtz Association of German Research Centres

Citation: Molecularly shutting down cancer cachexia (2016, August 30) retrieved 7 May 2024 from https://medicalxpress.com/news/2016-08-molecularly-cancer-cachexia.html

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.