



CECAD Excellence Cluster. University of Cologne

Press release

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New principle in cancer treatment found

Scientists of the Cluster of Excellence CECAD at the University of Cologne have developed a new strategy for cancer treatment. Tumor growth is dependent on attracting blood vessels, that supply nutrients and oxygen and dispose of metabolic waste. An insufficient blood supply results in significantly reduced tumor growth. The CECAD researchers were able to show that poisoning of the mitochondria, the cell's power-plants, inhibits blood vessel growth, but has no effects on existing vessels. For this purpose the scientists used the weak mitochondrial poison Embelin. Selective inhibition of mitochondrial function could represent a fundamentally new therapeutic approach that may help advance the development of cancer treatments.

Cologne, 2014, March 20. A team of researchers from five CECAD departments led by PD Dr. Hamid Kashkar (University Hospital of Cologne, Institute of Clinical Microbiology, Immunology and Hygiene) and Dr. Oliver Coutelle (University Hospital of Cologne, Department of Internal Medicine I) have found a new principle for the treatment of proliferating solid tumors. Tumors are highly dependent on the growth of blood vessels supplying nutrients and oxygen and excreting CO₂ and metabolic waste products. Accordingly, the inhibition of tumor blood vessels by blocking specific vascular growth factors is a strategy that is already being used successfully to treat tumors today. In close collaboration with Dr. Hue-Tran Hornig-Do and Prof. Dr. Rudolf Wiesner (University Hospital of Cologne, Institut of Vegetative Physiology), the CECAD team reports in a recent article in EMBO Mol Med, that Embelin, a substance that is used in African traditional medicine, inhibits vessel growth by a novel mechanism. They showed that Embelin acts as a weak poison for mitochondria, the power plants of cells. They demonstrated that growing blood vessels – but not resting normal blood vessels – are highly dependent on mitochondrial function and have little capacity to compensate for mitochondrial dysfunction induced by Embelin. Together their findings show that Embelin significantly slowed the growth of tumors by inhibiting their blood supply, but had little effect on existing normal blood vessels and other tissues at the concentrations required. The study was supported by further research in collaboration with Prof. Sabine Eming (University of Cologne, Dermatology). Wound healing experiments demonstrated

delayed closure of wounds in the presence of Embelin due to the lack of blood vessel in-growth, providing additional evidence for the effectiveness of Embelin in inhibiting new blood vessel formation.

Experiments in cooperation with Prof. Aleksandra Trifunovic (CECAD) provide additional support for the dependence of new blood vessels on adequate mitochondrial function. In particular, mitochondrial dysfunction induced by mutation in mitochondrial DNA severely impaired the capacity for vascularisation of implanted artificial plugs, designed to attract new blood vessels. In summary, the scientists were able to prove that impairment of mitochondrial function provides a fundamentally new approach to inhibit blood vessel growth in solid tumors with little side effects on normal body functions. Prof. Dr. Rudolf Wiesner: „We all feel excited about this new principle that will provide new approaches in the fight against cancer.“

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