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Manoa researchers collaborate on new anti-cancer drug

Researchers with the University of Hawai‘i Cancer Center and the Department of Chemistry at the University of Hawai‘i at Mānoa have collaborated on the development of a potential new anti-cancer drug that specifically kills renal cancer cells.

Florian Sulzmaier and Joe Ramos of the UH Cancer Center and William Chain (inset) of the UH Mānoa Department of Chemistry have collaborated on the development of a potential new anti-cancer drug.

Cancer Biologist and Associate Professor Joe Ramos of the UH Cancer Center and Assistant Professor William Chain from the Department of Chemistry are the first to describe the biological effects that a compound known as englerin A has on renal cancer cells, as described in the October 22, 2012 edition of the journal PLOS ONE of the Public Library of Science.

Englerin A is a natural product found in the bark of phyllanthus engleri, a plant indigenous to east Africa. This compound was previously reported to be a potent and selective inhibitor that prevents the growth of six human renal cancer cell lines while not affecting other cancer cell types. Chain closely examined the chemical structure of englerin A and was able to effectively synthesize the compound in his lab.

“Synthesizing this compound helped us gain access to reasonable amounts of material needed for biological testing,” said Chain. “Additionally, synthesizing the
natural products compound from scratch enabled us to focus directly on englerin A’s desired effect on human cancer cells.”

With direct access to the synthesized compound, Graduate Student **Florian Sulzmaier** began examining the biological effects englerin A had on renal cancer cells and evaluating its impact on healthy cells.

Their findings confirmed that englerin A is a potent and selective inhibitor in the growth of human renal cancer cells. They further showed that the compound kills tumor cells and has no adverse effects on normal kidney cells. In addition, their research defines some of the biological changes caused by englerin A that precede cell death, revealing that it functions via a mechanism distinct from the current standards of care in the treatment of renal cancer.

“This discovery provides an important basis for the evaluation and validation of the compound’s use as an anti-tumor drug,” said Sulzmaier.

“This work also provides new guidance in the search for the targets of englerin A in renal cancer cells,” added Ramos.

The next step will be to identify how englerin A specifically targets renal cancer cells and to improve the compound’s effectiveness. This will allow the researchers to move forward with the promise of developing a new renal anti-cancer drug.