New target plus new drug equals death of melanoma cells

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Collaborative research presented by the University of Colorado Cancer Center, the University of North Carolina at Chapel Hill, Harvard Medical School and the University of Pittsburgh, at the American Association for Cancer Research (AACR) Annual Conference, shows that the protein receptor Mer is overexpressed in melanoma and that the investigational drug UNC1062 blocks Mer survival signaling in these cells, killing them.

"It's exciting in that Mer receptor expression correlates so perfectly with disease progression. It's tiered – you see a bump in expression as you transition from nevus to melanoma and then again as you transition from melanoma to metastatic disease," says Doug Graham, MD, PhD, investigator at the CU Cancer Center and associate professor of Pediatrics and Immunology at the University of Colorado School of Medicine, the paper's senior author.

After proving this correlation between Mer receptor expression and disease stage in melanoma tissues from clinical patient samples, Graham and colleagues wondered what would happen if they interrupted this Mer signaling. Luckily, the University of North Carolina had recently developed a new compound that did just that – UNC1062. The results were dramatic.

"We showed decreased survival signaling, increased apoptosis and decreased growth of the melanoma cells in dishes and in mouse models," Graham says. It seems that Mer receptors are not only correlated with

melanoma progression but are in fact driving the aggressiveness of the disease.

"This is the first time there's been an association between Mer and melanoma and the first time to report about this new drug," Graham says.

Provided by University of Colorado Denver