

Systemic inflammation, age, cardiac risk linked

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(Medical Xpress) -- Systemic inflammation, the immune system's defense against disease or injury that can contribute to problems like cancer and diabetes over time, increases with age in people with heart-disease symptoms, while inflammation specific to vascular disease does not, a UC Davis study has found.

The outcome, published in the September issue of [Arteriosclerosis, Thrombosis and Vascular Biology](#), is an important step in better understanding the role of different types of inflammation in [heart disease](#). It also underscores the need to consider a broader range of immune-system factors in the quest to find accurate [biomarkers](#) of heart disease, especially in relation to age.

"Age is a known risk factor for heart disease, but the underlying disease processes that contribute to that risk are not well understood," said Lars Berglund, senior study author and senior associate dean for research at the UC Davis School of Medicine. A respected international expert in lipid disorders and [cardiovascular risk](#), Berglund is believed to be the first to look at how age influences both vascular and systemic [inflammatory markers](#).

"Our study shows that the search for answers should look beyond the inflammation linked particularly with vascular disease and instead evaluate the overall burden of inflammation," Berglund said.

Berglund explained that [lifestyle factors](#) like physical activity, diet and

[tobacco use](#) are currently the best predictors of heart disease, however these classic predictors become less reliable with age. Treatment usually begins when symptoms of blood-vessel conditions such as atherosclerosis become apparent, but by then the disease process is well under way. Finding an accurate immune-system biomarker or cluster of biomarkers could lead to earlier diagnosis and interventions that reduce the impact of heart disease, which affects more than 83 million Americans, costs an estimated \$444 billion annually and is the nation's leading cause of death.

For the study, Berglund and his team included 336 Caucasian and 224 African-American patients of two New York hospitals who were about to undergo diagnostic coronary arteriography, a test given to patients with heart-disease symptoms to detect blood flow to the heart and blockages. The participants' ages ranged from 24 to 72.

The researchers measured blood levels of markers for [systemic inflammation](#) — C-reactive protein, fibrinogen and serum amyloid-A — along with markers of vascular inflammation, lipoprotein-associated phospholipase A2 (Lp-PLA2) and pentraxin-3.

They found that the older a study participant was, the higher their blood levels of systemic inflammation markers. [Blood levels](#) of vascular inflammation markers remained stable across ages. These findings did not change among patients of different racial backgrounds, genders or body weights.

Berglund said the outcome suggests that inflammation factors beyond those that directly affect blood vessels may be responsible for increased heart-disease risk as we age.

"It could be that we accumulate an inflammatory load over a lifetime that eventually does harm to blood vessels. Now that we have an idea of

how systemic inflammation compounds over time, we can test that possibility," said Berglund.

Berglund added that developing heart-disease treatments that target the immune system is complicated, since the goal is to reverse harmful effects without altering protective qualities.

"We want to treat the cause but not shoot down the disease-fighting mechanisms," he said. "We still have work to do before we can define heart disease as more than a 'lifestyle disease' and make major breakthroughs in fighting what has become the plague of our time."

Additional UC Davis co-authors of the study include first author Erdembileg Anuurad, Byambaa Enkhmaa and Wei Zhang. Zeynep Gungor, now of the University of Istanbul, was at UC Davis when the study was conducted. Additional authors were Russell Tracy of the University of Vermont and Thomas Pearson of the University of Rochester.

Provided by UC Davis

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