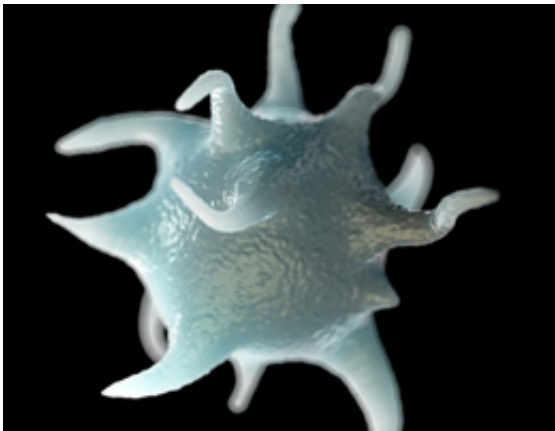


# Anticoagulants to reduce stroke risk may increase incidence of heart attacks

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Activated blood platelet. Credit: royaltystockphoto / fotolia.com

Medical researchers at the Ludwig Maximilian University of Munich have discovered why a new class of anticoagulants designed to reduce the risk of strokes can—in rare cases—increase the incidence of heart attacks.

Cardiac arrhythmias such as [atrial fibrillation](#) can lead to the formation of blood clots in the [heart](#). Rupture and subsequent transport of such clots into the brain can in turn result in strokes. To reduce the risk of stroke, patients who suffer from arrhythmias receive coagulation inhibitors designed to prevent clot formation. So-called new oral anticoagulants (NOACs)—which include the oral thrombin inhibitors (OTIs, e.g., dabigatran) - provide a valuable alternative to the vitamin K

antagonists (VKAs, e.g., marcumar) that are conventionally used for this purpose. However, clinical trials have shown that the use of OTIs in patients with atrial fibrillation is actually associated with a slight increase in the incidence of heart attacks. Medical researchers at LMU have investigated why this is so and, as they report in the journal *Science Translational Medicine*, their findings show that atherosclerotic alterations of the vasculature might predispose for the increase in myocardial infarction among patients who receive OTIs.

OTIs represent an entirely new class of agent, which directly inhibits thrombin, the central clotting factor. In the course of the new study, the LMU team carried out tests on blood drawn from patients who were being treated with OTIs. The tests revealed that, under certain circumstances, OTIs can promote the formation of clots. Thus, under flow conditions on atherosclerotic plaque material, the incidence of platelet aggregation is increased in the presence of OTIs—as is the risk of [clot formation](#) (which involves platelet aggregation) at sites of blood-vessel damage. "These features reflect what happens in the early phase of an acute heart attack precipitated by the rupture of an atherosclerotic plaque," explains Tobias Petzold, first author on the new study.

The researchers went on to show that OTIs alter the interaction of thrombin with the receptor that mediates platelet binding, which in turn stimulates platelet aggregation. This effect of OTIs could be prevented by the addition of aspirin. "The clinical advantages of an oral thrombin inhibitor relative to a VKA have been clearly demonstrated in several studies," says Petzold. "However, we conclude from our results that certain patients will benefit from a personalized therapy, and that one should ascertain whether the patient suffers from coronary heart disease and assess his or her risk of [heart attack](#) before beginning a course of treatment with oral thrombin inhibitors."

**More information:** T. Petzold et al. Oral thrombin inhibitor

aggravates platelet adhesion and aggregation during arterial thrombosis, *Science Translational Medicine* (2016). DOI: [10.1126/scitranslmed.aad6712](https://doi.org/10.1126/scitranslmed.aad6712)

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