If you’ve taken high school biology, you know that your body needs platelets to stop bleeding at the site of a wound. University of Rochester researcher Dr. Craig Morrell, however, sees these good guys of the circulation system in a little different light.

“When a platelet gets activated, it forms a blood clot so you don’t bleed to death,” he said. “It also secretes inflammatory molecules, to promote an immune response and recruit white blood cells to fight infection. When we look at things like cerebral malaria, platelets can do bad things by promoting inflammation.”

Many patients with vascular issues take cumadin or other “blood thinners” to limit the effects of platelets, but these medications require weekly blood tests to keep the levels appropriate for the patient’s health. Finding a lower-maintenance alternative would improve quality of life for millions of patients.

Dr. Morrell and his lab team used mice to discover that platelets can be both good and bad in fighting infections like malaria. “Immediately after we infect the mice, the platelets get activated, which helps activate the acute phase response,” he said. “But later, the platelets cause inflammation, which leads to lesions in the brain.”

Dr. Morrell’s lab includes a cardiology fellow who researches platelet activity after heart attack, revealing some important insights. “After a heart attack, platelets attract the white blood cells, which actually drive that heart attack,” said Dr. Morrell.

Antiplatelet therapy can alleviate some of this, but how much is too much? “The goal is to find the sweet spot where you’re slowing down platelet activation but not preventing it,” he explained. “If you can regulate the platelets in certain situations, you can slow them down without totally preventing activation. We’re looking at potential regulators.”