

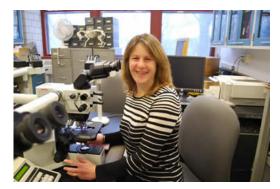
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Breaking down blood clots

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A clot cutting off blood to the wrong place can spell disaster or death for unborn foals and even adult horses. When the infectious disease equine herpes virus-1 (EHV-1) causes its infamous effects, abortions and adult neurological disease, blood clots are to blame.

Clinical pathologist Dr. Tracy Stokol has been investigating how the virus triggers these clots. Funded by the Harry M. Zweig Memorial Fund for Equine Research, her work investigating the role of platelets in the pathogenesis of EHV-1 infection has shown



that EHV-1 virus particles seem to be binding to platelets, small cells in blood involved in clotting.

When incubated together with platelets at a multiplicity of infection (MOI) of 1 - a ratio of 1 virus particle per platelet - particles of the neuropathogenic strain Ab4 and abortion-inducing strain RacL11 induced platelet activation within 10 minutes. Activation causes the release of P selectin, a protein that platelets use to bind to other cells, such as the cells lining blood vessels. The viral gene product glycoprotein B was also amplified from platelets, suggesting that the virus is binding to them directly.

"We are excited to discover that EHV-1 activates platelets," said Stokol. "Platelets play a crucial role in thrombosis, a major cause of abortion and neurological symptoms due to EHV-1. If platelets are involved in the pathogenesis of these EHV-1-associated disease syndromes, administration of platelet-inhibiting medications such as Plavix or aspirin may prove useful in the treatment of infected horses."

Stokol is planning several experiments to address the many remaining questions regarding how EHV-1 activates platelets. These include projects that will determine if virus-mediated platelet activation requires other clotting proteins in blood, if the virus uses known cell receptors, such as MHCI, to bind to and then activate platelets, and if inhibitors, such as Plavix and aspirin, can prevent the virus-induced platelet activation.

She is continuing an innovative technique involving growing equine endothelial cells, which line blood vessels, in a microfluidic device to determine whether virus-activated platelets show increased binding to these cells, which could spur clotting and potentially inflammation.

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