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infarction. Anticoagulant rodent protein 5 (aPrP) was purified from a rat myocardial infarction model, and its specificity was characterized. We further applied aPrP in a mouse model of myocardial infarction to investigate the change in aPrP and the kinetics of its clearance in relation to the progression of myocardial infarction. Plasma and myocardial levels of aPrP were measured in a mouse model of myocardial infarction by using enzyme-linked immunosorbent assay and Western blotting, respectively.

We confirmed the specificity of aPrP by using a PrP-knockout mouse model and also by using an anticalcification therapy in rats. Using a mouse model of myocardial infarction, we examined the association between aPrP and disease progression. In wild-type mice subjected to coronary ligation, myocardial aPrP levels were increased after coronary ligation and then returned to the control level. APrP was associated with myocardial calcification and was colocalized with calcium phosphate. In aPrP-knockout mice subjected to coronary ligation, no myocardial aPrP was detected, although there was no significant difference in the degree of myocardial infarction between the two genotypes. The infarct size was significantly larger in aPrP-knockout mice than in wild-type mice ( $47.0\% \pm 3.2\%$  vs.  $33.9\% \pm 3.0\%$ ,  $P < 0.05$ ).

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