Plasminogen activator inhibitor type 1 promote experimental aortic dissection in mice.

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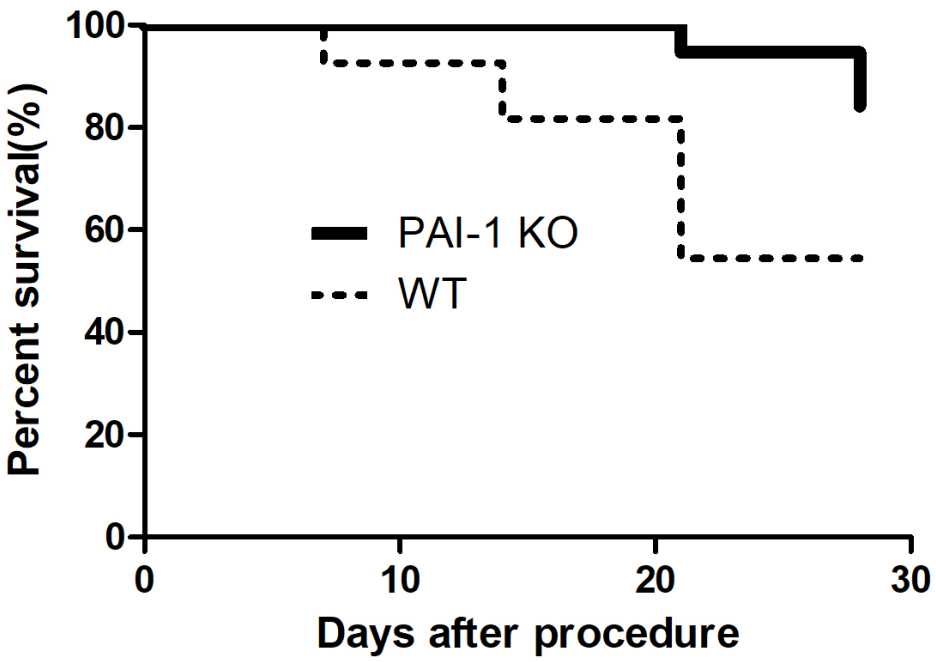
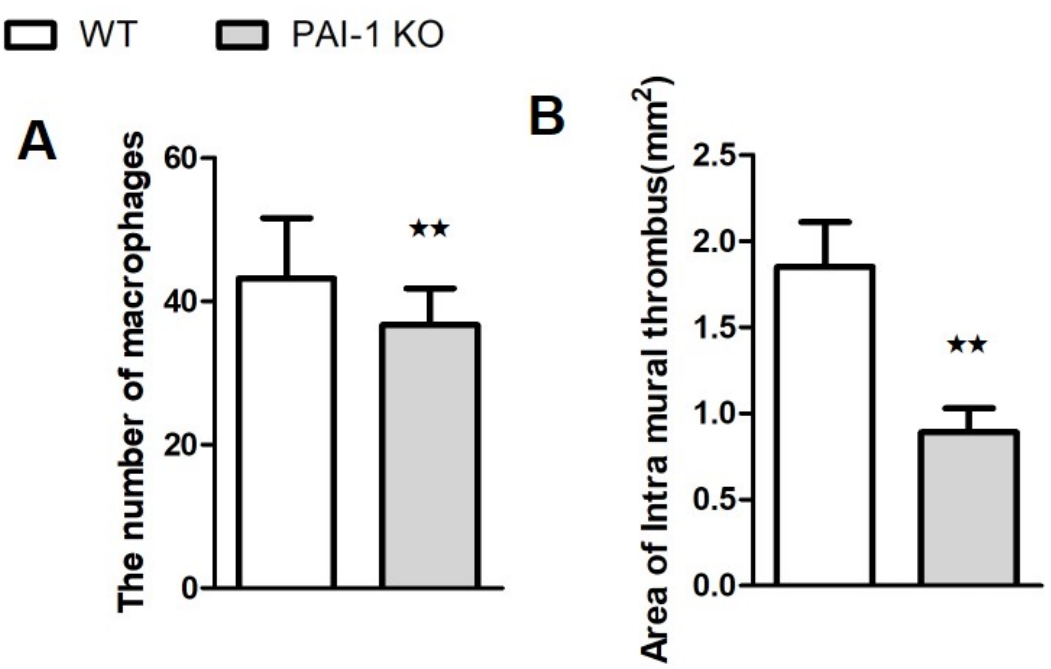
Method: Male 18–20-week-old PAI-1 deficiency mice and C57Bl/6J mice used as wild type (WT) were treated by angiotensin II infusion and were administered 0.2 % beta-aminopropionitrile in drinking water for 28 days.



PAI-1 deficiency significantly reduced the mortality caused by aortic rupture followed by dissection with bleeding.



Macrophage infiltration to the wall was decreased by PAI-1 deficiency compared to WT mice

Conclusions—Our results revealed a previously unknown pathogenic pathway involving PAI-1 that plays a key role in AAD occurrence.