

# Abstract

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## **Evaluation of lipoprotein(a) as a prothrombotic factor: progress from bench to bedside.**

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**PURPOSE OF REVIEW:** Since the homology between apolipoprotein(a) (apo(a)) and plasminogen was discovered in 1987, the role of lipoprotein(a) (Lp(a)) as an inhibitor of the normal fibrinolytic role of plasmin(ogen) has been a major research focus. In this review we summarize recent basic research aimed at identifying mechanisms by which Lp(a) can either inhibit fibrinolysis or promote coagulation, as well as recent clinical studies of Lp(a) as a risk factor for thrombosis either in the presence or absence of atherosclerosis.

**RECENT FINDINGS:** It has recently been reported that the inhibition of plasminogen activation by apo(a) results from the interaction of apo(a) with the ternary complex of tissue-type plasminogen activator, plasminogen and fibrin, rather than competition of apo(a) and plasminogen for binding sites on fibrin. Lp(a) species containing smaller apo(a) isoforms bind more avidly to fibrin and are better inhibitors of plasminogen activation. Recent clinical studies have provided strong evidence that Lp(a), either alone or in synergy with other thrombotic risk factors, significantly increases the risk of venous thromboembolism and ischemic stroke.

**SUMMARY:** Lp(a) both attenuates fibrinolysis, through inhibition of plasminogen activation, and promotes coagulation, through alleviation of extrinsic pathway inhibition. Further basic and clinical studies are required to more clearly define the role of Lp(a) in thrombotic disorders, and to determine the extent to which thrombotic risk is dependent on apo(a) isoform size.

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