



# Maternal Thrombophilic and Hypofibrinolytic Genetic Variants and Idiopathic Recurrent Pregnancy Loss: Correspondence

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Dear Editor, the article titled "Maternal Thrombophilic and Hypofibrinolytic Genetic Variants in Idiopathic Recurrent Pregnancy Loss (RPL): a Continuing Mystery [1]" caught our attention. No differences between control women and women with RPL were seen in the prevalence of the other studied variations, according to Younis et al. [1]. Younis et al. concluded that there was insufficient evidence to relate the RPL risk to the F5 1691G > A/4070A > G, MTHFR 677C > T/1298A > C, and APOE 388 T > C/526C > T haplotypes. It may be possible to identify women at risk of RPL and enhance RPL susceptibility prediction by using the F13A1 G103T, FGB—455G > A, and ITGB3 T1565C variations, which are linked to a higher possibility of developing RPL [1]. A promising method for more accurate RPL risk assessment could result from routine workup including molecular testing of thrombophilic and hypofibrinolytic genetic variations [1].

There may or may not be a role for thrombophilic and hypofibrinolytic genetic variations in the etiology of RPL. We must be aware of additional potential complicating issues, such as concurrent chronic infections and reproductive organ anatomical issues. Other non-thrombophilic and hypofibrinolytic genetic variants may potentially have an impact on the genetic variant effect. The genetic polymorphisms in the 3'-Untranslated Regions of SMAD5, FN3KRP, and RUNX-1, as well as interleukine—6 polymorphisms [2–4] are noteworthy examples. If the consequences of

additional genetic differences are further investigated, other research may offer more persuasive data.

## Declarations

**Conflict of Interest** None.

## References

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