VEGF expression by SES could affect and may be related to stent thrombosis. Although not proven, the inhibition of reduced stimulus for endothelial regeneration and may be attributed to parallel effects of rapamycin on cellular VEGF production. This possibility was explicitly mentioned in our manuscript, together with the hypothesis that a reduction in VEGF serum levels may be attributed to parallel effects of rapamycin, e.g. anti-inflammatory properties. Indeed, we did not go into the subject in detail, as this was not the aim of our study. Moreover, our results showing that there is no correlation between serum levels of VEGF and in-stent restenosis, did not provide the motivation to research the origin of serum VEGF changes further.

We certainly agree with the authors’ statement that other factors apart from monocytes, such as platelets, also make an important contribution to vascular endothelial growth factor (VEGF) concentrations in human blood, and that the reduced VEGF serum levels following sirolimus-eluting stent (SES) implantation may reflect a systemic effect of rapamycin on cellular VEGF production. This possibility was explicitly mentioned in our manuscript, together with the hypothesis that a reduction in VEGF serum levels may be attributed to parallel effects of rapamycin, e.g. anti-inflammatory properties. Indeed, we did not go into the subject in detail, as this was not the aim of our study. Moreover, our results showing that there is no correlation between serum levels of VEGF and in-stent restenosis, did not provide the motivation to research the origin of serum VEGF changes further.

We certainly agree with the authors’ comment that decreased VEGF levels following SES implantation may reflect a reduced stimulus for endothelial regeneration and may be related to stent thrombosis. Although not proven, the inhibition of VEGF expression by SES could affect and delay re-endothelization, creating conditions favourable for acute stent thrombosis. We also wished to indicate a possible implication of the above hypothetical mechanism in the case of the patient in the SES group who suffered from an acute myocardial infarction post-stenting. However, this unique case among the small number of participants of our study did not allow us to draw any further inferences. Further research efforts could focus on that direction.

We do not dispute that it would be interesting to look at the monocyte counts. On the other hand, the aim of our study was to investigate the link between angiogenic factors and restenosis as these are reflected through VEGF gene expression in monocytes and not the number of monocytes, since this is already known from previous reports to be correlated with late luminal loss. Besides, monocyte activation and their VEGF gene expression do not depend on the absolute number of monocytes. Nonetheless, we concur that future studies should focus on the functional aspects of monocytes, such as adhesion or chemotaxis, in order to obtain a clearer picture of the pathophysiology of neointima formation following coronary stenting.

**References**


**Corrigenda**

doi:10.1093/eurheartj/ehn316


On page D24, in Figure 18, the placement of fluorine groups and stereochemistry in the chemical structure of AZD6140 were incorrectly shown. The correct figure is reprinted below.

![Correct Figure](https://academic.oup.com/eurheartj/article-abstract/29/15/1925/510728/1)


Regrettably, on page 187, in the list of author names, the name of Dr Kiotsekoglou was incorrectly quoted as ‘Kiotsekolglou’.

**Letters to the Editor**

**Online publish-ahead-of-print 17 June 2008**

**Vascular endothelial growth factor protein levels and gene expression in peripheral monocytes after stenting: a randomized comparative study of sirolimus-eluting and bare-metal stents: reply**

We thank the authors for their interest in our work. We have read their comments carefully and are pleased to have the opportunity to reply.

Czepluch et al. correctly state that other factors apart from monocytes, such as platelets, also make an important contribution to vascular endothelial growth factor (VEGF) concentrations in human blood, and that the reduced VEGF serum levels following sirolimus-eluting stent (SES) implantation may reflect a systemic effect of rapamycin on cellular VEGF production. This possibility was explicitly mentioned in our manuscript, together with the hypothesis that a reduction in VEGF serum levels may be attributed to parallel effects of rapamycin, e.g. anti-inflammatory properties. Indeed, we did not go into the subject in detail, as this was not the aim of our study. Moreover, our results showing that there is no correlation between serum levels of VEGF and in-stent restenosis, did not provide the motivation to research the origin of serum VEGF changes further.

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**References**


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