Reviewer's report

Title: Curcumin supplementation could improve diabetes-induced endothelial dysfunction associated with decreased vascular superoxide production and PKC inhibition

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Reviewer: M Balasubramanyam

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The manuscript needs a thorough revision based on the following comments:

1. Curcumin has been recently shown to ameliorate high glucose-induced acute vascular endothelial dysfunction in rat thoracic aorta (Fang et al 2009 Clin.Expt.Pharmacol.Physiol.). This reference needs to be added and discussed in relation to the findings of the study.

2. An important clinical study has evaluated the effect of curcumin on endothelial function, oxidative stress and inflammatory markers in Type 2 diabetes patients (Usharani et al 2008; Drugs R D). Authors should also refer this and discuss.

3. There are some glaring omissions of references: for example (Balasubramanyam et al 2003 Journal of Biosciences; Premanand et al 2006) on curcumin work related reactive oxygen species generation and PKC inhibition, respectively.

4. In Figure 5, D – the lumen of artery seems constricted compared to other samples. What is the explanation for this?

5. In page 13 while discussing the possible mode of action of curcumin, authors states like this – “However, we need to confirm this up-coming hypothesis again by insulin monitoring. What is the rationale for insulin measurement in STZ treated model systems?”

6. PKC inhibition shown as an immunofluorescence micrography is not convincing. It would be rather important to show PKC translocation to membrane and its inhibition by curcumin.

7. What would have been expected if curcumin intervention is given soon after the STZ treatment? Current studies imply that poor glycemic control over a period imposes hyperglycemic memory at the tissue levels and this might have drastic consequences downstream. The only modest reductions in results of plasma glucose and HbA1c in curcumin administered STZ rats reinforces this. These need to be discussed in discussion.