Author's response to reviews

Title: Gene network analyses point to the importance of human tissue kallikreins in melanoma progression

Authors: Waleska K Martins (wkerllenmartins@gmail.com)
Gustavo H Esteves (gesteves@uepb.edu.br)
Otávio M Almeida (o.almeida@clinicanomina.com.br)
Gisele G Rezze (ggrezze@hotmail.com)
Gilles Landman (gilles.landman@gmail.com)
Sarah M Marques (sarah.marques@hsl.org.br)
Alex F Carvalho (alex.carvalho@hcancer.org.br)
Luiz Fernando L Reis (luiz.reis@hsl.org.br)
João P Duprat (jduprat@uol.com.br)
Beatriz S Stolf (biastolf@yahoo.com.br)

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Gene network analyses point to the importance of human tissue kallikreins in melanoma progression

Waleska K Martins, Gustavo H Esteves, Otávio M Almeida, Gisele G Rezze, Gilles Landman, Sarah M Marques, Alex F Carvalho, Luiz Fernando L Reis, João P Duprat and Beatriz S Stolf

Dear Editor,

First of all, we would like to thank the reviewers for the relevant critiques that helped us to improve our manuscript and for the recommendation of its publication. We have now addressed more clearly the question raised by one of the reviewers, including a new paragraph in page 21 (discussion section). We have also revised the text and corrected minor mistakes.

We believe that all the points raised by this referee are now included and that this new version of our manuscript is suitable for publication in BMC Medical Genomics.

Best regards,

Waleska K Martins
Response to reviewer Jaime Matta

1) I recommend that in the discussion be more critical of the limitations of the array they used. The answer to that point (#5) in the cover letter by-passes the fact that the technical platform used failed to select genes and pathways that are well established in the melanoma carcinogenesis process. This is not a fatal flaw that would hinder the publication of this manuscript, but need to be more critical of the limitations of their study.

The reviewer is correct to point that our customized array doesn’t include all genes that may be involved in melanoma carcinogenesis process. However, we found many altered modules in our study that were also identified in comparisons of melanoma gene expression using whole human genome expression arrays (GeneChip Human Genome U133 Plus 2.0 Array- Ricker et al, 2008).

We believe that even using a limited platform, we were able to add important information to melanoma progression due to the construction of a cancer related cDNA array and a careful selection of melanoma related modules. In fact, our strategy of analysis of altered modules (previously described by Segal et al., 2003) allowed the identification of known pathways that change during melanoma progression such as keratinocyte differentiation, epidermal development, cell adhesion, and cell-cell signaling in metastatic samples (Jaeger et al., 2007, Riker et al., 2008, Ren et al, 2008), as well as pathways involved in melanoma progression that hadn’t been described before, such as kallikrein related genes.

References

