LETTER TO THE EDITOR

The proteome differences – new trend of placenta examination

Svandova I¹, Volfova B¹, Zahumensky² J, Kucera E², Novotny J¹

Department of Physiology, Faculty of Science, Charles University in Prague, Czech Republic. iva@natur.cuni.cz

Comment and completion of our results on: Loncar "The comparison of ultrasonographic placenta examination with pathohistologic verification of fetal anomalies", published in Bratisl Lek Listy 2011, 112 (1).

To the Editor

Sir, we read with an interest the article by Loncar on the study cohort of 15 women with embryo abnormalities, the author correlates ultrasound placenta examination and pathohistological findings in placenta with different kinds of embryo abnormalities. Two main ultrasound placenta findings of those patients were hydrops placenta and cystic placenta degeneration.

With no doubts, ultrasound examination is the most comfort, but limited. For explanation, a powerful methodology of pathohistology and immunohistochemistry is excellent for describing changes of protein levels in terms correlated to tissue structure. There is a relatively new method dealing with visualisation of whole protein body of the tissue – 2-D electrophoretic protein separation. The proteome (= the entire set of proteins expressed by tissue) is divided according to Mr and pI of individual proteins and changes in level of every particular protein can be checked in one gel.

With consideration of the above-mentioned paper of Loncar, we introduce the first glance at differences in smokers and non-smokers placental proteome in term. Placentas and foetuses of smoking pregnant women also exhibit a broad range of abnormalities, some of them described in the work by Loncar. Although smoker placentas show no increased incidence of necrosis, there are detectable microscopic lesions reflecting a periodic low perfusion. Those lesions typically include obliterative endarteritis, slight cytotrophoblast hyperplasia, stromal fibrosis, and small villous infarcts. The most frequent lesions in smoker placenta are degeneration of the cytotrophoblast, cytotrophoblast hyperplasia, focal syncytial necrosis, reduced number of vasculo-syncytial membranes,

irregular thickness of the basement membrane, and increased collagen content in the villous stroma. Smoking during pregnancy also significantly increases villus surface and their calcification.

We present here the first visualisation of whole placenta proteome of non-smokers (n=5) and smokers (n=5) at term.

The proteome of 5 pooled samples of non-smoker and smoker placentas were compared. Firstly, we established the proteome map of normal human placenta as a standard for further proteomic analyses. The spots on 2-D gels were visualized by SPYRO-Ruby staining in the 4–7 pI range. About 400 distinct spots were detected in stained gels. Analysis of stained gels between the two groups revealed 19 spots with up or down changed expression. Expression of 11 spots was up- regulated in placentae of smoking women, 8 protein spots were down-regulated in them. Changes in expression of 4 spots were statistically significant.

Generation of placental proteome maps struggles with counterbalancing resolution of the maps and maps background to gain the best results. Finally, we performed 2-DGE with extraction protocol based on dichlormethane/methanol sample precipitation and 7M urea/2M thiourea/CHAPS solubilisation. Our work, performed on complex samples of total placental stromal tissue, revealed changes in expression of 19 placental proteins. We suppose that differentially expressed protein may belong to different functional protein classes.

Chronic hypoxia in smokers leads to changes in expression of many regulatory proteins, thus also affecting placental angiogenesis. Compensatory angiogensis resulting from adaptation to hypoxic stress can serve as protective phenomenon in smoking women against preeclampsia.

References

1. Loncar D. The comparison of ultrasonographic placenta examination with pathohistologic verification of fetal anomalies. Bratisl Lek Listy 2011; 112: 634–636.

Received February 28, 2012. Accepted August 10, 2012.

Address for correspondence: I. Svandova, Department of Physiology, Faculty of Science, Charles University in Prague, Vinicna 7, CZ-128 44 Praha 2, Czech Republic.

Acknowledgement: This work was supported by Ministry of Education, Youth, and Sports of the Czech Republic (grant MSM0021620858).

¹Department of Physiology, Faculty of Science, Charles University in Prague, and ²Department of Gynaecology and Obstetrics, 3rd Faculty of Medicine, Charles University in Prague, and Faculty Hospital Kralovske Vinohrady, Praha, Czech Republic