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Some Results of the Evaluation of Protein Activity in Placental Specimens During Normal and Post-Term Pregnancies

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Abstract

Background: However, the placental growth depends on from SHP-2 protein, its main duty is still undefined. P38 MARK pathway is one of the main transducers within placental IGFs' signal and is responsible for organogenesis and trophoblast differentiation.

Objective: To define the activation of SHP-2 and p38 protein in post-term pregnancy placenta.

Method: The protein concentrate from the placenta is defined through the BCA experiment kit and ELISA reader. SHP 2 protein and p-p38 protein activation are determined in normal and post-term pregnancy placental specimen using immunoblotting.

Results: As a result of our study, we noticed that SHP protein synthesis increased unevenly in normal and post-term pregnancy. P38 protein phosphorus which is an inflammation transcription factor, is not determined in both pregnancy groups.

Conclusion: SHP 2 protein synthesis is determined persistently in post-term pregnancy. P38 activation was detected in a placental tissue specimen.

Keywords: Postterm; Placenta; Immunoblotting

Background

For fetal development, the fetus receives all the necessary nutrition, oxygen from the mother through the placenta. If there is no change in size and thickness of the placenta the fetal development decreases and adversely results in adult life. There is some studies mentioned about the positive correlation between fetal and placental development [1].

During fetal growth, the SHP-2 protein acts as a leading role for placental cell development (trophoblastic cell development). (Yang et al. 2006) [2]. SHP-2 protein works for placental growth[3]. As a result of research in insulin-like growth factor (IGF), the SHP-2 molecule is defined as the main transducer of IGF function. In terms of placental insufficiency, there is a possible effect of IGF [2]. For example, the SHP-2 protein is involved in the pathogenesis of the disease with other IGF factors. However, the placental growth depends on SHP-2 protein, its main duty is still undefined [4]. In 7-9 weeks of gestation, increasing oxidation in results inactivation of the p38 MARK affects the placental early development and physiology and forms the placental disease [5]. P38 MARK pathway is one of the main transducers within placental IGFs' signal [2] and is responsible for organogenesis and trophoblast differentiation [6,7]. Pregnancy duration and placental apoptosis have a positive correlation [8]. The increasing apoptosis was confirmed during IUGR [9]. Increased apoptosis during post-term pregnancy accelerates the normal aging of the placenta. If apoptosis occurs initially, it may increase the risk of pregnancy-related morbidity [10]. Therefore, p38 MAPK may impact placental change such as placental formation, post-term pregnancy, some placental abnormality in gestational diseases [10].

Objective

Determination of SHP2 and p-p38 protein activity in placental specimens during normal and post-term pregnancies.

Method

The protein concentrate from the placenta defined through BCA experiment kit and ELISA reader. 2 mcl protein suspense liquified through 23mcl PBS and BCA solution added and light absorption is measured. The placental protein concentrate is in normal pregnancy measured between 43.17 –50.46 mcg/ml, in post-term pregnancy 50.61-66.64 mcg/ml. Furthermore, we have estimated 20-40 mcg protein in the gel hole.

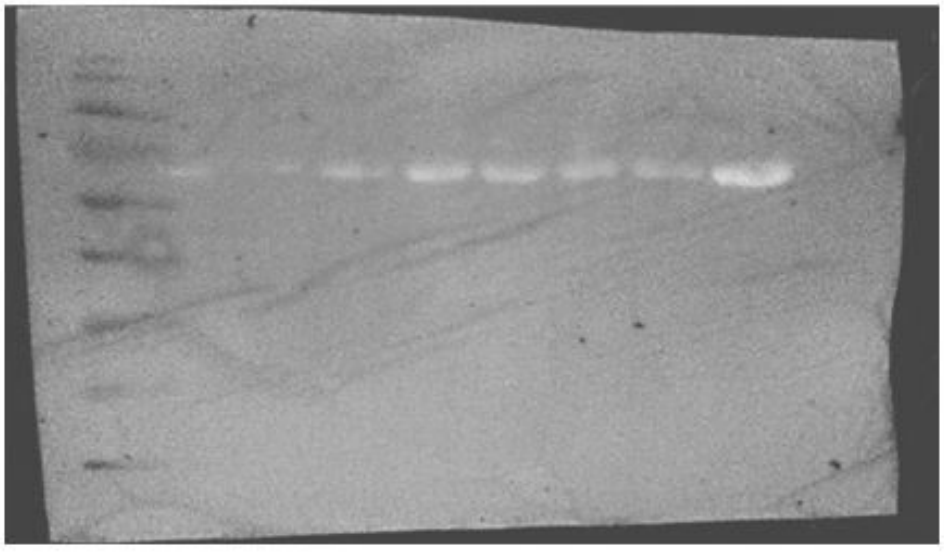

Figure 1: Evaluation of SHP_2 protein synthesis in the placenta of postterm and normal term pregnancy

Table 1: Determination of protein concentration in placental specimens

	Group	Measure-ment 1	Measure-ment 2	Measure-ment 3	BSA 1	BSA 2	BSA 3	Average	Dilution (×12.5)	Laemmli buffer (×5)	20 мкг	30 мкг	40 мкг
Normal term pregnancy group	Normal term pregnancy-placental specimens 1	0.37	0.38	0.42	4.22	4.32	4.96	4.50	56.27	45.02	0.44	0.67	0.89
	Normal term pregnancy-placental specimens 2	0.38	0.40	0.41	4.34	4.63	4.74	4.57	57.12	45.70	0.44	0.66	0.88
	Normal term pregnancy-placental specimens 3	0.40	0.36	0.38	4.58	4.02	4.35	4.32	53.97	43.17	0.46	0.69	0.93
	Normal term pregnancy-placental specimens 4	0.43	0.43	0.43	5.09	5.04	5.01	5.05	63.08	50.46	0.40	0.59	0.79
Postterm pregnancy group	Post-term pregnancy-placental specimens 1	0.43	0.43	0.43	5.01	5.07	5.11	5.06	63.26	50.61	0.40	0.59	0.79
	Post-term pregnancy-placental specimens 2	0.47	0.51	0.50	5.65	6.22	6.06	5.97	74.67	59.74	0.33	0.50	0.67

Note: Placental tissue was collected in the normal pregnant group, including 4 mothers with normal pregnancies and post-term pregnant women in the post-term pregnant group. Protein concentrations were determined by measuring light absorption three times and evaluating the results against the standard albumin curve (BSA, Bovine Serum Albumin). The mean was multiplied by the dilution factor (x12.5) to determine the final protein concentration in the x5Laemmli buffer. 20-40 μg of protein was used for each helium cell.

Immunoblotting of shp2 and p-p38 proteins in placental specimens

SHP 2 protein and p-p38 protein activation are determined in normal and post-term pregnancy placental specimen using antigen-specific primary antibody and immunoblotting. The Immunoblotting result showed in a graphic chart using ImageJ and audit band. From the results of immunoblotting, in normal pregnancy SHP protein synthesis increased unevenly. In contrast, post-term pregnancy, SHP 2 protein is increased in the whole placental specimen. However, p38 protein phosphorus, inflammation transcription factor, is not determined in both pregnancy groups. For example, SHP 2 protein synthesis was low in 1st and 2nd placental specimens while it was high in 3rd and 4th specimens. In contrast post-term pregnancy, SHP 2 protein synthesis was high in all postterm specimens. P-p38 protein phosphorus is not seen in both group.

In placental specimens of mothers with postterm and normal pregnancies SHP2 and p-p38 protein activation.

According to Yang et all SHP-2 protein effects on human placental cell development [2]. As a result of the study SHP-2 molecules in the placenta plays as the main conductor for IGF function [2]. To add more, SHP-2 protein supports placental growth [3]. However, SHP 2 protein's clear effect on placental growth confirmed, its postterm pregnancy influence is still unclear. Some research papers mentioned p38 MAPK involvement in placental differentiation and the possible effect on placental change during gestational diseases such as postterm pregnancy [10]. As a result of our study, we noticed that SHP protein synthesis increased unevenly in normal pregnancy. In contrast, in post-term pregnancy, SHP 2 protein is increased in the whole placental specimen. However, p38 protein phosphorus, an inflammation transcription factor, is not determined in both pregnancy groups. Consequently, we concluded that SHP 2 protein possibly increases in post-term pregnancy, which is similar to other research papers mentioned about SHP2 protein support on placental growth [3]. Furthermore, this protein should be determined in the umbilical cord.

Conclusion

SHP 2 protein synthesis is determined persistently in post-term pregnancy. P38 activation was not detected in a placental tissue specimen.

References

1. Li SM (2016) The biological function of SHP2 in human disease. Molecular Biology. 2016;50(1):22-27.

2. Forbes K, Westwood M (2010) Maternal growth factor regulation of human placental development and fetal growth. The J endocrinology 207: 1-16.

3. Scientists Discover Key Factor In Regulating Placenta And Fetal Growth. Biotechnology and Biological Sciences Research Council 2009.

4. Obnarujen kluchevoi factor regulytsi rosta placenta and embryo. News. Pregnancy, childbirth 2009.

5. Hernandez I, Fournier T, Chissey A (2019) NADPH oxidase is the major source of placental superoxide in early pregnancy: association with MAPK pathway activation. Scientific reports 9: 1-12.

6. Adams JM (2003) Ways of dying: multiple pathways to apoptosis. Genes & development 17: 2481-95.

7. Webster RP, Brockman D, Myatt L (2006) Nitration of p38 MAPK in the placenta: association of nitration with the reduced catalytic activity of p38 MAPK in pre-eclampsia. Molecular human reproduction 12: 677-85.

8. Smith S, Baker PN, Symonds EM (1997) Placental apoptosis in normal human pregnancy. Ame J obstetrics and gynecology 177: 57-65.

9. Smith SC, Baker PN, Symonds EM (1997) Increased placental apoptosis in intrauterine growth restriction. Ame J obstetrics and gynecology 177: 1395-1401.

10. Smith SC, Baker PN (1999) Placental apoptosis is increased in post-term pregnancies. BJOG: An Int J Obstetrics &Gynaecology 106: 861-2.