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ASPRE model for prediction and prevention of preeclampsia and molecular approach for a personalized diagnosis with an attempt for prevention in an in-vitro model with CRISPER/Cas9

Preeclampsia (PE) affects 2-8% of pregnant women and is a major cause of short and long-term maternal and perinatal morbidity and mortality. ASPRE (Combining first trimester risk prediction and evidence based prevention by low dose aspirin) has shown a 75% PE risk prediction by history, biochemical and biophysical markers at 10% false positive rate, identifying 10% of the high risk population of pregnant women. Aspirin provided to the high risk group from the first trimester prevents 62% of preterm preeclampsia (<37 weeks gestation), 89% of preeclampsia <32 weeks, and reduced by 67% the duration of stay and cost of NICU. Yet 25% of preterm cases remained un-identified and term PE are detected in only 44% of the cases. Placental protein 13 (PP13) preeclampsia is a placental specific protein that can be detected in the maternal blood from the 5th gestation week. Reduced PP13 RNA and low first trimester maternal blood level are PE biomarkers. The protein was found to prime the maternal pregnancy vascular system to pregnancy by expanding the pregnancy veins and arteries thus enabling the increase in blood flow and the supply of nutrients and oxygen to the placenta and the fetus. The effect is mediated by the eNOS system providing Oxygen and the prostaglandin system responsible for vessels vasodilation in the first trimester of pregnancy. Low PP13 will lead to insufficient blood and oxygen supply to the pregnancy. The low PP13 is derived of molecular polymorphism of the PP13 proteins with -98A/A promotor variant associated with low PP13 and high risk to term PE, especially among obese women. The 221 thymidine deletion generates a truncated variant is associated with a shorter PP13 and high risk for very sever early preeclampsia associated with fetal and mother mortality. Both mutated variants are more prevailed in women of African origin and may account in part to the higher PPE prevalence in Africa. The truncated variant fails to prime the blood vessels expansion. Using the Crispr/Cas9 system we have generated the thymidine 221 deletion and could generate its repair. The truncated mutation reduces significantly PP13 expression and blood vessels expansion. Mutation repairs by this system- renew both effect. This approach has the potential for major reduction in death and handicap for mothers and babies.

Biography

Hamutal Meiri holds a PhD in Neurobiology from the Hebrew University of Jerusalem (1979) and a MBA degree from Tel Aviv University, Recanati School of Business (1995). She was a faculty member in brain development in the medical schools of Tel Aviv University, Technion, and NYU (1982-1990), and a Visiting Professorship at Weil-Cornell Medical College, NY. In 1991 she was appointed to be the first Director of Israel National Committee of Biotech, and also served as the Research Advisor to UNESCO COBIOTECH Committee. In 1994-1999 she was the Consortium Director of Israel Chief Scientist Magnet program (Biotech). In 1996-1998 she was elected to be the Head of Israel Telemedicine Industry Forum, and also served as a consultant to the Scandinavians Prime Ministers on Telemedicine.

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