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Paternal Role in Preeclampsia

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1. Psychosocial and biological paternal role in pregnancy outcomes.

Author(s): Kashanian, Maryam; Faghankhani, Masoomeh; Hadizadeh, Hasti; Salehi, M Masoud; Roshan, Masoomeh Yousefzadeh; Pour, Mohammad Ehsani; Ensan, Ladan Sayyah; Sheikhansari, Narges

Source: The journal of maternal-fetal & neonatal medicine : the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians; Jan 2020; vol. 33 (no. 2); p. 243-252

Publication Date: Jan 2020

Publication Type(s): Journal Article

PubMedID: 29886805

Abstract: Background: Pregnancy outcomes are affected by many different factors. One of the influential factors on pregnancy outcomes is the male partner as an important person to mother's wellbeing.Objective: The aim of the present study was to investigate the effects of male partner's role including socioeconomic support, emotional support, accompanying pregnant women during prenatal care visits (PNC) and labor, and the level of pregnant women's satisfaction from their partners' support and involvement during pregnancy on pregnancy outcomes.Method: Two hundred first gravid pregnant women with mean age of 23.2 ± 4.3 were studied. Primary outcomes were total maternal and neonatal adverse outcome (TMNAO), total maternal adverse end result (TMAE), and total neonatal adverse outcome (TNAO), regardless of the type of outcomes. Preterm labor and delivery; premature rupture of membrane (PROM) and preterm premature rupture of membrane (PPROM); preeclampsia and eclampsia; placental abruption; chorioamnionitis; stillbirth; meconium passage; maternal death; postpartum hemorrhage; poor progression labor; abnormal vaginal bleeding in third trimester of pregnancy; low birth weight and neonatal need for CPR or intubation, neonatal anomaly, NICU admission, and neonatal mortality were also analyzed as subgroup outcomes.Results: One hundred twenty-seven (63.5%) participants showed a kind of total maternal and neonatal adverse outcome (TMNAO), 72 (36%) deliveries resulted in a kind of neonatal adverse outcome (TNAO), and 104 (52%) of participants had a kind of maternal adverse end result (TMAE). Iranian fathers showed a significantly higher rate of TMNAO than Afghan fathers did (82 versus 69%, odds ratio: 2.9, 95% CI 1.0-7.8, p: .01). Mother's nationality showed the same result (82 versus 64%, odds ratio: 2.6, 95% CI 0.9-6.8, p: .03). Iranian fathers showed a significantly higher rate of TMAE

than Afghan fathers did (79 versus 58%, odds ratio: 2.7, 95% CI 1.1-6.3, p: .01). Mother's nationality showed the same result (78 versus 60%, odds ratio: 2.4, 95% CI 1.0-5.6, p: 0.02). Neonates with Iranian fathers showed significantly more TNAO than those with Afghan fathers (50 versus 31%, odds ratio: 2.21, 95% CI 0.9-5.5, p: .04). The same trend was observed among Iranian mothers in comparison to Afghan mothers (50 versus 32%, odds ratio: 2.11, 95% CI 0.9-4.6, p: .06). Of mother's age, mother's BMI, father's age, father's BMI, and mother's nationality, only father's BMI contributed significantly to the binary logistic regression model (n = 116, R2: 9%, p: .028). It was found that for each decreased unit in BMI, the risk of TNAO was increased by 16%, p: .03. Moreover, Father's family history of preeclampsia resulted in a higher prevalence of total neonatal adverse outcome (TNAO) in comparison with lack of such family history (87 versus 43%, odds ratio: 8.9, 95% CI 1.1-74.5, p: .02). Besides, mothers' participation in prenatal care (PNC) visits, assessed by caregivers, was significantly more satisfactory in neonates without any adverse outcome than those with neonatal adverse outcomes (median (IQR) = 2 (1-2) versus 2 (2-3), p: .04). PROM, pre-eclampsia, NICU admission, neonatal intubation, low Apgar score minute 0, and low Apgar score minute 5 were significantly more prevalent in participants revealing positive father's family history of pre-eclampsia. Regarding psychosocial exposures, placental abruption was more prevalent in mothers with exposure to verbal aggression versus non-exposed ones (9 versus 2%, odds ratio: 4.0, 95% CI 0.9-24.6, p: .04). Moreover, a weak positive association between neonatal gestational age at birth and quality of mother's participation in PNC visits (r: +0.3, p: .01) as well as mother's satisfaction from father's commitment to PNC visits was found (r: +0.1, p: .03).Conclusion: Male partners may play a key role in pregnant women and fetus's heath.

Database: Medline

2. Paternal determinants in preeclampsia

Author(s): Galaviz-Hernandez C.; Sosa-Macias M.; Teran E.; Garcia-Ortiz J.E.; Lazalde-Ramos B.P.

Source: Frontiers in Physiology; 2019; vol. 10

Publication Date: 2019

Publication Type(s): Review

Available at Frontiers in physiology - from Europe PubMed Central - Open Access

Abstract: Preeclampsia is a condition associated with high rates of maternal-fetal morbidity and mortality. It usually occurs in 3-10% of nulliparous women and 18% of previously affected women. Different lines of evidence have demonstrated the role of the father in the onset of preeclampsia. The placenta is the cornerstone of preeclampsia and poses important paternal genetic determinants; in fact, the existence of a "paternal antigen" has been proposed. Nulliparity is a well-known risk factor. Change of partner to a woman without history of preeclampsia increases the risk; however, this change decreases in women with history of the condition. High interval between pregnancies, short sexual intercourse before pregnancy, and conception by intracytoplasmic sperm injection suggest a limited exposure to the so-called paternal antigen. A man who was born from a mother with preeclampsia also increases the risk to his partner. Not only maternal but also paternal obesity is a risk factor for preeclampsia. Fetal HLA-G variants from the father increased the immune incompatibility with the mother and are also significantly associated with preeclampsia in multigravida pregnancies. An analysis of a group of Swedish pregnant women showed that the risk for preeclampsia is attributable to paternal factors in 13% of cases, which could be related to genetic interactions with maternal genetic factors. This review aimed to evaluate the evidences of the father's contribution to the onset of preeclampsia and determine the importance of including them in future studies.

Database: EMBASE

3. Association of pre-eclampsia with SOD2 Ala16Val polymorphism among mother-father-infant triads.

Author(s): Luo, Zhong-Cheng; Julien, Pierre; Wei, Shu-Qin; Audibert, Francois; Fraser, William D; Maternal and Infant Research on Oxidative Stress (MIROS) study group

Source: International journal of gynaecology and obstetrics: the official organ of the International Federation of Gynaecology and Obstetrics; Aug 2018; vol. 142 (no. 2); p. 221-227

Publication Date: Aug 2018

Publication Type(s): Journal Article

PubMedID: 29745991

Abstract:OBJECTIVETo determine whether pre-eclampsia is associated with polymorphisms in superoxide dismutase (SOD) genes among mother-father-infant triads.METHODSWe did this follow-up cohort study at 17 urban hospitals in Canada between October 1, 2008, and September 30, 2010. We recruited Canadian participants who had participated in the International Trial of Antioxidant Supplementation for the Prevention of Pre-eclampsia. Saliva specimens were collected for DNA extraction. The SOD1 +35A/C (rs2234694) and SOD2 Ala16Val C/T (rs4880) single-nucleotide polymorphisms (SNPs) were genotyped.RESULTSDual presence of the SOD2 Ala16Val TT variant among mother-father pairs (n=657) was associated with an increased risk of pre-eclampsia when compared with the absence of the TT variant among the mother-father pairs (7/48 [14.6%] vs 11/339 [3.2%]; adjusted odds ratio 6.80, 95% confidence interval 2.32-19.95; P<0.001). By contrast, presence of a single T variant in mother-father pairs (16/270 [5.9%]) or mother-infant pairs (8/179 [4.5%]) was not associated with pre-eclampsia. The SOD1 +35A/CSNP was not associated with pre-eclampsia.CONCLUSIONThe SOD2 Ala16Val SNP might be involved in paternal influence on the maternal predisposition to pre-eclampsia. Genotyping of mother-father pairs could be a promising strategy to identify pre-eclampsia genes.

Database: Medline

4. Maternal and Paternal Height and the Risk of Preeclampsia.

Author(s): Lee, Yunsung; Magnus, Per

Source: Hypertension (Dallas, Tex. : 1979); Apr 2018; vol. 71 (no. 4); p. 666-670

Publication Date: Apr 2018

Publication Type(s): Research Support, Non-u.s. Gov't Research Support, N.i.h., Extramural Journal Article

PubMedID: 29463626

Abstract: The etiology of preeclampsia is unknown. Tall women have been found to have lower incidence of preeclampsia. This points to a possible biological causal effect but may be because of socioeconomic confounding. We used paternal height as an unexposed control to examine confounding. The MoBa (Norwegian Mother and Child Cohort Study) was used to extract data on parental heights, maternal prepregnancy weight, other background factors, and pregnancy outcomes for 99 968 singleton births. Multiple logistic regression was used to estimate odds ratios for preeclampsia according to parental height. The adjusted odds ratio for preeclampsia was 0.74 (95% CI, 0.66-0.82) for women >172 cm as compared with women 186 cm was 1.03 (95% CI, 0.93-1.15) compared with men <178 cm. The association between maternal height and preeclampsia is unlikely to be because of confounding by familial, socioeconomic factors or by fetal genes related to height. The observed association between maternal height and preeclampsia merits further investigation.

Database: Medline

5. Immunological Tolerance, Pregnancy, and Preeclampsia: The Roles of Semen Microbes and the Father.

Author(s): Kenny, Louise C; Kell, Douglas B

Source: Frontiers in medicine; 2017; vol. 4 ; p. 239

Publication Date: 2017

Publication Type(s): Journal Article

PubMedID: 29354635

Available at Frontiers in medicine - from Europe PubMed Central - Open Access

Abstract: Although it is widely considered, in many cases, to involve two separable stages (poor placentation followed by oxidative stress/inflammation), the precise originating causes of preeclampsia (PE) remain elusive. We have previously brought together some of the considerable evidence that a (dormant) microbial component is commonly a significant part of its etiology. However, apart from recognizing, consistent with this view, that the many inflammatory markers of PE are also increased in infection, we had little to say about immunity, whether innate or adaptive. In addition, we focused on the gut, oral and female urinary tract microbiomes as the main sources of the infection. We here marshall further evidence for an infectious component in PE, focusing on the immunological tolerance characteristic of pregnancy, and the well-established fact that increased exposure to the father's semen assists this immunological tolerance. As well as these benefits, however, semen is not sterile, microbial tolerance mechanisms may exist, and we also review the evidence that semen may be responsible for inoculating the developing conceptus (and maybe the placenta) with microbes, not all of which are benign. It is suggested that when they are not, this may be a significant cause of PE. A variety of epidemiological and other evidence is entirely consistent with this, not least correlations between semen infection, infertility and PE. Our view also leads to a series of other, testable predictions. Overall, we argue for a significant paternal role in the development of PE through microbial infection of the mother via insemination.

Database: Medline

6. Paternal contribution to the preeclampsia phenotype

Author(s): Tsunemi T.; Sado T.; Naruse K.; Kobayashi H.

Source: Clinical and Experimental Obstetrics and Gynecology; 2017; vol. 44 (no. 6); p. 914-922

Publication Date: 2017

Publication Type(s): Article

Abstract: Purpose of investigation: The aims of this study were to elucidate functional pathways of genes responsible for key events in trophoblast invasion and to compare differences in paternal and maternal fitness gene expression in preeclampsia (PE) placenta. Material(s) and Method(s): The authors combined data across seven studies published between 1995 and 2014. All genes downloaded from public web sites were analyzed using the metaprofiling and highlighted differentially expressed genes, the chromosomal location of the candidate genes, enriched pathways, and genomic conflicting situations that may play an important role in the trophoblast invasion. Result(s): The majority of differentially expressed genes and their downstream targets associated with trophoblast invasion were mediated through the activation of MMP signaling pathways. The paternal fitness genes that favor trophoblast invasion and fetal growth were reduced in the PE placenta. Half of the differentially expressed genes were located in close proximity to known imprinted genes. Several genes identified in PE were located in a cluster of imprinted genes on chromosomes 1p31, 9q34, and 11p15.4. Conclusion(s): PE may be recognized as a paternal/fetal imprinting disease.

Database: EMBASE

7. The paternal polymorphism rs5370 in the EDN1 gene decreases the risk of preeclampsia.

Author(s): Galaviz-Hernandez, Carlos; Arámbula-Meraz, Eliakym; Medina-Bastidas, Diana; Sosa-Macías, Martha; Lazalde-Ramos, Blanca P; Ortega-Chávez, Margarita; Hernandez-García, Lorena

Source: Pregnancy hypertension; Oct 2016; vol. 6 (no. 4); p. 327-332

Publication Date: Oct 2016

Publication Type(s): Journal Article

PubMedID: 27939477

Abstract:OBJECTIVETo evaluate whether the maternal, paternal or the combined maternal/paternal contribution of SNP rs5370 of the EDN1 gene is associated with preeclampsia and drove its expression in placenta.STUDY DESIGNThis case-control study included 61 preeclamptic patients and their partners and 49 healthy pregnant women and their partners. The population was sub-divided into three groups: women-only, men-only and combined (women/men). The analysis included genotyping of rs5370 in mothers and fathers and evaluating the expression profile of the EDN1 gene in placenta. Comparisons of categorical variables were performed using chi-square and/or Fisher's exact tests. The intergroup comparisons were analysed with the Mann-Whitney U test. The association between the polymorphism and the disease was evaluated through multivariate regression analysis. Spearman's correlation was performed to test the relationship between pre-gestational history and clinical features of the affected patients with EDN1 gene expression.RESULTSThe analysis of paternal risk factors associated with preeclampsia revealed no differences between groups. A negative association between SNP rs5370 and preeclampsia was found in men group (OR 0.42; CI 95% 0.18-0.94, p=0.034) but not in women or combined groups. The adjustment for paternal protective factors increased the observed negative association, and the opposite was observed in the presence of paternal risk factors. The expression of the EDN1 gene in the placenta was significantly higher in the group of cases and was not associated with the rs5370 polymorphism.CONCLUSIONThe paternal rs5370 polymorphism decreases the risk for preeclampsia and is not associated with placental expression of the EDN1 gene.

Database: Medline

8. Preeclampsia: What Does the Father Have to Do with It?

Author(s): Katsi, V; Felekos, I; Siristatidis, C; Kasioni, S; Drakontaidis, A; Farmakides, G; Makris, T; Aggeli, C; Nihoyannopoulos, P; Tousoulis, D; Kallikazaros, I

Source: Current hypertension reports; Aug 2015; vol. 17 (no. 8); p. 60

Publication Date: Aug 2015

Publication Type(s): Journal Article Review Systematic Review

PubMedID: 26088194

Available at Current hypertension reports - from SpringerLink - Medicine

Available at Current hypertension reports - from ProQuest (Health Research Premium) - NHS Version

Abstract: Preeclampsia (PE) is one of the leading causes of maternal and fetal morbidity and mortality, with incidence rates ranging between 2 and 5 % in the Western World. The exact causes of the disease remain largely unknown, because of the complex pathophysiologic mechanisms involved in the process. Genetic, environmental, and epigenetic parameters have been implicated by various authors as culprits for the pathogenesis of PE. Recent reports in the literature highlight the paternal role. Still, the exact extent and mechanism remain elusive. In this systematic review, we attempt to present data regarding the paternal role in a concise and comprehensive manner.

Database: Medline

9. Cardiovascular disease in a pregnant woman's partner as a risk factor for preeclampsia

Author(s): Parker C.E.; Doherty D.A.; Walters B.N.J.

Source: Pregnancy Hypertension; Jan 2015; vol. 5 (no. 1); p. 149

Publication Date: Jan 2015

Publication Type(s): Conference Abstract

Abstract: Objectives: Assessing risk of developing a hypertensive disorder of pregnancy (HDP) in nulliparous women is imprecise. Previous evidence suggests that self-reported family history of cardiovascular disease and risk (in particular risk in the woman's father) may improve identification of preeclampsia (Parker, ISOM 2012). The aim of this study was to evaluate maternal report of paternal family history of cardiovascular disease (CVD) or risk (CVR) as a risk factor for HDP including preeclampsia. Method(s): Women were recruited prospectively and reported on cardiovascular health in themselves, their partners and first degree relatives (n = 997). HDP diagnoses were assigned using SOMANZ (2008) criteria. Result(s): Preeclampsia was diagnosed in 12.6% of women, gestational hypertension in 6.2%. CVD/CVR was reported by 22.3% of mothers (1.7% CVD alone) and 9.3% of partners (1.7% CVD alone). Median age of the women was 27 years (range 16-45), median age of the partners was 30 years (range 26-34). All partners with CVD were under 45 years old (median 32, range 24-39). The median age of men with risk alone was 33 years (range 29-38). Partners' CVD increased risk of preeclampsia (5.6% vs. 1.7%; OR = 5.07, 95% CI 1.72-14.94, p = .003) adjusted for maternal age, BMI, smoking and maternal CVD/CVR. No increase in risk of gestational hypertension was evident. Partners' CVR did not appear to increase risk of HDP. Conclusion(s): History of CVD in the woman's partner may indicate elevated risk of preeclampsia however, the low frequency of CVD in partners at this young age impacts on the sample size and should be examined in future research.

Database: EMBASE

10. Preeclampsia: Paternal and fetal risk factors

Author(s): Dekker G.

Source: Journal of Perinatal Medicine; Jun 2013; vol. 41

Publication Date: Jun 2013

Publication Type(s): Conference Abstract

Available at Journal of Perinatal Medicine - from Unpaywall

Abstract: The etiology of preeclampsia is often considered to be purely maternal, i.e. maternal constitutional factors that impair maternal cardiovascular/endothelial mechanisms normally required to cope with the specific pregnancy demands, being primarily a generalised inflammatory response and a hyperdynamic circulation. Clinicians typically encounter preeclampsia as maternal disease with variable degrees of fetal involvement, often not recognizing that preeclampsia is actually a couple's disease with maternal and fetal manifestations. More and more the unique immunogenetic maternal-paternal relationship is appreciated, and as such also the specific 'genetic conflict' that is characteristic of haemochorial placentation. Over the past 15 years several studies have demonstrated the protective effect of lengthy sexual relationships and the effect of paternity change. Regarding intrinsic paternal risks, epidemiologic studies suggest that advanced paternal age is a risk factor with the risk for the mother to develop preeclampsia almost doubling if she has a partner aged > 45 yr. Also men with a familial history of early-onset cardiovascular disease and/or hypertension significantly (odds ratio > 3) increase the risk of preeclampsia, while obese men represent an independent risk factor for pregnancies resulting in small-for- gestational age infants. Paternal genetic risk factors include 'dangerous' paternal genes following the regular Mendelian laws, imprinted genes and genes affecting critical aspects of male reproductive physiology. Recent animal and human studies have demonstrated that the sex of the fetus can influence many aspects of the maternal physiology. In addition, large epidemiological studies and prospective cohort studies like SCOPE have demonstrated that male/female ratio differ greatly between several major pregnancy complications and in particular for preeclampsia by gestational age. The aim of this presentation is to provide an overview on how and to what extend paternal/relational and fetal factors play a role in the causation of preeclampsia.

Database: EMBASE

11. The paternal role in pre-eclampsia and giving birth to a small for gestational age infant; a population-based cohort study.

Author(s): Wikström, Anna-Karin; Gunnarsdóttir, Jóhanna; Cnattingius, Sven

Source: BMJ open; 2012; vol. 2 (no. 4)

Publication Date: 2012

Publication Type(s): Journal Article

PubMedID: 22936817

Abstract:OBJECTIVETo estimate the effect of partner change on risks of pre-eclampsia and giving birth to a small for gestational age infant.DESIGNProspective population study.SETTINGSweden.PARTICIPANTSWomen with their first and second successive singleton births in Sweden between 1990 and 2006 without pregestational diabetes and/or hypertension (n=446 459).OUTCOME MEASURESPreterm (<37 weeks) and term (≥37 weeks) pre-eclampsia, and giving birth to a small for gestational age (SGA) infant. Risks were adjusted for interpregnancy interval, maternal age, body mass index, height and smoking habits in second pregnancy, years of involuntary childlessness before second pregnancy, mother's country of birth, years of formal education and year of birth. Further, when we calculated risks of SGA we restricted the study population to women with non-pre-eclamptic pregnancies.RESULTSIn women who had a preterm pre-eclampsia in first pregnancy, partner change was associated with a strong protective effect for preterm pre-eclampsia recurrence (OR 0.24; 95% CI 0.07 to 0.88). Similarly, partner change was also associated with a protective effect of recurrence of SGA birth (OR 0.75; 95% CI 0.67 to 0.84). In contrast, among women without SGA in first birth, partner change was associated with an increased risk of SGA in second pregnancy. Risks of term pre-eclampsia were not affected by partner change.CONCLUSIONSThere is a paternal effect on risks of preterm pre-eclampsia and giving birth to an SGA infant.

Database: Medline

12. Association of 14 bp insertion/deletion polymorphism of the HLA-G gene in father with severe preeclampsia in Chinese.

Author(s): Zhang, Z; Li, Y; Zhang, L L; Jia, L T; Yang, X Q

Source: Tissue antigens; Aug 2012; vol. 80 (no. 2); p. 158-164

Publication Date: Aug 2012

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

PubMedID: 22708635

Abstract: Preeclampsia (PE), especially severe PE including early (before 34 weeks' gestation) and late (after 34 weeks' gestation) onset PE, is one of the leading causes of maternal and fetal mortality and morbidity. It is well known that abnormal human leukocyte antigen subtype G (HLA-G) expression may contribute to PE. In this study, we investigated allelic and genotypic frequencies of the 14 bp deletion/insert polymorphism in the 3(')-untranslated region (3(')-UTR) of the HLA-G gene in cases (120 pairs of mother-offspring, 82 couples, and 67 pairs of father-offspring with severe PE) and controls (158 pairs of mother-offspring, 87 couples, and 75 pairs of father-offspring with normal pregnancy). We found that the frequencies of the +14 bp/+14 bp HLA-G genotype of the offspring were significantly higher in the severe and early onset severe PE cases compared with controls, and the frequencies of the -14 bp/-14 bp HLA-G genotype of the offspring were significantly lower in the early onset severe PE cases compared with controls. The frequency of combined -14 bp/+14 bp mother/+14 bp/+14 bp offspring genotypes was significantly higher in the severe and early onset severe PE cases compared with controls, and the frequency of combined -14 bp/+14 bp mother/-14 bp/-14 bp offspring genotypes was significantly lower in the early onset severe PE cases compared with late onset severe PE cases. The frequency of combined -14 bp/-14 bp father/-14 bp/-14 bp offspring genotypes was significantly lower in the early onset severe PE cases compared with late onset severe PE cases and controls. In overview, the HLA-G 14 bp deletion/insert polymorphism is associated with severe PE in father-offspring, and its distribution is different between the early and late onset severe PE.

Database: Medline

13. Recurrence of placental dysfunction disorders across generations

Author(s): Wikstrom A.-K.; Cnattingius S.

Source: Acta Obstetricia et Gynecologica Scandinavica; Jun 2012; vol. 91 ; p. 29-30

Publication Date: Jun 2012

Publication Type(s): Conference Abstract

Available at Acta Obstetricia et Gynecologica Scandinavica - from Wiley Online Library

Abstract: Background: Knowledge about etiologies of placental dysfunctional disorders is limited. We performed an intergenerational study, focusing on risks of placental dysfunctional disorders in mothers and father born small-for-gestationalage (SGA). Method(s): Using linked generational data from the Swedish Medical Birth Register from 1973.2006we identified 321,383 mother-offspring units and 135,637 mother-father-offspring units. Result(s): Compared with mothers not born SGA, mothers born SGA had the following adjusted odds ratios (95% confidence intervals): late preeclampsia 1.41 (1.26.1.57), early preeclampsia 1.87 (1.38.2.35), placental abruption 1.60 (1.23.2.09), spontaneous pretermbirth 1.11 (1.00.1.23) and stillbirth 1.24 (0.84.1.82). Compared with parents not born SGA, the risk of preeclampsia was more than threefold increased if both parents were born SGA, whereas if only the mother was born SGA, corresponding risk was only increased by 50%. Conclusion(s): There is an intergenerational recurrence of placental dysfunctional disorders on thematernal side, andmost likely also on the paternal side.

Database: EMBASE

14. Hypertensive disorders in pregnancy and paternal cardiovascular risk: a population-based study.

Author(s): Myklestad, Kirsti; Vatten, Lars Johan; Salvesen, Kjell Åsmund; Davey Smith, George; Romundstad, Pål Richard

Source: Annals of epidemiology; Jun 2011; vol. 21 (no. 6); p. 407-412

Publication Date: Jun 2011

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

PubMedID: 21414802

Abstract:PURPOSEIt has been suggested that paternal genes may contribute to the risk of maternal hypertensive disorders in pregnancy and that genes associated with cardiovascular disease could be involved in the etiology of maternal hypertensive disorders in pregnancy. If these genes are of fetal origin, one would expect that paternal cardiovascular risk factors are associated with the fathering of pregnancies in which the mothers experience hypertensive disorders. Thus, we have studied 14,130 offspring and parents in Norway (1967-1997) to assess whether the fathering of pregnancies complicated by hypertensive disorders in the mother is associated with paternal cardiovascular risk factors.METHODSIn a population-based study of 14,130 family units, data on parental cardiovascular risk factors (blood pressure, body mass index, waist circumference, nonfasting serum lipids and glucose) collected in the Norwegian Hunt Study (1995-1997) were linked to pregnancy data from the Medical Birth Registry of Norway (1967-1997). Multiple linear regression methods were used, and all analyses were adjusted for lifestyle factors likely to be shared by the parents.RESULTSThere was no association between hypertensive disorders in pregnancy and paternal cardiovascular risk factors.CONCLUSIONSWe found no evidence that the fathering of pregnancies complicated by hypertensive disorders in the mother is associated with an unfavorable paternal cardiovascular risk profile.

Database: Medline

15. The etiology of preeclampsia: the role of the father.

Author(s): Dekker, Gus; Robillard, Pierre Yves; Roberts, Claire

Source: Journal of reproductive immunology; May 2011; vol. 89 (no. 2); p. 126-132

Publication Date: May 2011

Publication Type(s): Journal Article Review

PubMedID: 21529966

Abstract: Preeclampsia is often considered as simply a maternal disease with variable degrees of fetal involvement. More and more the unique immunogenetic maternal-paternal relationship is appreciated, and also the specific 'genetic conflict' that is characteristic of haemochorial placentation. From that perspective, pre-eclampsia can be seen as a disease of an individual couple with primarily maternal and fetal manifestations. The maternal and fetal genomes perform different roles during development. Heritable paternal, rather than maternal, imprinting of the genome is necessary for normal trophoblast development. Large population studies have estimated that 35% of the variance in susceptibility to preeclampsia is attributable to maternal genetic effects; 20% to fetal genetic effects (with similar contributions of both parents), 13% to the couple effect, less than 1% to the shared sibling environment and 32% to unmeasured factors. Not one of these large population studies focussed on the paternal contribution to preeclampsia, which is demonstrated by (1) the effect of the length of the sexual relationship; (2) the concept of primipaternity versus primigravidity; and (3) the existence of the so-called 'dangerous' father, as demonstrated in various large population studies. It is currently unknown how the father exerts this effect. Possible mechanisms include seminal cytokine levels and their effect on maternal immune deviation, specific paternal HLA characteristics and specific paternal single nucleotide polymorphisms (SNPs), in particular in the paternally expressed genes affecting placentation. Several large cohort studies, including the large international SCOPE consortium, have identified paternal SNPs with strong associations with preeclampsia.

Database: Medline

16. Variable effects of maternal and paternal-fetal contribution to the risk for preeclampsia combining GSTP1, eNOS, and LPL gene polymorphisms.

Author(s): Pappa, Kalliopi I; Roubelakis, Maria; Vlachos, George; Marinopoulos, Spyros; Zissou, Antonia; Anagnou, Nicholas P; Antsaklis, Aris

Source: The journal of maternal-fetal & neonatal medicine : the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstetricians; Apr 2011; vol. 24 (no. 4); p. 628-635

Publication Date: Apr 2011

Publication Type(s): Research Support, N.i.h., Intramural Journal Article

PubMedID: 20836743

Abstract:OBJECTIVETo evaluate the maternal, paternal, and fetal genotype contribution to preeclampsia. STUDY DESIGN, MATERIALS, AND METHODS: We combined the analysis of polymorphisms of the GSTP1, eNOS, and LPL genes - affecting biotransformation enzymes and endothelial function - in a cohort of 167 preeclamptic and normal control trios (mother, father, and child) comprising a total of 501 samples in the Greek population, never analyzed before by this approach.RESULTSFor the frequency of the GSTP1 Ile(105)/Val(105), the eNOS Glu298Asp and the LPL-93 polymorphisms, statistically significant differences were found between the two groups. However, the transmission rates of the parental alleles to neonates studied by the transmission disequilibrium test, disclosed no increased rate of transmission to preeclampsia children for the variant alleles of Val(105) GSTP1, 298Asp eNOS, and -93G LPL.CONCLUSIONSThese novel data, suggest that interaction of all three types of genotypes (mother, father and neonate), reveals no effects on the development of preeclampsia, but provide the impetus for further studies to decipher the individual contribution of each genetic parameter of preeclampsia.

Database: Medline

17. Pre-eclampsia: Definitions, paternal contributions and a four stage model.

Author(s): Redman, C W

Source: Pregnancy hypertension; Jan 2011; vol. 1 (no. 1); p. 2-5

Publication Date: Jan 2011

Publication Type(s): Journal Article

PubMedID: 26104227

Abstract: It is 40 years since I started researching pre-eclampsia. Much has changed but some old problems persist. These include the debate of how to define a syndrome, the inheritance and genetics of pre-eclampsia, why primiparae are so susceptible and is primipaternity important? If it is, in a multiparous pregnancy (after changing partners), the old hypothesis that pre-eclampsia is the outcome of failed maternal immunoregulation to accommodate nature's transplant - the fetus - must be confronted. These points are briefly reviewed and a four stage model of pre-eclampsia derived.

Database: Medline

18. Association of maternal, paternal and fetal single nucleotide polymorphisms in vascular endothelial growth factor family genes with preeclampsia

Author(s): Andraweera P.; Thompson S.; Zhang V.; Roberts C.; Dekker G.

Source: Pregnancy Hypertension; Oct 2010; vol. 1

Publication Date: Oct 2010

Publication Type(s): Conference Abstract

Abstract: Introduction: Preeclampsia (PE) is a systemic syndrome characterized by widespread maternal endothelial dysfunction. Altered expression of placental angiogenic factors are implicated in its pathophysiology. Vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) are angiogenic factors which act via Flt-1 (fms-like tyrosine kinase 1) and KDR (kinase insert domain receptor) receptors. Reduced maternal plasma VEGF and PlGF and elevated sFlt-1 are implicated in PE. We hypothesised that polymorphisms in VEGF (+2578C/A and +936 C/T), PlGF (+642C/A), KDR (+604T/C and +1192 C/T) and Flt-1 (+677C/T) genes associate with preeclampsia. Method(s): A prospective cohort study (SCOPE Study) was conducted where 1169 nulliparous pregnant women, their partners and babies were recruited in Adelaide. Preeclampsia was defined using international guidelines. Uncomplicated pregnancies served as controls. Peripheral blood from couples and cord blood from babies were collected. Caucasian pregnancies were selected to ensure ethnic homogeneity and all data relating to potential false paternity were excluded. DNA extraction and genotyping were performed using the Sequenom MassARRAY system. Genotypes for PE (n=84) were compared with controls (n=460) and analysed using Chi Squared test and logistic regression. Result(s): Neonatal VEGF+2578 CC genotype and paternal VEGF+2578 C allele were significantly increased in PE [p=0.02, OR (95%CI) = 2.08 (1.1-3.9) and (p=0.027, OR (95%CI) = 1.7 (1.1-2.7)]. Paternal PlGF+642 CA+AA genotypes were significantly increased in PE [p=0.04, OR (95% CI) = 1.5 (1.0-3.8)]. Paternal [p=0.04, OR (95%CI) = 1.5 (1.03-3.6)] and neonatal [p=0.03, OR (95%CI) = 2.09 (1.07-4.09)] KDR+604 CC genotype was significantly increased in PE. Neonatal KDR+1192 T allele was increased in PE [p=0.03, OR (95%CI) = 2.0 (1.0-3.7)]. The results remained significant after adjusting for potential confounding factors. Conclusion(s): Our results demonstrate that SNPs in the VEGF family genes confer an increased risk for the development of preeclampsia. The association of paternal SNPs with preeclampsia provides evidence for the partner's role in the pathogenesis of preeclampsia.

Database: EMBASE

19. Paternal factors involved in the causation of preeclampsia

Author(s): Dekker G.; Roberts C.; Furness D.; Andraweera P.

Source: Pregnancy Hypertension; Oct 2010; vol. 1

Publication Date: Oct 2010

Publication Type(s): Conference Abstract

Abstract: Preeclampsia is often considered as just a maternal disease with variable degrees of fetal involvement. More and more the unique immunogenetic maternal-paternal relationship is appreciated, and as such also the specific "genetic conflict" that is characteristic of haemochorial placentation. From that perspective, preeclampsia can also be seen as a disease of an individual couple with primarily maternal and fetal manifestations. The maternal and fetal genomes perform different roles during development. Heritable paternal, rather than maternal, imprinting of the genome is necessary for normal trophoblast development. Large population studies have estimated that 35% of the variance in susceptibility to preeclampsia was attributable to maternal genetic effects, 20% to fetal genetic effects (with similar contributions of both parents), 13% to the couple effect, less than 1% to shared sibling environment and 32% to unmeasured factors. Not one of these large population studies involved a real focus on the paternal contribution to preeclampsia which is demonstrated by: 1. The effect of the length of sexual relationship. 2. The concept of primipaternity versus primigravidity. 3. Existence of the so-called 'dangerous' father which has been demonstrated in various large population studies. It is currently unknown how the father exerts this effect. Possible mechanisms include seminal cytokine levels and their effect on maternal immune deviation, specific paternal HLA characteristics and specific paternal SNP's (in particular in the paternally expressed genes affecting placentation). Several large cohort studies, including the large international SCOPE consortium, have identified several paternal SNP's with strong associations with preeclampsia (Table presented).

Database: EMBASE

20. Preeclampsia and intra-uterine growth restriction: The role of the father

Author(s): Dekker G.A.; Roberts C.T.

Source: Journal of Reproductive Immunology; Aug 2010; vol. 86 (no. 1); p. 12

Publication Date: Aug 2010

Publication Type(s): Conference Abstract

Abstract: Preeclampsia is a disease of an individual couple with primarily maternal and fetal manifestations. Factors that are unique to a specific couple include the length and type of sexual relationship, the maternal acceptance of the invading cytotrophoblast and seminal fluid levels of transforming growth factor-beta and other cytokines. The maternal and fetal genomes perform different roles during development. Heritable paternal, rather than maternal, imprinting of the genome is necessary for normal trophoblast development. Preeclampsia may relate to extreme genetic conflict, or a mother unable to cope with a 'physiologic' genetic conflict. The paternal contribution to preeclampsia is demonstrated by four factors. (1) The effect of the length of sexual relationship. The SCOPE consortium has recently published the results of a large prospective study in nulliparous women providing further evidence that short duration of sexual relationship prior to conception is an independent risk factor for both preeclampsia and 'placental' small for gestational age. (2) The concept of primipaternity versus primigravidity. In 2002, the primipaternity concept was challenged by Skjaerven et al. According to these authors, prolonged birth interval and not paternity change was the explanation for the increased preeclampsia risk in multiparous women with new partners. More critical analysis of their data and the results of various subsequent studies have however clearly demonstrated that the primipaternity concept still stands. (3) Existence of the so-

called 'dangerous' father has been demonstrated in various large population studies. Men who have fathered one preeclamptic pregnancy are nearly twice as likely to father a preeclamptic pregnancy in a different woman. It is currently not certain how the father exerts this effect. (4) Paternal booking characteristics. Advanced paternal age (>45 years) has been demonstrated to almost double the risk of preeclampsia, while paternal obesity and central adiposity may be independent risk factors for infants who are small for gestational age by customized birth weight centiles.

Database: EMBASE

21. Paternal contribution of HLA-G*0106 significantly increases risk for pre-eclampsia in multigravid pregnancies.

Author(s): Tan, Chia Yee; Ho, Julia F V; Chong, Yap Seng; Loganath, Annamalai; Chan, Yiong Huak; Ravichandran, Jeganathan; Lee, Caroline G; Chong, Samuel S

Source: Molecular human reproduction; May 2008; vol. 14 (no. 5); p. 317-324

Publication Date: May 2008

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

PubMedID: 18353802

Available at Molecular human reproduction - from Oxford Journals - Medicine

Available at Molecular human reproduction - from HighWire - Free Full Text

Available at Molecular human reproduction - from Unpaywall

Abstract: Pre-eclampsia (PE) is a leading cause of maternal and fetal mortality and morbidity. Structural or functional alterations of human leukocyte antigen (HLA)-G present at the maternal-fetal interface may predispose women to PE. We tested the HLA-G gene for association with PE in a case-control study of 83 PE and 240 normotensive Malay women. HLA-G was amplified in a single-tube multiplex-PCR reaction and genotyped for 18 single nucleotide polymorphisms (SNPs) by multiplex-minisequencing. Case-control comparisons were performed, and associations with disease were expressed as odds ratios (ORs). Risk for PE was significantly associated with fetal allele G*0106 only in multigravid pregnancies (P = 0.002, OR = 5.0, 95% CI = 1.8-13.8). Among multigravid pregnancies, the frequency of PE babies heterozygous or homozygous for G*0106 was also significantly higher compared with normal control babies (P = 0.002, OR = 5.4, 95% CI = 1.9-15.4). Multivariate analyses with adjustment for factors associated with PE revealed similar results (P = 0.003, OR = 10.1, 95% CI = 2.2-46.8). Additionally, a significantly higher frequency of fetal-maternal G*0106 genotype mismatch was observed in PE compared with normal multigravid pregnancies (P = 0.001, OR = 9.6, 95% CI = 2.4-38.7). Thus, paternal HLA-G G*0106 contribution significantly increases risk for PE in multigravidas who do not carry this allele, potentially mediated by a gradual maternal alloimmune response to repeated exposure to the paternal HLA-G variant.

Database: Medline

22. Recurrence of pre-eclampsia across generations: exploring fetal and maternal genetic components in a population based cohort.

Author(s): Skjaerven, Rolv; Vatten, Lars J; Wilcox, Allen J; Rønning, Thorbjørn; Irgens, Lorentz M; Lie, Rolv Terje

Source: BMJ (Clinical research ed.); Oct 2005; vol. 331 (no. 7521); p. 877

Publication Date: Oct 2005

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

PubMedID: 16169871

Available at BMJ (Clinical research ed.) - from BMJ Journals - NHS

Available at BMJ (Clinical research ed.) - from ProQuest (Health Research Premium) - NHS Version

Abstract:OBJECTIVESTo assess the impact on risk of pre-eclampsia of genes that work through the mother, and genes of paternal origin that work through the fetus.DESIGNPopulation based cohort study.SETTINGRegistry data from Norway.PARTICIPANTSLinked generational data from the medical birth registry of Norway (1967-2003): 438,597 mother-offspring units and 286,945 father-offspring units.MAIN OUTCOME MEASURESPre-eclampsia in the second generation.RESULTSThe daughters of women who had pre-eclampsia during pregnancy had more than twice the risk of pre-eclampsia themselves (odds ratio 2.2, 95% confidence interval 2.0 to 2.4) compared with other women. Men born after a pregnancy complicated by pre-eclampsia had a moderately increased risk of fathering a pre-eclamptic pregnancy (1.5, 1.3 to 1.7). Sisters of affected men or women, who were themselves born after pregnancies not complicated by pre-eclampsia, also had an increased risk (2.0, 1.7 to 2.3). Women and men born after pre-eclamptic pregnancies were more likely to trigger severe pre-eclampsia in their own (or their partner's) pregnancy (3.0, 2.4 to 3.7, for mothers and 1.9, 1.4 to 2.5, for fathers).CONCLUSIONSMaternal genes and fetal genes from either the mother or father may trigger pre-eclampsia. The maternal association is stronger than the fetal association. The familial association predicts more severe pre-eclampsia.

Database: Medline

23. Maternal ethnicity, paternal ethnicity, and parental ethnic discordance: predictors of preeclampsia.

Author(s): Caughey, Aaron B; Stotland, Naomi E; Washington, A Eugene; Escobar, Gabriel J

Source: Obstetrics and gynecology; Jul 2005; vol. 106 (no. 1); p. 156-161

Publication Date: Jul 2005

Publication Type(s): Research Support, N.i.h., Extramural Journal Article Research Support, U.s. Gov't, P.h.s.

PubMedID: 15994632

Available at Obstetrics and gynecology - from Ovid (LWW Total Access Collection 2019 - with Neurology) .

Abstract:OBJECTIVETo examine the association of maternal and paternal ethnicity as well as parental ethnic discordance with preeclampsia.METHODSRetrospective cohort study of all low-risk women delivered from 1995 to 1999 within a mature managed care organization. Rates of preeclampsia were calculated for maternal, paternal, and combined ethnicity using both univariate and multivariate analyses.RESULTSAmong the 127,544 low-risk women, when examining maternal ethnicity in a multivariate model controlling for maternal age, parity, education, and gestational age, we found that the rates of preeclampsia were higher among African American (5.2%; odds ratio [OR] 1.41, 95% confidence interval [CI] 1.25-1.62) women and lower among Latina (4.0%; OR 0.90, 95% CI 0.84-0.97) and Asian women (3.5%; OR 0.79, 95% CI 0.72-0.88), with all results being statistically

significant as compared with white women. When paternal ethnicity was controlled for separately, however, the difference in the rate of preeclampsia among Asian women disappeared, the effect of African-American maternal ethnicity increased slightly (OR 1.49, 95% CI 1.33-1.72), and Asian paternity was found to be associated with the lowest rate of preeclampsia (3.2%; OR 0.76, 95% CI 0.68-0.85). Further, parental ethnic discordance was associated with an increase in the rate of preeclampsia (OR 1.13, 95% CI 1.02 - 1.26).CONCLUSIONWe found that rates of preeclampsia were lower with Asian paternal ethnicity. We also found that having a differing paternal and maternal ethnicity was associated with increased rates of preeclampsia. For every 1,000 pregnancies, there would be approximately 10 fewer cases of preeclampsia in the setting of Asian paternity and 5 more cases of preeclampsia in the setting of parental ethnic discordance. These differences may be useful in further investigation of the cause of preeclampsia.LEVEL OF EVIDENCEII-2.

Database: Medline

24. Maternal and paternal thrombophilia: Risk factors for perinatal mortality

Author(s): De Galan-Roosen A.E.M.; Kuijpers J.C.; Rosendaal F.R.; Steegers E.A.; Van Beers W.A.; Ponjee G.A.; Merkus H.M.

Source: BJOG: An International Journal of Obstetrics and Gynaecology; Mar 2005; vol. 112 (no. 3); p. 306-311

Publication Date: Mar 2005

Publication Type(s): Article

PubMedID: 15713144

Available at BJOG : an international journal of obstetrics and gynaecology - from Wiley Online Library

Available at BJOG : an international journal of obstetrics and gynaecology - from Unpaywall

Abstract: Background: Although some paternal components to the predisposition to pre-eclampsia have been demonstrated recently, it is not known whether such paternal factors play a role to thrombophilia-related perinatal mortality. Objective(s): To compare the paternal and maternal contribution to perinatal mortality. Study design: Data from a prospective registry of perinatal mortality in a Dutch healthcare region were used. Between December 1999 and May 2000, the prevalence of thrombophilia was studied in 74 women with a history of perinatal mortality (female cases) and 54 of their male partners (male cases). Seventy-one healthy unrelated women after uneventful pregnancies only and 66 of their male partners were used as controls. Setting(s): Obstetric outpatient clinic in a regional hospital (Remierde Graaf Group, Defit). Method(s): Presence of various coagulation abnormalities, hyperhomocysteinaemia and anticardiolipins was investigated. Result(s): The frequency of antithrombin deficiency (12% vs 0%), increased activated protein C (APC) resistance (32% vs 6%), total protein S deficiency (11% vs 1%) and elevated factor VIII:C activity (43% vs 17%) was significantly higher in female cases compared with controls. In male cases, the frequency of increased APC resistance was significantly higher compared with controls (22% vs 0%). In 30 of the 54 couples with a history of perinatal mortality, more than one thrombophilic abnormality was found (55%) compared with 10 of the 62 control couples (17%). Conclusion(s): The risk of having thrombophilia is doubled in men who have fathered pregnancies which ended in perinatal death as well as in the mothers of such pregnancies. © RCOG 2004.

Database: EMBASE

25. Paternal age and preeclampsia.

Author(s): Harlap, Susan; Paltiel, Ora; Deutsch, Lisa; Knaanie, Ariella; Masalha, Sausan; Tiram, Efrat; Caplan, Lee S; Malaspina, Dolores; Friedlander, Yechiel

Source: Epidemiology (Cambridge, Mass.); Nov 2002; vol. 13 (no. 6); p. 660-667

Publication Date: Nov 2002

Publication Type(s): Journal Article Research Support, U.s. Gov't, P.h.s.

PubMedID: 12410007

Available at Epidemiology (Cambridge, Mass.) - from Ovid (Journals @ Ovid) - London Health Libraries

Abstract:BACKGROUNDPaternal aging is associated with premeiotic damage to spermatogonia, a mechanism by which new point mutations are introduced into the gene pool. We hypothesized that paternal age might contribute to preeclampsia.METHODSWe studied the incidence of preeclampsia in 81,213 deliveries surveyed in 1964-1976 in the Jerusalem Perinatal Study. We controlled for maternal age, parity and other risk factors using logistic regression.RESULTSPreeclampsia was reported in 1303 deliveries (1.6%). Compared with fathers age 25-34 years, the odds ratios (ORs) for preeclampsia were 1.24 (95% confidence interval = 1.05-1.46) for age 35-44 and 1.80 (1.40-2.31) for age 45+. For fathers age <25, the OR was 1.25 (1.04-1.51). Although weaker than maternal age effects, paternal effects were consistent within subgroups of other variables.CONCLUSIONSThese findings support the hypothesis that a modest proportion of preeclampsia might be explained by new mutations acquired from fathers and add to a growing body of evidence for paternal age effects in birth defects, neuropsychiatric disease and neoplasia.

Database: Medline

26. Changing paternity and time since last pregnancy; the impact on pre-eclampsia risk. A study of 547 238 women with and without previous pre-eclampsia

Author(s): Trogstad L.I.S.; Eskild A.; Magnus P.; Samuelsen S.O.; Nesheim B.-I.

Source: International Journal of Epidemiology; 2001; vol. 30 (no. 6); p. 1317-1322

Publication Date: 2001

Publication Type(s): Article

PubMedID: 11821338

Available at International journal of epidemiology - from Oxford Journals - Medicine

Available at International journal of epidemiology - from HighWire - Free Full Text

Available at International journal of epidemiology - from Unpaywall

Abstract: Background. Long time interval between pregnancies has been found to increase the risk of pre-eclampsia in second pregnancy. Our aim was to investigate whether this effect is influenced by a history of pre-eclampsia or a change in paternity. Methods. We studied 547 238 women with a first and second pregnancy registered in the Medical Birth Registry of Norway, 1967-1998. The relative risk of pre-eclampsia in the second delivery according to time interval between deliveries was estimated as odds ratios (OR) in logistic regression models, controlling for changing paternity, maternal age and calendar time period in women with and without previous pre-eclampsia. Results. A change of paternity for the second pregnancy was associated with a reduced risk of pre-eclampsia after controlling for the time since first delivery (adjusted OR = 0.80, 95% CI : 0.72-0.90), but the interaction between change in paternity and time between deliveries was significant only for women with no previous pre-eclampsia. The interaction between history of pre-eclampsia and time interval between the two deliveries was highly significant, and for women with no previous pre-eclampsia

the risk of pre-eclampsia in second pregnancy increased with increasing time interval (for intervals longer than 15 years the adjusted OR was 2.11, 95% CI : 1.75-2.53). For women with previous pre-eclampsia the risk tended to decrease with increasing time interval between deliveries. Conclusions. The protective impact of a new father for the second pregnancy challenges the hypothesis of primipaternity, and implies that the increase in pre-eclampsia risk ascribed to new father by others is due to insufficient control for interpregnancy interval.

Database: EMBASE

27. Paternal and maternal components of the predisposition to preeclampsia.

Author(s): Esplin, M S; Fausett, M B; Fraser, A; Kerber, R; Mineau, G; Carrillo, J; Varner, M W

Source: The New England journal of medicine; Mar 2001; vol. 344 (no. 12); p. 867-872

Publication Date: Mar 2001

Publication Type(s): Research Support, Non-u.s. Gov't Journal Article

PubMedID: 11259719

Available at The New England journal of medicine - from Massachusetts Medical Society

Available at The New England journal of medicine - from ProQuest (Health Research Premium) - NHS Version

Available at The New England journal of medicine - from Patricia Bowen Library & Knowledge Service West Middlesex University Hospital NHS Trust (lib302631) Local Print Collection [location] : Patricia Bowen Library and Knowledge Service West Middlesex university Hospital.

Abstract:BACKGROUNDThere is an inherited maternal predisposition to preeclampsia. Whether there is a paternal component, however, is not known.METHODSWe used records of the Utah Population Database to identify 298 men and 237 women born in Utah between 1947 and 1957 whose mothers had had preeclampsia during their pregnancy. For each man and woman in the study group, we identified two matched, unrelated control subjects who were not the products of pregnancies complicated by preeclampsia. We then identified 947 children of the 298 male study subjects and 830 children of the 237 female study subjects who had been born between 1970 and 1992. These children were matched to offspring of the control subjects (1950 offspring of the male control group and 1658 offspring of the female control group). Factors associated with preeclampsia were identified, and odds ratios were calculated with the use of stepwise logistic-regression analysis.RESULTSIn the group whose mothers had had preeclampsia (the male study group), 2.7 percent of the offspring (26 of 947) were born of pregnancies complicated by preeclampsia, as compared with 1.3 percent of the offspring (26 of 1973) in the male control group. In the female study group, 4.7 percent of the pregnancies (39 of 830) were complicated by preeclampsia, as compared with 1.9 percent (32 of 1658) in the female control group. After adjustment for the offspring's year of birth, maternal parity, and the offspring's gestational age at delivery, the odds ratio for an adult whose mother had had preeclampsia having a child who was the product of a pregnancy complicated by preeclampsia was 2.1 (95 percent confidence interval, 1.0 to 4.3; P=0.04) in the male study group and 3.3 (95 percent confidence interval, 1.5 to 7.5; P=0.004) in the female study group.CONCLUSIONSBoth men and women who were the product of a pregnancy complicated by preeclampsia were significantly more likely than control men and women to have a child who was the product of a pregnancy complicated by preeclampsia.

Database: Medline

28. Father's genes influence risk of pre-eclampsia

Author(s):

Source: BMJ (Clinical research ed.); May 1998; vol. 316 (no. 7141); p. B

Publication Date: May 1998

Publication Type(s): Journal Article

PubMedID: 9564023

Available at BMJ (Clinical research ed.) - from BMJ Journals - NHS

Available at BMJ (Clinical research ed.) - from ProQuest (Health Research Premium) - NHS Version

Available at BMJ (Clinical research ed.) - from PubMed

Database: Medline

29. Pre-eclampsia/eclampsia: A fatal father factor

Author(s): Astin M.; Scott J.R.; Worley R.J.

Source: Lancet; 1981; vol. 2 (no. 8245); p. 533

Publication Date: 1981

Publication Type(s): Letter

PubMedID: 6115286

Available at Lancet (London, England) - from Patricia Bowen Library & Knowledge Service West Middlesex University Hospital NHS Trust (lib302631) Local Print Collection [location] : Patricia Bowen Library and Knowledge Service West Middlesex university Hospital.

Abstract: The chance that, as in these cases, a man might marry and lose two consecutive wives to severe forms of pre-eclampsia/eclampsia is remote. There is no increase in the incidence of eclampsia in daughters-in-law of eclamptic mothers, suggesting that there are no mother-to-son inheritance patterns, but there are no published reports of father-to-son inheritance patterns. Pre-eclampsia can occur in a woman who has had a normal pregnancy if she becomes pregnant by another mate, raising the possibility that the disease is the consequence of a pregnancy by the new husband rather than of specific underlying immunological abnormalities in the mother. Reduced fetomaternal incompatibility and immune response to fetoplacental antigens may contribute to the pathogenesis of pre eclampsia. Unfortunately, the circumstances of our cases provided us with only retrospective information. It was impossible to investigate whether HLA antigens were shared by this husband and his two wives. However, the existence of a genetically transmissible 'father factor' (which in these instances proved to be fatal) in cases of pre-eclampsia remains an interesting possibility.

Database: EMBASE

Strategy 793021

#	Database	Search term	Results
1	Medline	(paternal OR father*).ti	11288
2	Medline	exp FATHERS/	8709
3	Medline	(1 OR 2)	15975
4	Medline	(preeclamp* OR "pre eclamp*").ti,ab	30582
5	Medline	exp "PRE-ECLAMPSIA"/	29970
6	Medline	(4 OR 5)	41854
7	Medline	(3 AND 6)	62
8	EMBASE	(paternal OR father*).ti	12125
9	EMBASE	exp FATHERS/	26981
10	EMBASE	(8 OR 9)	32896
11	EMBASE	(preeclamp* OR "pre eclamp*").ti,ab	46424
12	EMBASE	exp "PRE-ECLAMPSIA"/	52937
13	EMBASE	(11 OR 12)	0
14	EMBASE	(10 AND 13)	183