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Roles of steroid hormones during post-partum depression

M Noveka*

Department of Psychology, University of Social Sciences and Humanities, Warsaw, Poland

*Corresponding author: E-mail: noveka123@gmail.com

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DESCRIPTION

Both pregnancy and childbirth are experiences associated with major hormonal changes which affect the physical and mental state of the mother by resulting in some mothers suffering from postpartum disorders such as Post-Partum Depression (PPD) may develop. Piglet crushing has been suggested to resemble postpartum psychosis crushing are associated with emotional problems. This may have long term implications for maternal behavior and future piglet development.

A theoretical perspective from evolutionary science offers an explanation for PPD as a psychological adaptation or as a by-product of modern civilization. Maternal infanticide is a counterintuitive and counter-evolutionary behavior with serious welfare and economic consequences. Predictors of her PPD in humans include poor infant and maternal health and lack of social support. These days, the current high rates of PPD in humans are a by-product of major maternal changes in which PPD may be a lifestyle disease. Some of these reflect the challenges faced by sows in commercial pig production. Examining parallels in experience with respect to changes or limitations of provides an opportunity to use human experience to study sow piglet grinding and possibly improve our understanding of both issues. Based on what we can learn from the sow, we extend our understanding of her PPD in humans.

Large social networks are good at protecting people from PPD. Both pregnancy and childbirth can be difficult and harrowing experiences. Members of animal kingdom which evolve social systems have the ability to learn socially transmitted information and the ability to communicate among all group members who can experience different emotions and feelings. Psychiatric conditions including depression. Social support from the partner in the first weeks after the birth and emotional intimacy with other mothers is important in predicting the development of PPD in mothers at the 8 weeks of age.

Steroid hormones play an important role in the development of PPD. During human pregnancy estriol increases 1000 fold, estradiol 50 fold, progesterone 10 fold, and prolactin 7 fold. There is a possibility in humans these rapid rises in hormone levels return to pre pregnancy levels within one to two weeks of life and abrupt withdrawal of this pregnancy hormone has been observed to stimulate depressive symptoms. Sudden withdrawal of estrogen, estrogen fluctuations, and persistent estrogen deficiency has been associated with mood disorders. It was associated with an increase, and a decrease in Brain Derived Neutrophil Factor (BDNF) levels. Recently, estrogen signaling sensitivity has been proposed as a biomarker for PPD indicating that hormone concentration may be less important than maternal sensitivity and ability to utilize available hormones.

Progesterone has anxiolytic and anesthetic properties and is thought to protect against depression by modulating serotonergic receptors. In a double-blind pregnancy simulation study, human women were given synthetic estradiol and progesterone and then discontinued. This induced symptoms of depression in women with a history of PPD but not in women without a history of PPD. Statistical differences in hormone levels between the two groups of women was not observed this study highlights that women with a history of PPD may be more sensitive to the mood destabilizing effects changes in gonadal steroids.

Low oxytocin levels during pregnancy or after childbirth may be a risk factor for PPD. Human women with low oxytocin levels during pregnancy and at 2 and 8 weeks postpartum had more symptoms of postpartum psychosis.

CONCLUSION

Postpartum depression is associated with abnormal HPA function and cognitive impairment, making women with PPD more likely to abuse and infantilize their children.

However, depression has many facets and can be caused by a combination of neurotransmitter disorders, hormonal imbalances, genetics, and psychosocial factors exhibit abnormal HPA axis function, including a flattened cortisol rhythm, prolonged recovery time from stress, decreased morning cortisol levels, and increased evening cortisol levels. Furthermore, depressed patients not only have aberrant responses to ACTH and CRH challenge, but also have atypical responses to dexamethasone suppression testing.