



Obstetric Complications and Aggression

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Topic

Aggression

Introduction

Obstetric complications refer to disruptions and disorders of pregnancy, labour and delivery, and the early neonatal period. Examples of such complications include prenatal drug exposure, poor maternal nutrition, minor physical anomalies (or MPAs: indicators of fetal neural maldevelopment, occurring near the end of the first trimester), and birth complications. Obstetric complications can have long-term effects on a child, including an increase in problematic behaviour. Research has identified links between obstetric complications and subsequent human aggression, and suggests that obstetric complications may elicit aggression by affecting brain development.

Recent Research Findings

Prenatal Substance Exposure

It is well documented that prenatal exposure to alcohol and other drugs has long-term effects on children. In addition to cognitive deficits,¹ fetal alcohol exposure is associated with social deficits such as disrupted attachment and emotion dysregulation in infancy;¹ increased anger, aggression, and distractibility in early childhood;² and inappropriate sexual behaviour, legal problems, depression, suicide, and poor caretaking of subsequent children in adulthood.¹ Similar deficits have been observed in children exposed to methadone³ and cocaine.⁴ Although it was initially unclear whether these effects occurred independent of prenatal alcohol exposure and other risk factors,⁴ preliminary findings from a well-designed study indicate that prenatal cocaine exposure independently relates to greater externalizing behaviour problems (relative to internalizing problems) in 6-year-old children.⁵ Children prenatally exposed to cigarette smoke are also at increased risk for conduct problems and criminal behaviour,⁶⁻⁹ and some research suggests that this risk is specific to aggression.¹⁰⁻¹²

Animal experiments indicate that prenatal substance exposure relates to aggression by interfering with the development of neurons (ie, cell bodies) and/or the functioning of different neurotransmitters (ie, chemical messengers in the brain that regulate behavioural, cognitive, and physiological functions). However, because prenatal drug exposure results in many cognitive and behavioural deficits, whether increased aggression is specifically observed in exposed children likely depends on the presence of

other social and biological risk factors. Such factors include delivery complications,¹⁰ adolescent motherhood, a single-parent family environment, unwanted pregnancy, and/or developmental motor lags.¹¹

Maternal Nutrition Deficiencies

Although cross-sectional research has been inconsistent, a unique and important study illustrates the potential causal role of malnutrition during pregnancy as a risk factor for antisocial behaviour. Near the end of World War II, Germany imposed a food blockade on Holland. The male offspring of women who *had* and *had not* been exposed to severe malnutrition while pregnant were assessed for antisocial personality disorder in adulthood. The adult offspring born to women who *had* suffered significant nutritional deficits during the first and/or second trimesters had 2.5 times the rates of antisocial personality disorder compared to the controls (who *had not* suffered any nutritional deficits).¹³

Maternal zinc deficiency, in particular, has also been linked to impaired DNA, RNA, and protein synthesis in fetal brain development, and to congenital brain abnormalities.^{14,15} Although the exact mechanisms through which zinc deficiency may relate to aggressive human behaviour is unknown, extensive animal research has shown that rats fed a diet marginal in zinc (or protein) during their pregnancy and lactation give birth to offspring with impaired brain development.^{16,17} Interestingly, the amygdala — which regulates certain human emotion processes and which functions abnormally in violent offenders^{18,19} — is densely innervated by zinc-containing neurons.²⁰

Minor Physical Anomalies

MPAs such as low-seated ears, adherent ear lobes, or a furrowed tongue are associated with heightened antisocial and aggressive behaviour in preschool and elementary school boys²¹⁻²³ and 17-year-old males.²⁴ MPAs appear most likely to elicit antisocial behaviour when another negative psychosocial factor is present (eg, family adversity, unstable home^{25,26}). However, there is some suggestion that MPAs are a general risk factor for disruptive behaviour rather than a factor specific to antisocial, aggressive behaviour.²⁷

Birth Complications

Several studies have shown that a combination of birth complications and psychosocial deficits, such as early maternal rejection,²⁸ a disadvantaged family environment,²⁹ or poor parenting³⁰ significantly increases the risk for serious criminal or violent behaviour in adulthood. Although a connection between birth complications and brain abnormalities has not been directly tested in aggressive individuals, fetal hypoxia (ie, a lack of oxygen) is associated with decreased cortical grey matter in schizophrenia patients.³¹ Thus, birth complications such as anoxia/hypoxia, pre-eclampsia (hypertension leading to anoxia), and forceps delivery may be one source of brain dysfunction observed in antisocial groups as well. When combined with a risk-filled environment that fails to foster socialization of the child through appropriate parenting practices, a child's predisposition to engaging in aggression may be substantially increased.

Conclusions

Overall, research supports the notion that obstetric complications such as prenatal alcohol, drug, and cigarette exposure, poor maternal nutrition, birth complications, and MPAs contribute to the development of aggression at different points in a lifespan. Thus, improved pre- and perinatal health care that reduces such complications may also help to reduce the development of aggressive behaviour problems. For children who nevertheless experience such complications, the amelioration of co-existing psychosocial stressors may reduce the risk of aggression, as it appears that obstetric complications increase the risk for later violence only when stressors such as maternal rejection, poor parenting practices, and a disadvantaged family environment are also present.

One should also note that the impact of these different obstetric complications on later behaviour is variable. Findings for prenatal alcohol exposure are far more extensive to date than those for maternal nutrition, for example. In addition, many obstetric complications (eg, fetal alcohol syndrome, prenatal cocaine exposure, and MPAs) are associated with other cognitive and behavioural problems such as mental retardation, significant attention deficits and schizophrenia. Thus, these risk factors should not be considered specific to aggression.

Future Research

Despite general research observations that obstetric complications relate to later aggression in affected children, many questions remain unanswered. First, animal experiments suggest that prenatal substance exposure, maternal malnutrition, and birth complications affect brain development, and that this brain dysfunction, in turn, results in aggressive behaviour. Therefore, promising directions for future research with humans include determining,

- 1) whether obstetric complications do result in identifiable brain abnormalities
- 2) whether brain abnormalities resulting from obstetric complications underlie persistent or severe forms of subsequent aggressive behaviour;
- 3) whether obstetric complications that are associated with aggressive and non-aggressive behaviour problems are each associated with different patterns of brain abnormalities.

In addition, research in all the different areas of obstetric complications has found that an obstetric complication (eg, prenatal cigarette exposure and MPAs) frequently requires an additional stressor to be present before an increase in aggression is observed. Thus, future studies may wish to seek a more complete understanding of how obstetric complications and the quality of the rearing environment interact to bring about an increased risk for aggression. Greater specificity in understanding these biological and social processes may serve to better inform policymaking and medical care standards in a way that ultimately helps reduce aggressive behaviour.

To learn more on this topic, consult the following sections of the Encyclopedia:

- [How important is it?](#)
- [What do we know?](#)
- [What can be done?](#)
- [According to experts](#)
- [Key messages](#)

REFERENCES

1. Kelly SJ, Day N, Streissguth AP. Effects of prenatal alcohol exposure on social behavior in humans and other species. *Neurotoxicology and Teratology* 2000;22(2):143-149.
2. Cohen S, Erwin EJ. Characteristics of children with prenatal drug exposure being served in preschool special education programs in New York City. *Topics in Early Childhood Special Education* 1994;14(2):232-253.
3. de Cubas MM, Field T. Children of methadone-dependent women: developmental outcomes. *American Journal of Orthopsychiatry* 1993;63(2):266-276.
4. Neuspil DR. The problem of confounding in research on prenatal cocaine effects on behavior and development. In: Lewis M, Bendersky M, eds. *Mothers, Babies, and Cocaine: the Role of Toxins in Development* Hillsdale, NJ: Lawrence Erlbaum Associates; 1995:95-110.
5. Delaney-Black V, Covington C, Templin T, Ager J, Nordstrom-Klee B, Martier S, Leddick L, Czerwinski RH, Sokol RJ. Teacher-assessed behavior of children prenatally exposed to cocaine. *Pediatrics* 2000;106(4):782-791.
6. Weissman MM, Warner V, Wickramaratne PJ, Kandel DB. Maternal smoking during pregnancy and psychopathology in offspring followed to adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry* 1999;38(7):892-899.
7. Wakschlag LS, Lahey BB, Loeber R, Green SM, Gordon RA, Leventhal BL. Maternal smoking during pregnancy and the risk of conduct disorder in boys. *Archives of General Psychiatry* 1997;54(7):670-676.
8. Fergusson DM, Woodward LJ, Horwood J. Maternal smoking during pregnancy and psychiatric adjustment in late adolescence. *Archives of General Psychiatry* 1998;55(8):721-727.
9. Rantakallio P, Läärä E, Isohanni M, Moilanen I. Maternal smoking during pregnancy and delinquency of the offspring: an association without causation? *International Journal of Epidemiology* 1992;21(6):1106-1113.
10. Brennan PA, Grekin ER, Mednick SA. Maternal smoking during pregnancy and adult male criminal outcomes. *Archives of General Psychiatry* 1999;56(3):215-219.
11. Räsänen P, Hakko H, Isohanni M, Hodgins S, Järvelin MR, Tiihonen J. Maternal smoking during pregnancy and risk of criminal behavior among adult male

- offspring in the Northern Finland 1966 birth cohort. *American Journal of Psychiatry* 1999;156(6):857-862.
12. Orlebeke JF, Knol DL, Verhulst FC. Child behavior problems increased by maternal smoking during pregnancy. *Archives of Environmental Health* 1999;54(1):15-19.
 13. Neugebauer R, Hoek HW, Susser E. Prenatal exposure to wartime famine and development of antisocial personality disorder in early adulthood. *JAMA-Journal of the American Medical Association* 1999;282(5):455-462.
 14. Pfeiffer CC, Braverman ER. Zinc, the brain, and behavior. *Biological Psychiatry* 1982;17(4):513-532.
 15. King JC. Determinants of maternal zinc status during pregnancy. *American Journal of Clinical Nutrition* 2000;71(5 suppl):1334S-1343S.
 16. Oteiza PI, Hurley LS, Lonnerdal B, Keen CL. Effects of marginal zinc deficiency on microtubule polymerization in the developing rat brain. *Biological Trace Element Research* 1990;24(1):13-23.
 17. Bennis-Taleb N, Remacle C, Hoet JJ, Reusens B. A low-protein isocaloric diet during gestation affects brain development and alters permanently cerebral cortex blood vessels in rat offspring. *Journal of Nutrition* 1999;129(8):1613-1619.
 18. Raine A, Buchsbaum M, LaCasse L. Brain abnormalities in murderers indicated by positron emission tomography. *Biological Psychiatry* 1997;42(6):495-508.
 19. Davidson RJ, Putnam KM, Larson CL. Dysfunction in the neural circuitry of emotion regulation: a possible prelude to violence. *Science* 2000;289(5479):591-594.
 20. Christensen MK, Frederickson CJ. Zinc-containing afferent projections to the rat corticomedial amygdaloid complex: a retrograde tracing study. *Journal of Comparative Neurology* 1998;400(3):375-390.
 21. Paulhus DL, Martin CL. Predicting adult temperament from minor physical anomalies. *Journal of Personality and Social Psychology* 1986;50(6):1235-1239.
 22. Waldrop MF, Bell RQ, McLaughlin B, Halverson CF Jr. Newborn minor physical anomalies predict short attention span, peer aggression, and impulsivity at age 3. *Science* 1978;199(3):563-564.
 23. Halverson CF Jr, Victor JB. Minor physical anomalies and problem behavior in elementary school children. *Child Development* 1976;47(1):281-285.
 24. Arseneault L, Tremblay RE, Boulerice B, Séguin JR, Saucier JF. Minor physical anomalies and family adversity as risk factors for violent delinquency in adolescence. *American Journal of Psychiatry* 2000;157(6):917-923.
 25. Brennan PA, Mednick SA, Raine A. Biosocial interactions and violence: a focus on perinatal factors. In: Raine A, Brennan PA, Farrington DP, Mednick SA, eds. *Biosocial Bases of Violence*. New York, NY: Plenum Press; 1997:163-174. *NATO ASI Series. Series A, Life Sciences*, Vol. 292.
 26. Pine DS, Wasserman G, Coplan J, Fried J, Sloan R, Myers M, Greenhill L, Shaffer D, Parsons B. Serotonergic and cardiac correlates of aggression in children. *Annals of the New York Academy of Sciences* 1996;794(1):391-393.
 27. Pomeroy JC, Sprafkin J, Gadow KD. Minor physical anomalies as a biologic marker for behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry* 1988;27(4):466-473.

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28. Raine A, Brennan P, Mednick SA. Birth complications combined with early maternal rejection at age 1 year predispose to violent crime at age 18 years. *Archives of General Psychiatry* 1994;51(12):984-988.
29. Piquero A, Tibbetts S. The impact of pre/perinatal disturbances and disadvantaged familial environment in predicting criminal offending. *Studies on Crime and Crime Prevention* 1999;8(1):52-70.
30. Hodgins S, Kratzer L, McNeil TF. Obstetric complications, parenting, and risk of criminal behavior. *Archives of General Psychiatry* 2001;58(8):746-752.
31. Cannon TD, van Erp TGM, Rosso IM, Huttunen M, Lönnqvist J, Pirkola T, Salonen O, Valanne L, Poutanen VP, Standertskjöld-Nordenstam CG. Fetal hypoxia and structural brain abnormalities in schizophrenic patients, their siblings, and controls. *Archives of General Psychiatry* 2002;59(1):35-41.

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