Anatomical and Physiological Changes to the Brain Due to Prenatal Alcohol Exposure (PAE)

EPSY 836: Neuropsychology
University of Regina
Kourtney Gorham

Alcohol and the Developing Fetus

- Alcohol is a teratogen that enters through the placenta (Brown, Conner, & Adler, 2012)
- Reuptake of amniotic fluid allows the blood-alcohol level to remain high for longer (Nash & Davies, 2017)
- The 2nd through 8th weeks are particularly critical due to Central Nervous System (CNS) development and DNA synthesis occurring (Brown et al., 2012; Kolb, Whishaw, & Teskey, 2019)

Prenatal Development

- **1st Trimester** - heart; digestive and nervous system; back and spinal cord; lungs; brain; formation of arms, legs, fingers, hands, and face; muscles and bones; stomach
- **2nd Trimester** - hears; bones get bigger/stronger; hair; fingernails; toenails; teeth under gums; brain continues to develop; arms/legs get longer; thin skin with fine hairs and wax; eyelashes; eyebrows; fingerprints and footprints form
- **3rd Trimester** - organs function mostly on their own; fat under skin; skin thickens; knows voice of caregiver; opens eyes; lungs fully developed; brain continues to develop; antibodies passed from mom to baby; reflexes learned; grasps own hands; gets stronger, bigger, and positioned for birth (Saskatchewan Prevention Institute, n.d.)

Prevalence of FASD

- PAE can cause Fetal Alcohol Spectrum Disorder (FASD) with both neurocognitive and neurobehavioral impairments due to CNS damage (Brown et al., 2012; CanFASD, 2019; Nash & Davies, 2017; Popova et al., 2015; Support Network, 2017).
- 4% or 1.4 million Canadians and at least 1% of individuals in Saskatchewan have FASD (CanFASD, 2019; Support Network, 2017).
- Approximately 50% of adult and 80% of adolescent pregnancies are unplanned; only 9.4% of adults and 13.4% of adolescents drink while pregnant with reductions in each trimester (Nash & Davies, 2017)

Factors

- PAE has lifelong impacts regardless of diagnosis
- Studying PAE can be complicated as it is hard to get specific information about the amount, frequency, and developmental time of use
- Comorbid conditions and other substances used prenatally can further complicate research (Lebel, Roussotte, & Sowell, 2011; Moore et al., 2017)

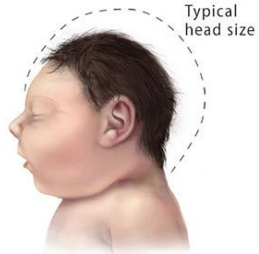
Anatomical and Physiological Abnormalities

- Microcephaly
- Reduced white and gray matter volumes
- Malformations in the frontal, parietal, and temporal lobes
- Abnormalities of the corpus callosum (CC)
- Neural loss and communication issues

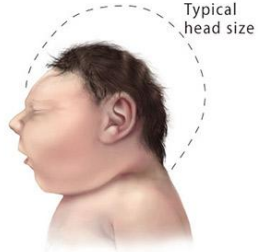
Anatomical Abnormalities: Microcephaly

- Reduced head and brain size and volume (Chen et al, n.d.; Fryer et al., 2012; Lebel et al., 2011; Nash & Davies, 2017; Stephen et al., 2012)
- Can lead to impairments such as developmental delays, seizures, intellectual disability, hearing and vision problems, and issues with movement and balance (CDC, 2019)

Baby with Typical Head Size



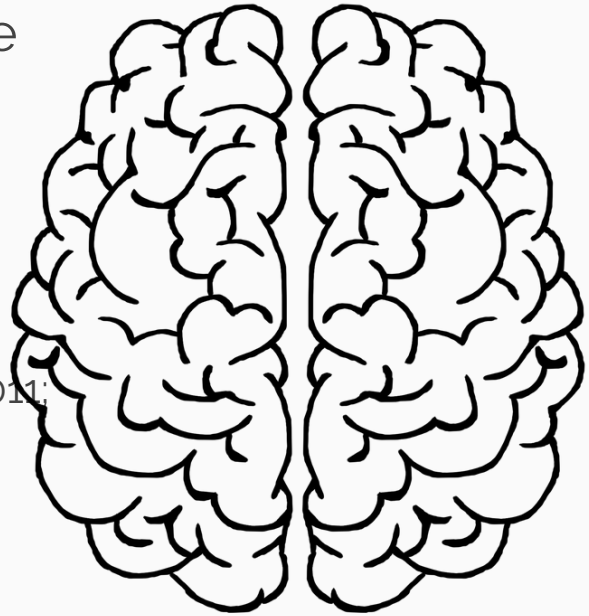
Baby with Microcephaly



Baby with
Severe Microcephaly

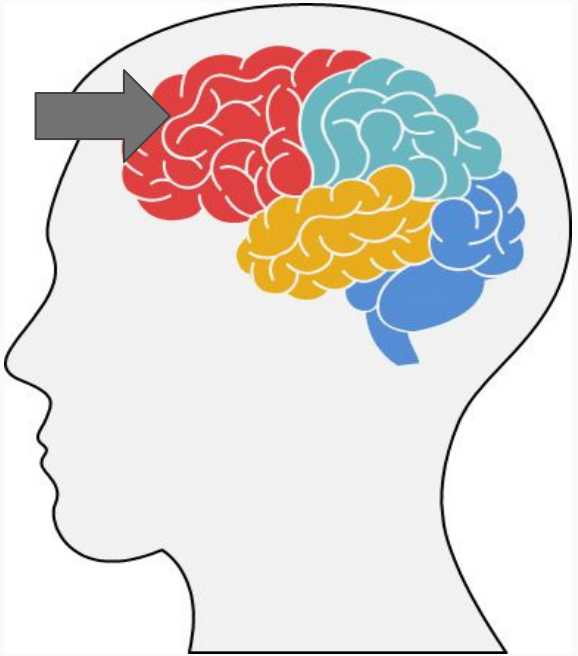
Anatomical Abnormalities: Reduced White and Gray Matter

- Even after accounting for microcephaly, there are reduced white and gray matter volumes (Lebel et al., 2011)
- Gray matter regions: thalamus, amygdala, caudate, hippocampus, basal ganglia, and pallidum are smaller (Fryer et al., 2012; Lebel et al., 2011; Sharma & Hill, 2017)
- White matter is reduced in the parietal lobe and cerebral cortex (Chen et al., n.d.)



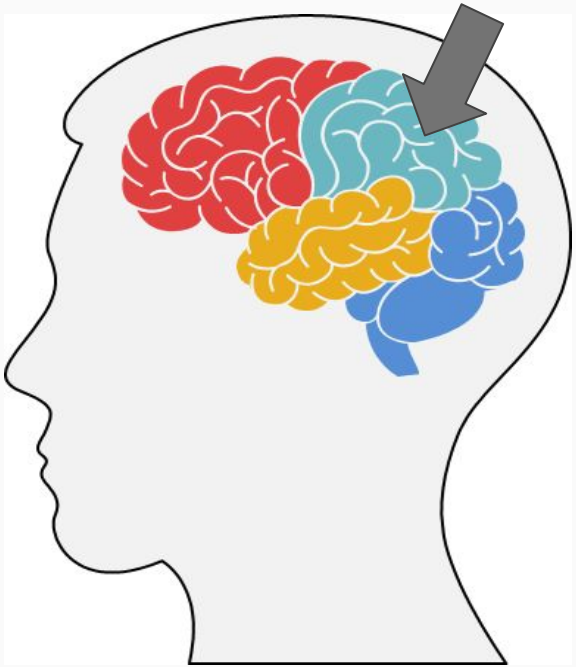
Anatomical Abnormalities: Frontal Lobe

- Less volume of total white and gray matter
- Reduced gyrification - cortical folding of the brain to create sulci and gyri to promote neuron connections and efficiency
- Abnormal cortical thickness



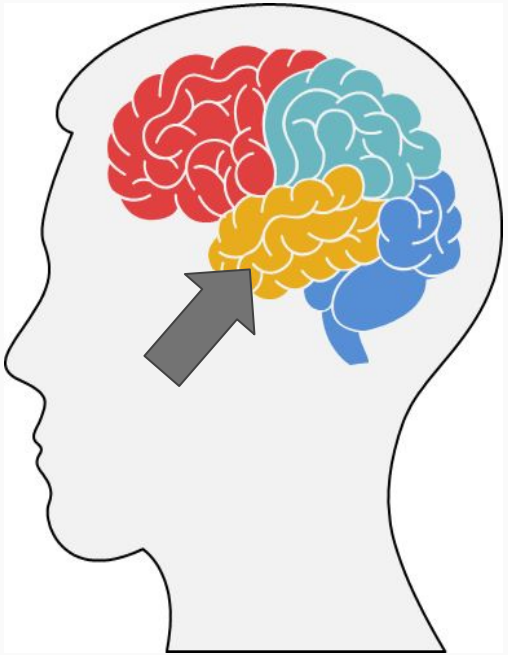
Anatomical Abnormalities: Parietal Lobe

- Less white and gray matter and volume due to narrowness of the lobes
- Thicker cortices, smaller fusiform gyrus, reduced temporal asymmetry, and displacement of inferior parietal and temporal regions (Lebel et al., 2011)
- Reduced gyrification
- Abnormalities related to high-order math difficulties (Moore et al., 2017)



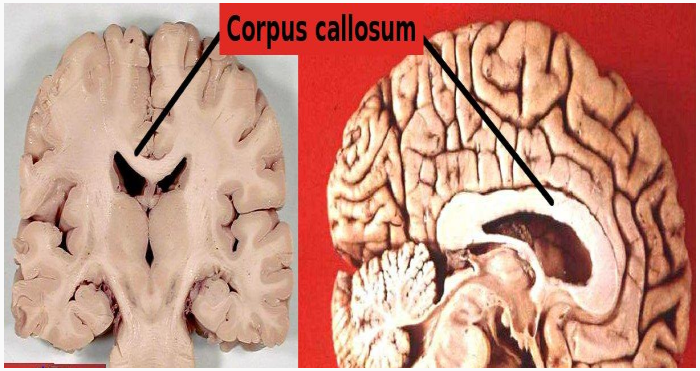
Anatomical Abnormalities: Temporal Lobe

- Less white and gray matter and volume due to narrowness of the lobes
- Thicker cortices, smaller fusiform gyrus, reduced temporal asymmetry, and displacement of inferior parietal and temporal regions (Lebel et al., 2011)
- Reduced gyrification
- Spelling difficulties were a result of abnormalities in the temporal lobe (Moore et al., 2017)



Anatomical and Physiological Abnormalities: Corpus Callosum

- Shape abnormalities and location displacement of 7mm (Lebel et al., 2011; Sowell et al., 2011)
- Complete or partial agenesis may occur (Eckstrand et al., 2012; Jacobson et al., 2017; Sowell et al., 2001; Stephen et al., 2012)
- Colossal thinning (Lebel et al., 2011)
- Smaller volume, area, and length
- White matter microstructural/frontostriatal connectivity issues (Donald et al., 2016)



Human brain

01 Frontal lobe

02 Limbic lobe

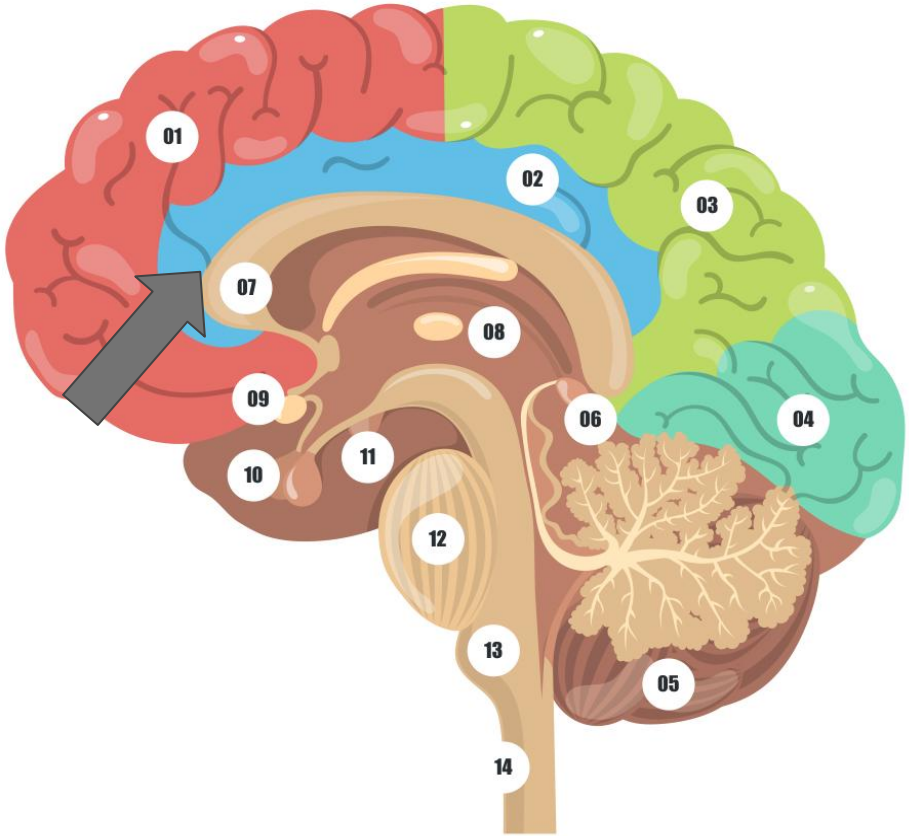
03 Parietal lobe

04 Occipital lobe

05 Cerebellum

06 Pineal gland

07 Corpus callous



08 Thalamus

09 Optic schiasma

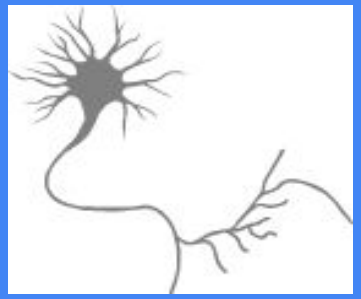
10 Pituitary gland

11 Mammillary body

12 Pons

13 Medulla oblongata

14 Spinal cord



Physiological Abnormalities: Neural Loss and Communication

- Cellular alterations and neural loss can occur (Chen et al., n.d.; Eckstrand et al., 2012)
- Abnormal cell growth and division (Nash & Davies, 2017)
- Alcohol disrupts cell migration from the production to end sites, impacting communication (Chen et al., n.d.)
- Cell migration disruptions occur due to agenesis, poor myelination, poor axonal integrity, or thinning, complicating transmission to dendrites in the cortex, hippocampus, and other important brain structures (Jacobson et al., 2017; Migliorini et al., 2015)

Conclusion

- The neurocognitive and neurobehavioral impairments - verbal learning, executive functioning, social deficits, cognitive and memory challenges, motor delays, etc. - are a result of CNS damage
- Support is essential!

Works Referenced

Brown, N., Connor, P., Adler, R., & Langton, C. (2012). Conduct-disordered adolescents with fetal alcohol spectrum disorder: Intervention in secure treatment settings. *Criminal Justice and Behavior, 39*(6), 770-793.

CanFASD: Canada FASD Research Network (2019). Diagnosis. Retrieved from: https://canfasd.ca/topics/diagnosis/

Center for Disease Control and Prevention (CDC) (2018). Facts about microcephaly. Retrieved from: https://www.cdc.gov/ncbddd/birthdefects/microcephaly.html

Chen, W. A., Maier, S. E., Parnell, S. E., & West, J. R. (2003). Alcohol and the developing brain: Neuroanatomical studies. *Alcohol Research & Health, 27*(2), 174-80.

Donald, K., Ipser, J., Howells, F., Roos, A., Fouche, J., Riley, E.,… Stein, D. (2016). Interhemispheric functional brain connectivity in neonates with prenatal alcohol exposure: Preliminary findings. *Alcoholism: Clinical and Experimental Research, 40*(1), 113-121.

Eckstrand, K. L., Ding, Z., Dodge, N. C., Cowan, R. L., Jacobson, J. L., Jacobson, S. W., & Avison, M. J. (2012). Persistent dose‐dependent changes in brain structure in young adults with low‐to‐moderate alcohol exposure in utero. *Alcoholism: Clinical and Experimental Research, 36*(11), 1892-1902

Works Referenced

FASD Network of Saskatchewan Inc. (2017). *Fetal alcohol spectrum disorder: A guide to awareness and understanding.*

Fryer, S., Mattson, S., Jernigan, T., Archibald, S., Jones, K., & Riley, E. (2012). Caudate volume predicts neurocognitive performance in youth with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research, 36*(11), 1932-1941.

Glass, L., Moore, E., Akshoomoff, N., Jones, K., Riley, E., & Mattson, S. (2017). Academic difficulties in children with prenatal alcohol exposure: Presence, profile, and neural correlates. *Alcoholism: Clinical and Experimental Research, 41*(5), 1024-1034.

Infante, M., Moore, E., Bischoff-Grethe, A., Migliorini, R., Mattson, S., & Riley, E. (2015). Atypical cortical gyrification in adolescents with histories of heavy prenatal alcohol exposure. *Brain Research, 1624*, 446-454.

Jacobson, S. W., Jacobson, J. L., Molteno, C. D., Warton, C. M. R., Wintermark, P., Hoyme H. E.,… Meintjes, E. M. (2017). Heavy prenatal alcohol exposure is related to smaller corpus callosum in newborn MRI scans. *Alcoholism: Clinical and Experimental Research, 41*(5), 965-975.

Kolb, B., Whishaw, I., & Teskey, G. (2016). *An introduction to brain and behavior* (6th ed.). New York: Worth Publishers.

Works Referenced

Lebel, C., Roussotte, F., & Sowell, E. (2011). Imaging the impact of prenatal alcohol exposure on the structure of the developing human brain. *Neuropsychology Review, 21*(2), 102-118.

Migliorini, R., Moore, E., Glass, L., Infante, M., Tapert, S., Jones, K.,… Riley, E. (2015). Anterior cingulate cortex surface area relates to behavioral inhibition in adolescents with and without heavy prenatal alcohol exposure. *Behavioural Brain Research, 292*, 26-35.

Nash, A., & Davies, L. (2017). Fetal alcohol spectrum disorders: What pediatric providers need to know. *Journal of Pediatric Health Care, 31*(5), 594-60.

Nguyen, T., Levy, S., Riley, E., Thomas, J., & Simmons, R. (2013). Children with heavy prenatal alcohol exposure experience reduced control of isotonic force. *Alcoholism: Clinical and Experimental Research, 37*(2), 315-324.

Osterman, R. (2011). Decreasing women's alcohol use during pregnancy. *Alcoholism Treatment Quarterly, 29*(4), 436-452.

Popova, S., Lange, S., Burd, L., & Rehm, J. (2015). Cost attributable to fetal alcohol spectrum disorder in the Canadian correctional system. *International Journal of Law and Psychiatry, 41*, 76.

Works Referenced

Roebuck, T., Mattson, S., & Riley, E. (2002). Interhemispheric transfer in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research, 26*(12), 1863-1871.

Saskatchewan Prevention Institute (n.d.). Your pregnancy month-by-month. Retrieved from: https://skprevention.ca/your-pregnancy-month-by-month/

Sharma, V., & Hill, S. (2017). Differentiating the effects of familial risk for alcohol dependence and prenatal exposure to alcohol on offspring brain morphology. *Alcoholism: Clinical and Experimental Research, 41*(2), 312-322.

Sowell, E., Mattson, S., Thompson, P., Jernigan, T., Riley, E., & Toga, A. (2001). Mapping callosal morphology and cognitive correlates: Effects of heavy prenatal alcohol exposure. *Neurology, 57*(2), 235-244.

Stephen, J. M., Kodituwakku, P. W., Kodituwakku, E. L., Romero, L., Peters, A. M., Sharadamma, N. M., . . . Coffman, B. A. (2012). Delays in auditory processing identified in preschool children with FASD. *Alcoholism, 36*(10), 1720-7.

Zhou, D., Rasmussen, C., Pei, J., Andrew, G., Reynolds, J., & Beaulieu, C. (2015). Thinner cortex and reduced brain volumes in children and adolescents with prenatal alcohol exposure. *International Journal of Developmental Neuroscience, 47*, 112-113.

thank you
danke
рахмат
謝謝
ngiyabonga
شكراً جزيلاً
teşekkür ederim
Баярлалаа
спасибо
faafetai lava
mersi
kia ora
barka
welalin
tack
spas
dank je
misaotra
matondo
paldies
grazzi
mahalo
gracias
tapadh leat
vinaka
спасибі
blagodaram
kiitos
dankie
nanni
nandri
dhanyavad
bedankt
bayarlalaa
gràcje
hvala
māuruuru
köszönöm
akun
dankon
aciū
хвала
asante
manana
obrigada
murakoze
tenki
enkosi
dziękuję
djiere dieuf
tau
mochchakkeram
дякую
mamnun
chokrane
chnorakaloutioun
gratias ago
gràcies
sulpāy
go raibh maith agat
sobodi
dēkuji
obrigado
sukriya
kop khun krap
taiku
sağolun
arigatô
takk
dakujem
trugarez
mèsi
didi madloba
najis tuke
ありがとう
tanemirt
grazie
terima kasih
rahmet
ευχαριστώ
diolch
dhanyavadagalu
shukriya
mercè
мерси
kam sah hamnida
rahmat
তোমাকে ধন্যবাদ
감사합니다
xièxie
merci

FASD AFFECTS MORE PEOPLE THAN AUTISM, CEREBRAL PALSY, AND DOWN'S SYNDROME COMBINED

FASD NETWORK