Dok 79

Monday, 11 November 2024

# Domestic Violence in review

## Proposal: An integrated approach system,

Despite significant progress in Malta's legal framework to address domestic violence, there are still several gaps and areas for improvement in how the law is applied and in the protection of victims. Here are some key gaps and challenges identified in Malta's domestic violence laws:

**Implementation and Enforcement Challenges:**

There is a need for more consistent enforcement and greater legal follow-up to ensure the safety of the victims.

While Malta has strong legal provisions for combating domestic violence, enforcement can sometimes be inconsistent, providing a window for abusers to fall through.

Although Maltese police have received training on handling domestic violence cases, there are concerns that some officers still lack sensitivity or fail to respond promptly and effectively in every case.

In some situations, victims may feel dismissed or not taken seriously when reporting abuse, which can lead to underreporting or delayed intervention.

Inconsistent Issuance of Protection Orders: While the law provides for the issuance of protection orders, there have been instances where such orders are not issued promptly or are violated without significant consequence.

**Limited Focus on Psychological and Emotional Abuse**

While Maltese law addresses physical violence and sexual abuse, **psychological and emotional abuse** are still relatively underrepresented in legal proceedings. The The Domestic Violence Act (2003) and its amendments recognize psychological abuse, but it is not always treated with the same gravity as physical violence, leading to a gap in protecting victims who suffer non-physical forms of abuse, which can be just as damaging.

**Difficulty in Proving Abuse**

Another issue is that **evidence of abuse** can be difficult to collect, especially when the violence is not physical or when the abuse occurs in private spaces. This

can make it harder to secure convictions or even protection orders, particularly when the victim is reluctant to testify or provide evidence against the perpetrator.

**Equal Support for Male Victims**

While domestic violence is predominantly experienced by women, male victims of domestic violence may face additional barriers to seeking help, including societal stigma or a lack of specific services tailored to their needs.

The Maltese system, like many others, tends to focus primarily on female victims, with fewer services and support networks available for male survivors of domestic violence.

**Social and Cultural Attitudes** :The legal and support system may not be fully equipped to deal with abusive relationships in different culture groups where women are not treated as equal to men.

**Limited Focus on Children as Victims**

housing policies and social welfare benefits into the domestic violence protection strategy could help address this gap.

**Perpetrator Rehabilitation**

Rehabilitation programs for abusers should be mandatory. Without addressing the root causes of abusive behavior and offering abusers the tools to change, there is a risk of perpetrators continuing the cycle of violence.

Whilst some NGOs and support services may offer rehabilitation programs for abusers, these are not always mandatory or universally accessible. In some cases, perpetrators may not be adequately held accountable for their actions, and interventions aimed at breaking the cycle of abuse are insufficient.

**Underreporting and Lack of Trust in the System**

Victims of domestic violence, especially women, often hesitate to seek legal protection due to fears of retaliation, lack of faith in the legal system, or concerns about the social stigma attached to being a victim of abuse.

Conclusion

While Malta's domestic violence laws have made significant progress in protecting victims and addressing various forms of abuse, there are still important gaps that need to be addressed including

1. Investing in education and associated behaviours in youths.
2. Training and detection of domestic violence to the general population
3. Protection of pregnant women
4. Including the protection of the child in the womb

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CEO Life Network Foundation

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THE CONVERSATION
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Women having a subsequent baby are more likely to disclose domestic violence than first time mothers. Vyshnova/Shutterstock

# Pregnant women are at increased risk of domestic violence in all cultural groups

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Domestic violence occurs across all age groups and life stages. Rather than reducing during pregnancy, expecting a child is a key risk factor for domestic violence beginning or escalating.

Our research, published today in the journal BMJ Open, found that 4.3% of pregnant women due to give birth in Western Sydney disclosed domestic violence when asked about it by a midwife at her first hospital visit. The study examined more than 33,000 ethnically diverse women who gave birth between 2006 and 2016, and found that these disclosures spanned all cultural groups.

Domestic violence in pregnancy not only causes distress and trauma for the mother and baby, it increases the risk of the baby having a low birth weight (very small baby) or being born prematurely (before 37 weeks), which is linked to jaundice, anaemia and respiratory distress in infancy, and diabetes and heart disease later in life.

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***Read more:*** ***Midwives can help detect domestic violence – here's how***

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## Abuse and trauma

Depending on the state or territory, women may receive a "psychosocial" assessment from midwives when they first book into a public hospital during pregnancy. This screens for depression, anxiety, childhood abuse, domestic violence, support and stress.

Using these assessments, we found that 4.3% of women disclosed domestic violence overall, but rates were higher among women having a subsequent baby, compared with first-time mothers.

We're unsure if this is because violence has escalated for these women with subsequent pregnancies; if they trust health providers more to disclose the violence; or if they seek help because they're becoming more aware of the impact of domestic violence on their children.

We found that domestic violence occurred across all cultural groups, but reported rates were highest among women from New Zealand and Sudan.

Previous research has shown high rates of domestic violence among Maori women in some parts of New Zealand.

There is also evidence of high rates of domestic violence among Sudanese women prior to migration.

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***Read more:*** ***Family violence victims need support, not mandatory reporting***

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We found that women born in India and China reported very low rates of domestic violence. This may reflect a cultural tendency not to discuss what is considered private family business with outsiders.

It's important that health professionals know how to ask about domestic violence in a culturally appropriate way so women feel comfortable disclosing abuse and can access appropriate support.

## What needs to be done?

Midwives need to consider cultural norms and acceptability when asking migrant women questions about domestic violence, and this must always be done in a way that keeps the woman safe. Partners should not be present when the questions are asked – and this may be done at another time in the pregnancy if necessary.

Where English is not the first language, interpreters should be used. But this can also present challenges if the interpreter comes from the same community and is known to the woman.

When women have continuity of midwifery care and get to know a midwife well throughout the pregnancy, it is easier for midwives to gain women's trust and to notice when things change. This style of care should be rolled out more widely in Australian public hospitals.

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***Read more:*** ***Acting on family violence: how the health system can step up***

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*The National Sexual Assault, Family & Domestic Violence Counselling Line – 1800 RESPECT (1800 737 732) – is available 24 hours a day, seven days a week for any Australian who has experienced, or is at risk of, family and domestic violence and/or sexual assault.*

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# Antenatal maternal intimate partner violence exposure is associated with sex-specific alterations in brain structure among young infants: Evidence from a South African birth cohort

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ABSTRACT

Maternal psychological distress during pregnancy has been linked to adverse outcomes in children with evidence of sex-specific effects on brain development. Here, we investigated whether *in utero* exposure to intimate partner violence (IPV), a particularly severe maternal stressor, is associated with brain structure in young infants from a South African birth cohort. Exposure to IPV during pregnancy was measured in 143 mothers at 28–32 weeks' gestation and infants underwent structural and diffusion magnetic resonance imaging (mean age 3 weeks). Subcortical volumetric estimates were compared between IPV-exposed ($n = 63$; 52% female) and unexposed infants ($n = 80$; 48% female), with white matter microstructure also examined in a subsample (IPV-exposed, $n = 28$, 54% female; unexposed infants, $n = 42$, 40% female). In confound adjusted analyses, maternal IPV exposure was associated with sexually dimorphic effects in brain volumes: IPV exposure predicted a larger caudate nucleus among males but not females, and smaller amygdala among females but not males. Diffusivity alterations within white matter tracts of interest were evident in males, but not females exposed to IPV. Results were robust to the removal of mother-infant pairs with pregnancy complications. Further research is required to understand how these early alterations are linked to the sex-bias in neuropsychiatric outcomes later observed in IPV-exposed children.

## 1. Introduction

Maternal psychological distress during pregnancy is associated with an increased risk of mental health problems in children (Davis and Sandman, 2012; Park et al., 2014) and predicts psychiatric disorders in adulthood (Betts et al., 2015; Van den Bergh et al., 2020). The experimental induction of prenatal stress in animal models has shown enduring effects on neurodevelopment of the offspring (Soares-Cunha et al., 2018; Zhang et al., 2021). Evidence points to how the excessive release of cortisol, serotonin (Bush et al., 2017; Sandman et al., 2011) or pro-inflammatory cytokines (Glover, 2015) may be one of numerous mediating factors that lead to alterations in neurochemistry and signalling pathways involved in regulating neuroplasticity (Lai and Huang, 2011; Lautarescu, Craig et al., 2020). Limbic brain regions may be

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particularly sensitive to prenatal stress due to their high content of glucocorticoid receptors (Teicher et al., 2003), while animal models also indicate sex-specific biological responses to prenatal stress (Gabory et al., 2009; Mueller and Bale, 2008) that may lead to different behavioural phenotypes (Glover and Hill, 2012; Weinstock, 2017).

Psychological distress in humans during pregnancy encompasses symptoms of anxiety, depression, as well as the effects of exposure to stressful or traumatic life events including intimate partner violence, bereavement, or natural disasters (Wu et al., 2020). The adverse outcomes observed in children later in life is presumably due to how psychological distress affects brain structure and biochemistry *in utero*. Brain imaging studies have assessed the relationship between variations in cortisol as a marker of antenatal maternal psychological distress and the subsequent development of limbic brain structures in children and adolescents many years later. Consistent with the preclinical literature, most evidence points to how antenatal maternal psychological distress may influence the offspring amygdala in a sex-specific manner, with likely indicators of antenatal maternal distress being associated with enlarged amygdala volumes in school-aged girls, but not boys (Acosta et al., 2019; Buss et al., 2012; Jones et al., 2019; Wen et al., 2017), albeit with some inconsistent findings (El Marroun et al. (2016). For example, higher maternal cortisol measured in pregnancy predicted larger right amygdala volumes in 7-year-old girls, which mediated later affective problems (Buss et al., 2012). Additionally, female children of mothers exposed to a natural disaster during pregnancy showed larger amygdala volumes at aged 11, which in part explained greater externalizing problems (Jones et al., 2019). In contrast, evidence for changes to the hippocampus due to prenatal stress has been mixed. While reduced hippocampal volume is evident in adults and children exposed to *early life stress* (Humphreys et al., 2019; Marečková et al., 2018; Woon and Hedges, 2008), there is little evidence for an association between hippocampal volume and *antenatal* stress in humans (Buss et al., 2012; Favaro et al., 2015; Marečková et al., 2018; Qiu et al., 2013). Importantly, few studies have examined the developing brain in very young infants to minimize the impact of the postnatal environment as, for example, parenting styles may also contribute to children's brain development (Suffren et al., 2022). One recent study reported no association between maternal cortisol

axon diameter (Alexander et al., 2007). Further studies have begun to consider the effects of prenatal maternal distress on the microstructural organization of other white matter tracts. For example, exposure to prenatal stress has been associated with disruptions to the anterior cingulate (i.e. cingulum) within 2–5 weeks after birth (Demers et al., 2021), and corpus callosum in 6-month-old infants (Borchers et al., 2021). Given this emerging evidence, the potential impacts of prenatal stress on white matter pathways in young infants, including possible sex-specific effects, warrants further investigation.

One particularly severe stressor that can occur antenatally is exposure to intimate partner violence (IPV) (Barnett et al., 2018). According to large population-based household and national health surveys, the prevalence of violence against women by their intimate partner varies widely across countries but tends to be substantially higher in low-income and middle-income countries versus high-income countries (Coll et al., 2020; Garcia-Moreno et al., 2006). Maternal IPV exposure during pregnancy not only has negative mental and physical health consequences for women (McKelvie et al., 2021), but is also associated with poor health and developmental outcomes for their children (Chai et al., 2016; Da Thi Tran et al., 2022) and is a serious public health concern. The present study utilizes data from the Drakenstein Child Health Study (DCHS), a multidisciplinary South African birth cohort study, to conduct the first examination of how antenatal IPV exposure affects early brain development. Based on previous DCHS findings which found an association between antenatal depression exposure and infant brain structure (Groenewold et al., 2022), we hypothesized that exposure to antenatal IPV would have a similar impact on neurodevelopment. Our primary objective was to establish whether maternal IPV exposure during pregnancy is associated with alterations in neonatal subcortical brain volumes and white matter microstructure in offspring within the first few weeks of life, including the testing of sex-specific effects. Specifically, we examined IPV in relation to volumes of the amygdala, hippocampus, and other key subcortical regions in the basal ganglia and thalamus in neonates. In a subset of the sample, we examined microstructural white matter integrity, measured [illegible] MD, of limbic white matter tracts implicated in the [illegible] psychopathology in older children

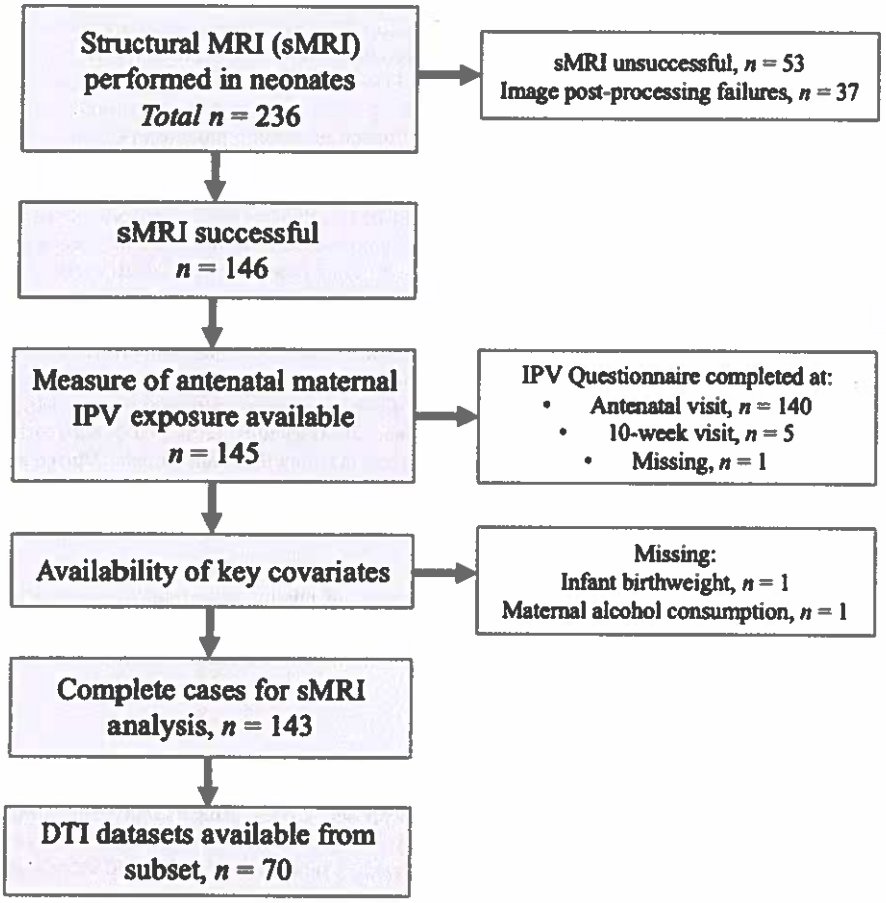


Fig. 1. Drakenstein Child Health Study cohort flow chart of neonates with neuroimaging. Structural MRI was unsuccessful in infants who did not sleep or because of artefacts evident during the scan. In the 183 infants with eligible $T_2$-weighted images, 37 did not pass quality control either due to poor image quality, normalization errors, or segmentation faults leaving a total of 146 infants with structural MRI data available. We were unable to account for missing birthweight ($n = 1$) and missing antenatal alcohol consumption ($n = 1$), as they are considered exogenous thus are not predicted by any of the other variables in the model.

approved by the Western Cape Provincial Health Research committee (2011RP45). All study procedures were carried out in accordance with the Declaration of Helsinki (World Medical Association, 2013).

### 2.2. Measures and variable calculation

Questionnaires were administered to the enrolled women at the primary health care clinics between 28- and 32-weeks' gestation. The questionnaires were offered in locally spoken languages in the Western Cape (English, Afrikaans, and Xhosa). Approximately 50% of questionnaires were administered in Afrikaans, whereas the others were administered in either English (~25%) or Xhosa (~25%).

#### 2.2.1. Intimate partner violence exposure

The Intimate Partner Violence (IPV) Questionnaire assessed mothers' recent exposure (past 12 months) to emotional, physical, and sexual abuse, and has been adapted from the WHO multi-country study on women's health and domestic violence against women (WHO, 2005). There are four questions relating to emotional violence (e.g., being purposefully scared or intimidated), five relating to physical abuse (e.g., being hit with a fist or with something else that could hurt), and three questions assessing sexual violence (e.g., being physically forced to have sex). A 4-point frequency of occurrence scale was used for each of the 12 questions: (1) never, (2) once, (3) few times, and (4) many times, resulting in a maximum score of 48. At the end of the emotional violence questions, mothers were then asked, *"Have any of these things happened in the past 12 months?"*. The same question was asked again at the end of both the physical and sexual violence questions. If the answer was *"Yes"* to any one of the three violence categories (emotional, physical, or sexual), mothers were classified as being exposed to recent IPV. Participants were designated into the control group if they were not exposed to any type of IPV within the previous 12 months. Therefore, antenatal exposure to IPV was used as a dichotomous variable (exposure to recent IPV/ no exposure to recent IPV) in all statistical models.

#### 2.2.2. Demographic and clinical variables

*Sociodemographic:* Maternal sociodemographic characteristics were collected by interview and questionnaires, including mother's age, employment status, marital status, household income, maternal education, as well as medical history including HIV status.

*Birth characteristics:* Gestational age at birth (in weeks) was recorded either through ultrasound, measurements of fundal height, or through self-reported last menstrual period. Birth weight was obtained at the hospital following delivery.

*Prenatal alcohol consumption and smoking status:* Prenatal alcohol exposure risk was classified as a binary variable, defined as an Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) total alcohol score > 10 (at least weekly alcohol use with negative consequences) or retrospective self-report of consuming alcohol on 2 or more occasions per week (Newcombe et al., 2005). Smoking status was assessed via prenatal cotinine measurements in maternal urine using the IMMULITE 1000 Nicotine Metabolite Kit (Siemens Medical Solutions Diagnostics, Glyn Rhonwy, Llanberis, UK). Cotinine is the major metabolite of nicotine and is often used as a proxy measure of recent tobacco use. Active smoking was defined by cotinine > = 500 ng/ml, with passive smoking > = 10–499 ng/ml, and nonsmoking < 10 ng/ml.

*Maternal depression:* Antenatal maternal depressive symptoms were assessed using the Beck Depression Inventory II (BDI-II; Beck et al. (1996), which has been validated and used in multiple studies of South African women (Kagee et al., 2014). The measure consists of 21 items,

each assessing major depressive symptoms over the past four weeks (rated 0–3, total score range 0–63). A cut off score of ≥ 20 was used for dichotomizing participants into "probable moderate/severe clinical cases" versus "sub-threshold participants", as previously described Stein et al. (2015).

### 2.3. Image acquisition

MR images were acquired on a 3 T Siemens Magnetom Allegra MRI scanner (Allegra MR2004A, Germany) at the Cape Universities Brain Imaging Centre (CUBIC), Tygerberg Hospital, Cape Town. Infants were fed, swaddled in a blanket, and encouraged to sleep. Earplugs and mini muffs were used for double ear protection and a qualified neonatal nurse or pediatrician monitored the infant in the scanner room for the duration of the scan (Wedderburn et al., 2022). To overcome limitations with scanning smaller tissue volumes, voltage was reduced to optimize signal, and a radiofrequency transmit/receive head coil was loaded with a wet clay inlay (40x40cm, 2 cm thickness, standard sculpting clay). The imaging protocol included a $T_2$-weighted anatomical scan with the following parameters: FOV = 160 × 160 mm, $TR = 3500$ *ms*, $TE = 354$ *ms*, 128 slices, in-plane resolution = 1.3 × 1.3 mm and a slice thickness of 1.0 mm. The acquisition time was 5 min 41 s. Diffusion weighted images were collected using a spin-echo echo-planar imaging (EPI) sequence with images collected in both the anterior–posterior (AP) and posterior–anterior (PA) phase encoding directions to correct for field inhomogeneities. Parameters were as follows: 30 diffusion directions; FOV = 160 × 160 mm, $TR = 7800$ *ms*, $TE = 91$ *ms*, voxel size 1.8 × 1.8 × 2.0 $mm^3$; *b*-value 1 of 0 $s/mm^2$ and b-value 2 of 1000 $s/mm^2$. Total diffusion scan time was 12 mins 54 s

### 2.4. Image processing

From the original sample of 236 infants, 53 infants did not sleep or were excluded due to artefacts leaving a sample of 183 infants (78%) for image processing. $T_2$-weighted images were skull stripped using the brain extraction tool (BET) from the FMRIB Software Library (FSL) v5.0 (Jenkinson et al., 2012). Each scan was checked visually following the initial BET and additional custom thresholds were applied to ensure non-brain tissue was adequately removed. Brain images were pre-processed further using Statistical Parametric Mapping software (SPM8, University College London, London, UK) with researchers blin

deemed usable for data pre-processing and statistical analyses (see Supplementary Methods). Usable diffusion data was preprocessed using TORTOISE v2.5.2 (Tolerably Obsessive Registration and Tensor Optimization Indolent Software Ensemble), which implements comprehensive correction and applies greater anatomical registration ability compared to mainstream diffusion processing pipelines (Taylor et al., 2016). The DiffPrep module was used to compute distortion corrections for participant motion, eddy currents and EPI distortions on each AP and PA encoded image. The DR BUDDI module merged the encoded sets and performed further EPI distortion corrections. Diffusion tensor parameter fitting was then performed with Tract-Based Spatial Statistics (TBSS) in FSL to extract parameters (Smith et al., 2006). A study-specific template was used to enhance registration quality, given the infant brain will differ from the adult MNI template. Fractional anisotropy (FA), a standardized measure of directional water diffusion, and mean diffusivity (MD), the average of water diffusion, were extracted for the five white matter tracts of interest (uncincate fasciculus, fornix, cingulum, corticospinal tract, and corpus callosum) using the Johns Hopkins University ICBM-DTI-81 atlas (Mori et al., 2008).

### 2.5. Missing data

Fig. 1 details the proportion of missing data related to potential confounding variables. Infants were included in the analysis if either structural or diffusion MRI data, recent IPV exposure, and all potential covariates were available.

### 2.6. Statistical analysis

Socio-demographic and clinical characteristics were compared between IPV-exposed and unexposed groups using independent *t*-tests (continuous variables), and chi-squared tests (categorical variables). Group differences in neuroimaging outcomes were assessed using a series of full factorial linear regression models, with each brain structure of interest as the dependent variable and recent IPV and infant sex as predictors. In the event of a significant interaction between IPV exposure and infant sex, sex-stratified analyses were conducted in boys and girls separately. A similar approach was taken for the DTI analyses. Confounders were selected *a priori* and included household income – as a proxy for socioeconomic status, maternal HIV status, maternal depression symptoms, gestation duration, infant birthweight, postnatal

($n = 1$), preeclampsia ($n = 4$), pelvic inflammatory disease ($n = 1$), and/or high blood pressure ($n = 5$).

Statistical analyses were conducted using STATA, v17.0 (StataCorp Inc, College Station, TX, USA). $p < 0.05$ was used as a threshold of statistical significance. To minimize the number of comparisons, unilateral volumes were added together to create bilateral volumes, whereas DTI outcomes were averaged across hemispheres. The standard False Discovery Rate method was used as an additional correction for multiple comparisons (Benjamini and Hochberg, 1995) ($n = 6$ for subcortical volumes; $n = 10$ for diffusion outcomes) with a false-positive rate of 5% ($q = 0.05$).

## 3. Results

### 3.1. Participant characteristics for IPV-exposed and unexposed infants

The final sample for the structural MRI analysis included 143 mother-child dyads. Sixty-three mothers (44%) reported IPV within the preceding 12 months, whereas 80 mothers (56%) who did not report IPV exposure served as a control group. Mothers in the two groups were similar in most sociodemographic characteristics including age at birth, education level, household income, and employment status (Table 1). However, mothers exposed to IPV were more likely to be married or living with a partner compared to controls (53.9% versus 33.8%, $\chi^2 = 6.34$, $p = 0.012$), whereas the majority of the control group were single. Antenatal alcohol consumption was more prevalent in the IPV exposed compared to unexposed group (27.0% versus 11.3%, $\chi^2 = 5.87$, $p = 0.015$), whereas IPV-exposed mothers were less likely to be HIV positive than controls (17.5% versus 32.5%, $\chi^2 = 4.16$, $p = 0.041$). Two mothers were on psychotropic medication at enrolment. One IPV-exposed mother reported antidepressant medication use (citalopram), whereas one unexposed mother reported pain medication use (tramadol). There were no group differences in infant birth characteristics with regards to sex, age at scan, gestation duration, birthweight, and head circumference, and the imaging subsample was largely representative of the wider DCHS sample (Wedderburn et al., 2022). Overall, sixteen infants (11%) were moderate to late preterm (between 33 and 37 weeks gestational age at birth), whereas the majority were born at full term. All pregnancies were uniparous. From the subset of 143 mother-child dyads with structural MRI data and all key covariates available, DTI data were available from 70 infants (See Supplementary Table 2 (ST2) for characteristics of this subsample).

### 3.2. Effects of antenatal IPV exposure on neonatal brain volumes

Given the exploratory nature of this study, all models were initially run without correction for multiple comparisons. There were no main effects of antenatal IPV exposure on total WM or GM volumes, and no IPV-by-sex interactions. However, infants who were exposed to IPV *in utero* had larger caudate nucleus volumes in comparison to controls (Table 2). This main effect was further qualified by a IPV-by-sex interaction. In sex-stratified analyses, IPV exposed boys had 5% larger caudate volumes compared to unexposed boys (3852 vs. 3665 $mm^3$; $p = 0.008$; Cohen's $d = -0.69$ [95% CI −1.17 to −0.20]). In contrast, girls exposed to IPV did not differ from unexposed girls (3687 vs. 3708 $mm^3$; $p = .211$; Cohen's $d = 0.06$ [95% CI 0.41 to 0.53]). There was no main IPV effect on amygdala volume; however, a significant IPV-by-sex interaction was observed. Analyses stratified by sex demonstrated 2% smaller amygdala volumes in IPV-exposed compared to unexposed female infants (1069 vs. 1086 $mm^3$; $p = 0.028$; Cohen's $d = 0.45$ [95% CI −0.03 to 0.92]), whereas no significant difference was present in male infants (1096 vs. 1081 $mm^3$; $p = 0.283$; Cohen's $d = -0.41$ [95% CI −0.88 to 0.07]) (see Fig. 2A). Neither the amygdala nor caudate results survived correction for multiple comparisons. There was no evidence that antenatal IPV exposure was associated with the volume of the hippocampus, pallidum, putamen, or thalamus. Results from

**Table 1**
Descriptive characteristics of IPV-exposed and unexposed groups.

| | IPV-exposed ($n = 63$) | Controls ($n = 80$) | Group differences ($\chi^2$ or $t$) | p-value |
|---|---|---|---|---|
| **Sociodemographic** | | | | |
| **Clinic (n, %)** | | | | |
| *Mbekweni* | 28, 44.4% | 42, 52.5% | 0.92 | 0.339 |
| *TC Newman* | 35, 55.6% | 38, 47.5% | | |
| *Maternal age at birth (M ±SD)* | 27.98 ± 5.72 | 26.92 ± 6.04 | -1.06 | 0.290 |
| **Household income per month (n, %)** | | | | |
| *<R1000* | 19, 30.2% | 28, 35.0% | 1.41 | 0.493 |
| *R1000 – R5000* | 36, 57.1% | 38, 47.5% | | |
| *>R5000* | 8, 12.7% | 14, 17.5% | | |
| **Employment status (n, %)** | | | | |
| *Employed* | 14, 22.2% | 26, 32.5% | 1.85 | 0.174 |
| *Unemployed* | 49, 77.8% | 54, 67.5% | | |
| **Maternal education (n, %)** | | | | |
| *Primary/some secondary* | 36, 57.1% | 41, 51.2% | 0.49 | 0.483 |
| *Completed secondary/ any tertiary* | 27, 42.9% | 39, 48.8% | | |
| **Marital status (n, %)** | | | | |
| *Single (never married)* | 28, 44.4% | 53, 66.2% | 6.34 | 0.012 |
| *Married/living with partner* | 34, 53.9% | 27 33.8% | | |
| **Maternal clinical characteristics** | | | | |
| **HIV status (n, %)** | | | | |
| *Positive* | 11, 17.5% | 26, 32.5% | 4.16 | 0.041 |
| *Negative* | 52, 82.5% | 54, 67.5% | | |
| **Beck Depression Inventory (n, %)** | | | | |
| *Above threshold* | 21, 33.3% | 20, 25.0% | 1.20 | 0.274 |
| *Below threshold* | 42, 66.7% | 60, 75.0% | | |
| **Antenatal alcohol exposure (n, %)** | | | | |
| *Exposure* | 17, 27.0% | 9, 11.3% | 5.87 | 0.015 |
| *No exposure* | 46, 73.0% | 71, 88.8% | | |
| **Antenatal tobacco use (n, %)** | | | | |
| *Non-smoker (<10 ng/ml)* | 13, 20.6% | 26, 32.5% | 3.80 | 0.150 |
| *Passive smoker (>=10–499 ng/ml)* | 26, 41.3% | 34, 42.5% | | |
| *Active smoker (>=500 ng/ml)* | 24, 38.1% | 20, 25.0% | | |
| **Infant characteristics** | | | | |
| **Sex (n, %)** | | | | |
| *Male* | 30, 47.6% | 42, 52.5% | 0.576 | 0.448 |
| *Female* | 33, 52.4% | 38, 47.5% | | |
| Age at scan (weeks) (*M ± SD*) | 3.22 ± 0.98 | 3.13 ± 0.76 | -0.62 | 0.534 |
| Gestation duration (weeks) (*M ± SD*) | 38.95 ± 1.96 | 38.95 ± 1.91 | -0.01 | 0.994 |
| Birthweight (grams) (*M ± SD*) | 3108 ± 469 | 3200 ± 454 | 1.19 | 0.236 |
| Head circumference (cm) (*M ± SD*) | 33.52 ± 1.72 | 33.99 ± 1.66 | 1.66 | 0.099 |

*Missing marital status ($n = 1$). R South African Rand. R1000 is approximately equal to US $60.

models unadjusted for covariates (Table S3) show effects of similar direction and magnitude.

### 3.3. Effects of antenatal IPV exposure on neonatal white matter microstructure

#### 3.3.1. Uncinate fasciculus

Infants who were exposed to IPV *in utero* had higher MD in the uncinate fasciculus compared to controls (Table 3), which was further qualified by an IPV-by-sex interaction. Analyses stratified by sex demonstrated that IPV exposure predicted 4% higher uncinate fasciculus MD in boys (2.00 vs. 1.92, $p = 0.002$; Cohen's $d = -0.94$ [95% CI −1.64 to −0.23]), whereas no association was found in girls (1.92 vs. 1.93, $p = 0.297$; Cohen's $d = 0.13$ [95% CI −0.57 to 0.82]). Both the main and interaction effects remained significant after multiple comparison correction. There were no significant associations between IPV or IPV-by-sex interactions on FA. Results from models unadjusted for covariates (Supplementary Table 4 [ST4]) show effects of similar magnitude and direction.

#### 3.3.2. Corpus callosum

Similarly, corpus callosum MD was higher in IPV-exposed infants compared to controls (Table 3). There was also a significant IPV-by-sex interaction; in sex-stratified analyses, IPV exposure predicted 4% higher corpus callosum MD in boys (1.12 vs. 1.08, $p = 0.004$; Cohen's $d = -0.90$ [95% CI −1.60 to −0.20]). In contrast, IPV did not predict corpus callosum MD in girls (1.08 vs. 1.08, $p = 0.389$; Cohen's $d = 0.04$ [95% CI −0.66 to 0.73]). The main effect survived FDR correction for multiple comparisons, although the interaction effect did not. There were no significant associations between IPV or IPV-by-sex interactions on FA.

#### 3.3.3. Corticospinal tract

Finally, corticospinal tract FA was lower in IPV-exposed infants compared to controls (Table 3) and was further qualified by a IPV-by-sex interaction. IPV exposure predicted 9% lower corticospinal tract FA in boys (0.312 vs. 0.342, $p = 0.005$; Cohen's $d = 0.97$ [95% CI 0.26–1.67]), but not girls (0.340 vs. 0.331, $p = 0.400$; Cohen's $d = -0.27$ [95% CI −0.97 to 0.44]). Neither the main effect nor the interaction effect remained significant after FDR correction. There were no significant associations between IPV or IPV-by-sex interactions on MD.

#### 3.3.4. Fornix and Cingulum

There were no significant effects of IPV or IPV-by-sex interactions on FA or MD in the fornix or cingulum.

### 3.4. Sensitivity analyses

Our findings were further supported by sensitivity analyses in which mother-infant pairs who had pregnancy complications including pre-eclampsia, gestational diabetes mellitus, pelvic inflammatory disease and/or high blood pressure were removed. Excluding these infants from the analysis ($n = 11$) did not meaningfully change the associations or effect sizes reported above (Supplementary Tables 5 and 6 [ST5 and ST6]).

## 4. Discussion

This prospective study of South African infants (aged 2–6 weeks) was a novel examination of subcortical brain volumes and white matter microstructure whose mothers had been exposed to IPV during pregnancy. We found tentative evidence for sex-specific effects of maternal antenatal IPV exposure on young infant subcortical volumes: IPV predicted a *smaller* amygdala among females but not males, and a *larger* caudate nucleus in males but not females. No main effects of IPV or IPV-by-sex interactions were observed for the hippocampus, or any other subcortical region. In a subsample ($n = 70$) with diffusion imaging data available, we found robust evidence that infants exposed to IPV *in utero* possessed higher mean diffusivity in the uncinate fasciculus and corpus callosum and lower fractional anisotropy in the corticospinal tract. Additional analyses suggest that these microstructural alterations also vary as a function of infant sex: diffusivity alterations were apparent in males but not females exposed to IPV. All the observed effects remained significant after excluding mother-infant dyads with pregnancy

Table 2
Adjusted associations between maternal IPV exposure and its interaction with neonatal sex on whole brain and subcortical grey matter volumes.

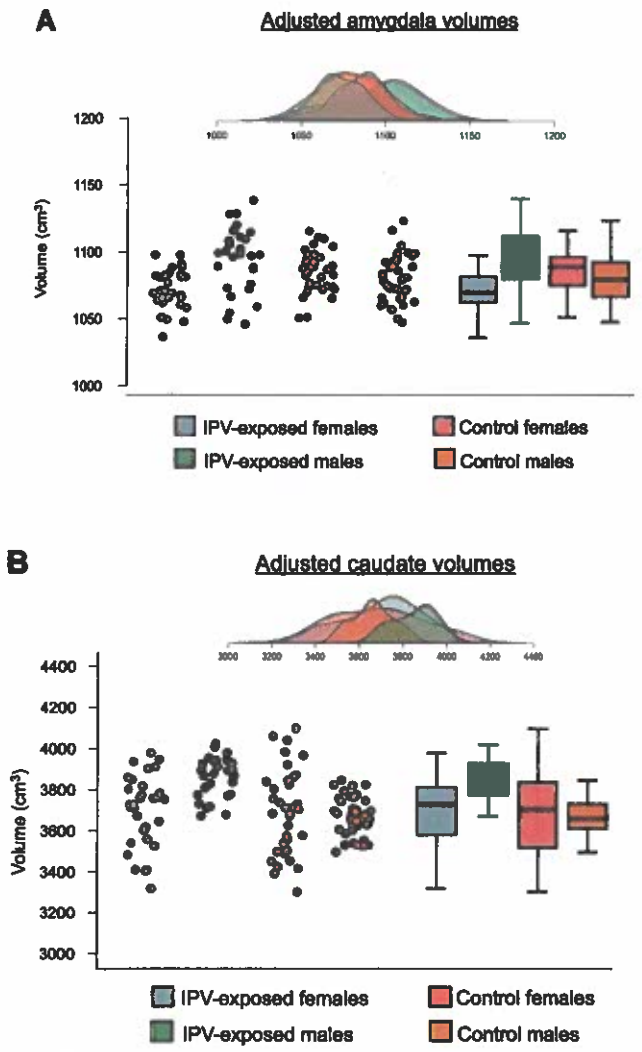


**Fig. 2.** Raincloud plots (jittered data points for all participants, boxplots, and probability distribution of the data) for predicted values of (a) amygdala volume, and (b) caudate volume. For the boxplots, the boxes and the horizontal line inside show the quartiles (1st to 3rd quartile) and the median, respectively. The whiskers denote 1.5 times the interquartile range.

complications. Taken together, our results suggest that volumetric and microstructural brain alterations are observed in IPV-exposed infants shortly after birth, implying that they occur in the intrauterine environment.

Our observations of sex-specific effects of IPV exposure on neonatal amygdala volume builds on previous findings that have reported sex differences in amygdala volume in children and adolescents whose mother's were exposed to antenatal psychological distress. However, the direction of effect was contrary to our initial prediction; amygdala volume was *lower* in IPV-exposed females in comparison to their unexposed counterparts, which contrasts with previous findings of enlarged amygdala volumes reported in older female children and adolescents (Acosta et al., 2019; Buss et al., 2012; Jones et al., 2019; Wen et al., 2017). Interestingly, the pattern emerging from recent studies of neonatal amygdala structural connectivity in relation to higher maternal cortisol is one of enhanced maturation and greater connections in female neonates, and potentially reduced amygdala development in males (Graham et al., 2019; Stoye et al., 2020). Taken together, these results suggest a possible delay in amygdala development in very young infant girls exposed to IPV and possible 'catch- up' growth thereafter, with brain growth patterns implicated in a vast range of psychiatric and developmental conditions (Brouwer et al., 2022). Longitudinal studies of brain development in this cohort will be able to shed light on this hypothesis.

In terms of other subcortical structures, we found no evidence that antenatal IPV exposure was related to variations to hippocampal volume in neonates, in support of previous literature (Buss et al., 2012; Favaro et al., 2015; Marečková et al., 2018), and there were also no effects on the thalamus, putamen, or pallidum. However, the caudate nucleus was found to be 5% larger among males, but not females, exposed to IPV. To our knowledge, this is the first report of caudate nucleus volume being associated with prenatal stress exposure. Alongside a key role in executive functioning, sensory integration, and socio-emotional processing (Choi et al., 2022; Lanciego et al., 2012), caudate structure and function may have important neurodevelopmental implications. For example, larger caudate volumes have been related to impaired problem solving and increased impulsivity in children with autism (Voelbel et al., 2006) and have recently been identified as a marker of severe neurodevelopmental delay within the first 2 years of life (Hüls et al., 2022). Given that our findings did not survive strict correction for multiple comparison testing suggests future studies may select the amygdala and caudate as *a priori* regions of interest in examining the neurological consequences of psychological distress during pregnancy.

Our examination of white matter microstructural integrity found that antenatal maternal IPV exposure was associated with higher MD in the uncinate fasciculus and corpus callosum, and lower FA in the corticospinal tract. Strikingly, only IPV-exposed boys, but not girls, showed changes in diffusivity in these tracts, with very large effect sizes (Cohen's $d = -0.90$ and 0.97). Lower FA/higher MD during development generally points to reduced microstructural integrity, including disrupted glial proliferation and maturation (Yoshida et al., 2013), as well as increased brain water content and decreases in axon density (Lautarescu, Pecheva et al., 2020; Wimberger et al., 1995). However, the direction of effect can also depend on developmental stage (Tamnes et al., 2018). The uncinate fasciculus is part of the temporo-amygdala-orbitofrontal network, and diffusivity alterations within this tract have previously been associated with disorders that are more prevalent in males, such as conduct problems (Passamonti et al., 2012; Sarkar et al., 2013) and autism spectrum conditions (Catani et al., 2016), suggesting that these early microstructural alterations may have adverse developmental consequences. The present study also supports previous literature which documents an association between prenatal psychological distress and changes to the diffusion of two major white matter pathways: the corpus callosum and corticospinal tract (Borchers et al., 2021; McCreary et al., 2016; Zwicker et al., 2013). Overall, our findings provide a picture of disrupted microstructural white matter integrity in tracts that are central to socioemotional functioning in association with IPV exposure during pregnancy, specifically in male infants.

Exposure to IPV is a particularly severe stressor, and intense or prolonged stress during pregnancy increases fetal exposure to stress biomarkers (e.g., cortisol and pro-inflammatory cytokines) which can alter the development of the nervous system (Goldstein et al., 2021). There are plausible biological mechanisms that can help explain how an infant's biological sex may modify the effect of IPV on brain structure and connectivity, with sex chromosomes in the placenta likely to produce sex-specific transplacental signals to the developing brain (Bale, 2016; Carpenter et al., 2017). Male and female fetuses also have different patterns of glucocorticoid expression during development, which may indicate different windows of vulnerability to cortisol exposure (Owen and Matthews, 2003). Further examination of these sex-specific effects may be key to understanding the sex bias in neurodevelopment disorders.

The present study has several strengths including its prospective design and the fact that the mothers and infants were well-characterized from a sociodemographic perspective. Statistical adjustment for plausible confounders that may impact fetal brain development is essential

Table 3
Adjusted associations between maternal antenatal IPV exposure and its interaction with neonatal sex on microstructural measures for white matter tracts.

| | IPV Mean (SD) | Control Mean (SD) | Unstandardized IPV β (SE) | IPV P value | Effect size, Cohen's d (95% CI) | Unstandardized IPV × sex β (SE) | IPV × sex P value | Partial eta-squared (95% CI) |
|---|---|---|---|---|---|---|---|---|
| *Uncinate fasciculus* | | | | | | | | |
| FA | 0.274 (0.028) | 0.282 (0.021) | -0.016 (0.008) | 0.057 | 0.31 (−0.18 to 0.79) | 0.014 (0.012) | 0.241 | 0.02 (0–0.15) |
| MD | 1.958 (0.094) | 1.925 (0.088) | 0.095 (0.029) | **0.002** *§ | -0.37 (−0.85 to 0.12) | -0.124 (0.042) | **0.004** *§ | 0.13 (0.01–0.30) |
| *Fornix* | | | | | | | | |
| FA | 0.318 (0.028) | 0.328 (0.022) | -0.016 (0.008) | 0.064 | 0.41 (−0.09 to 0.90) | 0.010 (0.012) | 0.414 | 0.01 (0–0.12) |
| MD | 1.923 (0.090) | 1.906 (0.069) | 0.043 (0.024) | 0.078 | -0.22 (−0.70 to 0.26) | -0.037 (0.035) | 0.293 | 0.02 (0–0.13) |
| *Cingulum* | | | | | | | | |
| FA | 0.232 (0.024) | 0.240 (0.024) | -0.010 (0.009) | 0.273 | 0.33 (−0.15 to 0.81) | 0.005 (0.013) | 0.695 | 0.01 (0–0.08) |
| MD | 1.773 (0.067) | 1.757 (0.058) | 0.018 (0.021) | 0.387 | -0.253 (−0.73 to 0.23) | 0.004 (0.030) | 0.898 | 0.01 (0–0.08) |
| *Corpus callosum* | | | | | | | | |
| FA | 0.299 (0.024) | 0.311 (0.017) | -0.012 (0.008) | 0.135 | 0.60 (0.11–1.09) | -0.002 (0.011) | 0.846 | 0.01 (0–0.06) |
| MD | 1.095 (0.042) | 1.079 (0.038) | 0.041 (0.013) | **0.002** *§ | -0.42 (−0.90–0.06) | -0.049 (0.019) | 0.010 * | 0.11 (0.01–0.27) |
| *Corticospinal tract* | | | | | | | | |
| FA | 0.327 (0.036) | 0.338 (0.032) | -0.034 (0.012) | 0.008 * | 0.32 (−0.16 to 0.81) | 0.044 (0.018) | 0.016 * | 0.10 (0.01–0.26) |
| MD | 1.580 (0.083) | 1.558 (0.075) | 0.043 (0.024) | 0.074 | -0.29 (−0.77 to 0.20) | -0.029 (0.034) | 0.403 | 0.01 (0–0.12) |

IPV exposure (0 =below threshold; 1 = above threshold), infant sex (male = 0; female = 1) and their interaction, as predictors of neonatal diffusion outcomes. All models are adjusted for household income, maternal HIV status, maternal depressive symptoms, antenatal smoking and alcohol consumption, duration of gestation, and infant birthweight. Positive IPV regression coefficients indicate that IPV exposure is associated with higher diffusion values for that region.
Notes: Diffusion coefficients are averaged across hemispheres ($\times 10^{-3}$ $mm^2/s$).
*Uncorrected $p < 0.05$.
§DTI comparisons to survive multiple comparison correction using the false discovery rate across the 5 regions and 2 metrics, which generated a corrected overall $p$-value of 0.005.
Abbreviations: β, unstandardized beta coefficient; CI, confidence interval; IPV, *in utero* exposure to intimate partner violence; MD, mean diffusivity; FA, fractional anisotropy.

to isolate the specific impact of IPV exposure, with influential prenatal factors often not reported comprehensively in infant neuroimaging studies (Pulli et al., 2019). These include intrauterine alcohol exposure (Archibald et al., 2001; Donald et al., 2015), HIV status (Wedderburn et al., 2022), smoking (Ekblad et al., 2015; El Marroun et al., 2014), and maternal depression (Barnett et al., 2021; Groenewold et al., 2022). The high rates of IPV exposure in this sample (44% of the sample) provide approximately equal group sizes meaning the cohort is well-suited for studying the impact of antenatal IPV. Crucially, scans were conducted at alcohol during pregnancy (27%) compared to the control group (11%). While we have adjusted for prenatal alcohol exposure in all models, we acknowledge that this is only a first-order linear approximation of alcohol effects on brain outcomes that may not fully capture the full extent of the association. Finally, only a sub-sample of neonates had diffusion imaging data available due to movement and technical artefacts (49% of the cohort with structural MRI), which minimized statistical power for detecting IPV-by-sex interactions on white matter microstructure. Moreover, while TBSS is regarded as a standard

growth trajectories. These findings have important implications for understanding whether antenatal maternal stress via IPV exposure contributes to a sex bias in cognitive, emotional, and behavioural disorders often observed in childhood.

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## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Data Availability

Data cannot be shared publicly because of ethical conditions with which study investigators are obliged to comply. Access to the project data is restricted to nominated investigators approved by the University of Cape Town Human Research Ethics Committee, as per the consent document. Interested, qualified researchers may request to access this data by contacting the Drakenstein Child Health Study (via **lesley.workman@uct.ac.za**) to submit a formal data use request and ensure required ethical approval received prior to use.

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## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2023.101210.

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# Domestic Violence and Pregnancy Fact Sheet

- Homicide is the leading cause of death among pregnant women in the United States.[1]
- 50-75% of women abused before pregnancy are abused during pregnancy.[2]
- It is estimated that up to 20% of pregnant women experience violence during their pregnancy, making it more common than gestational diabetes or preeclampsia, conditions routinely screened for in pregnant women by physicians.[3]
- 77% of pregnant homicide victims are killed early (during the first trimester) in the pregnancy.[4]
- Autopsy reports show that of the 34 deceased pregnant women examined in the District of Columbia between 1988 and 1996, 38% were murdered.[5]
- Women with unintended pregnancies are two to four times more likely to experience physical violence than women with planned pregnancies.[6]
- Women who reported having experienced abuse in their relationship were also more likely to report that their pregnancies were unintended.[7]
- In some cases, unintended pregnancies resulted directly from physical violence that included marital rape.[8]
- Among those women whose pregnancies were intended, 5.3 reported abuse around the time of the pregnancy, compared with 12.6% of whose pregnancy was mistimed and 15.3% whose pregnancy was unwanted.[9]
- Pregnant adolescents (ages 13-17) have an elevated risk of violence from their partners when compared with pregnant adults (ages 18+).[10]
- An estimated 5.4%-37.6% of all adolescent girls experience physical violence while pregnant or during the year preceding delivery.[11]
- Pregnant homicide victims are more likely to be killed with a gun than nonpregnant homicide victims.[12]
- Women who are abused during pregnancy are more likely to delay entry into prenatal care.[13]

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- Intimate partner violence during pregnancy is linked to depression, substance abuse, smoking, anemia, first and second trimester bleeding, less than optimal weight gain, and unhealthy eating patterns.[14]
- Intimate partner violence during pregnancy is associated with a reduction in birth weight.[15]
- Approximately 72% of U.S. women aged 15-44 years receive at least one reproductive health care service annual.[16]
- Only 18% of pregnant women examined at an urgent care triage unit reported having been asked by their physician about intimate partner violence.[17]
- Less than half of health care providers routinely screen for domestic violence or sexual assault[18] yet 47% of intimate partner homicide and attempted homicide victims were seen by health care professionals in the year prior to their deaths or attacks.[19]

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## How Porn Can Promote Sexual Violence

As few as 1 in 3 and as many as 9 in 10 porn videos depict sexual violence or aggression. That's especially concerning, considering that research indicates that these sexually violent narratives can bleed into consumers' attitudes and behaviors.

Trigger warning: This article contains descriptions of sexual violence. Reader discretion is advised.

Did you know the average internet user spends over 40% of their waking hours online?[1] 40%! That's a lot of time on the internet.

Whether streaming Netflix, scrolling social media, or sending memes, everything we consume communicates a message. So, does the media we consume online actually have an effect on us? Or do we passively consume it and then quickly forget? Countless researchers have been asking similar questions since the dawn of the internet. According to their findings, the short answer is 'yes.' Our internet consumption does affect the way we think and behave.

From poorer mental health[2] to more negative body image,[3][4] studies are increasingly clear that what people consume online has the potential to affect them—both positively, and negatively. So, with that in mind, considering that an estimated 91.5% of men and 60.2% of women consume porn, let's take a few moments to examine how porn may be affecting its consumers.[5]

### Violence in porn

To start, let's talk about the content of porn. Is it mainly sensual but explicit sex, or is extreme content common? One team of researchers had the same question.

They analyzed hundreds of the most popular porn scenes and found that 88.2% contained physical violence or aggression. They also found that 48.7% contained verbal aggression.[6]

Another study estimated that nearly 40% of videos analyzed on Pornhub contained visible aggression or violence, while 25% contained verbal aggression.[7] And yet another study suggested that 45.1% of Pornhub videos and 35.0% of videos on XVideos depicted violence or aggression.[8] And as each of these studies agreed, women were almost always the targets.

Even by the lowest estimate, that still means that more than 1 in every 3 porn videos depicts sexual violence or aggression.[9] In fact, according to a study that analyzed porn titles alone, 1 out of every 8 titles suggested to first-time users on porn sites described acts of sexual violence.[10]

Some studies examine violence in porn by analyzing the content of porn videos. Other studies estimate violence prevalence by asking consumers how often they see certain behaviors in the porn they watch.

For example, a recent Australian study found that 70% of young people reported frequently seeing men portrayed as dominant. 34% frequently saw women being called names or slurs. 11% reported frequently seeing violence or aggression toward women that was nonconsensual. Another 13% of young people reported seeing aggressive nonconsensual sex 'occasionally' when watching porn. Together with other data, the study found that 1 in 4 young people had repeated exposure to depictions of violent, nonconsensual sex within the last year.[11]

The amount of violence shown in porn is troubling. What is perhaps even more disturbing are the portrayed reactions to that violence. One study found that 95% of the targets of violence or aggression in porn appeared either neutral or appeared to respond with pleasure.[12]

In other words, porn is sending the message that sexual violence is just a part of sexual pleasure.

**Porn consumers' attitudes toward sexual violence**

So how does this normalization of sexual violence affect porn consumers? Well, according to neuroscientific studies, with repeated exposure to porn, consumers can become desensitized to some sexual content. They may need to consume increasingly extreme content to get the same rush as before.[13]

By watching scene after scene of dehumanizing or violent content, it can start to seem normal.[1415]In fact, research indicates that porn consumers are more likely to objectify and dehumanize others sexually,[161718] more likely to express an intent to rape,[19] less likely to intervene during a sexual assault,[2021] more likely to victim-blame survivors of sexual assault,[2223] more likely to support violence against women,[2425] more likely to forward sexts without consent,[26] and more likely to commit actual acts of sexual violence.[27282930]

In 2016, a team of leading researchers performed a meta-analysis of quality studies on the connection between porn and sexual violence. After analyzing relevant studies on the topic, they concluded that the research left "little doubt that, on the average, individuals who consume pornography more frequently are more likely to hold attitudes conducive to sexual aggression and engage in actual acts of sexual aggression."[31]

Research suggests that people who consume more pornography tend to enjoy degrading, uncommon, or aggressive sexual behaviors.[32] Another study indicated that

porn plays a role in normalizing sexual violence, which can have devastating real-world consequences. Regular porn consumers might tell themselves that they aren't personally affected by porn. They might also think they're not affected by the toxic messages it perpetuates. However, research suggests otherwise. There is no guarantee that porn won't affect a consumer's attitudes about sex in unhealthy ways.

Porn that millions of people consume every day reinforces the message that sexual violence is a normal part of 'good sex.' This makes it more challenging for many young people to prepare for healthy sexual relationships where they can have their consent and boundaries respected. As our society continues to reckon with rape culture and its perpetuators, we must recognize the role of porn. Porn normalizes sexual violence and contributes to these harmful attitudes.

Saying no to porn and its problematic narratives helps to build a healthier world.

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