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The Intergenerational Effects of Intimate Partner Violence in Pregnancy: Mediating Pathways and Implications for Prevention

Abstract

Intimate partner violence during pregnancy (P-IPV) can have significant adverse impacts on both mother and foetus. Existing P-IPV interventions focus on the safety of the mother and reducing re-victimisation; yet expanding these to address the adverse impact on the foetus has considerable potential for preventing long term negative developmental outcomes. In this review, we draw together evidence on major pathways linking exposure to P-IPV and child outcomes, arguing that these pathways represent potential targets to improve P-IPV intervention efforts. Using a narrative review of 113 articles, we discuss candidate pathways linking P-IPV to child outcomes, as well as their implications for intervention. Articles were identified via keyword searches of social science and medical databases and by inspection of reference lists of the most relevant articles, including recent reviews and meta-analyses. Articles were included if they addressed issues relevant to understanding the effects of P-IPV on child outcomes via six core pathways: maternal stress and mental illness, maternal-foetal attachment, maternal substance use, maternal nutritional intake, maternal antenatal healthcare utilisation, and infection. We also included articles relevant for linking these pathways to P-IPV interventions. We conclude that developing comprehensive P-IPV interventions that target immediate risk to the mother as well as long-term child outcomes via the candidate mediating pathways identified have significant potential to help reduce the global burden of P-IPV.

Keywords: intimate partner violence, pregnancy, prenatal development, maternal mental health, substance use, stress

The Intergenerational Effects of Intimate Partner Violence in Pregnancy: Mediating Pathways and Implications for Prevention

Defined as physical, sexual, or psychological harm by a current or former partner or spouse, intimate partner violence during pregnancy or 'prenatal IPV' (P-IPV) represents a significant global challenge. It is estimated that 13.8% of women are exposed to physical violence during pregnancy, 8% to sexual abuse, and 28.4% to emotional abuse (e.g. Devries et al., 2010; James, Brody, & Hamilton, 2013). IPV can have significant adverse effects on victims at any time in their life (e.g., Black, 2011) but has special significance during pregnancy because of the added potential harms to the unborn child. It is well known that the prenatal period is one of particular sensitivity to the effects of insults such as infective agents, substance use, stress, poor nutrition, and inadequate healthcare (e.g. Bale et al., 2010; Flinkkilä, Keski-Rahkonen, Marttunen, & Raevuori, 2016; Kim, Bale, & Epperson, 2015; Monk, Georgieff, & Osterholm, 2013). These exposures increase the risk of a range of child developmental outcomes including neurodevelopmental disorders, mental illness, and lower intellectual functioning (e.g., Bale, 2015; Flinkkilä et al. 2016; Kim et al., 2015; Simanek & Meier, 2015; Singer et al., 2016). While P-IPV is known to be associated with many of these same exposures andchild developmental outcomes (e.g., Alhusen, Lucea, Bullock & Sharps, 2013; Devries et al., 2013; Flach et al., 2011; Islam, Broidy, Baird, & Mazerolle, 2017; Martinez-Torteya et al., 2016; Silverman, Decker, Reed, & Raj, 2006), the intervention potential of these apparent links is yet to be fully exploited.

Illuminating the pathways by which P-IPV impacts on the developing foetus and consequent long-term developmental outcomes is critical for informing interventions to ameliorate its effects. At present, P-IPV interventions focus primarily on the immediate safety and well-being of the expectant mother, with only a few additionally considering intergenerational impacts of P-IPV (i.e., the impacts on the unborn child; Howell, Miller-

Graff, Hasselle & Scrafford, 2017). A primary focus on maternal safety and wellbeing is important; however, an exclusive focus on these outcomes misses valuable opportunities to improve outcomes for the developing foetus. This is particularly so because, while P-IPV interventions have made some gains in preventing P-IPV, many randomised controlled trials do not show significant reductions in victimisation (Howell et al., 2017; Van Parys, Verhamme, Temmerman, & Verstraelen, 2014). Sexual P-IPV appears particularly resistant to intervention (Van Parys et al., 2014). Thus, although there are some promising indications, eradicating P-IPV is likely to be a lengthy and challenging process. While P-IPV persists, current interventions leave room for further steps to be taken to at least minimise its effects on the unborn child. As such, P-IPV interventions that include components that aim to ameliorate the negative impacts of P-IPV on the foetus alongside their P-IPV reduction efforts are thus important for reducing its collective harmful effects. Identifying the mediating pathways that link P-IPV to child developmental outcomes will be central to identifying appropriate targets for intervention. An expanded focus of P-IPV interventions that emphasises pathways by which the development of the unborn child can be optimised could have the added benefit of helping to reduce stigma associated with P-IPV interventions.

Research into the pathways that link P-IPV exposure to long term outcomes for the child has, however, remained somewhat fragmented. Partly this is due to the issue of P-IPV effects on child outcomes falling in the gap between the research traditions concerned with violence against children (VAC), violence against women and girls (VAWG), and prenatal exposures. In this review we draw together extant research on the effects of P-IPV on child developmental outcomes. We present the current state of knowledge and provide recommendations for a research agenda that can provide a robust evidence base to best support interventions aimed at minimising the impacts of P-IPV on the long-term health, wellbeing and functioning of the child. In particular, we review core candidate pathways

from P-IPV to child developmental outcomes, highlighting areas for future research as well as opportunities to incorporate existing knowledge into P-IPV interventions.

Method

Our primary research question concerned the pathways by which P-IPV impacts child developmental outcomes and the implications of these pathways for intervention. We focused on psychosocial developmental outcomes, which we defined as encompassing mental health, neurodevelopmental, and behavioural issues. We began by conducting an initial scoping search to determine the main pathways by which P-IPV can impact child developmental outcomes. The scoping search highlighted five key inter-linked pathways: maternal stress and mental illness, maternal-foetal attachment, maternal substance use, maternal nutritional intake, and maternal antenatal healthcare utilisation. It also identified the heterogeneity of study types with the potential to contribute to illuminating these pathways, including randomised controlled trials, epidemiological studies, genetic and epigenetic human studies, and animal studies. A second scoping search was used to identify the number and nature of previous studies evaluating P-IPV interventions. Following the initial identification of mediating pathways and P-IPV interventions a narrative review was used to synthesise and connect these two literatures.

We elected to use a narrative rather than a systematic review methodology because while the latter is valuable when specific, well-researched questions using shared paradigms can be defined, illuminating the issue of the effects of P-IPV on child developmental outcomes draws on a diversity of research areas and methodological designs. Further, narrative reviews can be particularly valuable for identifying gaps and overlooked issues in the literature and for suggesting future research. Given the greater flexibility for including evidence diverse in terms of research area and methodology afforded by narrative reviews

(e.g., Guedes, Bott, Garcia-Moreno, & Colombini, 2016) and our goal of developing a new research agenda, we thus determined that a narrative review was best suited to our aims.

As we used a narrative review methodology, our search terms and inclusion/exclusion were not strictly defined a priori but evolved in a flexible manner as required to accommodate the heterogeneity of research approaches that emerged from early findings. We targeted English-language publications. Databases searched included PsycINFO, Medline, PubMed, Web of Science, and Google Scholar. Searches were typically of the form: (intimate partner violence OR domestic violence) AND (pregnancy OR prenatal OR antenatal OR perinatal) AND ("pathway"), where "pathway" represented keywords related to one of the five identified pathways. Separately, we searched for articles which met search criteria of the form: ("pathway") AND ("child outcome"), where "pathway" again represents keywords related to one of the five identified pathways and "child outcome" represented post-natal and long-term outcomes such as gestational age at birth, weight at birth, weight for gestational age at birth, neurodevelopmental conditions such as ADHD or autism spectrum disorder, and mental health issues such as externalising problems, internalising problems and psychosis. We conducted separate searches for, on the one hand P-IPV and the prenatal exposures representing the candidate pathways and, on the other, the same prenatal exposures and child outcomes because our initial scoping search identified only a handful of articles which addressed all the intersection of P-IPV, exposure, and outcome. Finally we conducted a search of the form: (intimate partner violence OR domestic violence) AND (pregnancy OR prenatal OR antenatal OR perinatal) AND (intervention OR prevention) in order to identify studies evaluating prevention programmes for P-IPV. Our database searches were supplemented by examining the reference lists and citing articles of key papers.

Several previous reviews and meta-analyses were identified in relation to specific sub-topics of our over-arching research question. These represented priority studies to include in our narrative review. No specific restrictions were placed on study methodology but decisions regarding which primary studies to include were typically based on identifying the studies with the strongest research designs from a given area. In addition, all else being equal, recent studies were prioritised for inclusion over older studies.

P-IPV and Child Developmental Outcomes

Many studies have documented an association between P-IPV and pregnancy complications and birth outcomes (e.g., Donovan, Spracklen, Schweizer, Ryckman, & Saftlas, 2016; Hillis, Mercy, Amobi & Kress, 2016; Shah & Shah, 2010; Shamu, Abrahams, Zarowsky, Shefer, & Temmerman, 2013). In a meta-analysis of 50 primary studies, for example, Donovan and colleagues (2016) reported that women who experienced P-IPV were approximately twice as likely to give birth prematurely and to an infant with a low birth weight. These birth outcomes are known, in turn, to be associated with a range of child developmental outcomes such as mental health, behavioural, emotional and neurodevelopmental problems (e.g. Arpi & Ferrari, 2013; Cassiano, Gaspardo, & Linhares, 2016; Class et al., 2014; Fjørtoft et al., 2015; Groen-Blokhuis et al., 201; Losh et al., 2012; Patton, Coffey, Carlin, Olsson, & Morley, 2004; Talge et al., 2006)

In contrast, only a small number of studies have directly addressed the effects of P-IPV exposure on nascent dimensions of psychosocial functioning in infancy and their developmental outcomes in childhood, adolescence and beyond. Most of these studies have identified an association between P-IPV and child psychosocial dimensions, including insecure attachment, internalising problems, externalising problems, dissociative symptoms, and language and neurological delay (Flach et al., 2011; Lannert et al., 2014; Levendosky, Leahy, Bogat, Davidson, & von Eye, 2006; Martinez-Torteya, Bogat, Levendosky, & Von

Eye, 2016; Udo, Lewis, Tobin, & Ickovics, 2016; Yalch, Black, Martin, & Levendosky, 2016). Martinez-Torteya and colleagues (2016), for example, used a prospective longitudinal design to examine the associations between P-IPV exposure and internalising and externalising problems in 119 ten-year old children. Controlling for post-natal exposure to violence, they found associations between P-IPV and child-reported internalising (β=0.28) and externalising problems (β=0.28) and mother-reported child externalising problems (β=0.29). Further studies are needed to rule out alternative explanations for the association, especially genetic confounding; however, current evidence certainly suggests that the links between P-IPV exposure and long-term psychosocial health deserve greater attention.

Pathways from P-IPV to Child Outcomes

A number of potential pathways linking P-IPV exposure to long term child developmental outcomes can be identified. Major candidate pathways include: maternal stress and mental illness, foetal attachment, health-related behaviours including substance use, nutrition, and healthcare utilisation, and infection. As we discuss below, these pathways are not only inter-connected but likely interactive in their effects on child developmental and behavioural outcomes.

Maternal Stress and Mental Illness

Maternal stress is arguably the best-researched pathway linking P-IPV to child outcomes. There is little doubt that P-IPV is stressful for an expectant mother, making P-IPV an important cause of prenatal stress exposure. At the same time, decades of research on prenatal development make it clear that prenatal stress is an important risk factor for child developmental outcomes such as ADHD, lower intellectual functioning, externalising problems and internalising problems (e.g., Bergman, Sarkar, O'connor, Modi, & Glover, 2007; Betts, Williams, Najman, & Alati, 2014; Laplante et al., 2004; O'connor, Heron, Golding, & Glover, 2003). Through triangulation across animal studies, human

epidemiological, and 'natural experiment' methodologies, there is reasonable confidence in the field that there are causal and practically significant effects of prenatal stress exposure on the child (e.g. Monk et al., 2013; Ping et al., 2015). In particular, complementing the suite of epidemiological associations, studies such as those from *Project Ice Storm* (King & Laplante, 2005) and the *Iowa Flood Study* (Ping et al., 2015) have evaluated the effects of prenatal stress exposures independent of maternal characteristics to rule out potential confounding factors. For example, Laplante and colleagues (2004) analysed data on 150 children whose mothers were affected by a major ice storm while pregnant and found that children prenatally exposed to higher levels of objective stress deriving from the event had poorer cognitive development at age 2. Objective stress accounted for 11.4%, 12.1% and 17.3% of the variation in Bayley mental development index (MDI), productive language, and receptive language scores respectively.

Prenatal stress is proposed to be linked to child outcomes primarily via the activation of the maternal hypothalamic-pituitary-adrenocortical (HPA) axis, resulting in foetal exposure to glucocorticoids (Bale, 2015; Cao-Lei, Laplante, & King, 2016; Kim et al., 2015; Tobon, Stransky, Ross, & Stevens, 2016). In turn, foetal cortisol exposure is assumed to lead to changes to the foetal HPA axis, including epigenetic modification of genomic sites relevant for HPA axis functioning which ultimately put the child at greater risk of mental health difficulties (e.g. Sosnowski et al., 2018). Indeed, P-IPV exposure has been associated with methylation in the promoter binding site of the NR3C1 gene, which codes for glucocorticoid receptors, a key component of the stress response system (Radtke et al., 2011). This 2011 study by Radtke and colleagues compared youth (n=25, aged 10-19) whose mothers were exposed to P-IPV to those whose mothers were exposed to IPV before or after their pregnancy. Though this particular study was small, its results are consistent with findings from the general prenatal stress literature (Sosnowski et al., 2018). Foetal exposure

to cortisol is also thought to be augmented by the effects of stress in suppressing the activity of the HSDB11B2 enzyme, which normally converts maternal cortisol to inactive corticosterone before it is transferred to the foetus (see Cao-Lei et al., 2016). Thus, under stress the foetus is also less insulated from cortisol exposure. Collectively, early calibrations of the HPA axis resulting from prenatal stress exposure are assumed to put the child at risk of poorer developmental outcomes in the psychosocial functioning domain, with HPA axis dysregulation been implicated as a risk for mental health problems (Baumeister, Lightman, & Pariente, 2014).

However, given that maternal cortisol levels may be only weakly associated with prenatal mood, recent work has also begun to explore the involvement of additional pathways mediating the effects of prenatal stress on child developmental outcomes (e.g., see Glover, 2014). Pathways mediated by the immune system are under investigation, with some studies reporting increased levels of inflammatory markers in pregnant women affected by stress (e.g., Coussons-Read, Okun & Nettles, 2007). Other studies have suggested that down-regulation of placental monoamine oxidase A (MAO-A) due to stress exposure may play a role, resulting in greater foetal exposure to serotonin and impacts on neurodevelopment (Bonnin et al., 2011; Blakeley, Capron, Jensen, O'Donnell & Glover, 2013). Finally, a role for noradrenaline has been suggested, with some evidence from animal studies that it contributes to the down-regulation of HSDB11B2 (Mairese et al., 2007).

Stress pathways are also relevant when considering the effects of P-IPV on child outcomes through maternal mental disorders. P-IPV has shown a robust association with maternal mental illness during pregnancy; an association that likely reflects a reciprocal relation between violent victimisation and mental health problems (Devries et al., 2013; Howard, Oram, Galley, Trevillion, & Feder, 2013). Evidence suggests that prenatal maternal mental illness may be associated with and affect child outcomes via the activation of the

same stress responses described above (O'Connor, Monk, & Burke, 2016). Flach and colleagues (2011) provided evidence for maternal mental illness as a mediator of the link between P-IPV and child behavioural outcomes. Using data from the Avon Longitudinal Study of Parents and Children (ALSPAC), they found that P-IPV was associated with higher levels of antenatal depression (OR=4.02 after adjustment for confounders) and child behavioural problems at 42 months (OR=1.52 after adjustment for confounders). Entering antenatal depression in the model reduced the odds ratio for behavioural problems to non-significance, leading the authors to conclude that depression mediates the association between P-IPV and child behavioural outcomes.

However, significant gaps remain in the evidence linking stress associated with P-IPV with child outcomes. Among the wealth of studies on the effects of prenatal stress and maternal mental illness, few studies include a measure of IPV (see e.g. Bergman et al., 2007 and Radtke et al., 2011 for exceptions). This is important because although studies of prenatal stress exposure are indicative of the likely impacts of P-IPV, IPV can be more chronic, unpredictable and severe than commonly studied stressful exposures during pregnancy (e.g., see Martinez-Torteya et al. 2016 for a discussion). Thus, the impact of P-IPV on child developmental outcomes could be greater than estimates extrapolated from the general literature on prenatal stress might suggest. Second, though HPA axis functioning and epigenetic modification of HPA (Martinez-Torteya et al., 2016; Radtke et al., 2011) have both been linked to P-IPV exposure, these measures were taken in late childhood or adolescence. Linking P-IPV with early post-natal measures of HPA axis functioning and associated epigenetic modifications will be essential to rule out post-natal confounds. Addressing these evidence gaps would further strengthen the arguments for prioritising P-IPV prevention as well as maternal stress and mental illness screening and treatment in exposed women. Finally, several potential modifiers of the effects of stress have been

identified in the general stress literature. These include: stressor timing, chronicity, type, and subjective impact, as well as the sex of the child (e.g. Bale, 2015; Kim et al., 2015; Tobon et al., 2016). Understanding the implications of these factors in the context of P-IPV will be important for targeting and enhancing P-IPV interventions.

Maternal-foetal Attachment

A pathway likely closely related to maternal stress and mental health is mother-foetus attachment. Mother-foetus attachment is typically operationalised in terms of how an expectant mother mentally represents the foetus as well as their role as a mother. On average, women who experience IPV during pregnancy express less adaptive maternal representations of their foetus and of themselves as mothers (e.g. Huth-Bocks et al., 2004; Pires de Almeida et al., 2013; Quinlivan & Evans, 2005; Theran, Levendosky, Bogat, & Huth-Bocks, 2005). For example, based on structured interview data, Huth-Bocks et al. (2004) found that IPV-exposed pregnant women were more likely to be display 'disengaged' or 'distorted' foetal representations, while unexposed women were more likely to display 'securely attached' or 'balanced' representations.

Disrupted mother-foetal attachment during pregnancy is important because of its long-term impact on bonding with the child (Foley & Hughes, 2018). Mother-foetal bonding is considered the foundation for post-natal mother-infant bonding, with weak or maladaptive bonding prenatally increasing the risk for poor attachment post-natally (e.g. Benoit, Parker & Zeanah, 1997; Huth-Bocks et al., 2011; Zeitlin, Dhanjal, & Colmsee, 1999). Early attachment problems have, in turn, been linked to a range of developmental outcomes for the child, with less secure forms of attachment (ambivalent, avoidant, disorganised) associated with outcomes such as child internalising and externalising problems (e.g. Groh et al., 2017).

However, in order to support mother-foetus attachment in the context of P-IPV, more needs to be known about how P-IPV affects foetal bonding. Several factors likely contribute. Mothers who experience P-IPV may have more trouble bonding with their child due to stress or mental illness associated with victimisation (e.g. Kita et al., 2016). Mothers may also come to resent their unborn child if they perceive that the behaviour of the abusive partner is related to hostility towards the unborn child (e.g. Zeitlin et al., 1999). This could also occur when the abusive partner is the father and the unborn child evokes unpleasant reminders of the abusive partner. Similarly, mothers whose own attachment-related emotions and cognitions are affected by exposure to dysfunctional relationships or interpersonal trauma may lack the appropriate internal working models to successful form adaptive attachment relationships their foetus (e.g., Schwerdtfeger & Goff, 2007). Finally, the association may be explained by a higher proportion of unwanted pregnancies in women who experience P-IPV as a result of coercion or lack of control over contraception (e.g., Pallitto et al., 2013). The above explanations require further testing and disentangling in future research.

Substance use

Exposure to P-IPV is associated with increased substance use in pregnancy (e.g. Alhusen, Lucea, Bullock, & Sharps, 2013; Bailey & Daughtery, 2007; Cheng, Salimi, Terplan, & Chisolm, 2015; Martin, Beaumont, & Kupper, 2003; Udo et al., 2016). The effect is typically explained in terms of a method of coping with the psychological and physical distress of IPV victimisation (e.g. Devries et al., 2014; Taillieu & Brownridge, 2010). Thus, substance use is likely to be closely linked to the above-outlined stress and mental illness pathway.

Effects of substance use on the developing child depend on the chemical composition of the drug and- to some extent- its method of delivery. However, almost all commonly used

illicit and legal drugs are thought to have teratogenic effects (e.g. Flak et al., 2014; Huizink & Mulder, 2006; Wright, Schuetter, Tellei, & Sauvage, 2015; Richardson, Hester, & McLemore, 2016; Singer et al., 2016). Particularly well-characterised are the effects of prenatal alcohol exposure, which represents the leading cause of preventable intellectual disability (American Academy of Pediatrics Committee on Substance Abuse and Committee on Children with Disabilities, 2000). However, evidence also implicates other drugs including tobacco, cannabis, opiates and amphetamines and other psychostimulants in adverse developmental outcomes (e.g. Flak et al., 2014; Huizink & Mulder, 2006; Wright et al., 2015; Parris, 2016; Richardson et al., 2016).

There are some challenges in attributing causality to the use of particular substances and in ultimately tracing this back to P-IPV. These include the commonality of poly-drug use (making individual drug pathways difficult to disentangle), stigma surrounding admitting drug use during pregnancy (e.g. Glass & Mattson, 2016), and the presence of confounding with psychosocial characteristics of the mother and family (e.g. D'Onofrio et al., 2014; Singer et al., 2016). Moreover, no study has to our knowledge yet examined whether substance use is a mediator of the effects of P-IPV on the child. Demonstrating mediation of P-IPV effects by substance use in a well-controlled longitudinal design that includes both objective and self-report measures of substance use (e.g. from hair samples; Lendoiro et al., 2013) would provide a strong argument for ensuring that substance use is screened for and targeted in P-IPV interventions.

Maternal nutrition

A second health-related behaviour pathway from P-IPV to child outcomes is maternal nutrition during pregnancy. The role and importance of this pathway is more ambiguous than that of substance use. Many authors have argued that P-IPV is likely to impact nutrition

through stress-related factors such as loss of appetite, comfort eating, or stress-induced changes in metabolism (e.g. Alhusen, Geller, Dreisbach, Constantoulakis, & Siega-Riz, 2017), but the empirical evidence is unclear. Of the small number of studies to examine the association between gestational weight and IPV, some report an association with inadequate weight gain, some with excessive weight gain, and some with neither. Part of the issue may be – as Alhusen and colleagues. (2017) argue - that most studies do not adequately control for potential confounds such as poverty. Perhaps more importantly, however, weight and its derivations (e.g., BMI) do not provide information on the specific nutritional deficiencies and imbalances that are likely to matter most for healthy prenatal development. These have been little-studied in the context of P-IPV.

The neglect of more direct measures of maternal nutrition in the context P-IPV is unfortunate. Despite some inconsistencies (e.g., Christian et al., 2016; Leung et al., 2011) a general link has been established between nutrient deficiencies and imbalances during pregnancy and infant outcomes. Here, animal studies and human epidemiological, randomised controlled trials and natural experiment studies converge in suggesting that an insufficiency of key macro- and micro-nutrients during pregnancy can adversely affect offspring neurodevelopment (e.g. Monk et al., 2013). Animal studies have, for example, identified biological effects of caloric restriction that are similar to those of prenatal stress, including increased cortisol (Lingas, Dean, & Matthews, 1999). Trials in humans have meanwhile shown positive effects of supplementation of key nutrients such as DHA during pregnancy on offspring neurocognitive performance in early childhood (Helland et al., 2003). Similarly, balanced protein energy supplementation programmes have shown promising effects on birth outcomes (e.g. Stevens et al., 2015) and by extension, for neurocognitive outcomes.

The role of nutrition in mediating the effects of P-IPV on the offspring should, we thus argue, be further explored as a number of gaps remain. It is unfortunate, for example, that in studies linking P-IPV to low birth weight (e.g., see Donovan et al., 2016 for a review), maternal nutrition has not been investigated as a mediator. Further, maternal nutrition represents a pathway that may be particularly amenable to intervention, given past success of programmes such as those designed to encourage prenatal folic acid intake. Future research in this area should focus on assessing not just maternal weight but also specific nutritional intake and absorption in the context of P-IPV. Moreover, as nutritional intake may interact with stress, affecting not only food choices but also nutrient absorption and metabolism (Coe et al., 2007), obtaining biomarkers of nutrition that reflect the prenatal milieu will be particularly important. As such, approaches combining food frequency questionnaires and assays for biomarkers of nutritional status (e.g., using dried blood spots) may hold particular potential for illuminating the role of nutrition in mediating the effects of P-IPV on the child (e.g., Hoeller et al., 2016).

Antenatal healthcare utilisation

A final health-related behaviour pathway by which P-IPV may affect the unborn child concerns receipt of adequate healthcare during pregnancy. The World Health Organisation (WHO) recommends that women attend at least 4 antenatal visits, but the actual number of visits varies considerably across and within countries. Those who experience P-IPV are more likely to initiate antenatal care later or to attend fewer appointments overall (e.g., Bailey & Daugherty, 2007; Chambliss, 2008; Goodwin et al., 2000; Islam, Broidy, Baird, & Mazerolle, 2017; Rahman, Nakamura, Seino, & Kizuki, 2012; Subramanian et al., 2012). Reasons may include restriction of access to healthcare by abusive partners (e.g. Taggart & Mattson, 1996; Dietz et al., 1997; Koski et al., 2011; Islam et al., 2017; Zakar et al., 2012), fear of exposing physical signs of IPV to medical practitioners (Dietz et al., 1997; Taggart & Mattson, 1996),

and isolation of women from support systems which may be a critical source of information or practical support in prompting and facilitating the uptake of antenatal care. There is currently no strong evidence that mental illness associated with P-IPV can explain healthcare under-utilisation (Bitew et al., 2016), but the scarcity of studies with evidence bearing on the question means that this possibility cannot currently be ruled out.

Antenatal healthcare uptake matters because women who attend fewer antenatal appointments experience more pregnancy complications and adverse birth outcomes, both of which are associated with later child developmental outcomes (e.g. Asundep et al., 2014; Gumede, Black, Naidoo, & Chersich, 2017; Raatikainen, Heiskanen, & Heinonen, 2007; Tucker, Ogutu, Yoong, Nauta, & Fakokunde, 2010). By failing to attend antenatal appointments, women also miss opportunities to address other pathways that link P-IPV with poorer child outcomes (e.g., infection, nutrition, mental illness, substance use disorders and injury). Contact with health services at this juncture provides a key opportunity to address IPV, provided that there are appropriate referral pathways in place (e.g. Chisholm et al., 2017). For some women this may be one of the few times in their life where they have health service contact.

Infection

Effects of P-IPV on child outcomes are also likely to be mediated by health problems, with infection during pregnancy being of particular concern. Women exposed to P-IPV are at substantially increased risk of urinary tract, kidney and sexually transmitted infections (Silverman, Decker, Reed, & Raj, 2006). Silverman and colleagues (2006), for example, compared women exposed versus not exposed to P-IPV in the US-based Pregnancy Risk Assessment Monitoring System (PRAMS) study of n=118,579 women, and reported an odds ratio of 30 for kidney/urinary tract infection.

One reason that women who experience IPV are more likely to contract an infection is that stress compromises immune function, increasing vulnerability to novel infectious agents and reducing both vaccine response and control of latent infections (e.g., Simanek & Meier, 2015). However, P-IPV can also be related to infections more directly. In particular, sexually transmitted infection risk is related to 'risky sexual practices' associated with P-IPV. For example, in a meta-analysis of 27 studies, Coker (2007) reported that 23 studies showed a significant association between sexual risk taking (there defined as inconsistent condom use, sexual risk taking of women and partners, and partner non-monogamy) and physical IPV. It should be borne in mind, however, that women may report engaging in risky sexual practices not because they themselves prefer to take risks but because their partner has primary control over when and how they engage in sexual practices.

Infection during pregnancy, especially in the second trimester, has been linked to a range of offspring neurodevelopmental and mental health outcomes, including ADHD, schizophrenia spectrum disorders, mood disorders and ASD (see Flinkkilä et al., 2016 and Simanek & Meier, 2015 for reviews). Diverse maternal bacterial and viral infections, including cytomegalovirus, herpes simplex, influenza, tonsillitis, and toxoplasma gondii all appear linked to these child outcomes, suggesting mechanisms that transcend specific infective agents.

Animal models have helped illuminate these mechanisms by showing that both the transmission of the infection and the maternal responses to infection (especially immunoglobulin antibodies and pro-inflammatory cytokines) can affect brain development (e.g., Estes & McAllister, 2016; Simanek & Meier, 2015). Infections may also indirectly affect risk of neurodevelopmental and affective disorders by programming an inflammatory immune phenotype. At the same time, maternal infection is thought to contribute to prenatal programming of the HPA axis. Similar to the effects of psychological stress discussed above,

maternal infection can inhibit the production of HSDB11B2 and so increase exposure to cortisol (see Simanek & Meier, 2015). This makes infection in the context of P-IPV a situation of particular risk for adverse offspring outcomes. Finally, alongside these general mechanisms, particular infective agents can have specific effects on neurodevelopment, for example, in the cases of cytomegalovirus (CMV) and zika virus (e.g. Navti et al., 2016).

Like many prenatal exposures related to P-IPV, no study has to our knowledge yet examined the extent to which infection mediates the association between P-IPV and child outcomes. However, unlike exposures such as stress, substance use, and diet which are likely to be chronic across pregnancy, infection can occur for a more circumscribed period. This is important because timing of infection appears to be an important determinant of outcome with the second trimester representing a time of particular risk (e.g. Mednick et al., 1994). Future studies could thus explore the utility of screening for common infections in women who may be experiencing P-IPV, especially sexual P-IPV, especially in the prenatal phases of highest risk.

Cross-Cutting Issues and Recommendations for Future Research

We have argued that there are a multitude of inter-connected and potentially interacting pathways linking P-IPV to child psychosocial outcomes. For example, stress associated with P-IPV may impact on health behaviours, foetal attachment, and infection susceptibility while also potentially moderating the effects of nutritional deficits and infection. These mediators could act at various points along multiple pathways from P-IPV. For example, stress could impact foetal attachment as a secondary mediator directly, or indirectly via increasing the risk of mental illness or substance abuse. At the same time, healthcare under-utilisation can reduce the likelihood that mental and physical health issues associated with P-IPV including depression and anxiety, substance use, nutrition, and infection are addressed in a timely manner. However, studies of the effects of P-IPV and of

prenatal exposures have traditionally focused on specific effects and neglected the broader links within and between the pathways that connect P-IPV to child outcomes. Integrative analyses of how factors such as stress, attachment, health behaviour and health connect and interact as mediators of the effects of P-IPV on child outcomes will be important for advancing knowledge in this area and optimising interventions.

A second issue concerns study design for the attribution of causality. Studies of P-IPV exposure are primarily observational, leaving open the possibility that associations between P-IPV and child outcomes reflect the effects of confounders that contribute to both risk of P-IPV exposure and child outcomes. An example is the transmission of genes from mother or father to child that influence risk both of P-IPV and of child psychosocial problems. For example, a father who perpetrates P-IPV may pass on a genetic risk for externalising behaviour. This kind of genetic confounding via passive gene-environment correlation is a common concern in studies seeking to illuminate the effects of prenatal exposures (e.g., D'Onofrio et al., 2014). Another challenge to causality attribution is the strong continuity of IPV into the post-natal period (e.g., Flach et al., 2011), making it difficult to separate the effects of P-IPV from post-natal violence exposures where the latter could include IPV effects mediated through the mother, the child bearing direct witness to IPV or the child themselves becoming a victim. Taking the example of maternal-foetal attachment, owing to the stability of IPV, it is difficult to disentangle the effects of prenatal IPV on post-natal attachment mediated via foetal attachment from the concurrent effects of post-natal IPV on post-natal attachment. Similarly, given that attachment is a dyadic construct driven by both mother and child, P-IPV effects via maternal attachment are also difficult to disentangle from the effects of child exposure to domestic violence on mother-child attachment (e.g., Carpenter & Stacks, 2009). Greater use of matching-based, sibling-comparison, surrogacy-

based, and intervention designs can help address issues surrounding the attribution of causality regarding the pathways discussed in this review.

Another issue concerns the locations in which research on P-IPV and child outcomes research is conducted. Even though P-IPV is an issue in both high and low- and middle-income countries (e.g. Devries et al., 2010; James et al., 2013), research remains heavily focused on high income countries. Expanding the field to include low- and middle- income countries is essential to understand society- and culture-specific linkages between P-IPV and child outcomes and to ensure evidence is available where it is most needed. Societies differ in a number of relevant features including availability and use of particular substances, gender inequality, service availability, family structures and norms around P-IPV. It thus cannot be assumed that the research that has been conducted in high income countries generalises to the range of contexts that are under-represented in this area of research.

A final recommendation concerns research incorporating the above-discussed pathways into P-IPV interventions. Few P-IPV interventions have been rigorously evaluated. Van Parys and colleagues (2014) identified only 9 randomised controlled trials; six in the US, one in Peru, one in China, and one in Australia. The most recent systematic review of P-IPV interventions identified only 17 evaluation studies in totality, with the majority of these being in the high-income countries (Howell et al., 2017). These interventions varied widely in their aims, approach, duration and intensity, mode of delivery, and effectiveness. The majority of interventions targeted the prevention of re-victimisation; however, some also addressed the mental health effects of P-IPV and a small number of interventions addressed risks to the unborn child. Some involved only a single brief session, while others involved multiple sessions with follow-up in the post-natal periods. Intervention components included psychoeducation, counselling, empowerment and safety planning, advocacy, home visitation, and parenting training. Generally, the biggest focus was on the immediate safety of the

mother. Mental health of the mother and/or the well-being of the unborn child were sometimes an additional target of intervention but this was relatively uncommon, especially the latter outcome.

Overall, there was little evidence for the effectiveness of P-IPV interventions, with the majority of interventions failing to show significant reductions on victimisation and related outcomes (Van Parys et al., 2014). One implication is that P-IPV interventions require further modification to improve effectiveness and previous reviews have discussed potentially fruitful strategies (e.g. Howell et al., 2017). However, in addition to the essential focus on reducing victimisation of the mother, integrated interventions that also target the pathways reviewed above may have benefits in minimising harm to the unborn child. Some interventions target some of these pathways already, including maternal mental health (e.g. Taft et al., 2011); health-related behaviours (e.g. Calderon et al., 2008; Eckenrode et al., 2000; El-Mohandes et al., 2008; Joseph et al., 2009); and attachment (Lavi et al., 2015) with at least some evidence of intervention-related improvements on these outcomes. However, at present, comprehensive interventions that address the full suite of possible effects of P-IPV on a woman and her unborn child are generally lacking. Adding brief screens for P-IPV and providing information on services and/or referrals for the range of problems associated with P-IPV that can impact the unborn child would be an appropriate first step to explore and have shown some promise in the broader IPV field (Feder & Sardinha, 2015).

Another key future direction in P-IPV interventions concerns their implementation and delivery. The widespread use of healthcare systems in P-IPV interventions has a number of advantages, such as reduced stigma associated with their utilisation (e.g. Howell et al., 2017). However, as discussed above, women exposed to P-IPV may present at health services later in their pregnancy and attend fewer appointments overall. A dissemination model that exclusively delivers interventions through the healthcare system is liable to miss these most

vulnerable women. Providers may need to explore alternative options to reach those who may be unable or unwilling to participate via health services.

Conclusion

Though there has been little work directly examining the pathways that link P-IPV with child outcomes, evidence points to harmful effects of prenatal P-IPV exposure mediated by maternal stress and mental illness, health-related behaviours, infection, and problems with mother-foetal attachment. The evidence for the harmful effects on exposed children - as well as their mothers - reinforces the idea that addressing P-IPV should be a public health priority. These pathways are, however, only partially reflected in existing P-IPV interventions. Further exploration of the utility of incorporating these as targets of interventions could potentially ultimately lead to significantly reducing the overall negative impact of P-IPV. Future research using rigorous designs that bring together the various mediators of its effects can help inform comprehensive P-IPV intervention approaches that maximise intervention benefits for both mother and child.

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Tables

Table 1: Summary of Key Findings

- Prenatal exposure to maternal intimate partner violence (P-IPV) has important adverse effects on child outcomes
- Key pathways include maternal stress and mental illness, maternal substance use, maternal infection, mother-foetal attachment, maternal nutrition, and maternal healthcare under-utilisation
- Current P-IPV interventions only partly or obliquely address these pathways
- Expanding the scope of current P-IPV interventions to address these pathways has significant potential to ameliorate the effects of P-IPV on the exposed child

Table 2: Implications for policy, practice and research

- More research is needed to examine the pathways by which P-IPV affects children exposed prenatally and how these pathways can be targeted in interventions
- This research should particularly focus on maternal substance, maternal infection, mother-foetal attachment, maternal nutrition, and maternal healthcare under-utilisation, identified as core pathways in the current review
- Current P-IPV interventions could be expanded to target these pathways in order to ameliorate the negative effects of P-IPV on the unborn child