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# Influence of exposure to perinatal risk factors and parental mental health related hospital admission on adolescent deliberate self-harm risk

Nan Hu<sup>1,2</sup> • Jianghong Li<sup>1,3,4</sup> • Rebecca A. Glauert<sup>1</sup> • Catherine L. Taylor<sup>1</sup>

## Abstract

Adolescent deliberate self-harm (DSH) has been found to be associated with a range of bio-psycho-social factors. Simultaneous investigations of these factors enable more robust estimation of the independent effect of a specific risk factor by adjusting for a more complete set of covariates. However, few studies have had the ability to examine all of these factors together. This study used the linkage of population-level de-identified data collections from government agencies to investigate a range of biological, psychological, and social risk factors and their effects on adolescent risk of DSH (with or without suicidal intent). The investigation was undertaken by progressively adjusting for plausible covariates, including fetal growth status and birth order, early familial social factors, parental hospital admissions due to psychiatric disorders or DSH, and parental all-cause death. Conditional logistic regression was used for data analysis. Children's psychiatric history was analysed to examine the extent to which it may account for the link between the risk factors and adolescent DSH risk. This study identified significant biological and perinatal social risk factors for adolescent DSH risk, including overdue birth, high birth order ( $\geq 2$ ), single or teen/young motherhood, high neighbourhood socioeconomic disadvantage, and parental psychiatric and/or DSH-related hospital admissions. Further, parental psychiatric and/or DSH-related admissions, and children's psychiatric admissions in particular, largely attenuated the effects of the perinatal social risk factors but not the biological factors on adolescent DSH risk. These results highlight the importance of taking joint actions involving both health and social services in the prevention of adolescent DSH.

## Keywords

Deliberate self-harm – Social and biological risk factors – Parental psychiatric disorders – Data linkage

## Introduction

Deliberate self-harm (DSH) refers to behaviours performed intentionally to cause physical or psychological injuries to oneself, regardless of suicidal intent [1]. It is a major public health issue in adolescents and young people, affecting 17% of those aged between 15 and 24 years in Australia [2], similar to the prevalence of DSH in other countries [3]. Additionally, DSH in adolescence is a strong predictor for suicidal death in adulthood [4].

Research has shown that 12–20% of all DSH episodes result in hospital admissions due to the urgent need for medical treatment [5, 6]. Previous studies show that adolescents with DSH-related hospital admissions tended to have higher suicidal intent, poorer social support, more violent and disruptive behaviours, and an increased use of illicit drugs, compared to their peers with DSH behaviours who did not end up being hospitalised [5–8]. Hospitalised DSH behaviours therefore represent the severe end of the spectrum of DSH behaviours.

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Previous studies have shown that DSH is associated with a range of biological, psychological, and social risk factors [9]. However, recall bias, under-reporting due to perceived stigma, the lack of a clinically validated diagnosis of psychiatric disorders and DSH have limited the capacities of survey studies to examine the influence of important perinatal exposures on the subsequent risk of DSH in adolescence. The linking of administrative data has begun to demonstrate great potential for understanding the complex aetiologies of DSH, due to the advantages that data linkage can offer [10]. First, study samples are drawn from whole populations to minimise selection bias and improve the estimation with a large sample size. Second, data are collected on a routine basis over many years, capturing risk exposures during early life stage such as perinatal period. Third, data linkage is sourced from hospital records, whereby diagnosis is clinically verified for psychiatric conditions. Fourth, data linkage assembles life-course events more precisely in chronological order, thus establishing the time sequence for making causal inferences. Fifth, data linkage enables genealogical linkage for the investigation of the intergenerational effect of parental risk factors, which is difficult to capture in surveys.

Previous studies based on linked administrative records have identified three groups of risk factors for DSH behaviours. The first group is fetal growth restriction and high birth order. Low birth weight (i.e. <2.5 kg) has been identified in several studies to be a risk factor for suicide in early adulthood [11, 12]. High birth order has been linked with increased risk of DSH [13–15]. The second group is familial social disadvantage, which plays an important role in the developmental pathways to DSH behaviours. These factors mainly include teenage motherhood [11, 12, 15], parental socioeconomic disadvantage characterised by low educational attainment or unemployment and unskilled occupational status, and parental marriage breakdown [13, 14, 16]. The third group of risk factors includes the familial history of psychiatric disorders and/or DSH behaviours. It has been found that children of parents with psychiatric disorders or DSH behaviours are at increased risk of having DSH behaviours [13, 17, 18].

However, the majority of these studies focussed on DSH among young people or adults, rather than specifically on adolescents aged between 10 and 19 years. Previous studies have clearly demonstrated that adolescence is the period where DSH behaviours start to develop and peak, followed by a gradual decrease in adulthood [19, 20]. Additionally, unlike DSH behaviours during adulthood, DSH behaviours among adolescents are usually non-suicidal [21]. However, without appropriate interventions, adolescents with DSH behaviours are much more likely to have suicidal attempts during adulthood than those without DSH behaviours [4]. Therefore, it is important to examine the effects of the major risk factors mentioned above specifically for adolescent DSH behaviours, which may provide insights into the bio-psycho-social origins of DSH behaviours.

To date, few studies have used linked data to investigate all the three groups of risk factors together. The lack of such investigations in existing literature may have limited the estimation robustness of the independent effect of the risk factors, and thus restricted the understanding of the mechanisms that underpin the effects of the risk factors on adolescent DSH. For example, studies of the effects of restricted fetal growth on DSH risk have not taken into account the parental history of psychiatric disorders, which affect both fetal growth and adolescent DSH. Therefore, the extent to which parental psychiatric disorders account for the effect of fetal growth restriction on adolescent DSH remains unknown [22]. More importantly, the majority of previous studies in this area adjusted for all plausible confounders or mediators simultaneously, and thus important confounders or mediators could not be clearly identified, which may restrict the understanding of the aetiological pathways linking the risk factors to adolescent DSH. This may have limited the development of intervention strategies to reduce adolescent DSH risk.

Therefore, this study aimed to bridge the gap in existing literature by focussing on the three main groups of risk factors together (i.e. fetal growth restriction and birth order, early social disadvantage, and parental mental health disorders and death) to identify the risk factors that may specifically predict the occurrence of DSH-related hospital admissions during adolescence. This study was based on population-level administrative linked data to overcome the major limitations in surveys, and progressive adjustment for covariates in the regression analyses was undertaken to determine the important covariates for the effects of the risk factors on adolescent DSH.

## **Methods**

### **Record linkages**

This study was based on the linkage of health-related data routinely collected by government agencies in Western Australia (WA). The Data Linkage Branch (DLB) in WA is responsible for creating linkage keys for records across different datasets, using the unique identifiers (e.g., hospital unique medical record number or electoral number), record date, and important demographic information, such as name, date of birth, residential address, and sex. The DLB extracts this information for linking records from different data collections that belong to the same person. Where the unique identifiers are not available across all data collections, the demographic information is compared using

probabilistic matching methods to determine the likelihood that records belong to the same person. Uncertain links are manually checked by the DLB, and a de-identified linkage key unique to each person is generated and stored in all data collections [23]. The linkage accuracy has been shown to be greater than 99% with 0.11% of the links being invalid and missed [24, 25].

The linkage keys provided by the DLB were used to merge the de-identified individual-level records across different data collections. Datasets that were linked in this study included: the Midwives' Notification System (MNS) and the Birth Registrations including all births in WA; the Hospital Morbidity Data System (HMDS) including inpatient admissions (including psychiatric inpatients) in all hospitals in WA; the Mental Health Information System (MHIS) including psychiatric outpatients in public hospitals; the Emergency Department Data Collection (EDDC) including emergency department presentations in all hospitals which are under contract with the WA government; and the Death Registrations including all deaths registered in WA. All the data collections date back to 1980 and earlier, except for the EDDC which includes records since 2002. The MHIS includes records that date back to 1966.

## **Study population**

The Birth Registrations and Midwives' Notification System datasets were utilised to establish the source population, which was a birth cohort including all individuals born alive between 1981 and 2001 in WA and who were identified as non-Aboriginal ( $n = 244,104$  males,  $n = 230,756$  females). Given that hospitalised DSH is a rare event, with the prevalence being less than 2% [9, 15], a nested case-control sample was compiled from the source population to improve the computational efficiency. Cases consisted of all the individuals who were identified to have a DSH-related hospital admission between 10 and 19 years of age that occurred no later than the year 2011 ( $n = 7151$ ). Each case was randomly matched with twenty controls of the same sex and the year of birth. These matched controls ( $n = 143,020$ ) were sampled from the source population, and they had not had a DSH-related hospital admission or a death record by the date of the first DSH-related hospital admission of the matched case. Parental records were linked to children's information through the Family Connection System facilitated by the DLB.

## **Adolescent first DSH-related hospital admission (outcome)**

The outcome in this study was the first DSH-related hospital admission that occurred between 10 and 19 years, during the study period (1981–2011). Hospital admissions due to DSH were mainly identified in the HMDS and the EDDC, and DSH-related deaths were identified from the Death Registrations. The identification of DSH admissions was based on the International Statistical Classification of Diseases and Related Health Problems, 8th/9th Revisions, Clinical Modification (ICD-8/9/9-CM: E950-E959), and the 10th Revision, Australian Modification (ICD-10-AM: X60-X84). "Events with undetermined intent" (ICD-8/9-CM: E980-E989; ICD-10-AM: Y10-Y34) were considered as DSH-related episodes in this study to take into account possible under-recording due to the misclassification of DSH in clinic settings [26]. Additional symptom-related items in the EDDC were also considered in the identification of any DSH-related emergency contact. These items include "presenting symptom" and "human intent of injury".

The majority of the first DSH-related hospital admissions were identified in the HMDS (52.3%), followed by the EDDC (45.9%). Of the adolescents with DSH-related admissions, nearly 1% died as a result of the admissions (2.0% for males, 0.3% for females). Of the first DSH records, 7.2% were coded as "event with undetermined intent" (8.7% for males, 6.4% for females).

## **Risk factors**

### *Fetal growth status and birth order*

Information about fetal growth was identified from the MNS dataset. This study focussed on two measures on fetal growth: birth weight by gestation, and gestational age. Six categories were constructed for birth weight by gestational age (Table 1), according to the Australian national birth weight chart by gestation [27, 28]. Birth weight lower than the 10th percentile was considered small for gestational age (SGA); and birth weight higher than the 90th percentile large for gestational age (LGA). Gestational age was recorded in completed weeks. Gestational age between 20 and 36 weeks were considered as pre-term birth, and gestational age between 42 and 45 weeks overdue birth. Birth order was available in the MNS, and it excluded miscarriage.

### *Early familial social factors*

Early familial social factors were identified from the MNS dataset. This information included maternal age and marital status at the child's birth, and maternal neighbourhood socioeconomic status during the child's early years. Maternal marital status was grouped into three categories: married or de facto, unmarried, and single (e.g. separated, divorced). Maternal age was grouped into five categories: <20 years (i.e., teenage motherhood),

**Table 1** Distribution of risk factors among cases and controls

Risk factors	All	
	Case	Control
	<i>N</i> (%)	<i>N</i> (%)
Total	7151 (100)	143,020 (100)
Birthweight percentile by gestation		
<10th (small for gestational age, SGA)	827 (11.56)	14,143 (9.89)
10th–25th	1163 (16.26)	22,859 (15.98)
25th–50th	1820 (25.45)	36,821 (25.75)
50th–75th	1700 (23.77)	35,019 (24.49)
75th–90th	973 (13.61)	20,726 (14.49)
≥90th (large for gestational age, LGA)	666 (9.31)	13,404 (9.37)
Missing	2 (0.03)	48 (0.03)
Gestational age (weeks)		
20–36 (pre-term birth)	426 (5.96)	7836 (5.48)
37–41 (term birth)	6488 (90.73)	131,377 (91.86)
42–45 (overdue birth)	207 (2.89)	3294 (2.30)
Missing	30 (0.42)	513 (0.36)
Birth order		
1	2750 (38.46)	56,978 (39.84)
2	2344 (32.78)	48,018 (33.57)
3–4	1779 (24.88)	33,840 (23.66)
5+	274 (3.83)	4135 (2.89)
Missing	4 (0.06)	49 (0.03)
Maternal marital status at childbirth		
Married/de facto	5889 (82.35)	130,276 (91.09)
Unmarried	1077 (15.06)	11,264 (7.88)
Single (e.g. divorced, separated)	179 (2.50)	1410 (0.99)
Missing	6 (0.08)	70 (0.05)
Maternal age at childbirth (year)		
<20 (teenage motherhood)	727 (10.17)	6818 (4.77)
20–24 (young motherhood)	1940 (27.13)	30,844 (21.57)
25–34	3902 (54.57)	91,631 (64.07)
35–39	499 (6.98)	11,924 (8.34)
≥40	82 (1.15)	1801 (1.26)
Missing	1 (0.01)	2 (0.00)
Maternal neighbourhood socioeconomic status		
<10% (the most disadvantaged)	995 (13.91)	12,474 (8.72)
10–25%	1145 (16.01)	19,816 (13.86)
25–50%	1699 (23.76)	33,538 (23.45)
50–75%	1405 (19.65)	30,237 (21.14)
75–90%	673 (9.41)	17,283 (12.08)
≥90% (the least disadvantaged)	445 (6.22)	11,141 (7.79)
Missing	789 (11.03)	18,531 (12.96)
Parental all-cause death <sup>a</sup>		
No parental death	6839 (95.64)	139,150 (97.29)
Paternal death only	195 (2.73)	2696 (1.89)
Maternal death only	111 (1.55)	1120 (0.78)
Both parents died	6 (0.08)	54 (0.04)

**Table 1** continued

Risk factors	All	
	Case	Control
	<i>N</i> (%)	<i>N</i> (%)
Parental psychiatric hospital admissions <sup>a</sup>		
No parental admissions	3589 (50.19)	105,333 (73.65)
Paternal admissions only	886 (12.39)	12,549 (8.77)
Maternal admissions only	1860 (26.01)	19,954 (13.95)
Both parents had admissions	816 (11.41)	5184 (3.62)
Parental DSH-related admissions <sup>a</sup>		
No parental admissions	5710 (79.85)	133,537 (93.37)
Paternal admissions only	376 (5.26)	3033 (2.12)
Maternal admissions only	935 (13.08)	5936 (4.15)
Both parents had admissions	130 (1.82)	514 (0.36)
Children's history of psychiatric admissions		
No	1886 (26.37)	134,197 (93.83)
Yes	5265 (73.63)	8823 (6.17)

<sup>a</sup> Fathers could not be identified for two cases and forty-eight controls in the data

20–24 years (i.e., young motherhood), 25–34, 35–39, and ≥40 years.

The level of maternal neighbourhood socioeconomic disadvantage was determined using the Index of Relative Socioeconomic Disadvantage (IRSD), which was developed by the Australian Bureau of Statistics using the Australian Census of Population and Housing data [29]. The IRSD reflects the average level of socioeconomic disadvantage of the people and the households within a census district area, including approximately 250 households. The IRSD score summarises a range of different aspects of disadvantage, including low income (e.g. less than the third decile of population income distribution), low education (e.g. no post-school qualifications for people above 15 years), and unemployment and unskilled occupation. Each child's record in the MNS dataset was linked with an IRSD score, representing the level of socioeconomic disadvantage of the census district area where the child's mother resided at the time of the child's birth. In this study, six levels of neighbourhood socioeconomic disadvantage were constructed, based on the distribution of the IRSD score, and a lower level represented a higher level of disadvantage (Table 1).

#### *Parental death, psychiatric or DSH-related hospital admissions*

This study examined parental deaths due to all causes, and psychiatric or DSH-related hospital admissions that occurred before the child's first DSH-related hospital admission for the cases or the matched date for the

controls. Parental deaths were determined from the Death Registrations data; parental hospital admissions due to psychiatric disorders or DSH were identified from the HMDS, the MHIS, and the EDDC datasets. Psychiatric admissions were determined using the mental health related ICD codes (ICD-8/9/9-CM: 290-319; ICD-10-AM: F01-F99), and DSH-related admissions using the same codes as those for children noted above. All parental deaths and admissions were classified by parental sex, to examine whether the effects of parental deaths, parental psychiatric disorders or DSH behaviours on adolescent DSH risk differ by the sex of the parent [13].

## Data analysis and statistical methods

Frequency distribution was calculated for each risk factor among cases and controls. Conditional logistic regression was used to estimate the odds ratios (ORs) associated with the exposure. We did not adjust for the matching factors (i.e., child's sex, the year of birth, and the age at the end of the study period) in the univariate or multivariate analyses. The univariate regression analysis produced ORs that were automatically adjusted for by the matching factors. A series of multivariate logistic regression analyses were progressively conducted to examine how the effect of one set of risk factors on adolescent DSH risk changed when other sets of risk factors and covariates were adjusted for.

For fetal growth status and birth order, their effects on adolescent DSH risk were progressively adjusted for by early familial social factors, parental deaths, and parental hospital admissions due to psychiatric disorders or DSH. For familial social factors (i.e., maternal marital status, maternal age, and maternal neighbourhood socioeconomic status) and parental factors (i.e., parental all-cause death, parental psychiatric hospital admissions, and parental DSH-related hospital admissions), their effects on adolescent DSH risk were adjusted for by all factors listed in Table 1. The effects of early familial social factors, parental deaths, and parental admissions remained largely unchanged after adjusting for fetal growth status and birth order.

All the effects were adjusted for children's history of psychiatric admissions, in order to control for the strong correlation between psychiatric disorders and DSH behaviours. The same approach were used to identify children's psychiatric admissions as those used to identify parental psychiatric admissions as described above. Because fathers could not be identified for two cases and forty-eight controls, these cases and controls were excluded from the analyses in relation to parental deaths and admissions. Further, there were less than 0.5% of cases and controls who had a missing value for the factors shown in Table 1 (i.e., birthweight percentile by gestation, gestational age, birth order, maternal marital status, and maternal age). We therefore treated missing values as an independent category for each factor in the data analyses. We did not find that these missing categories had a significant effect on adolescent DSH risk, thus we did not report their effects, except for maternal neighbourhood socioeconomic status, because of a relatively high prevalence of missing for this variable. All statistical analyses were conducted using SAS® (EG) statistical software version 6.1 (SAS Institute Inc., Cary, NC, USA).

## Results

### Distribution of risk factors among cases and controls

Of 7151 cases, nearly two thirds were female ( $n = 4462$ , 62.4%), and over three quarters of all cases (77.7%) had their first DSH-related hospital admission between the age of 15 and 19 (80.1% for males and 76.3% for females,  $p < 0.0001$ ).

Distribution of risk factors among cases and controls is shown in Table 1. Generally, compared to controls, cases were more likely to be born small for gestational age, born later, have an unmarried or single mother, or a teenage or young mother (20–24 years of age) at birth, or have a mother who resided in a more socioeconomically disadvantaged area (IRSD score locating in the lowest quartile). The parents of cases were more likely to have died, or have had a psychiatric or DSH-related hospital admission, compared to the parents of controls.

### Fetal growth status and birth order

#### *Fetal growth status*

Univariate analysis showed that small for gestational age (SGA) was associated with a 1.21-fold (95% CI 1.11–1.31,  $p < 0.001$ ) increase in the odds of having a DSH-related hospital admission among adolescents, compared to birth weight between 50th and 75th percentiles by gestation (Table 2). Overdue birth was associated with a 1.27-fold (95% CI 1.10–1.47,  $p < 0.001$ ) increase in the odds of having a DSH-related hospital admission, compared to term birth. After adjusting for other risk factors and covariates, the effect of SGA was reduced to be statistically nonsignificant (aOR = 1.03, 95% CI 0.93–1.14,  $p = 0.6073$ ); whereas the effect of overdue birth remained significant (aOR = 1.32, 95% CI 1.10–1.57,  $p < 0.01$ ).

#### *Birth order*

Univariate analysis showed that higher birth order was associated with increasing odds of having a DSH-related hospital admission among adolescents (Table 2).

**Table 2** Effect of fetal growth status and birth order on adolescent DSH risk

Risk factors	Adjustment in regression analysis			
	OR (95% CI) <sup>a</sup>	OR (95% CI) <sup>b</sup>	OR (95% CI) <sup>c,e</sup>	OR (95% CI) <sup>d,e</sup>
Birthweight percentile by gestation				
<10th (SGA)	1.21 (1.11, 1.31) <sup>h</sup>	1.13 (1.03, 1.23) <sup>g</sup>	1.10 (1.01, 1.20) <sup>f</sup>	1.03 (0.93, 1.14)
10th–25th	1.05 (0.97, 1.13)	1.02 (0.94, 1.10)	1.01 (0.94, 1.10)	1.01 (0.92, 1.11)
25th–50th	1.02 (0.95, 1.09)	1.00 (0.94, 1.07)	1.01 (0.94, 1.08)	1.03 (0.95, 1.12)
50th–75th (reference)				
75th–90th	0.97 (0.89, 1.05)	0.99 (0.91, 1.07)	0.99 (0.91, 1.07)	1.01 (0.92, 1.12)
≥90th (LGA)	1.02 (0.93, 1.12)	1.07 (0.98, 1.17)	1.08 (0.98, 1.19)	1.06 (0.95, 1.18)
Gestational age (weeks)				
20–36 (pre-term birth)	1.10 (1.00, 1.22)	1.06 (0.96, 1.18)	0.98 (0.89, 1.09)	0.82 (0.73, 0.93) <sup>g</sup>
37–41 (term birth) (reference)				
42–45 (overdue birth)	1.27 (1.10, 1.47) <sup>h</sup>	1.20 (1.04, 1.38) <sup>f</sup>	1.25 (1.08, 1.44) <sup>g</sup>	1.32 (1.10, 1.57) <sup>g</sup>
Birth order				
1 (reference)				
2	1.01 (0.96, 1.07)	1.24 (1.16, 1.31) <sup>h</sup>	1.18 (1.11, 1.25) <sup>h</sup>	1.20 (1.12, 1.29) <sup>h</sup>
3–4	1.09 (1.03, 1.16) <sup>g</sup>	1.44 (1.34, 1.54) <sup>h</sup>	1.31 (1.22, 1.40) <sup>h</sup>	1.40 (1.29, 1.51) <sup>h</sup>
5+	1.37 (1.21, 1.56) <sup>h</sup>	1.81 (1.58, 2.07) <sup>h</sup>	1.56 (1.36, 1.79) <sup>h</sup>	1.53 (1.30, 1.81) <sup>h</sup>

<sup>a</sup> ORs were derived from conditional univariate logistic regression models, automatically adjusting for children's sex, the year of birth, and age at the end of the study

<sup>b</sup> ORs were derived from multivariate regression analysis, further adjusting for all early familial social factors

<sup>c</sup> ORs were derived from multivariate regression analysis, further adjusting for parental deaths and admissions

<sup>d</sup> ORs were derived from multivariate regression analysis, further adjusting for children's history of psychiatric admissions

<sup>e</sup> Two cases and forty-eight controls were excluded from analysis, because their fathers could not be identified in the data

<sup>f</sup> <0.05

<sup>g</sup> <0.01

<sup>h</sup> < 0.001

Controlling for maternal marital status and maternal age at childbirth increased the effect of birth order  $\geq 2$  on adolescent DSH risk. This is because children born with higher birth order were more likely to have a married or a mature-aged mother, which is a protective factor for adolescent DSH. When this factor was not taken into account, the effect of high birth order was suppressed. Table 2 shows that after adjusting for other risk factors and covariates, a later-born child had a 1.20–1.53-fold ( $p < 0.001$ ) increase in the odds of having a DSH-related hospital admission during adolescence, compared to the first-born child.

### Early familial social factors

#### *Maternal marital status at childbirth*

Univariate analysis showed that compared to adolescents born to a married/de facto mother, the odds of having a DSH-related hospital admission doubled for those born to an unmarried mother (aOR = 2.12, 95% CI 1.98–2.26,  $p < 0.001$ ), and nearly tripled for those born to a single mother (aOR = 2.81, 95% CI 2.40–3.29,  $p < 0.001$ ) (Table 3). After controlling for other risk factors and covariates, having a single mother, but not an unmarried mother, at birth continued to have a significant effect on adolescent DSH risk (aOR = 1.52, 95% CI 1.24–1.87,  $p < 0.001$ ).

#### *Maternal age at childbirth*

Table 1 shows that more than 10% of cases were born to a teen mother, compared to approximately 5% of controls. Table 3 shows that children born to a teen mother had a 2.51-fold (95% CI 2.31–2.72,  $p < 0.001$ ) increase in the odds of having a DSH-related hospital admission during adolescence, compared to those born to a mother aged 25–34 years. Children born to a teen mother continued to have significantly increased risk of having a DSH-related hospital admission, after controlling for covariates (aOR = 1.37, 95% CI 1.22–1.53,  $p < 0.001$ ). Table 3 also shows that being born to a young mother (20–24 years of age at the child's birth) was associated with a significant increase in adolescent DSH risk (aOR = 1.49, 95%

**Table 3** Effect of early (perinatal) familial social factors on adolescent DSH risk

Risk factors	Adjustment in regression analysis			
	OR (95% CI) <sup>a</sup>	OR (95% CI) <sup>b</sup>	OR (95% CI) <sup>c,e</sup>	OR (95% CI) <sup>d,e</sup>
<b>Maternal marital status at childbirth</b>				
Married/de facto (reference)				
Unmarried	2.12 (1.98, 2.26) <sup>h</sup>	1.56 (1.44, 1.68) <sup>h</sup>	1.28 (1.19, 1.39) <sup>h</sup>	0.99 (0.90, 1.09)
Single (e.g. divorced, separated)	2.81 (2.40, 3.29) <sup>h</sup>	2.63 (2.24, 3.08) <sup>h</sup>	1.91 (1.62, 2.24) <sup>h</sup>	1.52 (1.24, 1.87) <sup>h</sup>
<b>Maternal age at childbirth (year)</b>				
<20 (teenage motherhood)	2.51 (2.31, 2.72) <sup>h</sup>	1.88 (1.71, 2.06) <sup>h</sup>	1.61 (1.46, 1.77) <sup>h</sup>	1.37 (1.22, 1.53) <sup>h</sup>
20–24 (young motherhood)	1.49 (1.40, 1.57) <sup>h</sup>	1.34 (1.27, 1.42) <sup>h</sup>	1.19 (1.12, 1.26) <sup>h</sup>	1.04 (0.97, 1.12)
25–34 (reference)				
35–39	0.98 (0.89, 1.07)	0.97 (0.88, 1.07)	0.91 (0.83, 1.00)	0.90 (0.80, 1.01)
≥40	1.07 (0.85, 1.33)	1.05 (0.84, 1.32)	0.97 (0.77, 1.22)	0.81 (0.63, 1.05)
<b>Maternal neighbourhood socioeconomic status</b>				
<10% (most disadvantaged)	1.72 (1.58, 1.87) <sup>h</sup>	1.44 (1.32, 1.57) <sup>h</sup>	1.21 (1.11, 1.32) <sup>h</sup>	1.16 (1.04, 1.28) <sup>g</sup>
10–25%	1.24 (1.15, 1.35) <sup>h</sup>	1.13 (1.04, 1.23) <sup>g</sup>	1.02 (0.94, 1.11)	0.94 (0.85, 1.03)
25–50%	1.09 (1.01, 1.17) <sup>f</sup>	1.05 (0.97, 1.12)	0.99 (0.92, 1.07)	1.04 (0.95, 1.14)
50–75% (reference)				
75–90%	0.84 (0.76, 0.92) <sup>h</sup>	0.88 (0.80, 0.96) <sup>g</sup>	0.90 (0.82, 0.99) <sup>f</sup>	1.03 (0.93, 1.16)
≥90% (least disadvantaged)	0.86 (0.77, 0.96) <sup>g</sup>	0.93 (0.84, 1.04)	0.99 (0.89, 1.10)	1.09 (0.95, 1.24)
Missing	0.92 (0.84, 1.00)	0.88 (0.81, 0.96) <sup>g</sup>	0.86 (0.78, 0.94) <sup>h</sup>	0.96 (0.86, 1.07)

<sup>a</sup> ORs were derived from conditional univariate logistic regression models, automatically adjusting for children's sex, the year of birth, and age at the end of the study

<sup>b</sup> ORs were derived from multivariate regression analysis, further adjusting for other early familial social factors. Note, ORs largely remained unchanged after adjusting for fetal growth status, therefore these factors were not adjusted for

<sup>c</sup> ORs were derived from multivariate regression analysis, further adjusting for parental deaths and admissions

<sup>d</sup> ORs were derived from multivariate regression analysis, further adjusting for children's history of psychiatric admissions

<sup>e</sup> Two cases and forty-eight controls were excluded from the analysis, because their fathers could not be identified in the data

<sup>f</sup> <0.05

<sup>g</sup> <0.01

<sup>h</sup> <0.001

CI 1.40–1.57,  $p < 0.001$ ); however, this effect became non-significant after controlling for children's history of psychiatric admissions (aOR = 1.04, 95% CI 0.97–1.12,  $p = 0.2657$ ).

#### Maternal neighbourhood socioeconomic status

Univariate analysis showed that higher neighbourhood socioeconomic disadvantage during children's early years was associated with increased odds of having a DSH-related hospital admission during adolescence (Table 3). Being born to a mother who resided in the most disadvantaged areas (IRSD score locating in the lowest 10% range) was associated with a 1.72-fold (95% CI 1.58–1.87,  $p < 0.001$ ) increase in the children's risk of having a DSH-related hospital admission during adolescence, compared to children born to mothers who resided in the areas where the socioeconomic status was between 50th and 75th percentiles. This effect remained significant after controlling for other risk factors and covariates (aOR = 1.16, 95% CI 1.04–1.28,  $p < 0.01$ ).

#### Parental deaths, psychiatric or DSH-related hospital admissions

##### Parental deaths

Univariate analysis showed that both paternal and maternal death had a significant effect on adolescent DSH risk, and maternal death (aOR = 2.02, 95% CI 1.66–2.46,  $p < 0.001$ ) had a stronger effect than paternal death (aOR = 1.47, 95% CI 1.27–1.71,  $p < 0.001$ ) (Table 4). However, after adjusting for covariates, maternal death had no significant effect on adolescent DSH risk.



**Table 4** Effect of parental death, psychiatric or DSH-related hospital admissions on adolescent DSH risk

Risk factors	Adjustment in regression analysis			
	OR (95% CI) <sup>a,e</sup>	OR (95% CI) <sup>b,e</sup>	OR (95% CI) <sup>c,e</sup>	OR (95% CI) <sup>d,e</sup>
Parental all-cause death				
No parental death (reference)				
Paternal death only	1.47 (1.27, 1.71) <sup>b</sup>	1.45 (1.25, 1.68) <sup>b</sup>	1.13 (0.97, 1.31)	0.82 (0.69, 0.99) <sup>f</sup>
Maternal death only	2.02 (1.66, 2.46) <sup>b</sup>	1.93 (1.58, 2.35) <sup>b</sup>	1.47 (1.20, 1.80) <sup>b</sup>	1.00 (0.78, 1.27)
Both parents died	2.26 (0.97, 5.26)	1.99 (0.85, 4.66)	1.24 (0.53, 2.95)	0.69 (0.26, 1.82)
Parental psychiatric hospital admissions				
No parental admissions (reference)				
Paternal admissions only	2.07 (1.92, 2.24) <sup>b</sup>	1.94 (1.79, 2.09) <sup>b</sup>	1.76 (1.62, 1.91) <sup>b</sup>	1.32 (1.20, 1.45) <sup>b</sup>
Maternal admissions only	2.74 (2.58, 2.90) <sup>b</sup>	2.43 (2.29, 2.58) <sup>b</sup>	2.06 (1.93, 2.20) <sup>b</sup>	1.18 (1.09, 1.28) <sup>b</sup>
Both parents had admissions	4.61 (4.25, 5.00) <sup>b</sup>	3.99 (3.67, 4.33) <sup>b</sup>	3.00 (2.73, 3.29) <sup>b</sup>	1.43 (1.27, 1.60) <sup>b</sup>
Parental DSH-related admissions				
No parental admissions (reference)				
Paternal admissions only	2.89 (2.59, 3.22) <sup>b</sup>	2.47 (2.21, 2.76) <sup>b</sup>	1.60 (1.41, 1.80) <sup>b</sup>	1.41 (1.21, 1.64) <sup>b</sup>
Maternal admissions only	3.69 (3.43, 3.98) <sup>b</sup>	3.04 (2.82, 3.29) <sup>b</sup>	1.87 (1.72, 2.04) <sup>b</sup>	1.38 (1.25, 1.54) <sup>b</sup>
Both parents had admissions	5.86 (4.82, 7.11) <sup>b</sup>	4.66 (3.82, 5.67) <sup>b</sup>	2.17 (1.76, 2.67) <sup>b</sup>	2.11 (1.60, 2.78) <sup>b</sup>

<sup>a</sup> ORs were derived from conditional univariate logistic regression models, automatically adjusting for children's sex, the year of birth, and age at the end of the study

<sup>b</sup> ORs were derived from multivariate regression analysis, further adjusting for all early familial social factors. Note, ORs largely remained unchanged after adjusting for fetal growth status, therefore these factors were not adjusted for

<sup>c</sup> ORs were derived from multivariate regression analysis, further adjusting for parental deaths or admission

<sup>d</sup> ORs were derived from multivariate regression analysis, further adjusting for children's history of psychiatric admissions

<sup>e</sup> Two cases and forty-eight controls were excluded from the analysis, because their fathers could not be identified in the data

<sup>f</sup> <0.05

<sup>g</sup> <0.01

<sup>h</sup> <0.001

#### Parental psychiatric hospital admissions

As shown in Table 1, half of the cases had at least one parent with a psychiatric hospital admission, and this percentage was nearly double that for controls (49.81 versus 26.34%). Univariate analysis showed that maternal psychiatric admissions (aOR = 2.74, 95% CI 2.58–2.90,  $p < 0.001$ ) had a significantly stronger effect on adolescent DSH risk than paternal psychiatric admissions (aOR = 2.07, 95% CI 1.92–2.24,  $p < 0.001$ ). When both parents had psychiatric admissions, the odds of the children having a DSH-related hospital admission increased 4.61-fold (95% CI 4.25–5.00,  $p < 0.001$ ), compared to those who had no parents with psychiatric admissions (Table 4). The effects of parental psychiatric admissions remained significant after taking into account familial social disadvantage, parental DSH-related admissions, and children's history of psychiatric admissions. Moreover, when these covariates were controlled for, paternal psychiatric admissions had a slightly stronger effect on adolescent DSH risk than maternal psychiatric admissions.

#### Parental DSH-related hospital admissions

Table 1 shows that over 20% of cases had at least one parent with a DSH-related hospital admission, and this percentage was more than triple that of controls (20.16 versus 6.63%). Table 4 shows that maternal DSH-related hospital admissions (aOR = 3.69, 95% CI 3.43–3.98,  $p < 0.001$ ) had a significantly stronger effect on adolescent DSH risk than paternal DSH-related admissions (aOR = 2.89, 95% CI 2.59–3.22,  $p < 0.001$ ). When both parents had DSH-related hospital admissions, the odds of the children having a DSH-related hospital admission increased approximately sixfold (aOR = 5.86, 95% CI 4.82–7.11,  $p < 0.001$ ), compared to those who had no parents with DSH-related admissions. The effects of parental DSH-related hospital admissions remained significant when familial social disadvantage, parental psychiatric and children's psychiatric

admissions were controlled for. Further, the effects of parental DSH-related hospital admissions were more pronounced than the effects of parental psychiatric admissions on adolescent DSH risk.

## Discussion

This study examined adolescent DSH risk related to a range of biological and social factors in perinatal period, as well as parental hospital admissions due to psychiatric disorders or DSH behaviours or death. The effects of these factors were adjusted progressively for other risk factors considered as important confounders, and were further adjusted for children's psychiatric disorders. This allowed for important confounders to be identified. This study has shown that overdue birth, high birth order ( $\geq 2$ ), early familial social disadvantage, and parental hospital admissions due to a psychiatric disorder or DSH all have an important effect on adolescent DSH risk.

A novel finding in this study was that overdue birth had a strong link with adolescent DSH risk, which was independent of perinatal exposure to social disadvantage and parental and children's psychiatric disorders. Overdue birth (or post-term birth) is associated with increased risk of both maternal and neonatal morbidity and mortality [30, 31]. However, few studies have examined the long-term psychosocial outcomes of overdue births. Existing evidence shows that compared to term born infants, overdue born infants may be more likely to have emotional and behavioural problems during preschool years [16, 32], which are associated with increased risk of psychopathologies during adolescence, and hence elevated risk of DSH behaviours [33]. A possible explanation for the association between overdue birth and increased risk of DSH is that "old" placenta at the end of a longer pregnancy offers fewer nutrients and oxygen than what a full-term foetus requires, and this may result in neurocognitive impairments, which may increase adolescent DSH risk [32].

Previous studies have shown that low birth weight defined as less than 2.5 kg is a risk factor for suicidal death in young adults [11, 12]. Birth weight by gestation was examined in this study, and SGA was not found to be associated with increased risk of DSH during adolescence. The difference in the effects between absolute low birth weight (i.e.,  $\leq 2.5$  kg) and low birth weight relative to gestation (i.e., SGA) on DSH may be attributed to two reasons. First, SGA is conditional on gestation, and three quarters of the infants born SGA had a birth weight greater than 2.5 kg in this study, resulting in a reduce effect than that of absolute low birthweight. Second, parental and children's psychiatric hospital admissions were adjusted for in this study, which affect children's DSH risk [22]. Previous research has shown that restricted fetal growth such as low birth weight is associated with increased neurobiological and psychological vulnerabilities to emotional problems [34]. Therefore, the increased risk of DSH among children born SGA may mainly be accounted for by the adverse outcomes related to familial (both parents and children) psychiatric disorders.

This study demonstrated that adolescent DSH risk progressively increased with birth order. This trend has been found in the association between birth order and suicidal death in populations with similar socioeconomic settings to Australia [12, 14]. Previous research has shown that both social and biological processes may be involved in the effect of higher birth order on children's development of mental health problems. The resource dilution theory suggests that parents do not pay as much attention to the signs of psychological deviations of later-born children during their early years of life, as they do to earlier-born children [35, 36]. Additionally, women may have increased maternal stress during later pregnancies, which may increase the prenatal exposure to high cortisol levels among later-born children, compared to earlier-born children. This intrauterine stress may elevate later-born children's risk of having depression and hence DSH risk [12].

This study showed a large effect of having a teen mother or separated parents on adolescent DSH behaviours. Teen or single mothers are more vulnerable to socioeconomic disadvantage during childrearing [37, 38]. They are more likely to experience prenatal and postnatal mental illnesses [22, 39, 40], and be more subjected to maltreatment [41] and intimate partner violence during pregnancy [42]. These pre-existing psychosocial difficulties, combined with childrearing responsibilities, may incur high levels of social and emotional distress to teen mothers during and after their pregnancy [43]. The socio-emotional distress may reduce teen mothers' parenting capacities of providing a nurturing environment for their children's development [44], and may also inhibit the children's development of attachment during their critical early years such as infancy and early childhood [45]. Therefore, the children of teen mothers are at higher risk of developing emotional and behavioural problems and maladaptive coping strategies [44, 46], which are all related to DSH behaviours during adolescence. Further, this study showed that the effects of teen or single mothers on adolescent DSH risk remained strong when controlling for high neighbourhood socioeconomic disadvantage and parental and children's psychiatric disorders. This result suggests that social support targeted at teen or single mothers should enhance their psychosocial functioning, in order to reduce their children's risk of DSH in adolescence.

Children born to a mother aged between 20 and 24 years (i.e., young mothers) were also found to have increased risk

of DSH during adolescence in this study. However, this study further showed that different from teenage motherhood, the effect of young motherhood on adolescent DSH risk may be fully accounted for by children's history of psychiatric admissions. This indicates that young motherhood may contribute to children's psychiatric disorders, which may then act as a main pathway to subsequent DSH behaviours of children. Young mothers are subjected to high socioeconomic disadvantage in Australia [47]. Previous research has shown that children born to a young mother have poorer educational (e.g., poorer schooling) and psychosocial outcomes in adolescence [48], and higher risk of substance use, mental health problems and suicide attempts [44], compared to children born to an older mother. It was reported in previous research that 17–18% of women who gave births during the study period (i.e., 1981–2001) were 20–24 years of age at the time of the birth of their first child [47]. This indicates that young motherhood could have a large population-level impact on the risk of adolescent psychiatric disorders and DSH. Therefore, DSH-related interventions should aim at reducing young motherhood by providing support and services to enhance the health, education, and career development of youth aged 20–24 [49].

Parental all-cause death has been found to be a risk factor for suicide attempt and death in children [50–52]. Consistent with previous studies, this study showed that maternal all-cause death had a larger effect on adolescent DSH risk than paternal all-cause death [53]. However, both the effects of maternal and paternal all-cause death on adolescent DSH risk were largely attenuated when children's psychiatric history was taken into account. This suggests that parental all-cause death may lead to psychiatric disorders in children, which may become a pathway to the occurrence of adolescent DSH behaviours. This result highlights the importance of promoting the mental health of bereaved children, to prevent the occurrence of mental health problems, and hence the occurrence of DSH behaviours.

In line with previous research, this study showed that the effect of maternal psychiatric admissions significantly outweighed the effect of paternal psychiatric admissions on adolescent DSH risk [17]. However, after adjusting for children's psychiatric history, the effect of paternal psychiatric admissions emerged to be slightly stronger than maternal psychiatric admissions. This may indicate that maternal psychiatric disorders increase adolescent DSH risk largely via the transmission of psychiatric disorders from mothers to children, whereas paternal psychiatric disorders increase adolescent children's DSH risk through additional pathways. One possible pathway is reduced social and adjustment skills among adolescent children, due to the compromised fathering capacities associated with paternal psychiatric disorders [54]. This study also showed that the effect of maternal DSH-related admissions was more pronounced than the effect of paternal DSH-related admissions on adolescent DSH risk. However, maternal and paternal DSH-related admissions had a similar effect when children's psychiatric history was taken into account. Similar to previous research [55], this study showed that the effect of parental DSH-related admissions on adolescent DSH risk remained significant, although it was largely attenuated after controlling for parental psychiatric admissions. This highlights the importance of the identification of the parental history of both psychiatric disorders and DSH behaviours in the risk assessment of adolescent DSH risk.

### **Strengths and limitations**

The greatest strength in this study is the use of population-level linked records routinely collected by government agencies. Accurate information on fetal growth status such as birth weight and gestation could be identified from administrative birth records. Early social disadvantage may also be difficult to be identified in surveys, as these factors occur early in life. However, administrative records can provide more reliable information on early life social disadvantage. Using hospital records enhances the diagnostic validity of psychiatric disorders and improves the identification of the history of psychiatric disorders for both children and their parents. Using administrative data is particularly useful in the identification of the past and current psychiatric disorders for people or families more difficult to reach in survey studies, such as fathers with mental health problems, people with more severe psychiatric disorders, and families where both parents have psychiatric disorders. The identification of psychiatric problems is usually carried out with psychometric scales in survey studies, and this process is often time-consuming.

There are several limitations that require caution when interpreting the results of this study. First, the identification of DSH-related hospital admissions in this study mainly relied on ICD codes, which contain no information about whether suicidal intent was involved in a DSH-related admission. However, there was a significant overlap in the associated risk factors for DSH behaviours with and without suicidal intent [9, 56]. Second, the parental history of psychiatric disorders and DSH was only able to be identified if a parent ever presented at a hospital, as records from general practice, private clinics, and prescription of medicines were not available in the linked data. As a result, this study showed the risk of adolescent DSH associated with moderate to severe parental psychiatric disorders and DSH behaviours [57], and the results may not adequately reflect the effect of mild parental

mental health problems on adolescent DSH risk. Third, only parental psychiatric admissions that occurred after the year 1966 could be captured in the data, which means that parental hospital admissions due to psychiatric disorders or DSH could not be identified, if they occurred prior to 1966. Fourth, a crude estimation of the attrition rate related to emigration from WA was less than 2% in general population [58]. However, we could not identify specific individuals who emigrated from WA during the study period, thus psychiatric or DSH-related admissions recorded outside WA could not be accessed. Fifth, factors could not be identified in the linked data regarding the degree of parental involvement in childrearing, particularly for fathers, as most children live with mothers during their early years [59]. The lack of this information may affect the interpretation of the effect of father's psychiatric disorders on adolescent DSH risk identified in this study.

## Implications

The results of this study have demonstrated that various biological, psychological, and social factors are independently associated with increased risk of having a DSH-related hospital admission during adolescence. Parental psychiatric disorders and children's psychiatric disorders may play an important role in the developmental trajectories linking perinatal biological and social risk factors to increased risk of DSH during adolescence. The results may suggest that mental health care and social services hold great promise in promoting the psychosocial functioning of the families where the children have a teen or single mother, or live in a socioeconomically disadvantaged area in perinatal period. These services may help the children to overcome the adverse consequences of the perinatal risk factors and parental psychiatric disorders, and use more effective coping strategies rather than DSH behaviours to deal with stress. Additionally, prenatal care may be needed to prevent post-term birth to reduce its potential negative influence on children's neurobiological development, which may increase adolescent DSH risk.

## Conclusion

Using population-level administrative linked data, this study has rigorously examined a range of bio-psychosocial factors associated with adolescent DSH risk. Using progressive adjustment in regression analysis, this study has shown that parental and children's psychiatric admissions are important factors that may underpin the effects of perinatal social factors on adolescent DSH risk. Therefore, joint actions based on mental health and social services are required by families with psychosocial disadvantages. These joint actions may enhance these families overall functioning, improve the children's early development, and reduce their risk of psychiatric disorders, and hence DSH during adolescence.

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## Compliance with ethical standards

**Ethics approval** This study was approved by the Human Research Ethics Committee of the Department of Health in Western Australia and the University of Western Australia Human Research Ethics Office. Because the complete datasets include records for both Aboriginal and non-Aboriginal people, this study has also been approved by the Western Australian Aboriginal Health Ethics Committee. However, this study only focused on non-Aboriginal people and it is the first study carried out using the dataset.

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