

PREDICTORS OF POSTPARTUM RETURN TO SMOKING: A SYSTEMATIC REVIEW

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COMPETING INTERESTS

The authors have no competing interests to declare.

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ABSTRACT

Background

Finding effective ways to help pregnant women quit smoking and remain abstinent is a major public health issue. Approximately half of UK women who smoke attempt cessation after conception; unfortunately, up to 75% return to smoking within 12 months postpartum. Interventions for preventing postpartum return to smoking (PPRS) have not been found to be effective. It is important to identify factors associated with PPRS, to inform development of alternative interventions.

Aims

Identify by systematic review factors associated with PPRS.

Methods

Systematic searches of electronic databases (MEDLINE, EMBASE, PsychINFO, CINAHL), trials registers and conference proceedings were conducted to November 2016. Studies statistically examining factors associated with PPRS were included. Modified versions of the Newcastle Ottawa Quality Assessment Scale were used to assess studies' quality and a narrative synthesis focussed on those judged of high quality.

Results

Thirty-nine studies, (12 trials, 27 observational studies), were included. Thirty-one (79.5%) studies were high-quality. Among these, the most common significant predictors of PPRS were being less well educated, younger, multiparous, living with a partner or household member who smoked, experiencing higher stress, depression or anxiety, not breastfeeding, intending to quit only for pregnancy and low confidence to remain abstinent postpartum.

Conclusions

Of the factors found to be associated with PPRS, intending to quit smoking only for the duration of pregnancy, partner/household member smoking and confidence to remain abstinent are those most likely to have a direct, causal impact on smoking behaviour after childbirth, and need to be considered when designing interventions to prevent PPRS.

IMPLICATIONS

This is the first systematic review of factors that may facilitate or inhibit PPRS. Considering how having a partner or household member who smokes, intending to quit smoking only for pregnancy,

having self-efficacy to quit long term, breastfeeding and depression exert direct or indirect impacts on women's relapse to smoking and how such impacts could successfully be manipulated will inform development of new interventions to prevent PPRS.

BACKGROUND

Maternal smoking in pregnancy harms both infants and mothers, increasing risks of miscarriage, stillbirth, prematurity, low birth weight, perinatal morbidity and mortality, neonatal or sudden infant death.¹ For adults, smoking is a leading cause of preventable morbidity and mortality.² Finding effective ways to help pregnant women quit smoking and remain abstinent is an important public health priority. In the UK, 26% of women smoke at some point in pregnancy;³ however, pregnancy has been identified as a life event which strongly motivates women to stop smoking, with approximately half of UK women who smoke attempting cessation after conception.⁴ Despite nearly all of these women wishing to remain abstinent after birth, up to 75% will return to smoking within 12 months postpartum.⁵⁻⁷

Smoking cessation interventions are effective in supporting pregnant women to quit;^{8,9} however, there is little evidence that evaluated interventions for preventing postpartum return to smoking (PPRS) are effective.¹⁰ A systematic review found that even among pregnant women participating in smoking cessation trials, of those who reported abstinence at end of pregnancy around 43% were smoking again 6 months later.¹¹ By improving women's and children's health, reducing maternal PPRS would be of significant social benefit,¹² and is likely to be extremely cost-effective.¹³⁻¹⁵ Most women giving birth are 16-44 years old, and therefore those who quit smoking will be young enough to minimise long-term health damage.¹⁶ Maintaining smoking abstinence postpartum also reduces the likelihood of smoking in future pregnancies. Furthermore, maternal smoking is the primary source of infant and child secondhand smoke exposure,¹⁷ a substantial cause of ill health and mortality,¹² and children of smoking mothers are twice as likely to become smokers themselves.¹⁸

A useful first step in the development of interventions which might prevent PPRS would be to identify factors which are associated with returning to smoking after pregnancy. In this study we attempt to do this and so describe those factors which may hinder or accelerate PPRS.

METHODS

This review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.¹⁹

Inclusion and exclusion criteria

Irrespective of study design, we identified for potential inclusion studies, conducted among women who quit smoking 3 months prior to or during pregnancy, which statistically examined factors associated with return to smoking during the first 12 months postpartum.

Intervention studies in which analyses did not control for intervention group allocation were excluded because one could not be certain of the extent to which these studies' findings might be influenced by intervention effects.

Strategies for searching the literature

We searched Medline, Embase, PsychINFO and CINAHL databases. Conference proceedings were searched through CAB abstracts, and hand searches of Society for Research on Nicotine and Tobacco (SRNT) and UK National Smoking Cessation (UKNSCC) conference outputs. Other sources of grey literature were searched using the following databases: European association for grey literature exploitation (EAGLE), Government health agencies, (Centre for disease control and prevention (USA), National Institute for Health (USA), UK Department of Health), World Health Organisation, The health care management information consortium (HMIC) database. Sources of on-going clinical trials were also searched: clinicaltrials.gov/, www.who.int/trialsearch, www.controlled-trials.com/ukctr/, portal.nihr.ac.uk/Pages/Portfolio.aspx. Hand searches of reference lists of identified relevant, eligible papers were conducted.

Searches had no date restrictions, were conducted up to November 2016 and were limited to English language papers. Databases were searched using combinations of the key words: smoking (smok*), tobacco, "smoking cessation", "tobacco cessation", pregnancy (pregnan*), postnatal (post*natal*), postpartum (post*partum), maternal, mother*, prenatal, relapse (relaps*), return, abstinence (abstinen*), cessation. Broad search terms were used to reflect the exploratory nature of the review aims. An example of a Medline search can be found in supplementary file S5.

Data extraction

Studies identified by search strategies were screened for eligibility; initially on the basis of title and abstract, and then by reading the full paper of the remaining studies. Where only abstracts were available, authors were contacted for full texts; if unsuccessful the abstract was excluded due to insufficient information. One reviewer (SO) screened all studies for inclusion with one-third also screened independently by another reviewer (TCH), with 100% agreement.

The following data was extracted from each eligible study by SO, and verified by MU or TCH: first author name, publication date, study location, methods (aims, design), data collection time frame, key sample characteristics, main outcome measure (definition of return to smoking, time of measurement), PPRS rate, factors examined (and controlled for in analysis), analysis method, analysis findings and effect estimate for significant findings.

Potential predictors of PPRS were categorised as infant-related, pregnancy-related, psychological, relationship and social activity, smoking and substance use related, sociodemographic characteristics and weight. The nature of the relationship (positive/negative/no association) between potential predictors and PPRS was noted.

Assessment of quality

Included studies were assessed for quality and risk of bias using modified versions of the Newcastle-Ottawa Quality Assessment Scale (NCOS).^{20,21}

For cross-sectional studies, we adapted Herzog and colleagues'²¹ version of the NCOS. For cohort studies, we adapted Wells et al.'s²² version of the NCOS. Both scales use a star-based system to assess quality on three domains: selection of study group, comparability of study group, and ascertainment of exposure/outcome.^{21,22} Similar changes were made to each scale, and so are described together below.

The 'ascertainment of the exposure' scale item was removed because it was not applicable to every study. Not all studies included explanatory variables which would be most appropriately measured using a validated scale (e.g. depression or anxiety). Additionally, many explanatory variables did not require validation (e.g. age).

The 'comparability' item was changed to 'design and analysis'; one star was awarded to studies that were judged to have used multivariable analyses appropriately to control for potential confounding factors in a manner consistent with the study aims and no stars were awarded to studies that failed to do this or which were described in insufficient detail for appropriateness to be assessed.

The 'assessment of outcome' was refined to reflect the two main methods used for assessing PPRS; greater methodological quality was assigned to studies using bio-chemically confirmed smoking status (e.g. by salivary cotinine, urinary cotinine or expired CO) over self-reported smoking status.

The 'demonstration that outcome of interest was not present at the start of the study' item on the cohort studies scale²² was irrelevant and consequently was removed from the scale.

Using these criteria, studies were awarded a quality rating score out of a maximum of five stars for cross sectional studies, and six for cohorts. An a priori cut off point of 4 stars for cohorts and 3 for cross sectional studies was used to categorise papers as of high or low methodological quality.

Two reviewers (SO and MU) independently completed quality assessments for included studies. Any discrepancies in scores were discussed and resolved.

Analysis

After inspection of included papers, meta-analysis was not considered appropriate as, for any one predictor, there were insufficient high quality studies (i.e., at least three) with the necessary level of homogeneity in study design, outcome measures and timing of outcome ascertainment.²³

Consequently, for ease of description, those factors found to be significantly associated with PPRS were categorised for presentation in a narrative data synthesis. These categories were developed by grouping thematically similar factors across the included papers, which were decided through discussion involving all authors. The agreed categories were sociodemographic (including personal and demographic characteristics, such as socioeconomic status, income, having health insurance, education, employment status, race/ethnicity, age, relationship status, place of birth, urban versus rural living), smoking and substance use (smoking behaviour and substance use, motivations and attitudes to smoking), psychological (depression, anxiety, stress), relationship and social activity (others' smoking in their social networks, parenting, childcare support), maternal/pregnancy related (parity, breastfeeding, antenatal care), weight (maternal weight, weight concerns) and infant related (birthweight, age, gender).

Given the exploratory nature of this review and much of the empirical literature, we planned to focus principally on findings from studies which were categorised as 'high' quality. Full details of study characteristics for both high and low quality papers are presented, however we only report findings from those categorised as high quality. For these high quality studies, we describe the factors which were significantly associated with PPRS and which had the strongest associations, and we also report which factors were found related to the timing of PPRS.

RESULTS

Searches identified 30,283 papers (10,166 after duplicate removal). Titles and abstract were assessed according to the above inclusion and exclusion criteria, which identified 102 papers of potential relevance for which full text retrieval was attempted. After contacting authors, full texts were not available for 24 abstracts which were excluded. Thirty-nine papers were included in the final review (figure 1). The combined total number of women included in the studies was 240,343. Study characteristics are presented in table S1.

Study characteristics

Study design

Twelve studies were trials²⁴⁻³⁵ and 27 were observational (14 cross sectional studies,³⁶⁻⁴⁹ 13 prospective cohort studies^{48,50-62}).

Study location

Twenty-four studies were conducted in the USA^{24-27,31,32,35-39,41,43,44,46,48,49,55-60,62}, three in Canada,^{29,34,40} two in the UK,^{45,50} two in Germany,^{30,33} two in Poland,^{28,61} two in Japan,^{42,47} and one each in Norway,⁵¹ France,⁵³ Switzerland,⁵⁴ and Hong Kong, China.⁵²

Outcome measures

Twenty-eight studies used self-reported measures of PPRS,^{26-28,30-33,36,37,40-54,58-60,62} and 11 used validated measures (e.g. expired CO or salivary cotinine).^{24,25,29,34,35,38,39,55-57,61} The use of self-reported or validated measures of PPRS are considered during assessment of quality.

Four studies^{39,50,55,62} had a maximum follow-up of <3 months postpartum, 18 studies of 3-6 months^{26,27,29,34-38,41,49,51,53,56-61}, 10 of 7-12 months^{24,25,28,30-33,44-46} and seven studies had a maximum follow-up of >12 months^{40,42,43,47,48,52,54}.

Study sample sizes

Eleven studies^{27,34,35,39,52,55,57,59-62} had sample sizes of 150 or less, 14^{24-26,28-33,42,44,50,54,56} samples sizes of 151-1000, and 14^{36-38,40,41,43,45-49,51,53,58} of more than 1001.

Participant characteristics

The majority of participants were aged 20-30 years. The predominant ethnic groups were Caucasian, African American and Hispanic.

Assessment of quality

Twenty cohort studies^{24-35,48,50,53,56-58,61,62} were of high methodological quality (scored ≥ 4). Lower-quality studies generally used non-validated measures of smoking status or had high attrition at follow-up. Eleven cross sectional studies^{37-43,45-47,49} were of high methodological quality (scored ≥ 3); lower quality studies used non-validated measures of smoking status, or had inappropriate/incomplete reporting of design or analysis (table S3 and S4).

Predictors of post-partum return to smoking in high-quality studies

For all studies, statistically significant predictors of PPRS are listed in Table S1 (column eight) and in Table S2 the direction of the association of these predictors is explained and effect estimates are presented.

Sociodemographic

The association between low socioeconomic status/level of deprivation and PPRS was examined in two studies, with one reporting a significant association,²⁴ and one reporting no significant association.⁴⁵ Low family income was examined in four studies, with a significant association with PPRS in one study,⁴⁸ and no association in three studies.^{30,40,43} One study⁴⁵ found a significant association between not managing financially among a subgroup of single mothers and PPRS. There was no significant association between being unemployed and PPRS, or partner unemployment in one study.⁵³ Three studies^{38,46,49} examined having no private health insurance during pregnancy, with none reporting a significant association with PPRS. There was no significant association between living with a grandparent and PPRS in one study.⁴²

Being of African American race/ethnicity,³⁷ being of black ethnicity,⁴⁶ and being of black non-Hispanic ethnicity⁴⁹ were significantly associated with PPRS in one study each. There were no significant association between race/ethnicity and PPRS in a further five studies.^{38,40,45,48,58,59}

Education and PPRS were examined in 16 studies, with four^{38,40,43,48} reporting a significant association between low education and PPRS, and 12^{30,31,33,37,41,42,45,46,49,53,58,62} reporting no significant association.

Marital/relationship status was examined in 11 studies,^{30,37,38,40,41,46,48,49,53,57,58}, with none reporting a significant association with PPRS.

Younger maternal age was studied in 17 studies; four^{38,42,43,49} reported a significant association with PPRS, and 13^{30,33,40,41,45-48,50,53,57,58,62} reported no association.

One study⁴⁵ found a significant association among married and single mothers, but no association among cohabiting mothers.

Place of birth (North America or outside North America), and region of residence, were not significantly associated with PPRS in one study.⁴⁰ Living in an urban region compared with a rural region was significantly associated with PPRS in one study.⁵⁰

Smoking and substance use

Heaviness of smoking was examined in eight studies. There was a significant association between increased cigarettes per day prior to pregnancy and PPRS in two studies,^{38 35} and no significant association in six studies.^{39,41,46,48,53,57} Younger age of smoking initiation was not found to be associated with PPRS in the three studies^{30,31,39} that looked at this factor.

Smoking any cigarettes during pregnancy was significantly associated with PPRS in one study.²⁷ Having quit smoking earlier in pregnancy was examined in three studies, with one³⁰ reporting a significant association with PPRS, and two^{45,61} reporting no significant association. Being a daily smoker prior to pregnancy was significantly associated with PPRS in one study;⁴⁹ however, it was not significantly associated with PPRS in a second study.³⁷

Higher prenatal nicotine dependence was significantly associated with PPRS in one study;²⁸ however, no significant association was reported in a further two studies.^{30,39} Higher prenatal smoking frequency was not associated with PPRS in one study that examined this factor.⁴⁰ High craving for cigarettes was associated with PPRS in one study;²⁴ however, a further study found no significant association between urges to smoke and PPRS.⁶¹

One study²⁶ found that having extrinsic motivations for quitting smoking, and changing from intrinsic to extrinsic motivations, were significantly associated with PPRS. Intending to quit only for pregnancy was significantly associated with PPRS in all three studies^{30,31,61} that examined this factor. Awareness of harmful effects of second hand smoke,⁴² belief of the benefits of smoking to the woman,⁵⁷ low confidence to not smoke in response to traditional smoking triggers,³⁹ thinking about own health to cope with urges to smoke⁵⁷ and avoiding situations where others are smoking⁵⁷ were not associated with PPRS. Low confidence not to smoke in response to infant crying,³⁹ smoking as a response to infant crying,³⁹ smoking to cope with stressful situations,⁶¹ snacking to resist urges to smoke⁵⁷ and not thinking about money saved to resist smoking⁵⁷ were all significantly associated with PPRS. Confidence in and desire for postpartum smoking abstinence were examined in three studies; one study²⁷ found a significant association with PPRS and one study⁵⁷ found no association. One study³¹ found that the association was dependent on time of measurement, with confidence measured at 1 month postpartum being significantly associated with PPRS at 6 months, but for confidence measured at 6 months postpartum the association was not significant.

Increased cigarette price, examined in one study,³⁸ was not significantly associated with PPRS.

Three studies explored the association between alcohol consumption and PPRS. One study⁴⁵ found drinking more than once a month, up to twice weekly in postpartum was associated with an increased risk of PPRS among married and cohabiting mothers. This study⁴⁵ also found that drinking less than once a month postpartum was associated with a reduced risk of PPRS among single mothers. A second study reported that not drinking alcohol at the time of becoming pregnant was significantly associated with a reduced risk of PPRS.⁴⁷ This study⁴⁷ found no significant association with drinking alcohol during pregnancy and PPRS. A third study found no significant association between drinking alcohol postpartum and PPRS.⁴⁸

Psychological

Three studies included depression or major depressive syndrome in their analyses, with a significant association with PPRS in two studies^{25,35} and no association in the other study.⁵⁶ Postpartum depression was examined in three studies, with one⁴⁶ reporting a significant association with PPRS, and two reporting no significant association.^{40,41} Low versus high maternal mood,⁴¹ positive versus negative affect⁵⁶, psychological distress⁴⁵ and perceived stress⁵⁶ were each examined in one paper, with none reporting a significant association with PPRS. Stressful life events were examined in five studies, with a significant association with PPRS in two studies,^{46,49} and no significant association in three studies.^{37,41,51} One study²⁵ reported a significant association between anxiety and PPRS.

One study⁶² looked at stage of change and decisional balance, reporting that being a member of 'high risk' or 'risk denial' groups was significantly associated with PPRS, whereas being members of 'ambivalent' or 'protected' groups was not. One study³² reported being a member of pre-contemplation, contemplation or preparation stages of change was significantly associated with PPRS. One study³⁴ found a significant association with baseline low delay discounting, a measure of impulsivity, and PPRS. One study³³ examined clusters of perceived advantages and disadvantages of non-smoking, and self-efficacy not to smoke; those in 'high risk', 'premature' or 'ambivalent' groups were significantly more likely to return to smoking postpartum.

Relationship and social activity

Having a high proportion of close associates who were smokers was examined in four studies, with a significant association with PPRS in two studies,^{27,35} and no association in the remaining two studies.^{39,57} Living with a smoker, or having other household members who were smokers was explored in six studies. Four studies^{31,40,48,50} found this to be significantly associated with an increased risk of PPRS, and one study²⁸ to be significantly associated with a reduced risk of PPRS. One study³³ found no significant association. Having a smoking environment at home was

significantly associated with PPRS in two studies that examined this factor.^{49,61} Exposure to passive smoking was not significantly associated with PPRS in one study.²⁷

Partner smoking was explored in 10 studies. Five^{27,32,45,47,53} of these reported partner smoking to be significantly associated with PPRS, and four^{30,42,57,62} found no association. One study³¹ found having a partner who smoked as much as before pregnancy was associated with PPRS at 12 months postpartum but not at 1 month postpartum.

One study⁴⁷ looked at decreased parenting satisfaction, lack of confidence in childrearing, not spending time with child in relaxed mood, low partner participation in childrearing, maltreatment of child, and having social support and people to talk to, finding no significant associations with PPRS. This study did report 'not talking about parenting on the internet' to be significantly associated with increased risk of PPRS.

One study³⁹ looked at increased hours per day that their infant cried, increased infant fussiness and intensity of infant fussiness, finding no association with PPRS.

Increased stress related to childcare,⁴² low partner positive support style,³¹ and low perceived helpfulness of spouse/best friend in early postpartum⁵⁷ were examined in one study each, with no significant associations. Having no one to share feelings with was significantly associated with PPRS in a subgroup of married mothers in one study.⁴⁵

Maternal/pregnancy related

Four studies examined pregnancy intention, with one⁴⁹ finding a significant association between unplanned pregnancy and PPRS, and three finding no association.^{38,41,45} Multiparity, examined in 13 papers, was significantly associated with PPRS in five studies.^{37,41,47,49,50} There was no significant association in seven studies.^{30,38,40,42,48,53,58} Beginning antenatal care in a late trimester was examined in three studies,^{37,41,45} with none finding a significant association with PPRS. Participation in an antenatal course was associated with PPRS in one study.⁵³ Receiving no advice from a health care worker about smoking,³⁷ delivery method,⁴² and being pregnant again at 12 months postpartum⁴⁸ were not associated with PPRS.

Breastfeeding was examined in 17 studies. Not breastfeeding, not intending to breastfeed and early weaning were associated with increased risk of PPRS in 13 studies,^{29,31,40-43,45,46,49,50,58,61,62} with no association in four studies.^{33,48,53,57}

Weight

In one study⁴⁶ being overweight or obese prior to pregnancy was significantly associated with a reduced risk of PPRS. Increased weight gain during pregnancy was examined in two studies, with one³⁷ reporting a significant association with PPRS, and one⁴⁸ finding no association. One study⁵⁶ found a significant association between smoking specific weight concerns and PPRS, but not with general weight concerns. One study³⁵ found general weight concerns to be associated with a reduced risk of PPRS. One study⁵⁷ found no significant association between perceived likelihood of returning to desired weight by 6 months postpartum and PPRS.

Infant related

Three studies^{42,48,49} examined infant birthweight, with none reporting a significant association with PPRS. Higher infant age at time of survey was explored in three studies, with one⁴⁶ reporting a significant association with PPRS, and two^{37,40} finding no association. Infant gender was not associated with PPRS in one study.⁴²

Strength of significant associations among high-quality studies

The factors with the strongest associations (OR/RR ≥ 3.00) to PPRS in high-quality papers are described below.

Smoking related factors with the strongest significant associations with PPRS were: partner/other household member smoking,^{45,47,48,50} having a smoking environment at home,⁶¹ higher nicotine dependence,²⁸ quit smoking later than 1 month after becoming pregnant,⁶¹ intending to quit smoking only for pregnancy,^{30,61} feeling the urge to smoke a few times a week or more,⁶¹ and smoking to help cope with stressful situations.⁶¹

Psychological factors with the strongest significant associations to PPRS were: the presence of an anxiety syndrome,⁵⁰ stage of change decisional balance cluster groups,⁶² and Transtheoretical Model of Behaviour Change cluster groups ('high risk group', 'Premature group', 'Ambivalent group').³³

Maternal/pregnancy related factors with the strongest associations with PPRS were: parity⁵⁰ and not breastfeeding.^{40,46,61}

One study²⁴ reported standardised structural coefficients (SSC), with strong significant associations observed for low socioeconomic status (SSC -0.215) and pre-partum cravings for cigarettes (SSC 0.428) and PPRS.

Identified associations and timing of PPRS

Four high-quality studies^{26,31,32,57} examined factors at more than one time point following childbirth. Simmons et al.³¹ reported that at 1 month postpartum the factors associated with returning to smoking were not planning to quit for good, lower confidence in not smoking at 6 months postpartum, the presence of another smoker in the household and not planning to breastfeed. At 12 months postpartum, not planning to quit for good and partner smoking as much as before pregnancy were associated with a return to smoking.³¹ In McBride et al.'s study,⁵⁷ at 6 weeks postpartum, participants reporting snacking to resist urges to smoke were less likely to return to smoking, whereas between 6 weeks to 6 months postpartum, reporting thinking about the money saved to resist smoking was significantly associated with not returning to smoking. Curry et al.²⁶ reported low intrinsic motivation, and moving from intrinsic to extrinsic motivation, was associated with return to smoking at 8 weeks postpartum, but not at 6 months postpartum. Stotts et al.³² found in univariate analysis that those in the pre-contemplation stage of change returned to smoking significantly sooner after birth, followed by those in the contemplation stage, preparation stage and action stage.

Discussion

Among high-quality studies, the factors most commonly associated with an increased risk of PPRS (i.e, reported by most studies) were: being less well educated, younger, multiparous, having a partner or other household member who smoked, higher levels of stress, depression or anxiety, not breastfeeding, intending to quit smoking only for pregnancy and lower confidence or desire to remain abstinent postpartum. Among these, the factors associated with the highest risk of PPRS were: not breastfeeding, intending to quit smoking only for pregnancy and partner/household member smoking.

A potential limitation of this review is the variable definition of PPRS across studies, with few using biochemical validation of smoking status. This may have introduced bias into individual studies as women may not have been truthful about their smoking status. However, we gave greater prominence to high-quality studies, which were more likely to have used validated measures of smoking status. Although we identified which factors are positively or negatively associated with PPRS, there were more studies which identified no association for many of these factors than which did. However, in most instances associations were in the same direction and we have not highlighted the relationship of any factors as being related to PPRS which were not. Consequently, as a means of identifying factors associated with PPRS we think our methods are robust. The use of observational

data means that causality between associations identified and PPRS cannot be assumed as they may have been influenced by unknown biases. However, the consistency in findings across studies reduced the likelihood of such biases being introduced. Additionally, a potential limitation is study heterogeneity; however, again, the consistency in the direction of findings across studies adds strength to the conclusions we have drawn. Furthermore, a number of tools exist for assessing the quality of observational studies, and it is possible that papers may have been classified differently had an alternative tool been used. The majority of studies included in this review were conducted in the USA, which may limit generalisability of findings to other countries, and should be taken into consideration in the development of future interventions to prevent PPRS.

To aid the description in the narrative review we created categories for the various factors. These categories were based on discussion and a consensus between the authors; however, we acknowledge that there are other ways of grouping the factors which might highlight other themes. We also appreciate that the factors and domains are not independent and studies are needed to explore direct and indirect pathways and interactions linking predictors and domains with postpartum return to smoking, thereby further focussing the priorities for intervention development. Only one of the reviewed studies explored this issue and showed that socioeconomic status indirectly influenced postpartum smoking relapse through increased pregnancy negative affect/stress, reduced sense of agency, and increased craving for cigarettes.²⁴

In several instances a single study provides evidence for a number of factors being significantly related to PPRS, each factor being reported in a separate categorical section of the narrative in this review. Consequently, there could be a risk that some studies, particularly those with large samples, will contribute more to the overall review. However, we have focussed on higher quality studies, so a large study with non-adjusted estimates or other biases will not feature prominently in the findings but a large study without such biases will feature. Thus, our focus on study quality is a strength in this instance.

A strength of this review is its novelty, and the systematic approach taken; we reviewed a large number of studies, and several researchers were involved in screening and data extraction, reducing the likelihood of bias being introduced. We conducted a thorough quality assessment, which is particularly important when studies are observational; however, over three quarters of studies were considered of high-quality and we give most prominence to findings from these. Inclusion of lower quality studies in the narrative synthesis would not have changed the overall conclusions of this review; just 21% of included studies were considered to be of lower quality, and there were no

notable differences between the direction or significance of associations with factors presented in these studies and PPRS compared with higher quality studies (Table S2). Further research is needed with factors where none or very few high quality studies have been conducted (e.g., occupation, employment status, number of previous quit attempts, success in previous quit attempts, cigarette craving). Although there are several studies which provide data on socio-economic characteristics these use diverse measures and it is hard to draw conclusions from their data, so future studies need to attempt to use measures of socio-economic status that are meaningful across different jurisdictions.

When considering our findings across the domains, they highlight the particular importance of the socio-demographic, relationship, psychological and pregnancy domains for identifying the characteristics of women who are at an increased risk of returning to smoking after childbirth. Caution needs to be taken in interpreting these findings, as whilst significant associations were observed in some papers, others reported no significant association. However, observed associations were generally in the same direction, and therefore these characteristics may be useful to target in future PPRS prevention interventions. As indicated above, work is now needed to fully integrate the findings across the various domains, for example, to uncover the relative extent to which socio-demographic, psychological, relationship and psychological predictors account for PPRS.

Some factors identified are likely to have a more direct impact on PPRS and hence may be more appropriate than others for incorporation into PPRS prevention interventions. Partner/household member smoking was found to be consistently and strongly associated with PPRS. Qualitative systematic reviews found partner smoking can be a barrier to women quitting and maintaining abstinence postpartum, with partner smoking increasing temptation to smoke, or quitting smoking changing the dynamics of the relationship.^{63,64} Previous couple-based interventional approaches to smoking cessation and PPRS prevention during pregnancy and postpartum have not been shown to be effective,^{32,65-68} suggesting that new evidence-based approaches are needed. A recent qualitative systematic review⁶⁹ of the experiences of smoking cessation by women's partners during pregnancy and postpartum identified barriers (e.g., smoking integral part of everyday life, smoking in the workplace and scepticism about risks associated with smoking) and facilitators (e.g., wanting to be a good father, supporting their partner) to quitting. While such findings can inform the development of novel interventions to target partner smoking, further research is needed as only a few, small studies have been conducted.

Other factors identified that are likely to have a direct impact on smoking were attitudinal in nature; for example, intending to quit only for pregnancy and low confidence for quitting. These findings reflect those of Notley et al.'s⁶⁴ qualitative review of postpartum return to smoking, which similarly found that intending to quit only for pregnancy was an important issue. These point to the possibility of developing an intervention component focused on changing attitudes; however, further research is needed to understand more about women's views on these topics. A further factor likely to have a direct impact on smoking is nicotine dependence; future interventions that provide women with effective strategies to avoid triggers to smoking and manage cravings, including potentially with nicotine replacement therapy,⁹ may be beneficial.

A number of factors identified in this review are likely to have an indirect effect on PPRS but still may have implications for the intervention design. Breastfeeding, or intention to breastfeed, was commonly and strongly associated with PPRS. Qualitative research suggests that breastfeeding can motivate women to maintain abstinence from smoking; however, due to concern that smoking contaminates breastmilk, many women who resume smoking stop breastfeeding.^{64,69} Providing encouragement and incentives for breastfeeding could be an indirect means of enhancing smoking cessation during pregnancy and reducing PPRS.^{70,71} Research should further explore women's perspective on the relationship between smoking and breastfeeding, to help inform how this may be incorporated into interventions, and to consider whether approaches that attempt to modify attitudes towards breastfeeding are of benefit.

Depression, anxiety and stress may also have an indirect effect on PPRS. Interventions which attempt to address these factors have reported mixed effectiveness; those using depression-focused treatment⁷² and mood management through cognitive behavioural techniques^{73,74} have not been found to be effective. However, a motivation and problem solving intervention that included a wellness plan focusing on individualised treatment goals for salient concerns, such as anxiety, stress or depression, reduced PPRS.⁷⁵ Also using financial incentives for smoking cessation among depression-prone women improved both abstinence rates and depression ratings up to 24 weeks postpartum.⁷⁶ There may therefore be some benefit to including components that target mood and mental health in future interventions.

Only four studies examined factors associated with PPRS at more than one time point postpartum; however, there is evidence that some factors may be more important depending on timing. For example, not intending to breastfeed was found to be significant in early postpartum, but not in late postpartum.³¹ Breastfeeding is initiated soon after birth, with the proportion of women breastfeeding declining over time;⁷⁷ intervention approaches targeting breastfeeding might

therefore be best timed during pregnancy and early postpartum. Another study⁵⁷ reported 'thinking about the money saved to resist smoking' being significantly associated with avoiding PPRS at around 6 months postpartum but not at 6 weeks postpartum. Financial incentives are effective in promoting smoking cessation during pregnancy^{76,78,79} and research is needed to establish whether they reduce PPRS. This finding emphasises the importance of finance to women, and identifies a potential time point postpartum to target such interventions.

Conclusion

The development of new interventions to prevent PPRS can be informed by considering the direct or indirect impacts that factors exert on women's return to smoking and how such impacts could be successfully manipulated. This review suggests that the notable factors to be considered in this context are: having a partner or household member who smokes, intending to quit smoking only for pregnancy, having self-efficacy to quit long term, breastfeeding and depression.

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REFERENCES

1. Royal College of Physicians. *Smoking and the young. A report of a working party of the Royal College of Physicians*. London 1992.
2. Rostron B. Smoking-attributable mortality by cause in the United States: revising the CDC's data and estimates. *Nicotine Tob. Res.* 2013;15(1):238-246.
3. The NHS Information Centre. *Infant Feeding Survey 2010. Early results*. The National Health Service Information Centre for Health and Social Care; 2011.
4. Taylor AE, Howe LD, Heron JE, Ware JJ, Hickman M, Munafo MR. Maternal smoking during pregnancy and offspring smoking initiation: assessing the role of intrauterine exposure. *Addiction*. Jun 2014;109(6):1013-1021.
5. Hajek P, West R, Lee A, et al. Randomized controlled trial of a midwife-delivered brief smoking cessation intervention in pregnancy. *Addiction*. 2001;96(3):485-494.
6. Fingerhut LA, Kleinman JC, Kendrick JS. Smoking before, during, and after pregnancy. *Am. J. Public Health*. May 1990;80(5):541-544.
7. Harmer C, Memon A. Factors associated with smoking relapse in the postpartum period: an analysis of the Child Health Surveillance System Data in southeast England. *Nicotine Tob. Res.* May 2012;15(5):904-909.
8. Chamberlain C, O'Mara-Eves A, Oliver S, et al. Psychosocial interventions for supporting women to stop smoking in pregnancy. *The Cochrane Library*. 2013.
9. Coleman T, Chamberlain C, Davey M, Cooper SE, Leonardi-Bee J. Pharmacological interventions for promoting smoking cessation during pregnancy. *Cochrane Database Syst. Rev.* 2012(9).
10. Hajek P, Stead LF, West R, Jarvis M, Hartmann-Boyce J, Lancaster T. Relapse prevention interventions for smoking cessation. *Cochrane Database Syst. Rev.* 2013(8).
11. Jones M, Lewis S, Parrott S, Wormall S, Coleman T. Re-starting smoking in the postpartum period after receiving a smoking cessation intervention: a systematic review. *Addiction*. 2016;111(6):981-990.
12. Royal College of Physicians. *Passive smoking and children. A report of the Tobacco Advisory Group of the Royal College of Physicians*. 2010; <https://www.rcplondon.ac.uk/sites/default/files/documents/passive-smoking-and-children.pdf>, accessed 12.01.2015.
13. Coleman T, Agboola S, Leonardi-Bee J, Taylor M, McEwen A, McNeill A. Relapse prevention in UK Stop Smoking Services: current practice, systematic reviews of effectiveness and cost-effectiveness analysis. 2010.
14. Taylor M, Leonardi-Bee J, Agboola S, McNeill A, Coleman T. Cost effectiveness of interventions to reduce relapse to smoking following smoking cessation. *Addiction*. 2011;106(10):1819-1826.
15. Agboola S, McNeill A, Coleman T, Leonardi Bee J. A systematic review of the effectiveness of smoking relapse prevention interventions for abstinent smokers. *Addiction*. Aug 2010;105(8):1362-1380.
16. Thun MJ, Carter BD, Feskanich D, et al. 50-year trends in smoking-related mortality in the United States. *N. Engl. J. Med.* 2013;368(4):351-364.
17. Sims M, Tomkins S, Judge K, Taylor G, Jarvis MJ, Gilmore A. Trends in and predictors of second-hand smoke exposure indexed by cotinine in children in England from 1996 to 2006. *Addiction*. Mar 2010;105(3):543-553.
18. Leonardi-Bee J, Jere ML, Britton J. Exposure to parental and sibling smoking and the risk of smoking uptake in childhood and adolescence: a systematic review and meta-analysis. *Thorax*. Oct 2011;66(10):847-855.
19. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Ann. Intern. Med.* 2009;151(4):264-269.

20. Wells G, Shea B, O'Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp, accessed 07.05.2014. Accessed 07.05.2014.
21. Herzog R, Álvarez-Pasquin MJ, Díaz C, Del Barrio JL, Estrada JM, Gil Á. Are healthcare workers' intentions to vaccinate related to their knowledge, beliefs and attitudes? A systematic review. *BMC Public Health*. 2013;13(1):1.
22. Wells G, Shea B, O'Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Paper presented at 3rd symposium on systematic reviews: beyond the basics. Oxford, 2000.2000.
23. Higgins J, Green S, (Editors). *Cochrane Handbook for Systematic Reviews of Interventions, Version 5.1.0*. The Cochrane Collaboration;2011.
24. Businelle MS, Kendzor DE, Reitzel LR, et al. Pathways linking socioeconomic status and postpartum smoking relapse. *Ann. Behav. Med.* Apr 2013;45(2):180-191.
25. Correa-Fernandez V, Ji L, Castro Y, et al. Mediators of the association of major depressive syndrome and anxiety syndrome with postpartum smoking relapse. *J. Consult. Clin. Psychol.* Aug 2012;80(4):636-648.
26. Curry SJ, Grothaus L, McBride C, Lando H, Pirie P. Motivation for smoking cessation among pregnant women. *Psychol. Addict. Behav.* 2001;15(2):126-132.
27. Mullen PD, Richardson MA, Quinn VP, Ershoff DH. Postpartum return to smoking: who is at risk and when. *Am. J. Health Promot.* 1997;11(5):323-330.
28. Polanska K, Hanke W, Sobala W. Smoking relapse one year after delivery among women who quit smoking during pregnancy. *Int. J. Occup. Med. Environ. Health*. 2005;18(2):159-165.
29. Ratner PA, Johnson JL, Bottorff JL. Smoking relapse and early weaning among postpartum women: is there an association? *Birth*. Jun 1999;26(2):76-82.
30. Röske K, Hannover W, Grempler J, et al. Post-partum intention to resume smoking. *Health Educ. Res.* 2006;21(3):386-392.
31. Simmons VN, Sutton SK, Quinn GP, Meade CD, Brandon TH. Prepartum and postpartum predictors of smoking. *Nicotine Tob. Res.* Apr 2014;16(4):461-468.
32. Stotts AL, DiClemente CC, Carbonari JP, Mullen PD. Postpartum return to smoking: staging a "suspended" behavior. *Health Psychol.* Jul 2000;19(4):324-332.
33. Thyrian JR, Hannover W, Roske K, Rumpf HJ, John U, Hapke U. Postpartum return to smoking: identifying different groups to tailor interventions. *Addict. Behav.* Oct 2006;31(10):1785-1796.
34. Yoon JH, Higgins ST, Heil SH, Sugarbaker RJ, Thomas CS, Badger GJ. Delay discounting predicts postpartum relapse to cigarette smoking among pregnant women. *Exp. Clin. Psychopharmacol.* Apr 2007;15(2):176-186.
35. Solomon LJ, Higgins ST, Heil SH, Badger GJ, Thomas CS, Bernstein IM. Predictors of postpartum relapse to smoking. *Drug Alcohol Depend.* 2007;90(2):224-227.
36. Allen AM, Prince CB, Dietz PM. Postpartum depressive symptoms and smoking relapse. *Am. J. Prev. Med.* Jan 2009;36(1):9-12.
37. Carmichael SL, Ahluwalia IB. Correlates of postpartum smoking relapse. Results from the Pregnancy Risk Assessment Monitoring System (PRAMS). *Am. J. Prev. Med.* Oct 2000;19(3):193-196.
38. Colman GJ, Joyce T. Trends in smoking before, during, and after pregnancy in ten states. *Am. J. Prev. Med.* Jan 2003;24(1):29-35.
39. Gaffney KF, Henry LL, Douglas CY, Goldberg PA. Tobacco use triggers for mothers of infants: implications for pediatric nursing practice. *Pediatr. Nurs.* May-Jun 2008;34(3):253-258.
40. Gilbert NL, Nelson CR, Greaves L. Smoking cessation during pregnancy and relapse after childbirth in Canada. *J. Obstet. Gynaecol. Can.* Jan 2015;37(1):32-39.

41. Gyllstrom M, Hellerstedt W, Hennrikus D. The Association of Maternal Mental Health with Prenatal Smoking Cessation and Postpartum Relapse in a Population-Based Sample. *Matern. Child Health J.* 2012;16(3):685-693.
42. Kaneko A, Kaneita Y, Yokoyama E, et al. Smoking trends before, during, and after pregnancy among women and their spouses. *Pediatr. Int.* Jun 2008;50(3):367-375.
43. Ko M, Schulken ED. Factors related to smoking cessation and relapse among pregnant smokers. *Am. J. Health Behav.* 1998;22(2):83-89.
44. McBride CM, Pirie PL. Postpartum smoking relapse. *Addict. Behav.* 1990;15(2):165-168.
45. Prady SL, Kiernan K, Bloor K, Pickett KE. Do risk factors for post-partum smoking relapse vary according to marital status? *Matern. Child Health J.* Oct 2012;16(7):1364-1373.
46. Tran T, Reeder A, Funke L, Richmond N. Association Between Smoking Cessation Interventions During Prenatal Care and Postpartum Relapse: Results from 2004 to 2008 Multi-State PRAMS Data. *Matern. Child Health J.* 2013;17(7):1269-1276.
47. Yasuda T, Ojima T, Nakamura M, et al. Postpartum smoking relapse among women who quit during pregnancy: cross-sectional study in Japan. *J. Obstet. Gynaecol. Res.* Nov 2013;39(11):1505-1512.
48. Kahn RS, Certain L, Whitaker RC. A reexamination of smoking before, during, and after pregnancy. *Am. J. Public Health.* Nov 2002;92(11):1801-1808.
49. Rockhill KM, Tong VT, Farr SL, Robbins CL, D'Angelo DV, England LJ. Postpartum Smoking Relapse After Quitting During Pregnancy: Pregnancy Risk Assessment Monitoring System, 2000-2011. *J Womens Health (Larchmt).* May 2016;25(5):480-488.
50. Harmer C, Memon A. Factors associated with smoking relapse in the postpartum period: an analysis of the child health surveillance system data in Southeast England. *Nicotine Tob. Res.* 2013;15(5):904-909.
51. Hauge LJ, Torgersen L, Vollrath M. Associations between maternal stress and smoking: findings from a population-based prospective cohort study. *Addiction.* Jun 2012;107(6):1168-1173.
52. Kong GWS, Tam WH, Sahota DS, Nelson EAS. Smoking pattern during pregnancy in Hong Kong Chinese. *Aust. N. Z. J. Obstet. Gynaecol.* June 2008;48(3):280-285.
53. Lelong N, Kaminski M, Saurel-Cubizolles M-J, Bouvier-Colle M-H. Postpartum return to smoking among usual smokers who quit during pregnancy. *Eur. J. Public Health.* Sep 2001;11(3):334-339.
54. Lemola S, Grob A. Smoking cessation during pregnancy and relapse after childbirth: the impact of the grandmother's smoking status. *Matern. Child Health J.* Jul 2008;12(4):525-533.
55. Letourneau AR, Sonja B, Mazure CM, O'Malley SS, James D, Colson ER. Timing and predictors of postpartum return to smoking in a group of inner-city women: an exploratory pilot study. *Birth.* Sep 2007;34(3):245-252.
56. Levine MD, Marcus MD, Kalarchian MA, Houck PR, Cheng Y. Weight concerns, mood, and postpartum smoking relapse. *Am. J. Prev. Med.* Oct 2010;39(4):345-351.
57. McBride C, Pirie P, Curry SJ. Postpartum relapse to smoking: A prospective study. *Health Educ. Res.* Sep 1992;7(3):381-390.
58. O'Campo P, Faden RR, Brown H, Gielen AC. The impact of pregnancy on women's prenatal and postpartum smoking behavior. *Am. J. Prev. Med.* Jan-Feb 1992;8(1):8-13.
59. Park ER, Chang Y, Quinn V, et al. The association of depressive, anxiety, and stress symptoms and postpartum relapse to smoking: a longitudinal study. *Nicotine Tob. Res.* Jun 2009;11(6):707-714.
60. Park ER, Chang Y, Quinn VP, Ross K, Rigotti NA. Perceived support to stay quit: what happens after delivery? *Addict. Behav.* Dec 2009;34(12):1000-1004.
61. Polanska K, Hanke W, Sobala W, Lowe JB, Jaakkola JJ. Predictors of smoking relapse after delivery: prospective study in central Poland. *Matern. Child Health J.* Jul 2011;15(5):579-586.

62. Simonelli MC, Velicer WF. Cluster subtypes appropriate for preventing postpartum smoking relapse. *Addict. Behav.* Mar 2012;37(3):280-286.
63. Flemming K, McCaughan D, Angus K, Graham H. Qualitative systematic review: barriers and facilitators to smoking cessation experienced by women in pregnancy and following childbirth. *J. Adv. Nurs.* 2015;71(6):1210-1226.
64. Notley C, Blyth A, Craig J, Edwards A, Holland R. Postpartum smoking relapse—a thematic synthesis of qualitative studies. *Addiction.* 2015;110(11):1712-1723.
65. de Vries H, Bakker M, Mullen PD, Van Breukelen G. The effects of smoking cessation counseling by midwives on Dutch pregnant women and their partners. *Patient Educ. Couns.* 2006;63(1):177-187.
66. Pollak KI, Lyna P, Bilheimer AK, et al. Efficacy of a couple-based randomized controlled trial to help Latino fathers quit smoking during pregnancy and postpartum: The Parejas Trial. *Cancer Epidemiology Biomarkers & Prevention.* 2015;24(2):379-385.
67. Aveyard P, Lawrence T, Evans O, Cheng K. The influence of in-pregnancy smoking cessation programmes on partner quitting and women's social support mobilization: a randomized controlled trial [ISRCTN89131885]. *BMC Public Health.* 2005;5(1):1.
68. Wakefield M, Jones W. Effects of a smoking cessation program for pregnant women and their partners attending a public hospital antenatal clinic. *Aust. N. Z. J. Public Health.* 1998;22(3):313-320.
69. Flemming K, Graham H, McCaughan D, Angus K, Bauld L. The barriers and facilitators to smoking cessation experienced by women's partners during pregnancy and the post-partum period: a systematic review of qualitative research. *BMC Public Health.* 2015;15(1):849.
70. Lowry R, Billett A, Buchanan C, Whiston S. Increasing breastfeeding and reducing smoking in pregnancy: a social marketing success improving life chances for children. *Perspectives in public health.* 2009;129(6):277-280.
71. Morgan H, Hoddinott P, Thomson G, et al. Benefits of Incentives for Breastfeeding and Smoking cessation in pregnancy (BIBS): a mixed-methods study to inform trial design. 2015.
72. Cinciripini PM, Blalock JA, Minnix JA, et al. Effects of an intensive depression-focused intervention for smoking cessation in pregnancy. *J. Consult. Clin. Psychol.* 2010;78(1):44.
73. Levine MD, Marcus MD, Kalarchian MA, Cheng Y. Strategies to Avoid Returning to Smoking (STARTS): A randomized controlled trial of postpartum smoking relapse prevention interventions. *Contemp. Clin. Trials.* 2013;36(2):565-573.
74. Levine MD, Cheng Y, Marcus MD, Kalarchian MA, Emery RL. Preventing Postpartum Smoking Relapse: A Randomized Clinical Trial. *JAMA internal medicine.* 2016;176(4):443-452.
75. Reitzel LR, Vidrine JI, Businelle MS, et al. Preventing postpartum smoking relapse among diverse low-income women: a randomized clinical trial. *Nicotine Tob. Res.* 2010;12(4):326-335.
76. Lopez AA, Skelly JM, Higgins ST. Financial incentives for smoking cessation among depression-prone pregnant and newly postpartum women: effects on smoking abstinence and depression ratings. *Nicotine Tob. Res.* 2015;17(4):455-462.
77. Donath S, Amir LH. Relationship between prenatal infant feeding intention and initiation and duration of breastfeeding: a cohort study. *Acta Paediatr.* 2003;92(3):352-356.
78. Higgins ST, Solomon LJ. Some Recent Developments on Financial Incentives for Smoking Cessation Among Pregnant and Newly Postpartum Women. *Current addiction reports.* 2016;3(1):9-18.
79. Cahill K, Hartmann-Boyce J, Perera R. Incentives for smoking cessation. *The Cochrane Library.* 2015.