Children and Passive Smoking: A Review

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Background. For the past 40 years, evidence has been accumulating on the effects of passive smoking on the fetus and on children. Over this period, research methods have become more precise and accurate, with confounding factors controlled for and actual exposure to smoke measured and validated by cotinine tests on body fluids. This review follows the progress of these studies and assesses the weight of evidence for various health risks of passive smoking on children from before birth to adolescence.

Methods. Nearly 200 research papers published worldwide were reviewed.

Results. Effects of maternal smoking on the fetus include low birthweight, increased risk of spontaneous abortion, and perinatal death. The effect of maternal smoking on breast feeding is still enigmatic. Breast-fed infants of mothers who smoke appear to be protected against respiratory diseases but are subjected to chemicals from the smoke transferred in the milk. It is difficult to separate prenatal and postnatal effects with regard to growth, development, and lung function retardation. There is, however, a definite increase in respiratory diseases, otitis media, and minor ailments, which are unequivocally related to parental, especially maternal, smoking.

Conclusions. There is now sufficient evidence that health problems in children are related to maternal, and to a lesser degree paternal, smoking during pregnancy, and, after birth, to exposure to environmental tobacco smoke (ETS) in the home and daycare centers. Exposure to ETS should be noted on pediatric patients' problem lists and addressed at each visit.

Key words. Smoking; tobacco smoke pollution; fetal development; fetal growth retardation; maternal child nursing; tobacco; child welfare; respiratory tract infections. (*J Fam Pract 1994; 38:267-277*)

The term "passive smoking" usually refers to the inhalation of smoke that is either exhaled by a smoker or released as sidestream smoke from a burning cigarette. These two kinds of smoke in the atmosphere constitute environmental tobacco smoke (ETS). Another name for passive smoking is "involuntary smoking," because the person who inhales it often has no choice in the matter. In no case is this more true than for children. If parents smoke in the home, the child has no alternative but to be a passive smoker.

The fetus of a smoking mother also is an involuntary smoker. Although not actually inhaling ETS, the baby is a captive recipient of tobacco chemicals passed across the placenta from the mother. For this reason, a review of passive smoking in children must begin with the fetus.

Concern about the effects of smoke on infants began in the 1930s and 1940s when smoking prevalence among women in the western world was on the rise. This trend was given added impetus during the World War II. The focus of concern and research at that time was the effect of breast-feeding by smoking mothers. A study published in 1942 concluded that, although chemicals from smoke were passed to the baby by the nursing mother, they were probably not a cause for concern.[1] This early conclusion has since been revised.

In the 1950s, the first of many studies on birthweight and prematurity related to maternal factors revealed a consistent pattern of low birthweight (small for dates) babies and premature birth in women who smoked. It was a crucial finding. Many of the problems

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subsequently found in babies whose mothers smoked during pregnancy appear to be related to low birthweight and the underlying retardation of fetal development.

As research on the topic of involuntary smoking evolved, methodologies have become more refined and a wide range of conditions and possible causative factors have been examined. Many early studies considered only the effect of the smoking factor. Later, the frequency of maternal smoking was included, along with many other background factors in the home, parents' physique and health problems, chemical or physiological processes involved, and biochemical measures of the absorption of smoke by the infant or older child.[2–11] Studies have become larger and more rigorously controlled. The resulting conclusion is that numerous conditions and health problems in infants and children are consistently related to passive smoking in the womb or in the environment. The purpose of this paper is to examine the evidence and to present a review of current knowledge with regard to children and passive smoking.

Fetal Growth and the Outcome of Pregnancy

Since 1957, evidence has been accumulating that has established that maternal smoking in pregnancy adversely affects the growth of the fetus and is associated with increased risk of miscarriage, premature birth, and perinatal death.[12] Low birthweight, which refers to infant size that is small for the gestational age, is always a disadvantage.

Smoking in pregnancy appears to increase the risk of having a small-for-gestational-age baby by as much as three times,[13] and babies of mothers who smoke weigh an average of 200 g less than those of nonsmoking mothers.[14,15] Over the past 35 years, many investigations of smoking in pregnancy and low birthweight have been carried out, and smoking has consistently emerged as the major factor related to increased risk.[16–19]

Although reservations have been expressed that socioeconomic status and other sociodemographic conditions could lead to a false implication of smoking,[20] smoking remains a significant variable even when background is taken into account. Studies have included socioeconomic status and psychosocial stress,[21–25] and other habits such as alcohol or caffeine consumption[26–29] during pregnancy, but smoking is the only factor consistently associated with low birthweight. One study showed that the risk of low birthweight increased even further in women smokers who also had a high intake of caffeine and alcohol during pregnancy.[28] Another study suggested a combined effect of smoking and drinking. The mean reduction in birthweight was 500 g in mothers who smoked and drank heavily during pregnancy as compared with those of nonsmoking, nondrinking mothers.[30]

Smoking in older mothers appears to be more harmful to the fetus than in younger women.[13,31,32] One study showed that the risk of a smoking mother over 35 years old having a small-for-gestational-age baby is five times that of a nonsmoking mother of the same age.[13] In mothers under the age of 17 years, the risk of a smoking mother having a low birthweight baby is at least twice as great as that of a nonsmoker. Because the increase in risk is significantly higher for all age groups, this discrepancy does not imply that it is safe for younger mothers to smoke.

Mothers who stopped smoking 6 to 16 weeks into pregnancy gave birth to heavier babies than those who stopped later than 16 weeks, who, in turn, gave birth to heavier babies than those who smoked throughout their pregnancy.[33] Therefore, it appears that the greatest effect of smoking on birthweight is in the second and third trimesters of pregnancy.

There is now evidence that passive smoking by the mother during her pregnancy as a result of her partner's smoking is also related to the baby being small for gestational age.[19,34–36] There has also been at least one report of sperm damage caused by the father's smoking.[37]

The effects of maternal smoking on fetal growth could include chemical or biochemical toxicity, physical damage to the placenta or other tissues and organs, secondary effects related to poor dietary habits, changes in blood flow, or a combination of these.

The ultrastructure of the placenta itself has been examined, along with functional changes that occur as a result of smoking.[38–40] No clear pattern of structural alterations has emerged except for uneven thickening of the trophoblastic basement membrane and narrowing of the fetal capillaries.[41] Although these changes could reduce oxygen and nutrient diffusion, it seems unlikely that the major reason for the low birthweight of smokers' babies lies in placental structure or function.

It is more likely that placental functional changes are biochemical, ie, the effect of carbon monoxide on oxygen availability or the effect of nicotine as a vasoconstrictor in the uteroplacental vasculature.[42] A dose-response relation has been shown, with smoking rates of 10 to 20 cigarettes a day being associated with lower birthweight than rates of under 10 per day.[43] The type of cigarette smoked also affects the risk of low birthweight.[44] Recent studies have measured maternal nicotine intake by assessing the level of cotinine in body fluids. Cotinine is the principal breakdown product of nicotine in the body. Measured as two different indices, a reduction of 25 g in

birthweight has been found for each microgram of urinary cotinine per milligram of creatinine, or a reduction of 12 g for every cigarette smoked per day.[45]

Delayed development has been associated not only with low birthweight but also with spontaneous abortion or miscarriage up to 28 weeks' gestation.[46–48] It has been calculated that the rate of spontaneous abortions in smoking mothers is 27% higher than in nonsmoking mothers even when other variables are taken into account.[49] Unlike other spontaneous abortions, in which chromosomal abnormalities are often found in the fetus, miscarriages in younger women smokers are likely to be normal fetuses in this respect.

Developmental factors also could be involved in miscarriage or preterm delivery in smokers. Several studies over the past decade have investigated possible mechanisms for spontaneous abortion and for perinatal mortality, which is also more frequent in mothers who smoke.[51–53] Preterm delivery is closely related to an increased risk of perinatal death.[54] As with low birthweight, preterm delivery is more frequent in older than in younger mothers who smoke and is related in some cases to early placental separation or displacement.[55–57]

A recent study has shown a significantly increased risk in the second and third trimester associated only with the mother's, not the father's, smoking.[58] Research measuring the serum cotinine of smoking mothers has shown a clear link between smoking and perinatal death, even when other factors associated with increased risk, such as socioeconomic status, maternal age, and previous perinatal deaths, are taken into account.[59,60]

Other investigations of causes of low birthweight, miscarriage, and preterm delivery have considered DNA damage in the placenta,[61] placental cadmium and zinc,[62] reduced umbilical artery prostacyclin formation,[63] and histological changes in the placenta, cholesterol levels,[64] and IgE levels.[65,66] More research is needed to identify the exact mechanisms.

It is often impossible to separate the effects of exposure of the fetus to smoke from those of passive smoking after birth because problems of early childhood can be both developmental and environmentally induced. Nevertheless, neonates of smoking mothers have been shown to be shorter and to have a smaller head circumference than those of nonsmoking mothers,[67] which could be a factor in the growth and developmental delays occurring in childhood. There is also evidence of lung growth impairment in fetuses of smoking mothers.[68] If babies start life with this disadvantage and subsequently are exposed to parental smoke in the home, the problem is compounded.

Subsequent Progress of the Child Related to Smoking During Pregnancy

Maternal smoking in pregnancy appears to have numerous effects on child development. At least three studies have shown an effect on mental development,[69] including an association with a reduced cognitive functioning by age 3 and a reduction in vigilance in the young child.[70–71]

Child growth has been shown in some studies to be reduced when the mother smoked during pregnancy,[72–76] which might be related to lower birthweight.[77] As with other smoking-related problems, many other factors, including height of the parents, socioeconomic status,[78] and passive smoking in childhood,[79,80] also must be taken into account.

Sudden infant death syndrome (SIDS) is a case in point.[81–86] Some diseases of childhood appear to be related to smoke exposure in utero as well as at home. Although many factors are known to be involved in SIDS, it has been calculated that one quarter of the risk is related to maternal smoking.[87] Research in the past few years has linked both smoking during pregnancy and exposure of the infant to environmental tobacco smoke with SIDS. Recent evidence indicates a relation between maturation of the fetal lungs,[68] infant lung function,[88] and maternal smoking during pregnancy. Since newborn babies of smoking mothers have lower Apgar scores,[89] it seems quite probable that some of the risk of crib death could lie in prenatal development.

There is also evidence of an increased risk of various cancers in children whose mothers smoked during pregnancy.[90–93] In some cases, the association was weak or the studies involved small numbers. A cumulative effect of exposure to passive smoking has been observed to increase the risk of cancer, especially of lung cancer, as might be expected.

Apart from physical harm to infant and mother, health care related to smoking in pregnancy is very costly[94] as well as anxiety-producing for the family.

There is no doubt that smoking during pregnancy increases the risk of low birthweight, miscarriage, and perinatal deaths, and that some of the harmful effects related to perinatal development and low birthweight are carried forward and become cumulative if the parents smoke in the home.

Breast-feeding

About two thirds of the women who quit smoking during pregnancy resume the habit after their baby is born,[95] which raises not only the question of the effect of

ETS on the infant but also the old issues of breast feeding by smoking mothers.

There is no doubt that chemicals are passed to the baby in the breast milk of mothers who smoke.[96–99] Evidence of this process was substantiated by one study in which the marker cotinine was present in the blood and urine of babies who were breast-fed by smoking mothers but not exposed to ETS.[97] The relation between cotinine in the baby's body fluids and the mother's milk supplies further evidence.[98,99] It is also known that if the mother smokes while nursing, the level of cotinine in the baby's fluids is high.[100]

A recent review[101] considers whether maternal smoking is independently associated with breast- or bottle-feeding, and whether health and development differ in breast- and bottle-fed children of smokers. Nonsmoking mothers have been shown to be more likely than smokers to breast-feed their children and to continue doing so longer.[102,103] The review suggests that breast-feeding even in smoking mothers is associated with a reduced risk of respiratory diseases in the baby,[104,105] although some studies have not shown this protective effect.[106] Breast-feeding by mothers who smoke has been found to have little or no association with most problems. In cases of squint and educational achievement deficiencies, for example, it might be associated with a slight exacerbation of a preexisting condition. Nevertheless, breast-feeding should be recommended to all mothers. It should be noted that smoking appears to reduce lactational capacity,[107] and a smoke-free pregnancy and nursing period should be encouraged rather than focusing on the pregnancy alone.

Children's Exposure to Environmental Tobacco Smoke in the Home

Children whose parents smoke often have also been exposed to smoke during fetal development, thus making it difficult to separate the effects of those two periods of passive smoking. The closeness of mother to infant compounds this problem. Because in most households, children are at home with their mother for a majority of their infancy and childhood, it is not surprising that most studies on the effect of passive smoking in the home have shown the mother's smoking to be most significantly related to the child's health problems.

There is evidence, however, that the physical distance between the new baby and the smoking mother or other family members[108] as well as the amount smoked correlates with the amount of cotinine in the baby's urine.[2] Schoolchildren in Japan were found to have increasing amounts of cotinine in their urine directly related to the number of cigarettes they were exposed to in their homes each day.[11] The correlation between the amount of exposure to passive smoking and the amount of cotinine in the saliva[3] or plasma[109] has even been found in adolescents, and with regard to respiratory diseases, otitis media, and several other health problems, the amount of exposure to passive smoking has been associated with the risk of these diseases.

Respiratory Diseases

Two of the earliest studies on passive smoking and infants conducted in 1974 found an increased risk of hospital admission for bronchitis and pneumonia in infants whose parents smoked.[110,111] The risk was highest when both parents smoked and lowest when neither smoked. These and other studies on hospital admissions suggest the possibility that smoking parents who have respiratory diseases themselves infect their children by contaminating the air through coughing.[112] If this is true, and it seems likely in view of the similarities between parent and infant symptoms,[113,114] it would further increase the risk of respiratory problems in infants of smoking parents. Nonsmoking parents also may be more health conscious than smokers are. A study has shown that nonsmoking parents and their children were more likely to attend clinics for immunization and take other preventive measures than were smokers and their children.[115]

With regard to hospitalization, a large study in Finland showed that infants of smoking parents were hospitalized more often and for longer periods than those with nonsmoking parents.[116] This study also showed that the death rate among children of smokers was higher than that of nonsmokers' children between the ages of 1 month and 5 years.

It is in the area of respiratory disorders and symptoms that the case against passive smoking is probably most conclusive.[117] By the early 1980s, general problems associated with the lungs and the respiratory tract of infants and young children with smoking parents were confirmed by numerous research studies.[118–122]

Although early studies associated passive smoking with respiratory illness only in infants,[123] research is now showing a continued effect through primary school to adolescence. For example, in children aged 5 to 11 years, wheezing, coughing, and bronchitis were shown to be related to the number of cigarettes smoked by the parents.[124,125] Coughing has been found in children whose parents smoke.[126,127] The effect of passive smoking is most marked in children under the age of 11 years but is still observable in adolescents up to age 18. The risk of frequent coughing increases with the number of smokers in the household.

When assessing the effect of passive smoking, it is important to take into account factors such as type and size of house, urban or rural location, type of cooking fuel used, and health history of the family. Even when all these confounding elements are accounted for, most research has shown that passive smoking is still associated with a higher risk of respiratory problems.

There is little evidence that passive smoking causes asthma in children,[128,129] but considerable evidence that it exacerbates a preexisting asthmatic condition. A survey of 4000 children showed that the incidence of recurrent wheezing increased by 14% when the mothers smoked more than four cigarettes per day and by 49% when they smoked 15 or more cigarettes daily.[130] Hospital visits, but not admissions, for asthmatic children increased by 63% if one or more family members smoked.[131] Studies of asthmatic children have shown that those of smoking mothers had 13% to 23% lower lung function indices than those of nonsmoking mothers,[132] and that the maternal smoking effect was greater on boys than girls, on older than younger children,[133] and in children with atopic dermatitis,[134] and that the effect severity was subject to seasonal variations.[135] Higher urinary cotinine levels have been found in asthmatic children compared with those of same-age nonasthmatics, suggesting greater exposure to passive smoking.[136] Other factors, such as living in a damp home, use of gas for cooking, use of a humidifier, and exposure to ETS, also contribute to the severity of asthma in children.[137] More current research into the topic of asthma and passive smoking has confirmed the association found in earlier studies.[138]

Although asthma is the most common chronic illness of childhood, bronchitis with wheezing also is fairly frequent. The risk of severity of bronchitis in children has repeatedly been shown to increase or the disease to be exacerbated when parents smoke.[139–146] Wheezing is more frequent in children who live in smoking households, with maternal smoking having a greater influence than that of fathers. A study in Canada showed that the risk of bronchitis in children whose mothers smoked was three times greater when compared with that of children with nonsmoking mothers.[147] Various research regarding the interaction between breast-feeding and risk of bronchitis in children has found indicators that breast-feeding may have a protective effect with regard to wheezing and other childhood respiratory disorders, even in smoking households.[148,149] Only a few studies have failed to show an association between respiratory symptoms and family smoking.[150–152]

Respiratory problems often can be associated with lung function.[153] Lung development starts in utero, and some research has addressed the carry-over effects while others have not separated antenatal and postnatal influences.[154] One study showed that there appeared to be a greater association between respiratory disease in children and fetal exposure to smoke than with exposure to ETS after birth.[155] This finding leads to a consideration of lung function and passive smoking.[156]

Lung Development and Function

There have been more than a dozen studies on lung function in children in relation to exposure to parental smoking, which have revealed an overall reduction of 1% to 5% lung volume. The findings are varied, however, largely as a result of the difficulty of establishing a meaningful measure for lung function. The usual indices are forced expiratory volume (FEV) and bronchial hyperresponsiveness, and, of course, many other factors also affect these conditions. Longitudinal and long-term studies, which are the most useful, have enabled a baseline FEV to be established and changes measured. Another problem in lung function studies is that of setting levels of exposure to tobacco smoke. Salivary cotinine has been used to assess this factor,[149] and it seems likely that future longitudinal studies might use cotinine measures as an ongoing record of smoke exposure.

The changes in lung function observed so far are not large enough to be considered illnesses,[153] but definitely are associated with passive smoking. Growth in lung volume was found to be impaired,[156–159] but recent studies indicate that the impairment appears to vary with gender. For example, the FEV/VC (ventilatory capacity) ratio was found to be affected in boys,[160] and the FEV in boys aged 13 to 16 showed slower growth if parents smoked.[161] This effect was not found in girls.

Bronchial hyper-responsiveness has been found in some studies,[162,163] but not in others.[11] Two recent papers provide a valuable up-to-date review of the relation between lung function in children and exposure to ETS.[11,87]

Growth and Development

The effect of ETS on a child's long-term growth and development is still relatively unclear. There are a number of reasons for this uncertainty, including the initial effect of low birthweight and the genetic, social, and cultural factors that interact in the developmental processes. As in studies on lung development, there is also the issue of how exposure to smoke is measured. A review of all the major studies on these aspects of growth and development through 1990 that was published recently[164] provides a balanced assessment of the situation. It appears that exposure to maternal smoking during pregnancy is

associated with 1 cm to 2 cm less height in children when other factors are taken into account.

Studies on exposure to smoke after birth have met with varied results. A recent large-scale analysis of three samples found no such effect.[10] It seems likely that some of the apparent effect is associated with pregnancy. For example, 3-year-old children whose mothers stopped smoking in pregnancy scored higher on certain ability tests than those of mothers who continued to smoke during pregnancy. Although 3-year-old children of mothers who had stopped smoking in pregnancy were taller and heavier than those of mothers who had continued to smoke, the apparent effect was not so great when the size of the baby at birth was taken into account. Another study showed that the height of 6- to 10-year-olds was related to the number of cigarettes smoked by the mothers, but the significant factor appeared to be in utero exposure rather than ETS after birth. Another study linked the number of cigarettes smoked in the home and the height of children, but this finding is also influenced by factors other than passive smoking.[79]

Studies have shown lower cognitive scores,[165] lower ability of less advanced reading, verbal, or mathematical skills, and more behavioral problems,[166] especially in children whose mothers smoked during pregnancy.[68–70,77] However, social class and the interaction of lifestyle factors must be untangled from the smoking issue before definite conclusions can be reached.

Otitis Media

The most frequent cause of deafness in children is secretory otitis media, middle ear effusion, or "glue ear." Not only does it often require surgery for the insertion of grommets, but also it can cause delay in language development and sometimes result in delay in educational progress.[167] One of the early studies to demonstrate the role of passive smoking showed that of children aged 2 to 3 years who had had three or more attacks of otitis media, only one third had nonsmoking parents. The reverse was true for children who had no middle ear diseases.[168]

There are a number of factors associated with an increased risk of middle ear disease and these have been taken into account from the earliest investigations.[169] A 1983 survey showed that when 76 children who were admitted to a hospital for the insertion of grommets were compared with a control group, the children with middle ear disease were significantly more likely to have at least one adult smoker in their homes. The increased risk appeared to be a combination of atopy and catarrh with passive smoking.[170] Other studies have also shown that children with at least one smoking parent were at increased risk of requiring surgery for otitis media.[171]

The increased risk is not only in infants. It has also been shown in children up to age 9. Black[172] showed an increased risk in 150 children aged 4 to 9 years of requiring surgery for "glue ear" related to parental smoking. Iverson and associates[173] found that parental smoking was the only home factor increasing the risk of middle ear effusion, accounting for 36% of cases in 6- and 7-year-olds.

Earlier studies counted the number of smokers in the household and the number of cigarettes smoked, but more recent research has measured the cotinine levels in the children's saliva and shown a convincing link between high levels of cotinine and increased risk of middle ear effusion in 7-year-olds.[174] Recent studies continue to confirm the association between passive smoking exposure and increased risk of middle ear effusion.[175]

Very few of the numerous studies carried out in the last 10 years on the risk of middle ear effusion related to passive smoking have not found a significant association,[176] and it can now be stated conclusively that exposure to environmental tobacco smoke in the home and in daycare[177] increases the risk of middle ear effusion in children. Overall, one third of the cases of "glue ear" can be attributed to parental smoking.

Other Health Problems

Repeated minor ailments can present real problems to children's educational progress. It is possible that some of the underachievement that appears to be related to passive smoking is actually caused by missing school or feeling unwell in class. At least one study has shown that whatever children's own smoking habits, parental smoking increased their risk of school absence on a randomly selected day.[178] Another showed that a "pack-a-day" smoker in a household increased the number of bed-disability days for young children by 20%.[179]

The effect of parental smoking on children has been associated with an increased risk of rhinitis, coughs, sputum, and having to have tonsils extracted.[180] The percentage of children experiencing four or more sore throats a year was directly related to the number of cigarettes smoked per day by the mother.[181] The latter study found that about half the children of nonsmokers, two thirds of those whose mothers smoked from 1 to 19 cigarettes per day, and three quarters whose mothers smoked 20 or more per day had four or more sore throats per year. Colic and alimentary problems,[182,183] tonsillitis,[184] and febrile seizures[185] have also been shown to be more frequent in children exposed to ETS in the home.

There is preliminary evidence of changes in the

cardiovascular system of children exposed to passive smoke. Changes in the lipoproteins[109] and oxygen transport in the blood of adolescents has also been shown. These changes could increase the risk of premature cardiovascular diseases.

Although cystic fibrosis is not caused by passive smoking, it can to be exacerbated by it.[186] One study found that children with cystic fibrosis whose mothers smoked needed more treatment than those of nonsmoking mothers,[187] while a second study showed that children with cystic fibrosis fared better in a nonsmoking atmosphere.[188]

Cancer Risk

A question frequently asked is whether passive smoking in childhood increases the risk of cancer in later life. The section on smoking during pregnancy includes a discussion of the initial tentative findings of an increased risk of childhood cancers in children born to these smoking mothers. There is also retrospective evidence that ETS exposure during childhood increases risk of cancers, but it is difficult to assess because of the extremely long latent period, especially with regard to lung cancer and the actual appearance of lesions and symptoms.[189]

When as many factors as possible were accounted for, at least three studies have shown positive links between passive smoking in childhood and lung cancer in later life. In the first piece of research, maternal smoking was the critical factor.[190,191] In both, the backgrounds of the lung cancer patients were compared with those of a matched control group.

Household exposure to 25 or more "smoker years" (the number of years living with smokers multiplied by the number of smokers; eg, 12½ years with two smokers) in childhood and adolescence appears to double the risk of lung cancer in lung cancer patients who never smoked.[192] Less than 25 "smoker year" exposure did not appear to increase the risk.

Conclusions

Based on the numerous excellent and detailed reviews of passive smoking effects in general[193–197] and on the fetus, infants and children in particular[198] that have been published over the past 10 years, the fact that children's health is at risk because of passive smoking is unquestionable. As public awareness grows and more assistance and advice is required by patient populations and the community in general, family physicians should recognize and be prepared to deal with the significant health implications of passive smoke on our society.[199]

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