# Passive smoking in children: facts and public health implications

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**التدخين القسري بين الأطفال: الحقائق والعواقب**

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**الخلاصة:** تقيس هذه الدراسة نسبة الكوتينين إلى الكرياتينين لدى ستين طفلاً ممن يدخن أبواهم، وعشرين طفلاً ممن يمتنع آباؤهم عن التدخين. وقد أظهرت الدراسة أن الأطفال المعرضين للتدخين القسري أكثر تعرضاً من غيرهم من الأطفال الشواهد، ومن بين هؤلاء الأطفال المعرضين للتدخين القسري كانت الإناث أكثر تعرضاً من غيرهن. وكان 35٪ من عبء الكوتينين على السكان يتركز في المجموعة التي تقل أعمارها عن السنتين وكان التعرض يتناقص بتقدم عمر الطفل. إن الأطفال المعرضين للتدخين القسري ممن يدخن آباؤهم أنواعاً (خفيفة) لديهم مستوى تعرض أكبر ممن يدخن آباؤهم أنواعاً عادية. وينبغي أن تزيد البرامج التثقيفية من توعية الناس حول التأثيرات الضارة لدخان التبغ في البيئة، ويجب على الأطباء تقديم النصح للوالدين المدخنين للتوقف عن التدخين بدلاً من التحول إلى الأنواع "الخفيفة" من الدخان.

ABSTRACT This study measured cotinine/creatinine ratios among 60 children in Alexandria, Egypt whose parent(s) smoked and 20 control children whose parents reported not smoking to show that passive smoker children were more exposed than controls. Among the passive smoker children, girls were more exposed, 35% of the population burden of cotinine was among those aged 0–2 years, and exposure significantly decreased with the child's age. Passive smoker children whose fathers smoked "lighter" cigarettes had higher exposure levels than those whose fathers smoked regular brands. Educational programmes should increase awareness of the ill-effects of environmental tobacco smoke, and health professionals should advise parents who smoke on quitting smoking rather than switching to a "lighter" cigarette brand.

**Le tabagisme passif chez les enfants : faits et implications**

RESUME Le tabagisme parental est la source la plus importante d'exposition passive à la fumée de tabac chez les jeunes enfants. Cette étude a mesuré objectivement le taux de cotinine/créatinine chez 60 enfants dont le père/la mère où les deux parents fumaient et 20 enfants témoins dont les parents déclaraient ne pas fumer, afin de montrer que les enfants de parents fumeurs étaient davantage exposés que les enfants témoins. Parmi les enfants de parents fumeurs, les filles étaient davantage exposées, 35 % du poids de la cotinine dans la population se trouvant dans le groupe d'âge de 0 à 2 ans, et l'exposition diminuait considérablement avec l'âge de l'enfant. Chez les enfants dont le père fumait des cigarettes « légères », le niveau d'exposition était plus élevé que chez ceux dont le père fumait des cigarettes normales. Des programmes éducatifs devraient accroître la sensibilisation vis-à-vis des effets néfastes de la fumée de tabac dans l'environnement, et les professionnels de la santé devraient conseiller aux parents qui fument d'arrêter de fumer au lieu de changer pour un type de cigarettes « légères ».

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## Introduction

*Nicotiana is named after Jean Nicot (1530-1600), French consul to Portugal, who is said to have first presented tobacco to the courts of Portugal and France.* Tabacum *is the latinization of an American name from which comes the word tobacco* [1]

Smoking, chewing and nasally insufflating (as snuff) the dried leaves of the tobacco plant Nicotiana tabacum are the only significant sources of nicotine in man, and tobacco smoke is probably the most important source of air pollution in the home [2]. Programmes and policies to reduce exposure to environmental tobacco smoke (ETS) in public places and work-places have been implemented in many countries [3]. Less attention has been directed to minimizing ETS exposure in home environments, in particular, the exposure of children [4].

The Eastern Mediterranean Region of the World Health Organization (WHO) consists of 22 Member States incorporating North African countries, countries in the Arabian peninsula and some Asian countries. According to 1998 surveys, smoking among adult men was highest in Tunisia, closely followed by the Republic of Yemen, and it was lowest in Oman [5]. Although cigarette smoking among women in the Eastern Mediterranean is less common than among women from other regions, Lebanon recorded the highest smoking prevalence among women at about 35%. Apparent increases in cigarette consumption have been associated with times of national crisis and increased access to the western media throughout the Region (television, cinema, magazines and satellite television channels) [6]. In Egypt, where the present study was undertaken, the prevalence of smoking in adult males (18 years and older) was about 43.6% in 1997 [6], with a per capita annual cigarette consumption of 1275 cigarette sticks [7].

Ill health effects of ETS in children, such as upper respiratory tract irritation, acute bronchitis and pneumonia, and wheezing illnesses [8] have been linked to passive smoking. Involuntary smoking causes new cases, additional episodes and increased severity of asthma, fluid in the middle ear, and a small but significant reduction in lung function [4]. Some studies have associated lung cancer and ETS [9], and the combination of ETS and a missing gene may make some women up to six times more likely to develop cancer if they are exposed to second-hand smoke. A recent comprehensive meta-analysis [10] documented that the public health impact of children's exposure to ETS is substantial and that the economic impact is considerable [11,12].

Nicotine is a powerful psychoactive drug and, fundamentally, smoking is drug taking. Thus, for the consumer, low-yield cigarettes raise the issue of drug "cutting". Despite the publicity about the disease risks of smoking, there seems to be a widespread belief that low-yield cigarettes are less hazardous [13]

The aim of this study was to investigate the levels of urinary cotinine [employing the urinary cotinine/creatinine ratio (CCR)] in young children exposed to passive smoking and to explore its relation to the nicotine yield of the cigarette that the parent/s smoke.

## Methods

### Participants and setting

A controlled study was conducted in 1991 on 60 children exposed to passive smoking in their homes, and their smoker parent/s. These were compared to 20 control children, reported not be exposed to ETS, and their non-smoker parents. The families under investigation were low-income families, living in standard-sized government apartments located in a single, high-rise building in the urban fringe of the city of Alexandria, Egypt. This setting was chosen in order to minimize potential confounding variables due to social class differences, parental education, or apartment size factors and it's the related ventilation characteristics that might indirectly influence the results [*14*].

The study received ethical clearance from the Research Committee of the Faculty of Medicine at the University of Alexandria and verbal consent was obtained from the participating families.

Sixty (60) passive smoker children (PSC) from 48 families, comprising three age brackets (0–2, > 2–6 and > 6–10 years), were included in the study (20 children in each bracket). From the same apartment building, another 20 children of matched age and sex not exposed to household tobacco smoke and their non-smoker parents were included as a control group. Breastfed infants of smoker mothers were excluded from the study due to the documented excretion of nicotine in mother's milk. A general medical examination of all children was undertaken prior to the study in order to exclude those with hepatic and renal problems because of the effects of such conditions on the metabolism and excretion of nicotine and hence its urinary levels.

### Measures of passive smoking: urinary CCR

Several measures of passive smoking are cited in the literature. Carbon monoxide concentration in expired air detects acute exposure but does not reflect the longer-term exposure because of its short half-life. Nicotine levels are also a poor exposure guide due its rapid metabolism to cotinine and hence its short half-life. Although the longer half-life of thiocyanate makes it an attractive indicator, the concentration in the body is influenced by diet, rendering its use as a marker for passive smoke exposure limited. On the other hand, cotinine, the major metabolite of nicotine, is specific for tobacco exposure, has high blood concentrations that fluctuate little making the time of measurement less critical, remains long after the cessation of smoking, and its urinary excretion is less influenced by urinary flow and pH [*15*]. Collectively, these characteristics render urinary cotinine a valid, reliable and specific marker for nicotine exposure [*16*]. Accordingly, urinary cotinine was used in this study as a non-invasive objective measure of passive smoke exposure in order to assess: 1) whether PSC had higher levels of cotinine, and 2) the relation between the nicotine yield of the cigarette that the parent/s smoked and the child's cotinine level. The biochemical measures employed in this study allowed recent exposure to be estimated directly.

### Procedures

All participating families completed a short questionnaire verifying the smoking status of the father and other family members. This supplied information on the presence of other smokers in the home other than the parent/s and the number and the nicotine

content of the cigarettes smoked (further verified from the reported brand). Total daily nicotine intake was calculated by multiplying the number of cigarettes smoked by the nicotine content of the brand. Most parents in the sample smoked a well-known cigarette brand manufactured locally in two different strengths (low and high nicotine contents of 1 or 2 mg/cigarette respectively).

Virtually all the participating smoker families reported that only the father smoked and that there were no other smokers in the household. Hence urine samples were obtained from the children and their fathers and were frozen. Creatinine was measured colorimetrically [*17*] using a Boehringer Mannheim Test-Combination (Code No. 124192) and expressed in mg/100mL. Quantitative measurement of cotinine was carried out using a double antibody I125 radioimmunoassay [*18*] (Double Nicotine Metabolite, Diagnostic Products Corporation, Code No. KCTD), read using a gamma camera and expressed in ng/mL. Finally CCR was calculated and expressed in ng/mg.

### Statistical analysis

The *t*-test, the *F*-statistic and the correlation coefficient ($r$) were used as tests of significance. The level of significance was considered at $P < 0.05$.

## Results

### Children

The age and sex distribution of the controls and PSC are shown in Table 1. In both groups, males were generally slightly overrepresented (about 60%). However, in the older age bracket (> 6–10 years) both sexes were equally represented in the PSC and more females were represented in the controls.

Table 1 **Age and sex distribution of controls and passive smoker children (PSC)**

| Age group (years) | Controls | | | PSC | | |
|---|---|---|---|---|---|---|
| | M | F | Total | M | F | Total |
| 0–2 | 3 | 2 | 5 | 14 | 6 | 20 |
| >2–6 | 5 | 1 | 6 | 13 | 7 | 20 |
| >6–10 | 4 | 5 | 9 | 10 | 10 | 20 |
| Total | 12 | 8 | 20 | 37 | 23 | 60 |

*M = males.*
*F = females.*

The variables under investigation, namely the levels of creatinine and cotinine and CCR were then compared (Table 2). There was no difference in the creatinine levels between PSC and controls, but PSC had significantly higher levels of cotinine and CCR was significantly higher. When the same comparison was broken down by age group (Table 3), no difference in creatinine level was found, but again PSC had significantly higher levels of cotinine and CCR for all the age brackets that were examined. The only exception was in the cotinine levels of the youngest age group (0–2 years), where no significant difference was observed between the controls and PSC.

When the same comparison was broken down by sex (Table 4), creatinine levels were no different, but PSC had significantly higher cotinine and CCR than controls regardless of sex. Further gender analysis was then undertaken separately for each of the PSC and controls. Within the controls, there were no creatinine, cotinine and CCR differences according to sex. However, within the PSC, females had higher cotinine levels, and the difference was close to statistical significance ($P = 0.06$) and significantly higher CCR levels

Table 2 **Levels of creatinine, cotinine and cotinine/creatinine ratio (CCR) of controls and passive smoker children (PSC)**

| Parameter | Controls Mean (*s*) | PSC Mean (*s*) | *P*-value |
|---|---|---|---|
| Creatinine (mg/100mL) | 0.664 (0.1643) | 0.658 (0.359) | 0.95 |
| Cotinine (ng/mL) | 22.58 (17) | 78.67 (63) | 0.0001 |
| CCR (ng/mg) | 32.51 (20.8) | 142.89 (115.1) | 0.0001 |

s = *standard deviation.*

Table 3 **Levels of creatinine, cotinine and cotinine/creatinine ratio (CCR) of controls and passive smoker children (PSC) by age group**

| Parameter | Age group (years) | Controls Mean (*s*) | PSC Mean (*s*) | *t*-test |
|---|---|---|---|---|
| Creatinine (mg/100mL) | 0–2 | 0.56 (0.2) | 0.49 (0.2) | 0.6 |
| | >2–6 | 0.7 (0.1) | 0.74 (0.3) | 0.26 |
| | >6–10 | 0.7 (0.2) | 0.76 (0.5) | 0.35 |
| Cotinine (ng/mL) | 0–2 | 17.32 (13.4) | 87.4 (96.9) | 1.59 |
| | >2–6 | 37.5 (21.6) | 74.3 (37.5) | 2.27* |
| | >6–10 | 15.56 (8.4) | 67.3 (55.9) | 2.73* |
| CCR (ng/mg) | 0–2 | 26.96 (16.9) | 191.19 (164.9) | 2.19* |
| | >2–6 | 51.05 (47.5) | 118.74 (71.7) | 2.25* |
| | >6–10 | 23.23 (1.6) | 100.33 (76.6) | 2.97* |

**Significant at P < 0.05*
s = *standard deviation.*

Table 4 **Levels of creatinine, cotinine and cotinine/creatinine ratio (CCR) of controls and passive smoker children (PSC) by sex**

| Parameter | Sex | Controls Mean (*s*) | PSC Mean (*s*) | *P*-value |
|---|---|---|---|---|
| Creatinine (mg/100mL) | Male | 0.7 (0.2) | 0.7 (0.3) | 0.95 |
| | Female | 0.62 (0.17) | 0.63 (0.43) | 0.93 |
| Cotinine (ng/mL) | Male | 25.55 (14.3) | 68.05 (50.7) | 0.006 |
| | Female | 18.13 (20.8) | 99.9 (79.6) | 0.009 |
| CCR (ng/mg) | Male | 37.1 (17.6) | 122.4 (98.9) | 0.005 |
| | Female | 25.65 (24.3) | 183.81 (135.7) | 0.003 |

s = *standard deviation.*

Table 5 **Levels of cotinine and cotinine/creatinine ratio (CCR) of passive smoker children (PSC) by age group and sex**

| Parameter | Age group (years) | Males Mean (s) | Females Mean (s) | *t*-test |
|---|---|---|---|---|
| Cotinine (mg/mL) | 0–2 | 81.43 (81.3) | 101.3 (134.9) | 0.41 |
| | > 2–6 | 60.8 (21) | 99.3 (49.6) | 2.46* |
| | > 6–10 | 62 (34.9) | 72.6 (72.9) | 0.42 |
| CCR (ng/mg) | 0–2 | 26.96 (16.9) | 191.19 (164.9) | 0.47 |
| | > 2–6 | 51.05 (47.5) | 118.74 (71.7) | 2.64* |
| | > 6–10 | 23.23 (1.6) | 100.33 (76.6) | 0.11 |

**Significant at P < 0.05.*
s = *standard deviation.*

($P = 0.05$). To further explore this gender difference, the PSC group was broken down by age group and sex (Table 5). No differences in creatinine levels by age and sex were found (results not presented), but within the middle (> 2–6 years) age bracket, females had significantly higher cotinine levels and CCR than males.

## Fathers

Fathers of PSC had significantly higher creatinine, cotinine and CCR levels than the control fathers. CCR showed no correlation with either the number of cigarettes smoked or by the nicotine intake (calculated by multiplying the number of cigarettes smoked with the nicotine content of the cigarette). Similarly, no significant correlation was found between CCR of PSC and either the fathers' CCR or the number of cigarettes smoked by the father. When the fathers were categorized according to the nicotine content of the cigarettes smoked into low ($n = 25$) and high content ($n = 13$), no difference was found between fathers' levels of CCR. However, an interesting finding was that PSC whose fathers smoked low nicotine content cigarettes had a significantly higher CCR than the children whose fathers smoked high nicotine content cigarettes (4102 ng/mg and 3806 ng/mg respectively, $P < 0.05$). This inverse relationship between the CCR values of PSC and the strength of the father's cigarette was augmented by another finding: a significant negative correlation between the CCR of PSC and their fathers' nicotine intake ($r = -0.37$, $P < 0.05$). This suggests that a "lighter" cigarette is not necessarily a "healthier" one.

## Contribution to population burden of cotinine

A measure of the population burden of passive exposure to tobacco smoke is given by the sum of the cotinine values for all children in this study. For a given group of children, it is then possible to calculate the percentage of the total population burden found in that group by calculating the sum of their cotinine values and expressing it as a percentage of the total [*14*].

Employing this technique, the percentage population burden of cotinine for the four mutually exclusive groups was as follows: non-exposed children (both pa-

rents non-smokers, irrespective of whether another external carer smoked) 9%; exposed children (only the father smoked, irrespective of whether the mother or external carer smoked) and child's age was 0–2 years 35%; child's age was 2–6 years 29%; child's age was 6–10 years 28%. As each of the four mutually exclusive groups comprised 20 children, these differences were a true reflection in groups' population burden of cotinine and not due to the differences in the number of children in each group.

## Discussion

Precise and objective biochemical measures of passive smoking are required to identify its risks and quantify the benefits of antismoking interventions [*2*]. This study employed cotinine and CCR measures to estimate the exposures directly. A cut-off CCR of 30 ng/mg reportedly identified children exposed to smoking at home with 80% sensitivity and 100% specificity [*16*]. This study found that employing the same cut-off yielded a higher sensitivity (96%), but at the same time a substantially lower specificity (55%). The implication is that the 30 ng/mg cut-off value might be more effective in identifying the proportion of true positives that are correctly identified as such, but less effective in identifying the proportion of true negatives that are correctly identified as such. Hence such a cut-off might be more useful as a confirmatory rather than an exclusion tool for environmental tobacco exposure.

Other studies on children who were either known or not known to be exposed to home tobacco smoke (verified by air sampling for nicotine) detected small amounts of cotinine in some unexposed children's urine samples [*16*]. In this study, although the parents of the control group reported the absence of household tobacco exposure, the CCR for the control children ranged from 4.4 to 80 ng/mg with a mean of 32.5 ± 20.8 ng/mg, which exceeds the 30 ng/mg cut-off level of home exposure. This imprecise reporting of exposure could be due to the parents having become sensitized to the issue of passive smoke exposure by the study questionnaire itself and thus minimizing the reported exposures. Another possible explanation for the discrepancy could be outdoor accidental exposure, which was not included in our questionnaire. Our results are consistent with other studies [*2,19*] that have reported that high cotinine levels are sometimes demonstrated in children with no reported exposure at home. In Egypt, such unavoidable exposure could have resulted from both the high and uncontrolled prevalence of passive smoking in public places (no-smoking buildings and transport had not been implemented), and the high prevalence of smokers in the community in general. Other studies have also reported that children not exposed at home have low cotinine concentration, the level depending on the prevalence of smoking in the community [*14*].

Employing the 30 ng/mg cut-off level, about 86% of the children in this study were classified as exposed to household tobacco smoke (regardless of exposure status reported by the parents). This was higher than the 78% exposure level reported by El-Sawy et al. [*2*] in a study also undertaken in Alexandria. However, both studies document a disturbingly high rate of household cigarette smoke exposure emphasizing the need for preventive antismoking measures and interventions. Infants and very young children cannot complain; older children may not complain, or

may be ignored when they do. Children often cannot remove themselves from exposure and, therefore, are dependent on other measures for protection [*4*].

The findings of this study also suggest an inverse relationship between CCR and the age of the PSC, where there was a steady decrease in CCR with increase in age. Taking the three age brackets of PSC in ascending order, CCR levels significantly decreased in a stepwise fashion from 191.19 ng/mg to 118.74 ng/mg to 100.33 ng/mg ($F = 3.626$, $P = 0.03$) (Table 3). A similar decline in children's lower respiratory tract infections related to passive smoking at home with increasing age has been noted in the literature [*20,21*]. Such dose-response relationships have been attributed to the amount of time that younger children (0-2 years) spend with the mother, who is frequently one of the smokers in the house. By the age of 2 years, most children attend day care centres and consequently time is spent outdoors away from smoke exposure at home. Although no mother in our sample reported to be a smoker (index smoker parent was always the father), there could have been an element of underreporting of maternal smoking habits due to cultural factors and stigma in the Egyptian community regarding women's smoking. However, other studies have demonstrated that, despite the lower levels of smoking by mothers, maternal smoking is more important than that of the father [*14*].

In a study of passive smoking and lower respiratory tract infections in Egypt [*2*], the authors stated that mothers in their sample did not report any smoking. However, El-Sawy and colleagues confirmed the absence of significant differences in the degree of exposure between children below or above 2 years of age, and attributed it to the fact that in their sample, children over the age of 2 years did not attend day care centres and thus were usually at home. Although our study was implemented in the same city as that of El-Sawy and coworkers, contrary to their findings, we documented a significant inverse dose-response relationship between age and CCR. This is consistent with our North American [*20*] and European [*21*] colleagues. The implication is that even when children are not attending day care centres, with increasing age, they move around more, either within the confines of the apartment to areas that might harbour lower concentrations of ambient smoke, or in the immediate vicinity of the apartments playing with siblings/friends, and thus might be exposed to lower smoke concentrations. The same argument might explain why, in this study, female cotinine and CCR values were higher than those of males, especially in the 2–6-year age bracket (Table 5). In Egypt, it is more common for male children than their female counterparts to be regularly outdoors playing with their peers. As exposure mainly depends on proximity to smokers, younger children who spend much of their time with parents that smoke are particularly vulnerable [*22*], and a decrease in cotinine concentration would be expected with the ageing of the children [*23*]. Similarly, Cook et al. reported that that there were small but significant age and sex differences, with cotinine concentrations being higher in younger children and in girls [*14*].

This investigation demonstrates that children exposed to parental smoking at home had significantly higher urinary cotinine and CCR than control children. However, consistent with other studies [*2,24*], there was no correlation between CCR of PSC and the number of cigarettes smoked

in the household. One salient finding is that PSC whose fathers smoked cigarettes with low nicotine content had a significantly higher CCR than those whose fathers smoked high nicotine content cigarettes. This inverse relationship between the CCR values of PSC and the strength of the father's cigarette is similar to that observed by Sepkovic and Haley [25], who reported higher saliva and plasma cotinine concentrations in individuals smoking low nicotine content cigarettes as compared with those smoking high nicotine content cigarettes. This was attributed to the fact that when smokers smoke, it is for two reasons: a habit and the need for nicotine intake. With low nicotine content cigarettes, the number of cigarettes, the puff volume, the frequency and the degree of inhalation of the smoke are all subconsciously increased by the smoker in order to extract a higher nicotine yield per puff, which is not the case when the same smoker is smoking a high nicotine content cigarette [25]. The standard nicotine yields of cigarettes are very poor indicators of plasma nicotine concentrations in smokers, and the widespread and often officially sanctioned turn to low-yield cigarettes may be ill-advised and have unexpected casualties [26]. Low-nicotine cigarettes have been offered as a "prescription" for less hazardous smoking. Diagnostic procedures should be developed to distinguish those who may be benefiting from a switch to low-yield cigarettes from those who only mistakenly believe they are. Hence those smoking a 'weaker' cigarette might smoke more to get the same yield of nicotine, with the consequent emission of more smoke to their households. The image frequently promoted by the tobacco industry that a 'weaker' or 'lighter' cigarette is a 'healthier' cigarette for smokers and those around them is thus thrown into doubt.

## Public health implications

The harmful effects of active smoking are now well known, but whether the risks from passive smoking are appreciated is unproved [22]. In relation to asthmatic children, the more the parent reported smoking in front of the child, the fewer health care visits that were made for the child's asthma, perhaps due to a lack of awareness of asthma symptoms among heavy smokers or a reluctance to visit the general practitioner [27]. Such findings are alarming, especially since studies have confirmed significant associations between reported quantitative exposure of children to ETS and urine cotinine levels [28].

Although clinicians have been advised to counsel parents about the harmful effects of passive smoking on their children, studies have shown that many do not give such advice routinely [29]. Some are even uncertain about the effect of counselling smokers at every opportunity [30], and whether advice would encourage parents to reduce their children's exposure to tobacco smoke [22]. Some parents might be less inclined to stop smoking after a brief advice, consistent with the theory that patients are resistant to information or advice when it is not being sought: "Telling patients what to do can make them feel challenged and provoke them to assert control by continuing their unhealthy behaviours with renewed vigour" [31].

Two recent meta-analyses reported that unaided smoking cessation is about 7% [32,33]. Poor success is associated with young age, being female and low social class. The home is the only place where parents can make free choices about their smoking, and informing them of the harmful effects of passive smoking might be insufficient to reduce the exposure of their children to ETS. When a child's health is

being affected by parental smoking, the parent's smoking needs to be addressed as a separate issue from the child's health [*22*]. Targeting smoking cessation interventions at the smoker's health may have a modest impact [*34*], but interventions aimed at the health of a third party (the parent's child) seem ineffective.

The role of advertising requires consideration. Advertisements comparing the risk of lung cancer from passive smoking with a variety of other apparent risks from everyday activities can be misleading [*35*]. The 20% increased risk of lung cancer among those exposed to other people's tobacco smoke might appear minute in comparison with the 500% increased risk of lung cancer associated with a high saturated fat diet, the 180% increase with frequent cooking with rape seed oil and the 60% increase with drinking 1–2 glasses of whole milk per day [*35*]. Such cleverly tailored advertisements stimulate the public's scepticism about the apparent health risks of everyday activities.

Another related point is the advertising of low-yield cigarettes. With the dramatic shift from high-tar to low-tar cigarettes, nearly 60% of smokers believed that cigarette advertising using terms like low tar, low nicotine or lower yield was trying to communicate that the brand was safer, healthier or less harmful. However, authorities agree that there is no safe cigarette, and that the tar ratings bear almost no relationship to cardiovascular illness [*36*]. Despite this, an implied "safer cigarette" message continues to be communicated as a means of retaining health-concerned smokers. Several studies have identified smokers' compensatory mechanisms associated with nicotine intake to obtain the "desired" nicotine delivery including a change in the puffing patterns and depth of inhaling [*36*]. Hence terms such as "light" provide connotations that may be interpreted as "beneficial," when, in fact, even exposure to the smoke of a few cigarettes per day has well documented adverse health effects.

This study demonstrates that the very young children (0–2 years) carried the greatest population burden of cotinine due to passive smoking, and that females generally had higher urinary cotinine concentrations than males. As the investigation was undertaken in a less affluent part of Alexandria, the families participating in the study could be categorized as social class IV or V (all living in the same high-rise building, fathers mostly employed in manual labour). In the United Kingdom, Cook and colleagues [*14*] found that social class effects were strong, and that cotinine concentrations were eight to nine times greater in children of social classes IV and V compared with social class I. Hence, the danger triad of being of very young, female and from a less advantaged social class needs to be the immediate focus of any anti-passive smoking health promotion campaigns. Box 1 summarizes the study findings and their public health implications.

## Conclusion

Children's exposure to ETS has a substantial public health and economic impact. Children are more likely than adults to suffer health effects from ETS exposure, and the home is the most likely site of exposure [*4*]. Due to the higher relative breathing rates of children, they have higher internal exposure to ETS, as measured by urinary cotinine, for the same level of external exposure.

- Children exposed to parental smoking at home had significantly higher urinary cotinine and cotinine/creatinine ratio (CCR) than controls. Implication: besides self-completed questionnaires, cotinine concentration should be employed as an objective measure of environmental tobacco smoke (ETS) exposure.
- 86% of children were exposed to cigarette smoke in the home. Implication: programmes and legislation need to be directed at reducing ETS exposure in the home
- 35% of the population burden of cotinine was among passive smoker children aged 0-2 years. Exposure to cigarette smoke in the home significantly decreased in a stepwise fashion as the child aged. Implication: younger children need to be the focus of policies to decrease ETS exposure
- Female cotinine and CCR values were higher than those of males, especially in the 2-6 year age group. Implication: ETS awareness needs to be especially targeted to female children.
- Passive smoker children whose fathers smoked lower nicotine content cigarettes showed a significantly higher CCR than those whose fathers smoked high nicotine content cigarettes. Implication: community-based programmes need to remind smokers that lowering the nicotine content does not expose their children to less harm.
- Low-nicotine or lower-yield cigarettes are not safer, healthier, or less harmful than regular cigarettes in relation to their effect on passive smokers. Implication: health professionals should advise smokers to quit smoking rather than switching to a "lighter" brand of cigarette.

Box 1 **Findings of the study and their public health implications**

The average cotinine concentration in adult cigarette smokers is around 300 ng/mL. The mean levels observed in this study in PSC were 87 ng/mL, 74 ng/mL and 67 ng/mL in the three age brackets respectively. Such concentrations have been associated with small decreases in lung function, increases in respiratory infections and glue ear, and may be associated with a raised risk of lung cancer [*15*]. The average concentrations in non-exposed children were considerably lower, at 22.5 ng/mL. Nevertheless, 9% of the population burden of cotinine was among these children.

Supported by two decades of evidence, the World Health Organization (WHO) has widely publicized that second-hand smoke is a real and significant threat to public health [*37*]. The scientific community now agrees that there is no safe level of exposure to second-hand smoke [*37*]. In line with WHO's World No-Tobacco Day 2001 initiative, programmes and strategies to reduce ETS exposure in the home need to be based on multipronged approaches [*4*].

The public needs to be educated about the health effects of ETS and effective ways of controlling exposure. Electronic and print media information, billboards, and warnings on cigarette packages could stir up appropriate responses, while tobacco education programmes in schools might raise ETS awareness. Community-based programmes need to remind smokers to extend the benefits of abstinence to their children and partners, as they do not expose their coworkers or the general public in many places. Health professionals' knowledge and skills should be targeted and upgraded with regard to smoking to ensure effective interventions with parents, other caregivers and children. Voluntary health associations, expert groups, professional associations and government agencies need to work together towards public positions advocating strategies to protect children from ETS exposure. Policy and advocacy statements create awareness and set agendas for action. Finally, evaluation components are critical if we are to learn which programmes and policies are effective in reducing ETS exposure and protecting children. Such multipronged strategies, which need to be evidence-based, should help to 'clear the air'.

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**EMRO' s Tobacco Free Initiative (TFI) website**

We would like to draw our readers' attention to the Tobacco Free Initiative (TFI) website which provides a wealth of information on WHO's work to fight the tobacco epidemic with particular reference to the work being done in the Eastern Mediterranean Region (EMR). TFI is a priority programme. This means that it has to move and move fast. There are currently many areas of work underway on tobacco control most important of which is the Framework Convention on Tobacco Control, which is the flagship of WHO's efforts to control tobacco. The website provides information on: TFI developments, EMRO's work related to TFI, publications in the area of tobacco control, legislation, facts and FAQs, country profiles for EMR countries, World No Tobacco Days, and useful links to other sites on tobacco control. The website can be accessed at: http://www.emro.who.int/TFI/TFI.htm