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Evolution and characteristics of studies estimating attributable mortality to second-hand smoke: a systematic review

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Background: Several studies have estimated the impact of second-hand smoke (SHS) exposure on mortality in the population of different countries. This study aimed to identify and describe studies that have estimated the attributable mortality (AM) associated with SHS exposure in the adult population. **Methods:** A literature search was conducted in MEDLINE, EMBASE, Cochrane Library and CINAHL databases up to January 2023. Studies that estimated the AM associated with SHS exposure in the adult population and used a prevalence-dependent method were included. The main characteristics of the studies and their results were described. **Results:** Fifty-three studies were included. Most of them were conducted in North America ($n = 13$), Europe ($n = 14$) and China ($n = 6$) and included lung cancer ($n = 46$) or ischaemic heart disease ($n = 22$) as causes of death. There was considerable variety in the population under study regarding the relationship with tobacco: non-smokers ($n = 30$); never-smokers ($n = 9$); both non and never-smokers ($n = 2$); the whole population ($n = 1$) and not known ($n = 11$). The age at which AM was estimated also varied between studies, ranging from 15 to 40 years and older. **Conclusions:** Studies estimating AM associated with SHS exposure are heterogeneous in terms of the causes of death studied, the age at which mortality is attributed, or the population to which mortality referred: consensus should be reached. Despite their importance, studies assessing AM to SHS are infrequent in low- and middle-income countries.

Introduction

In 2004, the International Agency for Research on Cancer classified second-hand smoke (SHS) as carcinogenic to humans.[1] In 2007, the Surgeon General's report 'The Health Consequences of Involuntary Exposure to Tobacco Smoke' provided a synthesis of the available evidence on the health effects of SHS exposure. This report established a causal relationship between SHS exposure and mortality from lung cancer and ischaemic heart disease in healthy non-smoking adults.[2] Since then, almost 20 years have passed, and various studies have assessed the impact of SHS exposure on the health of the population by estimating the attributable mortality (AM) associated to it.[3] Having different estimates at different time points allows us to monitor the impact of SHS exposure on mortality, an important measure in controlling the tobacco epidemic.[4]

To the best of our knowledge, no work has systematically reviewed the different studies estimating AM to SHS exposure and described the basic characteristics of the estimation. This review is relevant from two perspectives: one methodological, oriented to identify variations in the populations to which mortality referred, or, among other factors, the causes of death assessed[5]; and the other, a public health perspective, oriented to report the evolution of the global tobacco epidemic.

Therefore, this study aimed to identify the different studies that have estimated the AM associated with SHS exposure in adults by applying a prevalence-dependent method.

Methods

Search strategy and inclusion criteria

A systematic review was conducted in MEDLINE, EMBASE (OVID), Cochrane Library and CINAHL (EBSCO) databases according to the PRISMA guidelines.[6] The search strategy was adapted for each database and can be consulted in the Supplementary tables S1–S4. The initial search was performed on 12 June 2022 and updated on a monthly basis until 12 January 2023. The review was registered in PROSPERO (CRD4202222377606).

We selected studies that estimated AM to SHS exposure in the adult population using a prevalence-dependent method based on the calculation of population attributable fractions (PAF). We excluded studies that calculated PAFs but did not estimate the number of attributed deaths, studies that estimated the joint impact of active and passive smoking or morbidity associated with SHS exposure, studies that did not describe the calculation method, studies that provided estimates for only paediatric population and simulation

studies. In addition, conference papers, letters, editorials and studies derived from the Global Burden of Disease initiative were excluded. The reason for this latter exclusion was that the source of data from the different countries included in the estimates was not detailed. Moreover, reports produced by public and private organizations were excluded because the peer review process could not be guaranteed.

Study selection

In the first phase, titles and abstracts were reviewed by two researchers (M.P.R. and D.C.L.M.) and discrepancies were resolved by consensus. In the second phase, the two researchers read the full text of the records selected in the first phase to identify those studies that met the inclusion criteria. The references of the selected articles were reviewed, and the identified records that met the inclusion criteria were included.

Data collection and synthesis of information

An *ad hoc* data collection table was designed to summarize information of interest in the following domains: year of estimation; population studied: geographical location, age, relationship with tobacco use and causes of mortality analyzed; data characteristics: prevalence of exposure to SHS, number and percentage of deaths attributable to SHS (total and/or by sex), source of risks (meta-analysis vs. individual studies) and relative risk data. Six researchers extracted this information independently, and discrepancies were resolved by consensus. Results are presented by decades.

Results

The search, after removing duplicates, yielded 6510 records, of which 6080 were eliminated after reading the titles and abstracts. A total of 430 full-text records were reviewed, of which 70 met the inclusion criteria. From the manual literature review of the selected records, six studies were identified and included. Finally, 53 studies were included (figure 1). The main characteristics on these studies are shown in Supplementary table S5 and the complete reference list can be consulted in Supplementary table S6.

Regarding the prevalence of SHS exposure, the figures applied in the AM analysis are included in 36 studies, in 23 of them they are presented by sex. The source of the risks applied in the AM analysis are stated in 48 studies, deriving in 30 from meta-analyses, 11 from individual studies and 7 from both sources. Regarding deaths attributed to SHS exposure, the estimation obtained is presented by sex in 35 studies (table 1).

1980–1989

The first three studies estimating AM to SHS exposure based on the prevalence dependent method were published in the 1980s. These estimates referred to Canada (Wigle et al.), the United States (Wells et al.) and New Zealand (Kawachi et al.) The Wells study, estimated AM for 1984 in non-smokers, whereas the other two estimated AM for 1985 in never-smokers. All three studies combined the calculation of attributable fractions with mortality rates in smokers and never-smokers. Wigle, in Canada, set the lower age limit for estimating AM at 40 years, whereas Wells, in the United States, set it at 35 years. Wigle and Wells did not set an upper age limit.

In terms of causes of death related to exposure, Wigle et al. included lung cancer, with spousal smoking being the source of SHS prevalence, thus assessing exposure at home. The study by Wells included lung cancer, other cancers and cardiovascular diseases as causes of death related to exposure to SHS. In this study, the prevalence of exposure referred to home and work and was derived from the self-reported SHS exposure of controls in several case-control studies. Finally, the study by Kawachi analyzed lung cancer and ischaemic heart disease as causes of death related to exposure to SHS, assessing exposure to SHS at home and at work.

1990–1999

Two studies were identified in the 1990s. One referred to the impact of SHS exposure, derived from spousal smoking, on lung cancer mortality. In this study, the estimate was made for each of the 15 European countries included in the study (Tredaniel et al.). This is the first time that AM to SHS was estimated in different countries, and the last time that the prevalence of exposure in women derived completely from the consumption of their spouses. This study was the first applying, with a prevalence-dependent method, a 20-year latency period between exposure and mortality. During this decade, another study (Wen et al.) was published estimating AM in Taiwan for both SHS exposure and tobacco consumption, although very little information was provided on the calculation procedure.

2000–2009

At the beginning of the 21st century, the number of studies estimating AM to SHS increased. A total of 12 studies were identified. Six of these estimated AM in European countries, including two in Germany, two in the United Kingdom, one in Spain and one in Finland. Three estimated AM in North America, two in the United States and one in Canada; two in Asia, one in China and one in Hong Kong; and one in Oceania, conducted in New Zealand.

Three of the studies estimated the burden of AM associated with SHS exposure exclusively on workers. One of these studies focussed on the impact of SHS exposure on lung cancer (Rushton et al.); another added cardiovascular disease (Steenland et al.), and the last, in addition to lung cancer, also assessed the impact on ischaemic heart disease, stroke, chronic obstructive pulmonary disease (COPD), asthma and infections (Nurminen et al.). Two of these studies (Rushton et al.; Steenland et al.) estimated AM for different risk factors, whereas the other focussed only on SHS exposure. The remaining studies were referred to the general population, although the Woodward study included a differentiated estimate of AM in children, and the study by Jamrozik included a differentiated estimate for hospitality workers.

Except for three studies (Nurminen et al.; McGhee et al.; Zollinger et al.), the number of causes of death included in the mortality attribution analysis varied between one and three. In general, the AM estimates made during this decade assessed the impact of SHS exposure on lung cancer and cardiovascular disease. With regard to the causes of death analyzed in each study, the two studies conducted in Germany assessed AM for coronary heart disease (Heidrich et al.) and cerebrovascular disease (Heuschmann et al.); the study in Canada for cardiovascular disease (de Groh et al.), and the study in the United Kingdom for lung cancer (Rushton et al.). Studies from Spain (Lopez et al.) and China (Gan et al.) estimated AM for lung cancer and ischaemic heart disease, whereas the one from United Kingdom (Jamrozik et al.) incorporated stroke and the one from New Zealand (Woodward et al.) also included coronary heart disease. Studies in Hong Kong (McGhee et al.) and Finland (Nurminen et al.) included COPD for the first time.

The studies varied in terms of whether they estimated AM in the whole population, in non-smokers or in never-smokers, although it was not always clear whether the estimate referred to non-smokers or never-smokers. From the 10 studies that identified the population in which the estimate was made, five referred to never-smokers and one to the whole population regardless of their smoking status. Three studies took into account a latency period between exposure and death.

In this decade, the first cost studies were published, which assessed both SHS (Zollinger et al.) and SHS in combination with others risk factors (McGhee et al.). In the Zollinger study, the number of pathologies associated with SHS exposure reached seven.

2010–2019

In the 2010s, 31 estimates of AM associated with SHS exposure were published, including 15 from Asian countries and six from the United States. In 2011, the first global estimate of AM associated

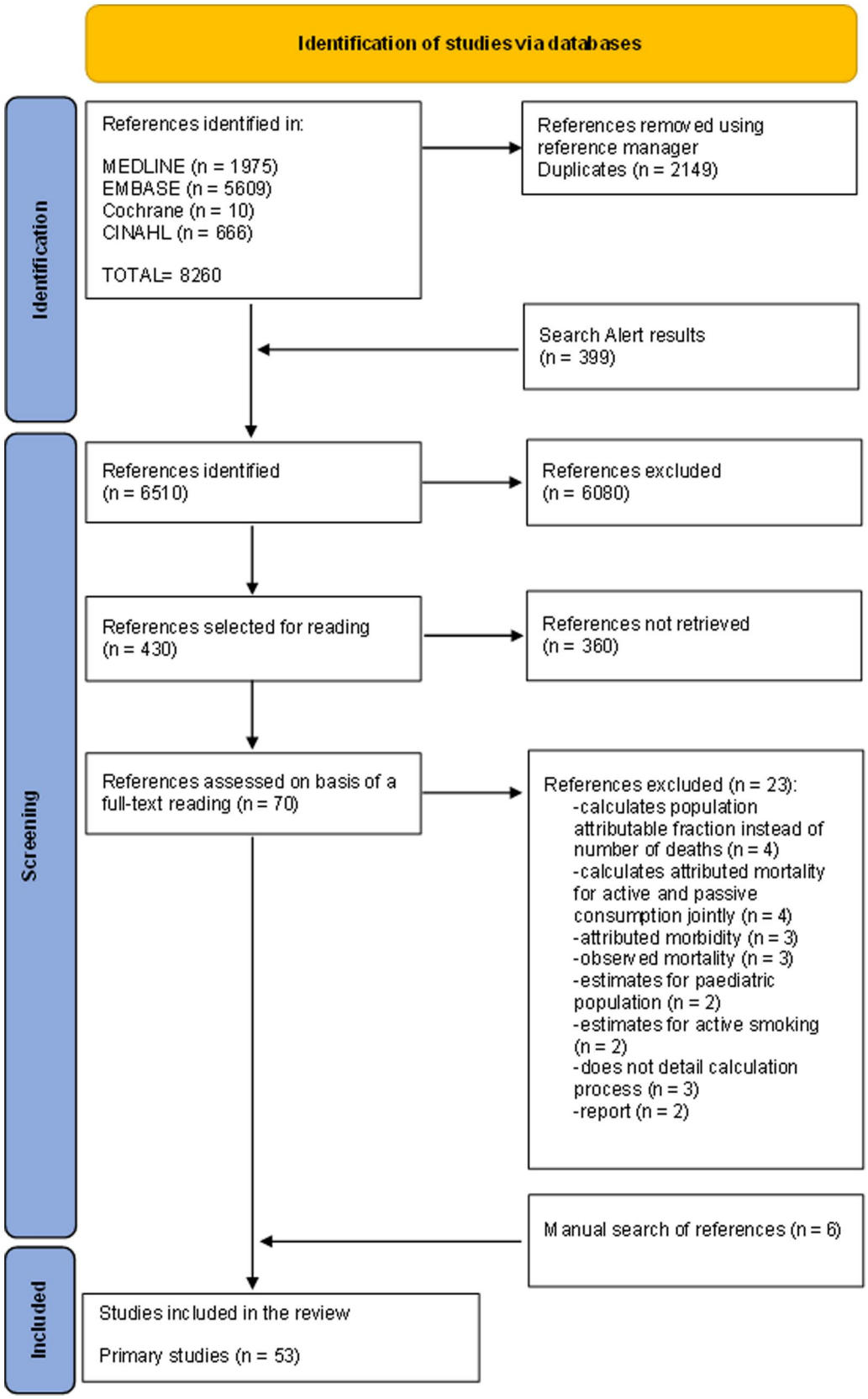

Figure 1 Flowchart of the selection of studies

with SHS exposure in non-smokers greater or equal to 15 years of age was published (Oberg et al.). In this study, in addition to estimate AM to lung cancer and ischaemic heart disease, asthma was included, and different latencies periods were applied according to the cause of death.

In this decade, it was common that the same study included AM estimates to different risk factor or combined the estimations with cost studies. In addition, the first study including projections of AM related to SHS exposure was published (Adam et al.). The first estimate for a country in Africa (Tachfouti et al.), the effects of

Table 1 Prevalence of second-hand smoke (SHS) exposure and number of deaths attributable to SHS exposure global and by sex; and relative risk source

Author and year of publication	Estimation area	Prevalence of SHS exposure (%)			Number of deaths attributable to SHS			Percentage of deaths attributable to SHS			Relative risk source	Relative risk data
		Overall	Men	Women	Overall	Men	Women	Overall	Men	Women		
1980–1989 Period												
Wigle et al. (1987)	Canada	37.0%	18.0%	48.0%	56	7	9	10.6%[a]	5.1%[a]	12.6%[a]	Meta-analysis	Lung cancer: 1.3
Wells et al. (1988)	USA	N/S	61.0%	76.0%	46 000	24 966	28 507	N/S	N/S	N/S	N/S	Multiple data
Kawachi et al. (1989)	New Zealand	N/S	At home: 12.7% Work: 33.6%	At home: 16.1% Work: 23.4%	At home: 95 Work: 177	At home: 54 Work: 145	At home: 41 Work: 33	At home: 1.1%[a] Work: lung cancer: 10.2%[a]	At home: 1.1% [a] Work: 10.7%[a]	At home: 1.2% [a] Work: 8.4%[a]	Individual and meta-analysis	Multiple data
1990–1999 Period												
Wen et al. (1994)	Taiwan	N/S	Multiple data	Multiple data	3144 (1980) 4785 (1990)	798 (1980) 1426 (1980)	2348 (1980) 3359 (1990)	N/S 18.6%[a]	N/S 22.0%[a]	N/S 13.0%[a]	Individual	Multiple data
Tredaniel et al. (1997)	Europe	N/S	N/S	N/S	1146	307	839	0.7%[a]	0.22%[a]	2.5%[a]	Meta-analysis	Lung cancer: 1.3
2000–2009 Period												
Woodward et al. (2001)	New Zealand	24.0%	At home: 14.7% Work: 19.1%	At home: 16.5% Work: 6.2%	297	191	105	N/S	N/S	N/S	N/S	Multiple data
de Groh et al. (2002)	Canada	8.0%	7.0%	8.0%	803	N/S	N/S	1.8%[a]	N/S	N/S	Meta-analysis	Coronary heart disease: 1.24
Steenland et al. (2003)	USA	21.0%	N/S	N/S	58 640	N/S	N/S	4.2–6.8%[a]	N/S	N/S	Meta-analysis	Range: 1.21–1.35
Nurminen et al. (2001)	Finland	10.0%	12.0%	8.0%	252	210	42	N/S	N/S	N/S	Meta-analysis	Multiple data
Zollinger et al. (2004)	USA	N/S	N/S	N/S	247	N/S	N/S	10.8%[a]	N/S	N/S	Meta-analysis	Multiple data
Jamrovzik et al. (2005)	UK	11.0–13.0%	N/S	N/S	11 302	N/S	N/S	5.2%[a]	N/S	N/S	Individual and meta-analysis	Multiple data
McGhee et al. (2006)	Hong Kong-China	N/S	56.0%	46.0%	1324	N/S	N/S	4.0%[a]	N/S	N/S	Individual	Multiple data
Gan et al. (2007)	China	N/S	N/S	N/S	56 000	12 000	44 000	N/S	N/S	N/S	Meta-analysis	Multiple data
Lopez et al. (2007)	Spain	N/S	3.2–9.5%	2.1–12.0%	1228–3237	408–1703	820–1534	N/S	Multiple data[a]	Multiple data[a]	Meta-analysis	Multiple data
Heuschmann et al. (2007)	Germany	N/S	10.0%	13.6%	774	189	585	N/S	N/S	N/S	Meta-analysis	Stroke: 1.18
Heidrich et al. (2007)	Germany	12.0%	N/S	N/S	Multiple data	725	1423	1.7%[a]	N/S	N/S	Meta-analysis	Coronary heart disease: 1.25
Rushton et al. (2008)	UK	N/S	N/S	N/S	254	144	110	N/S	N/S	N/S	Meta-analysis	N/S
2010–2019 Period												
Oberg et al. (2011)	World	N/S	33.0%	35.0%	437 000	156 000	281 000	N/S	N/S	N/S	Individual and meta-analysis	Multiple data
Wang et al. (2011)	China	N/A	N/A	At home: 36.7% Work: 8.4%	N/A	N/A	11 507	N/A N/A	N/A N/A	11.1%[a]	Individual	At home: 1.15 At work: 1.79

(continued)

Table 1 Continued

Author and year of publication	Estimation area	Prevalence of SHS exposure (%)			Number of deaths attributable to SHS			Percentage of deaths attributable to SHS			Relative risk source	Relative risk data
		Overall	Men	Women	Overall	Men	Women	Overall	Men	Women		
Ha et al. (2011)	Korea	N/S	19.0%	11.3%	163	133	30	N/S	3.5%[a]	2.4%[a]	Meta-analysis	Men: 1.19 Women: 1.22
Inoue et al. (2012)	Japan	N/S	At home: 8.0% Work: 58.0%	At home: 35.0% Work: 32.0%	2842	708	2133	N/S	17.9%[a]	18.9%[a]	Individual	At home: 1.34 Work: 1.32
Max et al. (2012)	USA	39.1%	44.5%	35.0%	41 284	23 526	17 758	N/S	N/S	N/S	Individual	Lung cancer: 1.29 Coronary heart disease: 1.32
Adam et al. (2013)	Hungary	N/S	N/S	N/S	1852	N/S	N/S	N/S	N/S	N/S	Individual and meta-analysis	N/S
Jarvholm et al. (2013)	Sweden	N/A	N/A	5.0%	N/A	N/A	5	N/S	N/S	1.2%[a]	Individual	Lung cancer: 1.25
Saywell et al. (2013)	USA	N/S	N/S	N/S	1409	N/S	N/S	8.3%[a]	N/S	N/S	Individual and meta-analysis	Multiple data
Liu et al. (2014)	China	54.0%	46.0%	57.0%	67 970	14 860	53 109	6.29%[a]	2.40%[a]	11.53%[a]	Individual	Multiple data
Liu et al. (2014)	USA	N/S	52.0%	44.0%	36 555	N/S	N/S	11.28%[a]	N/S	N/S	Meta-analysis	Lung cancer: 1.22; ischemic heart disease: 1.27
Sung et al. (2014)	Taiwan	25.0%	24.0%	25.0%	1196	613	583	N/S	N/S	N/S	Meta-analysis	Multiple data
Ginsbert et al. (2014)	Israel	N/S	N/S	N/S	793	N/S	N/S	N/S	N/S	N/S	Meta-analysis	N/S
Max et al. (2015)	USA	5.0%	N/S	N/S	794	N/S	N/S	1.70%[a]	N/S	N/S	Meta-analysis	Multiple data
Schram-Bijkerk et al. (2013)	The Netherlands	18.0–40.0%	N/S	N/S	120–480	N/S	N/S	N/S	N/S	N/S	Individual and meta-analysis	Lung cancer:1.21; ischemic heart disease: 1.27
Heo et al. (2014)	Korea	N/S	22.2%	19.9%	1130	420	710	N/S	Multiple data[a]	Multiple data[a]	Individual and meta-analysis	Multiple data
Mason et al. (2015)	USA	Multiple data	N/S	N/S	LOD 0.015 ng/ml: 215 LOD 0.05 ng/ml: 116	N/S	N/S	Multiple data[a]	N/S	N/S	Meta-analysis	Multiple data
Mason et al. (2016)	New Zealand	3.7%	N/S	N/S	98	54	44	1.37%[a]	N/S	N/S	Meta-analysis	Multiple data
Lopez et al. (2016)	Spain	Multiple data	N/S	N/S	1028	586	442	Multiple data[a]	Multiple data[a]	Multiple data[a]	Meta-analysis	Lung cancer: N/S. Cardiovascular disease 1.3 (home);1.21 (work)
Tachfouti et al. (2016)	Morocco	N/S	N/S	N/S	233	77	156	Multiple data[a]	Multiple data[a]	Multiple data[a]	Meta-analysis	Multiple data
Cui et al. (2016)	Hubei-China	N/S	N/S	N/S	9301	6452	2849	2.59%[b]	3.06%[b]	1.92%[b]	N/S	N/S
Zahra et al. (2016)	Korea	N/S	46.9%	27.7%	N/S	N/S	N/S	Multiple data[a]	Multiple data[a]	Multiple data[a]	Individual	Lung cancer: 1.5; Ischemic health disease: Multiple data; Stroke: Multiple data

(continued)

Table 1 Continued

Author and year of publication	Estimation area	Prevalence of SHS exposure (%)			Number of deaths attributable to SHS			Percentage of deaths attributable to SHS			Relative risk source	Relative risk data
		Overall	Men	Women	Overall	Men	Women	Overall	Men	Women		
Islami et al. (2017)	China	N/S	N/S	N/S	52 160	12 140	40 000	2.4%[a]	0.9%[a]	4.9%[a]	N/S	Lung cancer men: 1.58 Lung cancer women: 1.34
Chen et al. (2017)	Hong Kong-China	20.0%	27.8%	14.9%	672	416	256	N/S	N/S	N/S	Individual	Multiple data
Wilson et al. (2018)	Australia	N/S	N/S	N/S	9921	N/S	N/S	22.5%[c]	26.0%[c]	18.0%[c]	Meta-analysis	Multiple data
Becher et al. (2018)	Germany	N/S	39.5%	23.5%	Approach I: 190	Approach I: 107	Approach I: 83	2.8%[a]	4.0%[a]	2.0%[a]	Meta-analysis	Lung cancer: 1.21
					Approach II: 143	Approach II: 71	Approach II: 72	2.8%[a]	4.0%[a]	2.1%[a]		Multiple data
Xia et al. (2018)	China	46.0%	N/S	N/S	26 350	6180	20 170	3.2%[a]	1.4%[a]	10.4%[a]	Individual	N/S
Islami et al. (2018)	USA	N/S	32.8%	22.9%	4370	2680	1660	0.7%[a]	0.9%[a]	0.6%[a]	Individual	Lung cancer: 1.29
Permitasari et al. (2018)	Indonesia	N/S	N/S	N/S	17 905	12 113	5792	N/S	49.2%[a]	38.6%[a]	Meta-analysis	Multiple data
Kristina et al. (2019)	Southeast Asia	Multiple data	N/S	N/S	323 284	234 287	88 997	2.8%[a]	3.2%[a]	2.3%[a]	Meta-analysis	Multiple data
Kristina et al. (2019)	Indonesia	N/S	N/S	N/S	3354	2263	1091	44.7%[b]	50.3%[b]	36.4%[b]	Meta-analysis	Multiple data
Rezende et al. (2019)	Brazil	N/S	N/S	N/S	431	264	167	1.8%[a]	1.9%[a]	1.8%[a]	Meta-analysis	N/S
2020–2023 Period												
Carreras et al. (2020)	Europe	N/A	N/A	7.6%	N/A	N/A	379	NA	NA	Multiple data	Meta-analysis	Breast cancer: 1.07
Rezende et al. (2020)	Chile	N/S	17.3%	13.0%	70	46	24	2.0%[a]	2.2%[a]	1.6%[a]	Meta-analysis	N/S
Nguyen et al. (2020)	Vietnam	N/S	N/S	N/S	9812	3290	6522	6.0%[a]	3.6%[a]	8.8%[a]	Meta-analysis	N/S
Carreras et al. (2021)	Europe	N/S	N/S	N/S	24 000	N/S	N/S	Multiple data	N/S	N/S	Meta-analysis	Multiple data
Inoue et al. (2022)	Japan	N/S	N/S	N/S	2667	735	1932	0.7%[a]	0.3%[a]	1.3%[a]	N/S	N/S

a: Percentage of attributed mortality over observed mortality from the causes studied.
b: Percentage of attributed mortality over observed mortality from all causes.
c: Percentage of attributed mortality over observed mortality due to cancer.
N/S, not specified; N/A, not applicable. LOD, Serum cotinine limit of detection measured by nanograms/ml.

exposure in hospitality workers (Liu et al.; Liu et al.); and an update of the AM in Germany (Becher et al.), Spain (Lopez et al.) and New Zealand (Mason et al.) were also published in this decade. Nine of the published studies estimated only the impact of SHS exposure on lung cancer in non-smokers or never-smokers. Although not all studies set the age at which the impact of SHS exposure on AM was considered, most studies made estimations in subjects aged 30 years and above (four studies) or 35 years and above (five studies). Estimates of AM were most commonly made in non-smokers, with 21 of the 25 studies reporting the smoking status of the population analyzed. Latency periods between exposure and death were considered in 10 studies, although these periods were variable.

2020–2023

Five estimations of AM associated with SHS exposure were published in the first 4 years of this decade. Two of them referred to Europe as a whole but did not give a global estimate of SHS impact either because they referred to a particular disease or because they referred to a particular setting of exposure (Carreras et al.; Carreras et al.). The other three studies, performed in Chile, Vietnam and Japan (Rezende et al.; Nguyen et al.; Inoue et al.), referred to the effect of different risk factors on cancer mortality, with two of them focussing on lung cancer. Consistency was lacking with regard to the age of the population included or whether the estimates focussed on non-smokers or never-smokers.

Discussion

To date, different estimates of mortality attributable to SHS exposure are available. Estimates are most frequent in Europe and North America. Differences in the data applied in the estimation procedure, such as in the causes of death studied or the age at which mortality is attributed, do not allow a clear assessment of how the impact of SHS exposure on mortality has evolved or the differences between countries.

The first studies estimating AM to SHS exposure were performed in the 1980s. In these early studies, the estimation was based on differences in mortality rates between smokers and never-smokers.[7] However, during this decade, the calculation procedure was modified by using prevalence-dependent methods based on the calculation of PAF. Since then, there has been an increase in the number of studies estimating AM to SHS in different countries. One reason for this increase, especially in the United States, could have been related to the publication of the Surgeon General's monograph 'The Health Consequences of Involuntary Smoking' in 1986,[8] which highlighted SHS as a risk factor affecting the health of those exposed to it. Also, this increase could be partly explained because the data needed for the calculation (observed mortality, exposure prevalence and risk) became more readily available. However, these early estimations of AM to SHS exposure were met with strong criticism,[9–13] and attempts were made to discredit the estimates obtained. This led some authors to publish papers attempting to confirm the validity of the estimates[14–16] or even to publish estimates years later correcting and updating them.[15]

During almost 45 years since the first estimation applying a prevalence-dependent method, no consensus has been reached on how to carry out these studies. An examination of studies estimating the effect of SHS exposure on mortality reveals that although the process is of low demand in terms of the information required, the uncertainty in the comparability of the estimates is high owing to several factors. There is no consensus on the age from which the effect of SHS exposure on mortality should be assessed. However, the most recent studies tend to estimate AM from younger ages. This is in contrast to the estimation of AM to tobacco consumption, which is generally assessed from the age of 35 years, justifying this age by the time required to induce cancer in smokers. In the case of SHS exposure, it is not so easy to establish a lower limit for the age at which exposure begins, as it can start in the prenatal period. There is evidence of a possible causal relationship between SHS exposure and COPD or stroke.[17,18] However, recent estimates are 'simplistic' by focussing mainly on AM to a specific cancer, usually lung, and neglect heart or respiratory diseases.

The causes of death included in the studies are often large in those that do not exclusively focus on AM related to SHS exposure. In this case, diseases that are not even causally related to exposure to SHS, such as cervical cancer, are included.[2] Some studies are more conservative and include only the causes identified in the 2006 Surgeon General's report.[2] The source of evidence establishing a causal relationship between exposure to SHS and disease is not clear in all the studies reviewed.

It is, particularly, interesting to note that an increasing number of countries have estimates of AM to SHS exposure. This indicator, together with the prevalence of exposure, makes it possible to assess the burden of this carcinogen on the health of the population. However, it should be noted that estimates in Africa and South America are anecdotal, which suggests that this risk factor is probably not a priority in these countries, where the burden of disease associated with communicable diseases is still relevant.[19] More studies on AM to SHS are necessary in low- and middle-income countries. These studies will allow us to characterize the health impact of SHS in populations where both the prevalence of tobacco use, and the cancer burden are high.

The United States and China are the countries with the highest number of AM to SHS estimations. The first publication in China dates back to 2007,[20] whereas in the United States it dates back to 1988.[21] New Zealand notably published three estimates at various time points since the 1990s.[22–24]

The limitations of this review are mainly related to the difficulty in identifying the studies, as AM to SHS may not be the only objective in the analysis; this means that the identifiability of studies may have been compromised. In addition, studies that did not describe the calculation procedure were not included.[25] These include studies that are an important source of population-level data. An example are the CDC reports in which the calculation procedure is included without detailing specific aspects of the calculation procedure.[26,27] Similarly, our review excluded studies that did not estimate the burden in terms of the number of deaths attributed to SHS exposure.[28–31] Reporting the burden of mortality associated with a risk factor in terms of the number of deaths attributed, provides for simple and easily understandable information for the population. Although correct from an epidemiological point of view, e.g. PAF is not easily understood by the general population.

One of the strengths of this study is that an exhaustive systematic review was performed across multiple sources of information without limitation by date of publication or language. In addition, all stages of the study selection and collection of information were conducted by peers.

In recent years, there has been a global shift in the focus of tobacco control legislation towards protecting non-smokers from exposure to SHS, without losing sight of sales, promotion and consumption regulation. However, the information available to assess the impact of legislation on AM to SHS in the population is limited and difficult to compare. At this point, the lack of AM estimates in low- and middle-income countries should be noted, which makes it difficult to advance in tobacco control policies in these countries. It would be possible to approximate the impact of tobacco control legislation by periodically estimating AM associated with SHS exposure. To this end, having comparable information would be quite valuable. It is, therefore, essential to determine the necessary and appropriate inputs for the calculation procedure, to make comparable the estimations of AM in different countries, making possible to have a clear picture of the burden of AM to SHS worldwide.

Research ethics approval: human participants

Ethical approval was not required as this study is not conducted on human participants including identifiable human material or identifiable data and no human intervention is performed.

Supplementary data

Supplementary data are available at *EURPUB* online.

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Conflicts of interest: None declared.

Data availability

The derived data generated in this research will be shared on reasonable request to the corresponding author.

Author contributions

D.C.L.M. has participated in the formal analysis, investigation, methodology, writing of the individual draft and visualization. C. C.P. has participated in the investigation, methodology, writing-review & editing. J.R.B. has participated in the conceptualization, formal analysis, investigation, methodology, and writing-review & editing. C.G.T. has participated in the formal analysis, methodology and writing-review & editing. G.G. has participated in the formal analysis, writing-review & editing. L.M.G. has participated in the formal analysis, methodology and writing-review & editing. B.C.A. has participated in the methodology (search management) and writing-review & editing. A.R.R. has participated in the conceptualization and writing-review & editing. L.V.L. has participated in the conceptualization and writing-review & editing. M.P.R. has participated in the conceptualization, formal analysis, investigation, methodology, supervision, project administration, writing-review & editing and visualization.

Key points

- Estimates of mortality attributed to SHS are more frequent in Europe and North America, however, they are scarce in low- and middle-income countries.
- More recent studies estimating mortality attributed to SHS include fewer causes of death, and focus mostly on lung cancer.
- Studies estimating mortality attributed to SHS exposure are heterogeneous in terms of the causes of death studied, the age at which mortality is attributed or the population to which mortality referred.

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