Open Access Library Journal
2024, Volume 11, e1083
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Neighbourhood-Level Differences in Mortality Attributable to Behavioural Risk Factors in the City of Milan, Italy

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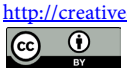
How to cite this paper: Consolazio, D., Benassi, D. and Russo A.G. (2024) Neighbourhood-Level Differences in Mortality Attributable to Behavioural Risk Factors in the City of Milan, Italy. *Open Access Library Journal*, **11**: e1083.
https://doi.org/10.4236/oalib.1111083

Received: December 6, 2023
Accepted: January 27, 2024
Published: January 30, 2024



Abstract

This paper investigates the impact of behavioural risk factors on non-communicable diseases mortality in Milan, focusing on their neighbourhood variation, with the scope to provide context-specific information to guide the development of effective health promotion interventions. Using administrative healthcare data, population attributable fractions were calculated based on information provided by the Global Burden of Disease project to estimate the number and proportion of deaths attributed to smoking, high body mass index, alcohol consumption, physical inactivity, and dietary risk. The findings revealed distinct territorial patterns of risk factors based on sex/gender, as territorial differences along the centre-periphery axis were observed in men but not in women. Smoking emerged as the primary risk factor for avoidable mortality, particularly in men whilst in females metabolic-related risk factors played a larger role. The proposed methodology provided valuable insights into the distribution of risky health behaviours at the neighbourhood level and underscored the need for context-specific interventions. Overall, the study emphasized the intertwined nature of territorial, socioeconomic, and gender dimensions in shaping health outcomes. It called for targeted interventions that address the specific risk profiles and challenges of each neighbourhood, promoting health equity and reducing the burden of non-communicable diseases. By understanding these complex dynamics, policymakers and public health professionals can develop effective strategies to improve population health and reduce health inequalities.

Subject Areas

Epidemiology, Public Health, Sociology

Keywords

Attributable Fraction, Attributable Risk, Behavioural Risk Factors, Social Epidemiology, Territorial Health Inequalities, Non-Communicable Diseases

1. Introduction

This study examines the geographical variation in mortality related to preventable non-communicable diseases (NCDs) in the Municipality of Milan, located in Northern Italy. In 2019, NCDs accounted for approximately 90% of deaths in Western Europe, with over one-third of these deaths attributed to behavioral risk factors such as unhealthy diet, tobacco smoking, excessive alcohol use, and physical inactivity (IHME, 2023) [1]. These risk factors are largely modifiable and consequently adopting healthier lifestyles can significantly prevent or delay the onset of these diseases.

From a public health perspective, understanding the distribution of health-related risk factors and their impact on mortality is essential for identifying intervention priorities and implementing effective prevention strategies (Lopez *et al.*, 2006) [2]. Information on health status and risk factors is typically collected through survey research, which in Italy is limited to provincial or district levels and provides, at most, municipality-level data for larger cities. On the other hand, administrative healthcare databases contain detailed information on individual health that can be geographically referenced to various local units such as neighbourhoods, census tracts, and census blocks. However, these data sources do not include information on risk factors and primarily focus on health outcomes and services. This limitation prevents the study of the association between behavioural risk factors and mortality at a granular level, including the exploration of heterogeneity within the urban environment. Consequently, it hinders the development of data-driven preventive interventions tailored to specific local contexts. Like many metropolitan areas, Milan exhibits significant socioeconomic inequality (d'Ovidio, 2009) [3], resulting in a heterogeneous population composition across neighbourhoods, with a distinct divide between a prosperous city centre and increasingly disadvantaged peripheral areas (Consolazio *et al.*, 2021 [4], 2023 [5]; Petsimeris & Rimoldi, 2015 [6]). Knowing from an extensive body of evidence that the prevalence of health damaging lifestyles follows a social gradient (Marmot, 2015) [7], increasing further down the social ladder (Braveman & Gottlieb, 2014 [8]; Phelan *et al.*, 2010 [9]; Solar & Irwin, 2010 [10]), it is reasonable to expect differences in the share of behavioural risk factors across neighbourhoods, because of their distinct socioeconomic composition. Moreover, built environmental and social features of the urban environment are likely to influence the adoption of healthy lifestyles, contributing to increase the exposure to the risk of NCDs over and above individual people's position in the social hierarchy (Frohlich *et al.*, 2001 [11]; Macintyre *et al.*, 2002

[12]). Accordingly, locally tailored public health interventions aimed at reducing the burden of NCDs by tackling the adoption of health damaging lifestyles cannot disregard the territorial heterogeneity in the distribution of such risky behaviours across urban areas, which should instead constitute a starting point to grasp the context-specific leveraging factors fundamental to improve population health conditions. Given the lack of submunicipal-level data on the distribution of behavioural risk factors associated with NCD mortality, we propose estimating this information by applying *Population Attributable Fractions* (PAFs) derived from the *Global Burden of Disease* (GBD) project (GBD, 2015) [13] to mortality data obtained from administrative healthcare databases.

PAFs—also known as population attributable risks, refer to the proportion of cases of a specific health outcome in a population that can be attributed to a particular exposure. The term "attributable" implies a causal interpretation, indicating that PAFs estimate the fraction of cases that would not have occurred if the exposure had been absent (Mansournia & Altman, 2018) [14]. In essence, they represent the proportion of disease cases or mortality that could be prevented or avoided if the associated risk factors were reduced or eliminated. To illustrate this concept, if lung cancer in Italy in 2019 accounted for 5.7% of the total 642,341 deaths, and smoking was responsible for 69% of lung cancer deaths, it can be estimated that 25,665 deaths could have been avoided if there was no exposure to smoking. However, it is important to interpret PAFs in conjunction with the disease's significance in terms of absolute mortality (Northridge, 1995) [15]. For example, although dietary risk factors accounted for "only" 50% of deaths from ischemic heart disease, they still resulted in 51,229 preventable deaths, as ischemic heart disease itself was responsible for nearly 16% of total deaths.

Therefore, PAFs serve as a powerful tool to determine both the rate and absolute numbers of avoidable mortality within a population, enabling policymakers to prioritize interventions and design effective health promotion strategies (Rosen, 2013) [16].

In the Italian context, previous studies have conducted analyses based on the linkage between PAFs and mortality data from national statistics as an indirect method to estimate the number and proportion of deaths attributable to behavioural risk factors, aiming to assess regional differences in cancer-related deaths (Battisti *et al.*, 2017) [17] and deaths from NCDs (Carreras *et al.*, 2019) [18]. According to the former study, at least 45,000 cancer deaths in men and 21,000 in women in 2013 were attributed to modifiable risk factors. The latter study identified that 37% of deaths in men and 26% in women could have been prevented through the implementation of primary prevention interventions, with significant regional variations detected in both studies. Consistent with these findings, our study focuses on five risk factors identified by the Italian health promotion initiative "Guadagnare Salute" (Gaining Health, https://www.epicentro.iss.it/guadagnare-salute/) as targets for reducing the bur-

den of preventable morbidity, mortality, and disability from NCDs. These risk factors include tobacco smoking, excessive alcohol use, unhealthy diet, overweight, and physical inactivity. They are commonly associated with the NCDs responsible for the highest proportion of mortality in Italy, namely cardiovascular diseases (37% of total deaths in 2019), neoplasms (30%), diabetes (3%), and respiratory diseases (2%). The use of PAFs has been recognized as a valuable tool for detecting and understanding spatial disease clusters, which can inform place-based public health interventions (Yiannakoulias, 2009) [19]. However, to the best of our knowledge, our study represents the first attempt to determine the territorial distribution of behavioural risk factors within a specific urban setting by combining PAFs with administrative healthcare data. This approach may offer an alternative strategy for designing and implementing context-specific health promotion interventions to reduce health risk behaviours in the absence of comprehensive data.

2. Data

The study utilized the complete population residing in Milan as of January 1st, 2019, comprising a total of 1,397,494 individuals, with 52% females and 48% males. The population data was extracted from the *Administrative Healthcare Databases* (AHD) maintained by the Agency for Health Protection of the Metropolitan City of Milan. Each individual's record included information on age, sex, and residential address. The residential addresses were geographically referenced to one of the 88 neighbourhoods that make up the city, based on the administrative boundaries defining the *Local Identity Cores* (*Nuclei di Identità Locale*), which are specific neighbourhoods characterized by historical or project-defined features that distinguish them from one another (**Figure A1** in the **Appendix**). Mortality data, obtained from the Register of Causes of Death (*Registro Nominativo delle Cause di Morte*, ReNCaM), were linked to the AHD data using deterministic record linkage, facilitated by the unique tax code assigned to each individual.

3. Methods

Within the study population, deaths that occurred in the year 2019 were identified, and the primary cause of death was determined based on the first code listed on the death certificate, using the ICD-10 coding system. To exclude any distortions related to the COVID-19 pandemic that occurred from the beginning of 2020, the mortality follow-up was limited to one year. Following the methodology of the GBD study, deaths potentially attributed to the five risk factors were identified by linking each risk factor to a specific set of causes of death associated with it (**Table A1** in the **Appendix**). Since the focus of the study was on behavioural risk factors, the selection of causes of death was limited to non-communicable diseases (excluding deaths caused by road accidents, violence, drowning, and other causes that may be related to alcohol use). The exposure to each risk factor was

defined as follows: active smoking (daily or occasional tobacco product use), alcohol use (measured in average grams of pure alcohol consumed per day), dietary risk (characterized by a diet low in fruits, vegetables, legumes, whole grains, nuts and seeds, milk, fiber, calcium, seafood omega-3 fatty acids, polyunsaturated fatty acids, and high in red meat, processed meat, sugar-sweetened beverages, trans fatty acids, and sodium), high body-mass index (BMI greater than 25 kg/m^2), and physical inactivity (measured in total metabolic equivalents—METs, representing the average weekly physical activity at work, home, transportation, and recreational activities, with less than 3000 - 4500 MET minutes per week considered as physically inactive). For each cause of death, the risk-, sex-, and age-specific PAFs for Italy in 2019 were obtained from the Institute for Health Metrics and Evaluation's (IHME) website (https://vizhub.healthdata.org/gbd-results/). These PAFs were calculated in the GBD study (GBD, 2015) [13] using a specific formula:

$$\mathrm{PAF_r} = \frac{p_r\left(RR_r - 1\right)}{p_r\left(RR_r - 1\right) + 1} \tag{1}$$

where p_r is the prevalence of the risk factor r in the study population; RR_r is the relative risk of death for the exposed to the risk factor r compared to the non-exposed.

For each neighbourhood, the absolute number of deaths occurred in the study population associated with a risk factor were then multiplied by the corresponded PAF, identifying the absolute number of deaths attributable to the specific risk factor, which divided by the overall number of deaths in the neighbourhood led to the estimation of the proportion of deaths attributable to the risk factor in the area (see **Table A2** in the **Appendix** for clarification about the procedure followed for the calculation of absolute deaths and proportion over total deaths for mortality caused by each risk factor).

The final PAFs calculated represent the proportion of deaths attributable to each specific risk assuming that there is no interdependence between risk factors associated with multiple exposures (Carreras, 2019) [18]. However, it is important to note that certain causes of death may share one or more risk factors. As a result, the sum of PAFs for each risk factor may exceed 100%. To address this issue, we calculated the combined PAF using the methodology established by the GBD study. This approach assumes independence between risk factors and does not account for possible mediation, correlation, or effect modifications between the exposures (Battisti, 2017 [17]; Ezzati *et al.*, 2003 [20]). The formula used to calculate the combined PAF is as follows:

$$\mathrm{PAF_{combined}} = 1 - \prod_{r=1}^{5}\left(1 - \mathrm{PAF_r}\right) \tag{2}$$

where $\mathrm{PAF_r}$ is the PAF relative to the risk factor r.

The six PAFs calculated for each neighbourhood (five related to specific risk factors and one combined) were visualized on a map to examine the territorial variations in attributable mortality. The PAFs were categorized into five classes

using *Jenks' Natural Break Classification* (Jenks, 1967) [21], a method that minimizes the average deviation within classes while maximizing the deviation from the means of other classes. This approach aims to highlight significant differences in the distribution of the variable based on inherent groupings in the data. It is a preferable alternative to quantile division, which assigns an equal number of units to each class and may not capture the true distribution patterns in non-linearly distributed data (Osaragi, 2002) [22]. The analysis was stratified by sex, and neighbourhoods with fewer than 10 deaths (all causes) for each sex category in the year were excluded from the analysis to ensure reliable estimates. This exclusion criterion aimed to avoid unreliable estimates in areas with low population density, such as urban parks and rural areas on the outskirts, where the occurrence of a small number of deaths could lead to distorted measures (e.g., a high attributable mortality from smoking based on only 2 lung cancer deaths out of 4 total deaths in the year). As a result, 18 neighbourhoods meeting this criterion were excluded from the analysis. The PAFs were also provided age-adjusted (direct standardization method), with the five-year age classes distribution of the overall city as reference population. Age-adjusted estimates, although less informative in terms of the absolute impact of each risk factor on neighbourhood-specific mortality patterns, allow for comparisons between different areas of the city, independent of their age composition.

In addition, we conducted bivariate correlations between the calculated PAFs and the average number of years of formal education per person in each neighbourhood, used as a proxy for neighbourhood deprivation. This analysis served as an initial exploratory step to evaluate whether the distribution of attributable mortality aligns with the pattern of socioeconomic inequality in the city.

4. Results

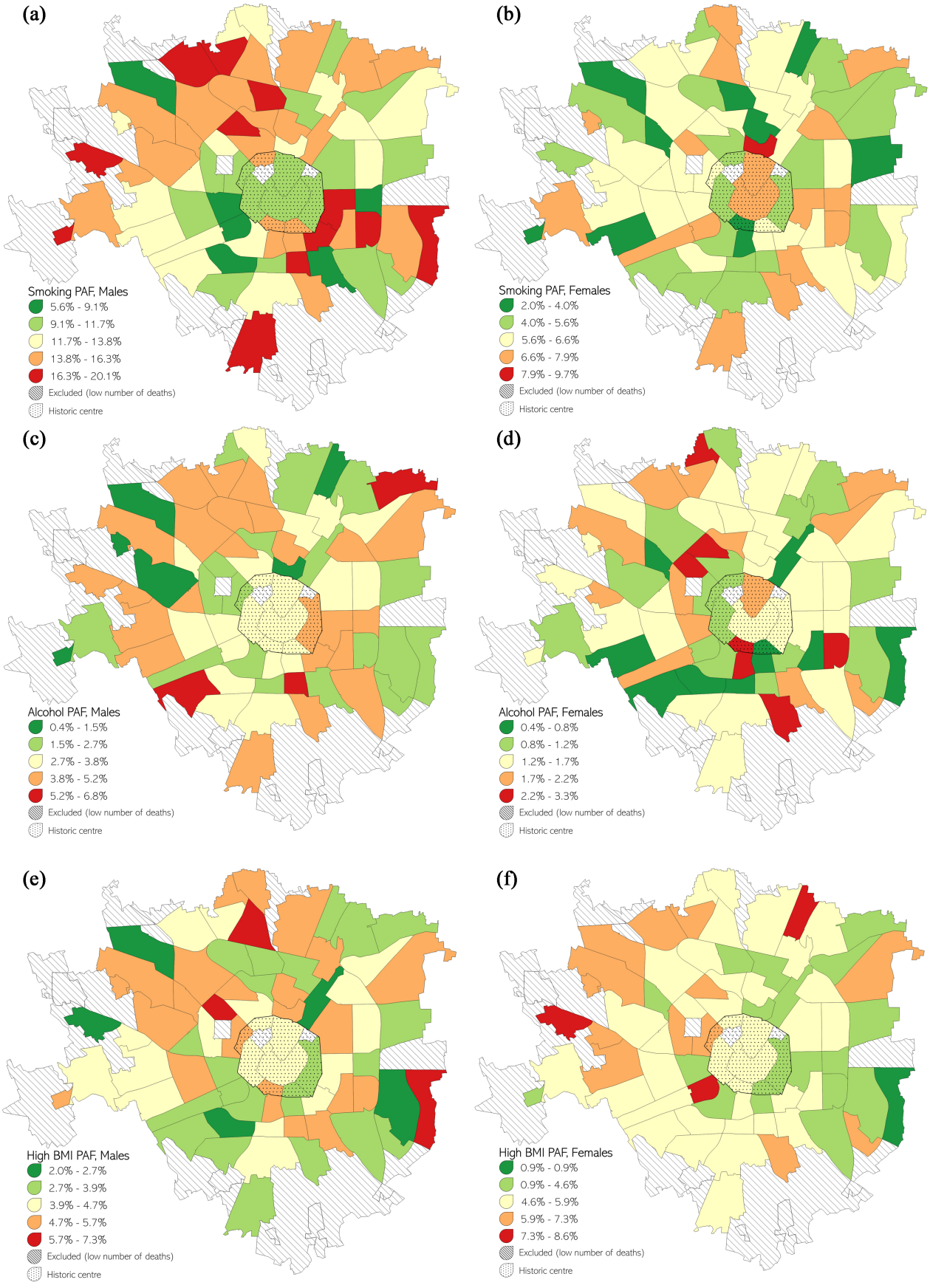
Table 1 displays the distribution of mortality and PAFs for each risk factor across the entire Municipality of Milan. The absolute number of deaths attributable to each risk factor represents the number of deaths that could have been prevented if there had been no exposure to the specific risk. Overall, these risk factors accounted for 2828 deaths, equivalent to 20.1% of the total mortality rate in the population (18.0% for females and 22.6% for males). Among men, smoking was identified as the most significant risk factor, contributing to 13.4% of total deaths. In women, dietary risk (6.0%), smoking (5.9%), and high BMI (5.3%) had similar contributions to mortality. Alcohol use was responsible for more than twice as many deaths in men (3.5%) compared to women (1.5%), while physical inactivity accounted for 1.0% of deaths in men and 1.7% in women.

Figure 1 presents the territorial distribution of each risk factor's PAFs. The most noticeable difference between sexes is the territorial gradient present in relation to each risk factor—though less pronounced for alcohol (**Figure 1(c)**) and high BMI (**Figure 1(e)**)—in males, which does not find correspondence in

Table 1. Deaths, mortality, and PAFs in the study population, by sex. Milan, 2019.

	Females	Males	Total
Population (n)	730,218	667,276	1,397,494
Number of deaths (n)	7784	6315	14,099
Mortality rate (‰)	10.7	9.5	10.1
Smoking—Attributable deaths (n)	460	847	1308
Smoking—PAF (%)	5.9	13.4	9.3
Alcohol use—Attributable deaths (n)	113	221	335
Alcohol use—PAF (%)	1.5	3.5	2.4
High BMI—Attributable deaths (n)	416	270	686
High BMI—PAF (%)	5.3	4.3	4.9
Dietary risk—Attributable deaths (n)	464	455	919
Dietary risk—PAF (%)	6.0	7.2	6.5
Physical inactivity—Attributable deaths (n)	130	62	192
Physical inactivity—PAF (%)	1.7	1.0	1.4
Combined risk factors—Attributable deaths (n)	1399	1429	2828
Combined risk factors—PAF (%)	18.0	22.6	20.1

females. In the former, the central areas exhibit lower mortality attributable to the five risk factors, while most peripheral neighbourhoods, albeit with different patterns for specific risk factors, consistently show higher rates. The most evident case is that of smoking (**Figure 1(a)**), which stands out with a clustering of high PAF in the north-western and south-eastern peripheral belts. In contrast, females do not display a marked centre-periphery dualism in the distribution of PAFs. This is particularly evident in relation to smoking (**Figure 1(b)**), where affluent central neighbourhoods like Duomo, Breda, and Garibaldi-Repubblica fall into the same category as some disadvantaged suburban areas. Considering all risk factors simultaneously, while some areas show similarities in having a low combined PAF for both sexes (e.g., Guastalla, Lambrate, Lodi-Corvetto, Maggiore-Musocco, Ortomercato, Padova, Rogoredo, Ronchetto sul Naviglio, Umbria-Molise), or conversely, a concentration of excess risk (e.g., Affori, Bovisasca, Gallaratese, Garibaldi-Repubblica, Ghisolfa, Niguarda, Quarto Cagnino, Quarto Oggiaro, Quinto Romano, Ticinese), it is clear that the overall configuration of attributable mortality differs substantially between sexes. In males, the central neighbourhoods mostly exhibit average or below-average PAFs, with two clusters of well-performing areas in the southwestern and southeastern periphery (**Figure 1(m)**). In contrast, for females, the central neighbourhoods align with higher attributable mortality found in a western and a northern cluster of areas, without any spatial grouping of neighbourhoods with below-average PAFs (**Figure 1(n)**).


(a)
Smoking PAF, Males
5.6% - 9.1%
9.1% - 11.7%
11.7% - 13.8%
13.8% - 16.3%
16.3% - 20.1%
Excluded (low number of deaths)
Historic centre
(b)
Smoking PAF, Females
2.0% - 4.0%
4.0% - 5.6%
5.6% - 6.6%
6.6% - 7.9%
7.9% - 9.7%
Excluded (low number of deaths)
Historic centre
(c)
Alcohol PAF, Males
0.4% - 1.5%
1.5% - 2.7%
2.7% - 3.8%
3.8% - 5.2%
5.2% - 6.8%
Excluded (low number of deaths)
Historic centre
(d)
Alcohol PAF, Females
0.4% - 0.8%
0.8% - 1.2%
1.2% - 1.7%
1.7% - 2.2%
2.2% - 3.3%
Excluded (low number of deaths)
Historic centre
(e)
High BMI PAF, Males
2.0% - 2.7%
2.7% - 3.9%
3.9% - 4.7%
4.7% - 5.7%
5.7% - 7.3%
Excluded (low number of deaths)
Historic centre
(f)
High BMI PAF, Females
0.9% - 0.9%
0.9% - 4.6%
4.6% - 5.9%
5.9% - 7.3%
7.3% - 8.6%
Excluded (low number of deaths)
Historic centre

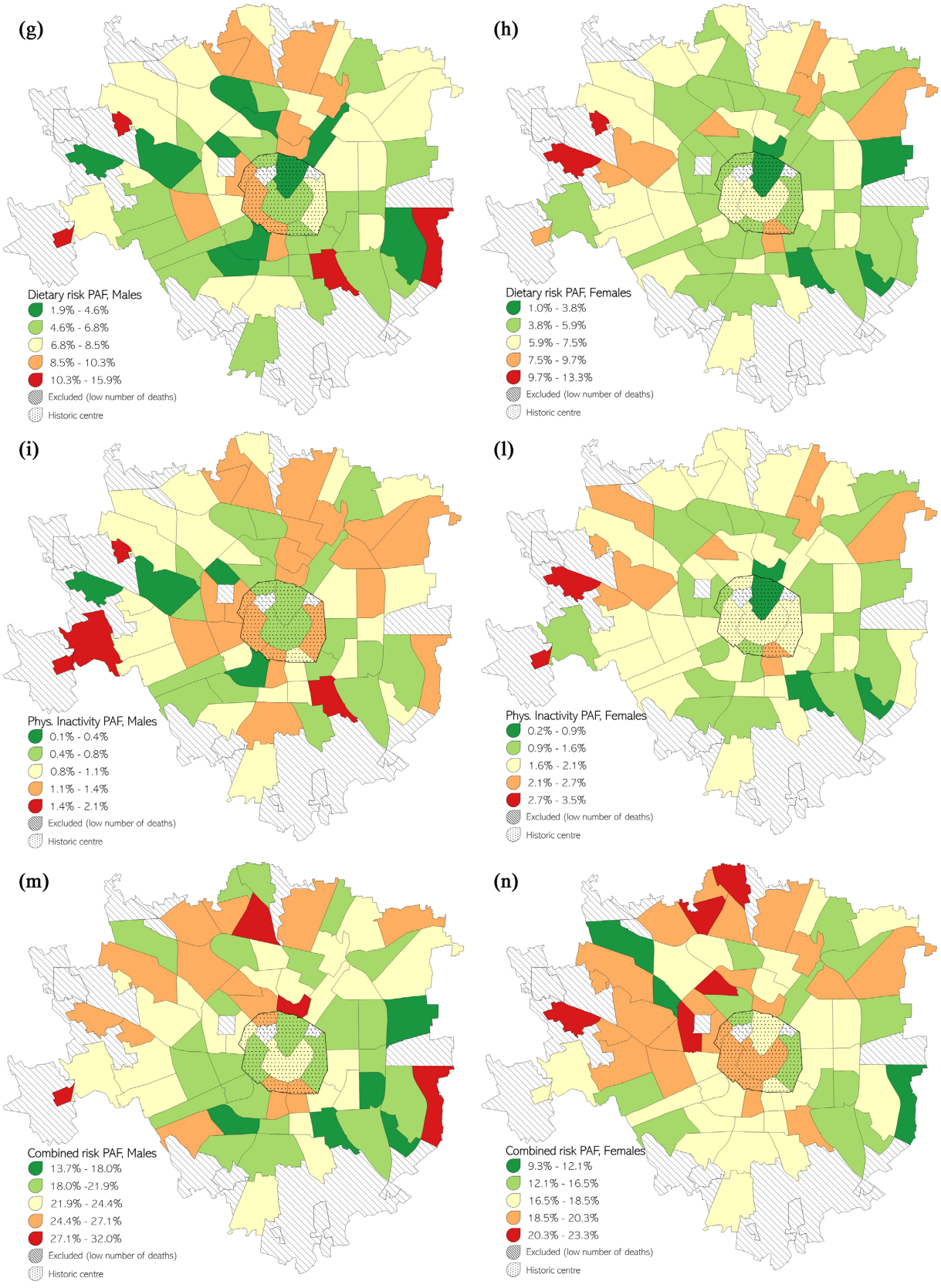


Figure 1. Males and Females' PAFs for each risk factor and for combined risk factors, by neighbourhood. Neighbourhoods with less than 10 deaths were excluded. Milan, 2019.

Age-adjusted PAFs were calculated to allow for the comparison of neighbourhoods with different age compositions. The standardization procedure helped to mitigate the differences between sexes regarding the PAFs, as shown in **Table A3** in the **Appendix**. At the sub-municipal level, although the standardization process introduced some changes in the territorial pattern of the PAFs, the fact that differences between neighbourhoods still persisted even after age adjustment indicates that the observed territorial heterogeneity was not solely attributable to variations in the age profiles of the neighbourhoods (refer to **Figure A3** in the **Appendix**), excluding the possibility of a compositional effect. Regarding the relationship between neighbourhood-level educational attainment (**Figure A2** in the **Appendix**) and the calculated PAFs, weak inverse correlations were found in males, indicating that higher neighbourhood education was associated with lower risk-specific attributable mortality. However, these correlations were extremely weak, except for smoking PAF, which exhibited a moderate correlation with an R^2 value of 0.11. No significant correlations were found in females (**Figure A4** in the **Appendix**).

5. Discussion

This study applied PAFs for five risk factors to administrative healthcare data to detect territorial differences in the distribution of risky health-related behaviours within the City of Milan. The goal was to generate valuable insights for planning and implementing targeted health promotion interventions that consider the specific characteristics of sub-municipal areas. Often, public health campaigns are conducted without precise information on the prevalence of detrimental lifestyles within a population, leading to a lack of targeting towards higher-risk groups. In this study, we employed an indirect method to assess the distribution of behavioral risk factors within an urban context, aiming to assist decision-makers in addressing health inequalities present in a specific setting (e.g., Friel *et al.*, 2007) [23]. Out of the total of 2828 preventable deaths, smoking accounted for nearly half, making it the most significant risk factor to address for reducing avoidable mortality, particularly among men. In women, metabolic-related risk factors played a larger role in shaping mortality patterns. This finding aligns with existing knowledge that cardiovascular diseases have been replaced by neoplasms as the primary cause of death in men, while this transition has not yet occurred in women (IHME, 2023) [1].

The most intriguing findings of this study pertain to the sub-municipal differences. Given previous empirical research indicating that socioeconomic inequality in Milan is concentrated along the centre-periphery axis (Consolazio *et al.*, 2021 [4], 2023 [5]; Petsimeris & Rimoldi, 2015 [6]), it was possible to investigate whether there was a socioeconomic pattern of attributable mortality in the city, focusing specifically on the neighbourhoods most affected by mortality due to behavioural risk factors. Interestingly, this pattern was observed exclusively among males, while it was not evident among females. Although this may seem

contradictory, it aligns with existing evidence regarding differences in the distribution of health behaviours across social strata between sexes in Italy. For example, women exhibit a reversed social gradient in smoking, with higher tobacco consumption observed among those with higher education compared to those with lower education (Alicandro *et al.*, 2018 [24]; Federico *et al.,* 2004 [25]; Tramacere *et al.*, 2009 [26]). This phenomenon could be attributed to smoking being perceived as a means of empowerment among younger women. However, other risk factors examined in the study, such as dietary risk, alcohol use, and high BMI, are known to follow a social gradient in both males and females. Hence, the presence of a territorial gradient among males but not females could be partially linked to the greater socioeconomic inequality in overall mortality observed among men compared to women in Italy (Alicandro *et al.*, 2018) [24], consistent with trends observed in most European populations (Mackenbach *et al.*, 2008) [27]. Supporting this interpretation, a previous study (Carreras *et al.*, 2019) [18] revealed that the Lombardy region, where Milan is located, ranks fourth to last among the 20 Italian regions in terms of the proportion of NCD deaths attributable to the combination of the five risk factors in females, while it aligns with the national average for males.

As an exploratory endeavour, the examination of the correlation between the PAFs and neighbourhood-level education provided some evidence in line with these findings. In males, a weak inverse association was observed between each PAF (except for high BMI) and education. Although these associations were minimal, they suggested the presence of a pattern. It is important to note that the weak correlations may be influenced by the limited number of observations (70 neighbourhoods), with few outliers contributing to weaken the overall pattern, as well as the internal heterogeneity in educational attainment within the neighbourhoods. In females, the absence of a consistent relationship between the PAFs and average education was consistent with the previously mentioned findings.

Indeed, the term "gender" may be more appropriate than "sex" when discussing the differences observed in this study. While we used the epidemiological terminology of "sex", it is essential to acknowledge that the divergent exposure to risk factors leading to distinct mortality patterns between males and females is a result of differences deeply rooted in the social and cultural environment in which individuals exist. Gender differences can manifest at various stages of prevention (Gordon, 1983) [18]. Beyond the adoption of unhealthy lifestyles (Oncini & Guetto, 2018) [29], socioeconomic and cultural factors can also influence access to healthcare services and preventive screening (Davis *et al.*, 2012) [30], as well as therapeutic compliance (Manteuffel *et al.*, 2014) [31]. However, it is important to recognize that certain health outcome disparities between men and women arise from physiological processes, primarily related to sex as a biological condition rather than a social and cultural category. For example, in the case of hypertension, women experience the protective effects of oestrogen be-

fore the onset of menopause, which makes them less susceptible to salt sensitivity and contributes to a delayed onset of the disease compared to men (Consolazio *et al.*, 2022) [32]. Since we do not have the means to determine the relative contributions of sex-related inherent characteristics and gender-driven sociocultural processes to mortality patterns, it is crucial to recognize the significance of both factors and conduct further research to better understand how to address inequalities and reduce the burden of NCDs by addressing the prevalence of health-damaging lifestyles.

The variation in territorial heterogeneity among different risk factors is evident, emphasizing the need to develop tailored strategies that go beyond the conventional concept of "neighbourhood disadvantage" often used in the field. It is clear that certain risk factors exhibit a higher degree of variation across neighbourhoods, highlighting the importance of focusing on the prevalent health-damaging lifestyles within each local area. This consideration holds particular significance for females, as relying solely on interventions targeted at deprived areas may prove ineffective if the specific risk profiles of individual neighborhoods are not taken into account.

An additional issue worth discussing is that of a possible contextual effect (Macintyre *et al.*, 2002) [12] in the phenomenon studied. The observed differences in PAFs across neighbourhoods may not solely be attributed to the socioeconomic composition of each area, but also to the material and psychosocial characteristics of the living environment. While individual factors such as education, occupation, and income are known to impact the adoption of risky health behaviours (Braveman & Gottlieb, 2014) [8] the presence of neighbourhoods with high PAFs might not be solely due to the aggregation of individual profiles, being also affected by environmental characteristics influencing individual behaviours. For example, the availability of walkable, cyclable, and green spaces (Rao *et al.*, 2007) [33], as well access to a healthy local food environment (Caspi *et al.*, 2012) [34], and the proximity to healthcare services (Brondeel *et al.*, 2014) [35] can all influence individual actions and behaviours. Additionally, less tangible neighbourhood features such as perceived crime, air and noise pollution, litter, and urban decay can contribute to a sense of unsafety, which may deter individuals from going outside if avoidable and affect their inclination for physical activity (Baum *et al.*, 2009) [36]. Moreover, these stress-inducing factors can also lead individuals to adopt risky behaviors as a coping mechanism for dealing with stressful conditions (Algren *et al.*, 2018 [37]; Pearce *et al.*, 2012 [38]). In some cases, stress can have direct effects on health outcomes independent of health-related behaviors. Prolonged exposure to multiple environmental stressors can result in allostatic load, which refers to the wear and tear on the body's physiological systems (Prior *et al.*, 2018) [39]. When individuals perceive their living environment as unsafe, it can lead to chronic stress, triggering the release of cortisol and cytokines that can cause damage to the immune and body systems. This, in turn, can impact insulin resistance and accelerate the

progression of chronic diseases, including T2DM (McEwen & Wingfield, 2003) [40].

Despite the importance of neighbourhood characteristics, the literature generally suggests that their role is weaker compared to individual socioeconomic conditions, with contextual effects being modest and smaller than compositional effects (Pickett & Pearl, 2001) [41]. This pattern holds true for the case of Milan as well (Consolazio *et al.*, 2021) [4]. Related to this, it is worth noting that the different measures of PAFs provided in the study (crude and age-adjusted) serve different purposes. The crude PAFs presented in the main text reflect a compositional effect, showing the actual impact of a specific risk factor on neighbourhood mortality and reflecting the population profile. These measures are useful for designing public health interventions based on the burden of NCDs in a particular local area. On the other hand, the use of age-adjusted measures allows for more appropriate comparisons between neighbourhoods but comes at the cost of losing information regarding the specific impact of a risk factor on mortality patterns. Age-adjusted measures help to assess the relative importance of each risk factor if all neighbourhoods had the same age profile, potentially highlighting the impact of other neighbourhood characteristics such as socioeconomic arrangements or contextual features that promote the adoption of risk behaviours. However, without measuring these factors directly, it is challenging to identify proper contextual effects.

Among all, this study has two limitations that should be highlighted. Firstly, in determining the number of deaths attributed to each risk factor, we relied on the GBD project (GBD, 2015) [13], which provided a single value for each risk factor and age group at the country level. Therefore, the PAFs used in this study represent the Italian average and do not account for specific geographic differences, such as regional or North/South divide variations. This could potentially overlook important variations in risk factor prevalence across different areas of Italy. Secondly, the classification of mortality data in the AHD is subject to some degree of bias, as death certificates may have low specificity or misclassification of causes of death (Alicandro *et al.*, 2018 [24]; Capocaccia *et al.*, 2009 [42]; Minelli & Marchetti, 2013 [43]). This could impact the accuracy of the estimates derived from the data and introduce potential measurement errors. Furthermore, it is important to note that comparing the results of this study with estimates from other Italian studies that used a similar methodology (e.g., Battisti *et al.*, 2017 [17]; Carreras *et al.*, 2019 [18]) may be misleading. The methods used to calculate PAFs within the GBD project are periodically updated, including changes in the thresholds used to determine exposure to risky behaviours, such as the number of cigarettes smoked or grams of alcohol consumed daily, portions of food categories for dietary risk, or the MET for identifying physical inactivity. These methodological updates could lead to discrepancies when comparing results from different time periods or studies using different versions of the methodology.

6. Conclusion

In this study, we aimed to estimate the number and proportion of deaths attributed to behavioural risk factors in the neighbourhoods of Milan in 2019. These risk factors are modifiable, meaning that a certain portion of the mortality associated with them is potentially preventable. Therefore, they are a focal point for primary and secondary prevention efforts in healthcare. However, the lack of context-specific information on the distribution of these risk factors hampers the development of effective interventions. To address this data gap, we proposed a methodology to indirectly estimate the distribution of risky health behaviours at the neighbourhood level, using PAFs applied to administrative healthcare data. By doing so, we were able to uncover distinct territorial patterns in the distribution of risk factors based on sex/gender. These findings suggest the presence of multiple avenues for intervention to reduce the burden of NCDs and address the associated inequalities, which stem from the interplay of territorial, socioeconomic, and sex/gender dimensions. By identifying these patterns and understanding the complex interrelationships between various factors, policymakers and public health professionals can develop targeted interventions that address the specific needs and challenges of different neighbourhoods, with the aim of reducing NCDs and promoting health equity.

Funding

The study has been conducted in the context of the project "Enhancing healthcare and well-being through the potential of big data: an integration of survey, administrative, and open data to assess health risk in the city of Milan with data science" (Project Code 2020-4269) funded by Fondazione Cariplo (https://www.fondazionecariplo.it/en/index.html) within the call "Data Science for science and society". The funder had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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Appendix Tables and Figures

Table A1. Causes of death, correspondent ICD 10 codes, and their association with risk factors, as codified in the GBD study. Numbers in brackets indicate the number of causes of death related to each risk factor.

Causes of death	ICD-10 Code	Smoking (32)	Alcohol use (11)	High BMI (22)	Dietary risk (18)	Physical inactivity (5)
Alcohol use disorders	E24.4, F10-F10.9, G31.2, G62.1, G72.1, P04.3, Q86.0, R78.0, X45-X45.9, X65-X65.9, Y15-Y15.9		X			
Alzheimer's disease and other dementias	F00-F02.0, F02.8-F03.9, G30-G31.1, G31.8-G31.9	X		X		
Aortic aneurysm	I71-I71.9	X			X	
Asthma	J45-J46.9	X		X		
Atrial fibrillation and flutter	I48-I48.9	X	X	X	X	
Bladder cancer	C67-C67.9, D09.0, D30.3, D41.4-D41.8, D49.4	X				
Breast cancer	C50-C50.9, D05-D05.9, D24-D24.9, D48.6, D49.3	X	X	X	X	X
Cardiomyopathy and myocarditis	B33.2, I40-I41.9, I42.1-I42.8, I43-I43.9, I51.4		X		X	
Cervical cancer	C53-C53.9, D06-D06.9, D26.0	X				
Chronic kidney disease	D63.1, E10.2, E11.2, I12-I13.9, N02-N08.8, N15.0, N18-N18.9, Q61-Q62.8			X	X	
Chronic obstructive pulmonary disease	J41-J44.9	X				
Cirrhosis and other chronic liver diseases	B18-B18.9, I85-I85.9, I98.2, K70-K70.3, K71.7, K73-K75, K75.2, K75.4-K76.2, K76.4-K76.9, K77.8		X			
Colon and rectum cancer	C18-C21.9, D01.0-D01.3, D12-D12.9, D37.3-D37.5	X	X	X	X	X
Diabetes mellitus	E10-E10.1, E10.3-E11.1, E11.3-E11.9, P70.2	X	X	X	X	X
Endocarditis	I33-I33.9, I38-I39.9				X	
Oesophageal cancer	C15-C15.9, D00.1, D13.0	X	X	X	X	
Gallbladder and biliary diseases	K80-K83.9	X		X		
Gallbladder and biliary tract cancer	C23-C24.9, D13.5			X		
Hypertensive heart disease	I11-I11.9		X	X	X	
Idiopathic epilepsy	G40-G41.9		X			
Ischaemic heart disease	I20-I25.9	X	X	X	X	X
Kidney cancer	C64-C65.9, D30.0-D30.1, D41.0-D41.1	X		X		
Larynx cancer	C32-C32.9, D02.0, D14.1, D38.0	X	X			
Leukaemia	C91-C91.0, C91.2-C91.3, C91.6, C92-C92.6, C93-C93.1, C93.3, C93.8, C94-C95.9	X		X		

Continued

Lip and oral cavity cancer	C00-C08.9, D10.0-D10.5, D11-D11.9	X	X			
Liver cancer	C22-C22.8, D13.4	X	X	X		
Lower respiratory infections	A48.1, A70, B97.4-B97.6, J09-J15.8, J16-J16.9, J20-J21.9, J91.0, P23.0-P23.4, U04-U04.9	X	X			
Multiple myeloma	C88-C90.9			X		
Multiple sclerosis	G35-G35.9	X				
Nasopharynx cancer	C11-C11.9, D10.6	X	X			
Non-Hodgkin lymphoma	C82-C86.6, C96-C96.9			X		
Non-rheumatic valvular heart disease	I34-I37.8				X	
Other cardiovascular and circulatory diseases	I28-I28.9, I30-I31.1, I31.8-I32.8, I47-I47.9, I51.0-I51.3, I68.0, I72-I72.9, I77-I83.9, I86-I89.0, I89.9, I98, K75.1				X	
Other pharynx cancer	C09-C10.9, C12-C13.9, D10.7	X	X			
Ovarian cancer	C56-C56.9, D27-D27.9, D39.1			X		
Pancreatic cancer	C25-C25.9, D13.6-D13.7	X		X		
Pancreatitis	K85-K86.9		X			
Parkinson's disease	F02.3, G20-G20.9	X				
Peripheral artery disease	I70.2-I70.8, I73-I73.9	X			X	
Prostate cancer	C61-C61.9, D07.5, D29.1, D40.0	X				
Rheumatic heart disease	I01-I01.9, I02.0, I05-I09.9				X	
Rheumatoid arthritis	M05-M06.9, M08.0-M08.8	X				
Stomach cancer	C16-C16.9, D00.2, D13.1, D37.1	X			X	
Stroke	G45-G46.8, I60-I63.9, I65-I66.9, I67.0-I67.3, I67.5-I67.6, I68.1-I68.2, I69.0-I69.3	X	X	X	X	X
Thyroid cancer	C73-C73.9, D09.3, D09.8, D34-D34.9, D44.0			X		
Tracheal, bronchus, and lung cancer	C33-C34.9, D02.1-D02.3, D14.2-D14.3, D38.1	X			X	
Tuberculosis	A10-A14, A15-A19.9, B90-B90.9, K67.3, K93.0, M49.0, N74.1, P37.0, U84.3	X	X			
Upper digestive system diseases	K25-K29.9	X				
Uterine cancer	C54-C54.9, D07.0-D07.2, D26.1-D26.9			X		

Table A2. Example of calculation of absolute deaths and proportion over total deaths for mortality caused by a hypothetical risk factor associated with two causes of death.

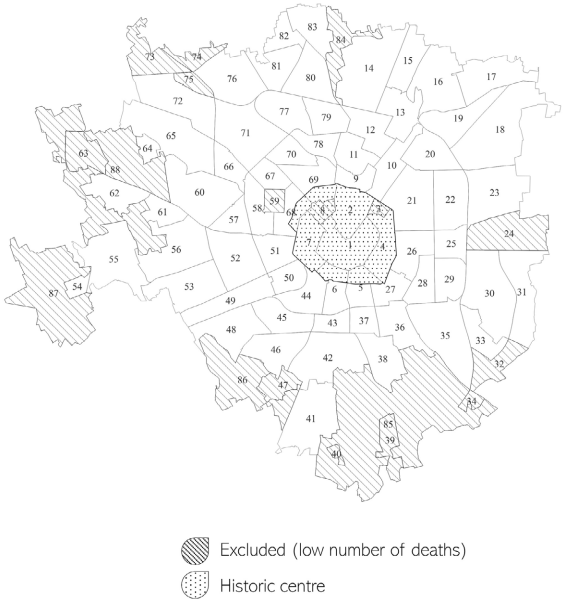
	NIL code	Sex	Age class	Total deaths (n)	Deaths by cause 1 (n)	Deaths by cause 2 (n)	PAFs cause 1	PAFs cause 2	Deaths from cause 1 attributable to risk factor (n)	Deaths from cause 2 attributable to risk factor (n)	Total deaths attributable to risk factor (n)	Proportion of deaths attributable to risk factor (%)
n	A	B	C	D	E	F	G	H	I	L	M	N
1	57	M	50 - 54	7	1	3	0.15	0.21	0.15	0.63		
2	57	M	55 - 59	9	3	4	0.23	0.23	0.69	0.92		
3	57	M	60 - 64	13	5	7	0.34	0.26	1.70	1.82		
4	57	M	65 - 69	17	6	4	0.37	0.32	2.22	1.28		
5	57	M	70 - 74	24	9	3	0.42	0.34	3.78	1.02		
6	57	M	sum	70	24	21	1.51	1.36	8.54	5.67	**14.21**	**20.30**
7	57	F	50 - 54	5	1	3	0.07	0.11	0.07	0.33		
8	57	F	55 - 59	8	2	4	0.09	0.13	0.18	0.52		
9	57	F	60 - 64	11	7	5	0.10	0.14	0.70	0.70		
10	57	F	65 - 69	15	6	3	0.11	0.16	0.66	0.48		
11	57	F	70 - 74	21	3	1	0.13	0.21	0.39	0.21		
12	57	F	sum	60	19	16	0.50	0.75	20	2.24	**4.24**	**7.07**
	Source: Administrative Healhtcare Databases (AHD)			Source: Register of Causes of Death (ReNCaM)			Source: GBD study		$E_n * G_n$	$F_n * H_n$	$I_{n+} L_n$	$M_n / C_n * 100$

Population characteristics in columns A-C come from *Administrative Healhtcare Databases* (AHD). Mortality data in columns D-F come from *Register of Causes of Death* (ReNCaM). Population Attributable Fractions (PAFs) in columns G-H come from *Global Burden of Disease* (GBD) study. Data in columns I-N are calculated with formulas reported in the bottom line.

Table A3. Comparison between crude and age-adjusted PAFs in the study population, by sex. Milan, 2019.

	Crude PAFs			Age-adjusted PAFs			$\Delta M\text{-}F_{crude}$	$\Delta M\text{-}F_{age\text{-}adj.}$
	Females	Males	Total	Females	Males	Total		
Smoking—PAF (%)	5.9	13.4	9.3	10.7	10.9	10.8	7.5	0.2
Alcohol use—PAF (%)	1.5	3.5	2.4	4.2	4.0	4.1	2.0	−0.2
High BMI—PAF (%)	5.3	4.3	4.9	8.2	3.3	5.8	−1.0	−4.9
Dietary risk—PAF (%)	6.0	7.2	6.5	6.5	6.0	6.2	1.2	−0.5
Physical inactivity—PAF (%)	1.7	1.0	1.4	2.8	0.3	1.6	−0.7	−2.5
Combined risk factors—PAF (%)	18.’	22.6	20.1	14.3	14.2	14.3	4.6	−0.1

Δ = PAFs difference between males and males.



1 Duomo
2 Brera
3 Giardini Porta Venezia
4 Guastalla
5 Porta Vigentina - Porta Lodovica
6 Porta Ticinese - Conca del Naviglio
7 Magenta- San Vittore
8 Parco Sempione
9 Porta Garibaldi - Porta Nuova
10 Stazione Centrale - Ponte Seveso
11 Isola
12 Maciachini-Maggiolina
13 Greco - Segnano
14 Niguarda - Ca' Granda - Prato Centenaro - Q.re Fulvio Testi
15 Bicocca
16 Gorla - Precotto
17 Adriano
18 Cimiano - Rottole - Q.re Feltre
19 Padova - Turro - Crescenzago
20 Loreto - Casoretto - NoLo
21 Buenos Aires - Porta Venezia - Porta Monforte
22 Città studi
23 Lambrate - Ortica
24 Parco Forlanini - Cavriano
25 Corsica
26 XXII Marzo
27 Porta Romana
28 Umbria - Molise - Calvairate
29 Ortomercato
30 Taliedo - Morsenchio - Q.re Forlanini
31 Monluè - Ponte Lambro
32 Triulzo Superiore
33 Rogoredo - Santa Giulia
34 Chiaravalle
35 Lodi-Corvetto
36 Scalo Romana
37 Morivione
38 Vigentino - Q.re Fatima
39 Quintosole
40 Ronchetto delle Rane
41 Gratosoglio - Q.re Missaglia - Q.re Terrazze
42 Stadera - Chiesa Rossa - Q.re Torretta - Conca Fallata
43 Tibaldi
44 Porta Ticinese - Conchetta
45 Moncucco - San Cristoforo
46 Barona
47 Cantalupa
48 Ronchetto sul Naviglio - Q.re Lodovico il Moro
49 Giambellino
50 Porta Genova
51 Porta Magenta
52 Bande Nere
53 Lorenteggio
54 Muggiano
55 Baggio - Q.re degli Olmi - Q.re Valsesia
56 Forze Armate
57 San Siro
58 De Angeli-Monte Rosa
59 Tre Torri
60 Stadio - Ippodromi
61 Quarto Cagnino
62 Quinto Romano
63 Figino
64 Trenno
65 Q.re Gallaratese - Q.re San Leonardo - Lampugnano
66 QT8
67 Portello
68 Pagano
69 Sarpi
70 Ghisolfa
71 Villapizzone - Cagnola - Boldinasco
72 Maggiore - Musocco - Certosa
73 MIND - Cascina Triulza 74
74 Roserio
75 Stephenson
76 Quarto Oggiaro - Vialba - Musocco
77 Bovisa
78 Farini
79 Dergano
80 Affori
81 Bovisasca
82 Comasina
83 Bruzzano
84 Parco Nord
85 Parco delle Abbazie
86 Parco dei Navigli
87 Assiano
88 Parco Bosco in città

Figure A1. Subdivision of Milan into its 88 neighbourhoods.

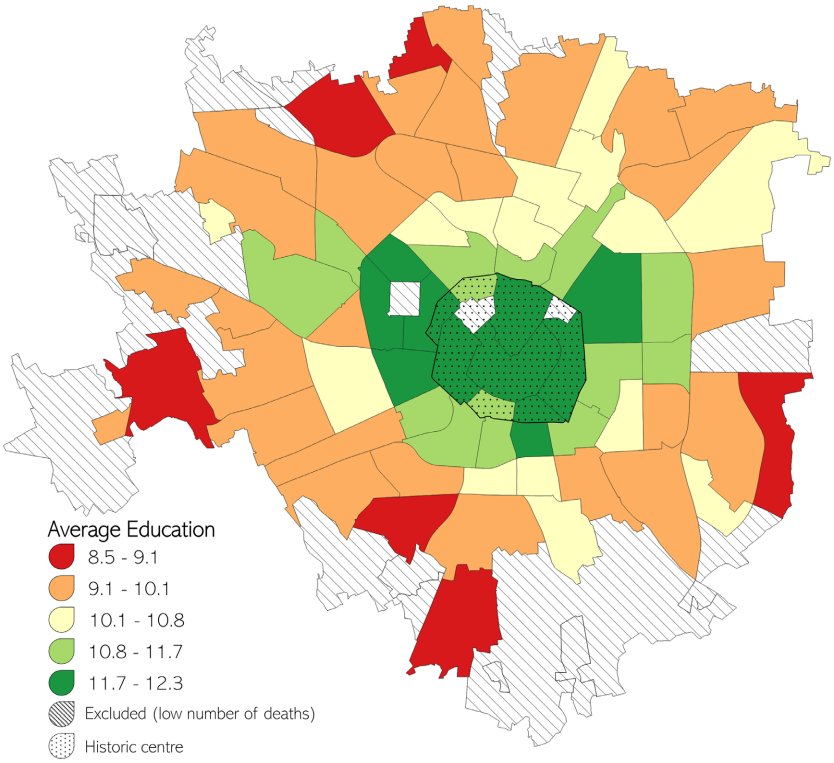
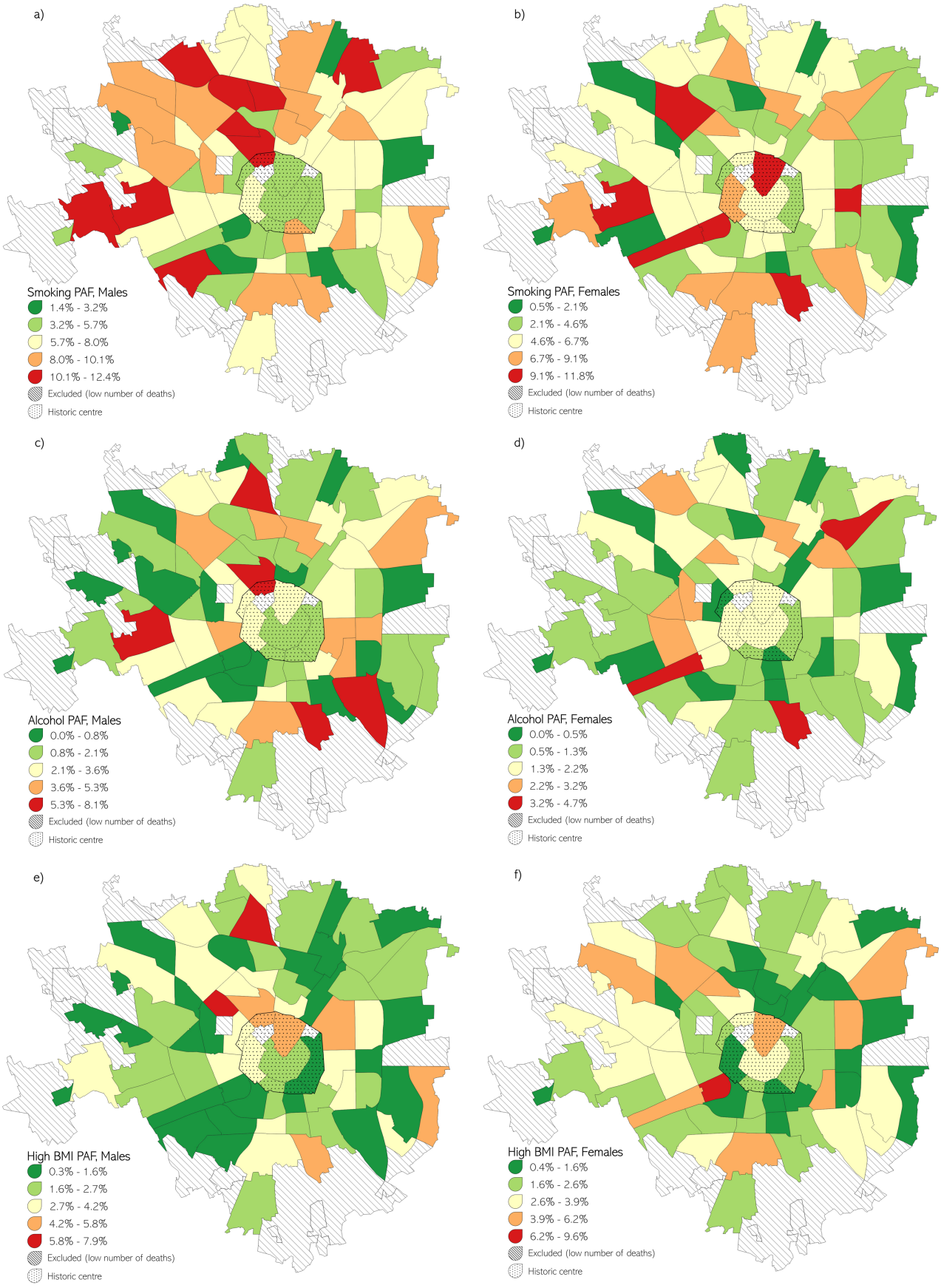


Figure A2. Average years of schooling per person in each neighbourhood.


a)
Smoking PAF, Males
1.4% - 3.2%
3.2% - 5.7%
5.7% - 8.0%
8.0% - 10.1%
10.1% - 12.4%
Excluded (low number of deaths)
Historic centre
b)
Smoking PAF, Females
0.5% - 2.1%
2.1% - 4.6%
4.6% - 6.7%
6.7% - 9.1%
9.1% - 11.8%
Excluded (low number of deaths)
Historic centre
c)
Alcohol PAF, Males
0.0% - 0.8%
0.8% - 2.1%
2.1% - 3.6%
3.6% - 5.3%
5.3% - 8.1%
Excluded (low number of deaths)
Historic centre
d)
Alcohol PAF, Females
0.0% - 0.5%
0.5% - 1.3%
1.3% - 2.2%
2.2% - 3.2%
3.2% - 4.7%
Excluded (low number of deaths)
Historic centre
e)
High BMI PAF, Males
0.3% - 1.6%
1.6% - 2.7%
2.7% - 4.2%
4.2% - 5.8%
5.8% - 7.9%
Excluded (low number of deaths)
Historic centre
f)
High BMI PAF, Females
0.4% - 1.6%
1.6% - 2.6%
2.6% - 3.9%
3.9% - 6.2%
6.2% - 9.6%
Excluded (low number of deaths)
Historic centre

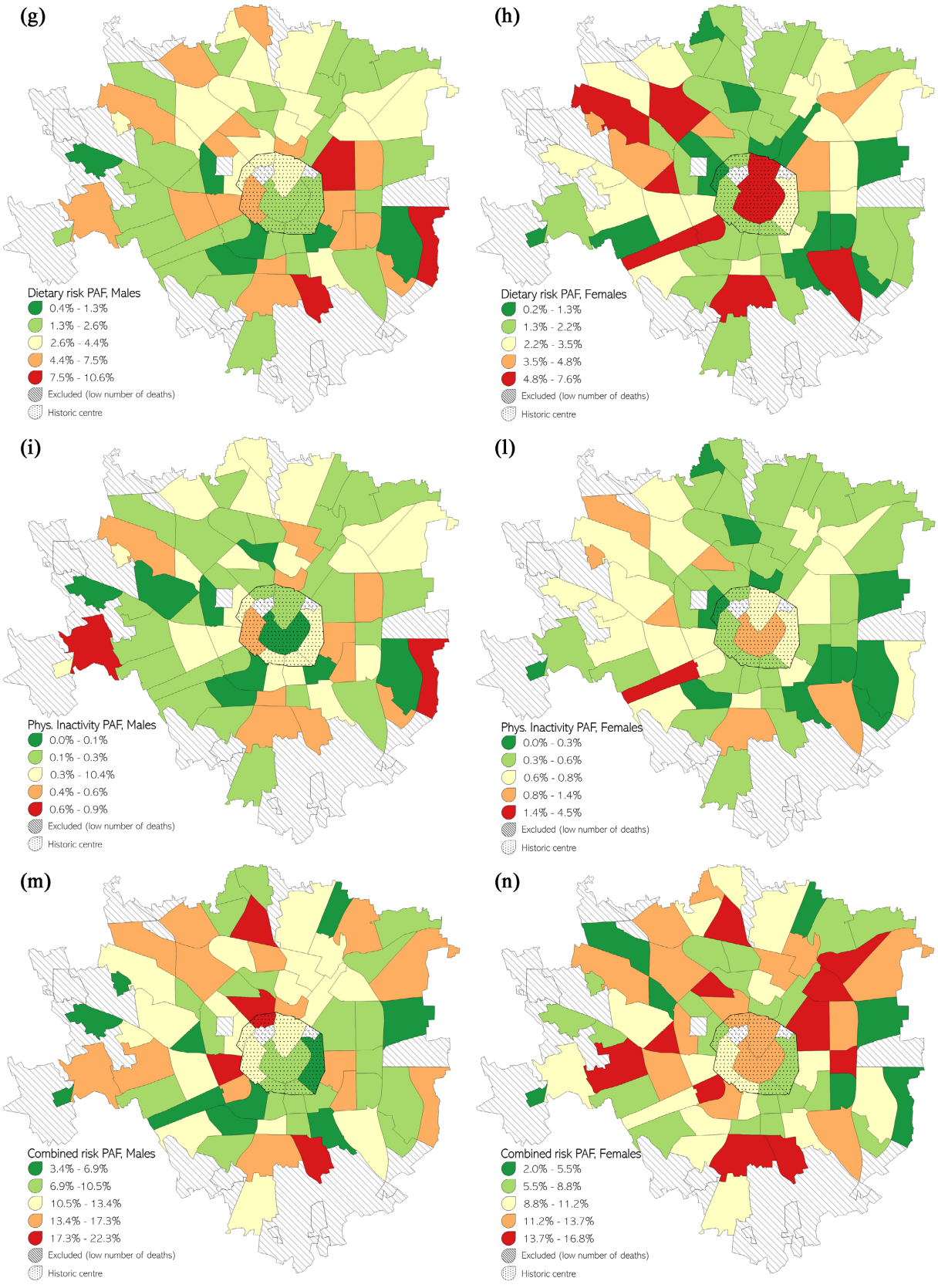
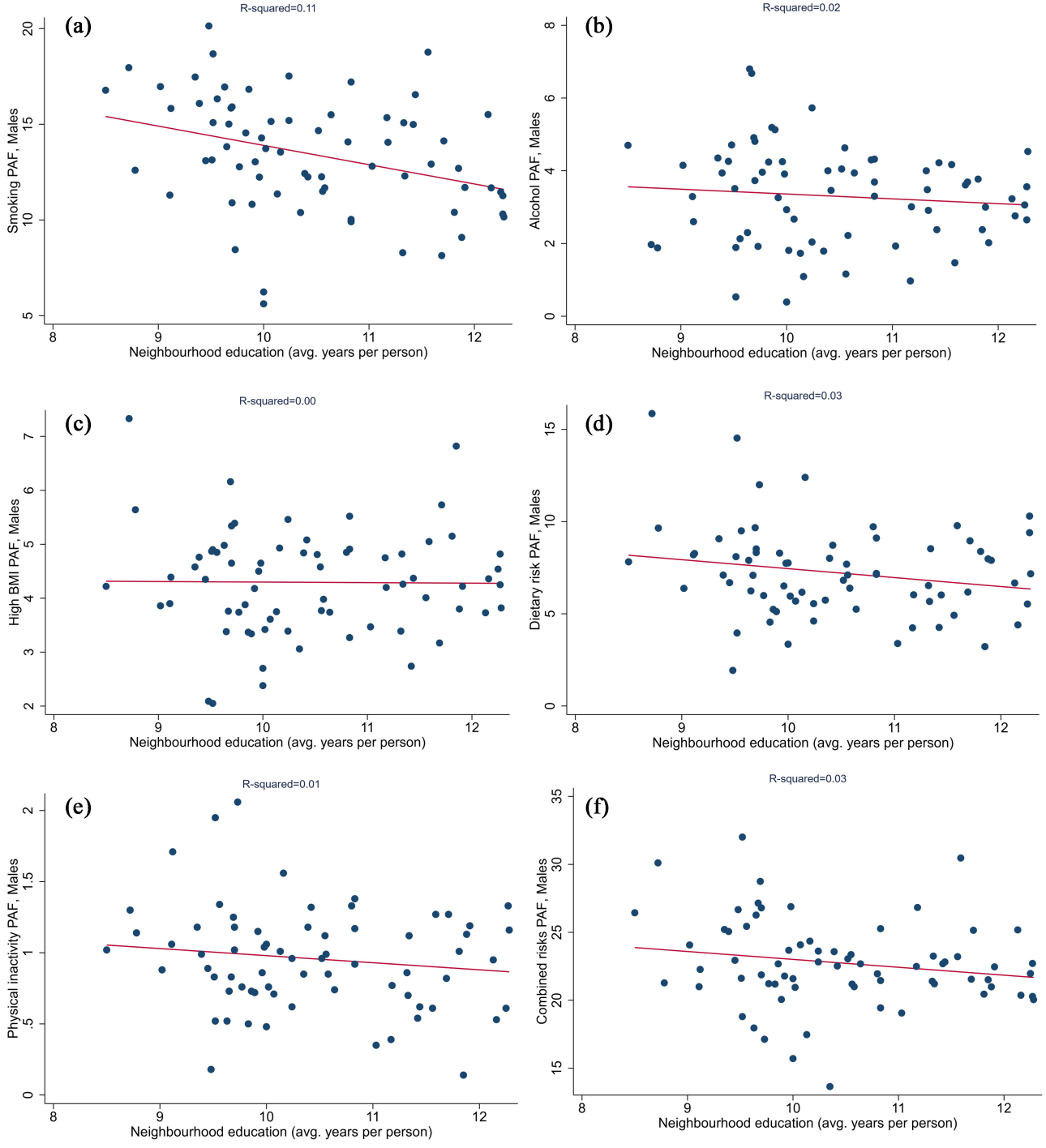


Figure A3. Males and Females' age-adjusted PAFs (five-year age classes) for each risk factor and for combined risk factors, by neighbourhood. Neighbourhoods with less than 10 deaths were excluded. Milan, 2019.


(a)
R-squared=0.11
Smoking PAF, Males
Neighbourhood education (avg. years per person)
(b)
R-squared=0.02
Alcohol PAF, Males
Neighbourhood education (avg. years per person)
(c)
R-squared=0.00
High BMI PAF, Males
Neighbourhood education (avg. years per person)
(d)
R-squared=0.03
Dietary risk PAF, Males
Neighbourhood education (avg. years per person)
(e)
R-squared=0.01
Physical inactivity PAF, Males
Neighbourhood education (avg. years per person)
(f)
R-squared=0.03
Combined risks PAF, Males
Neighbourhood education (avg. years per person)

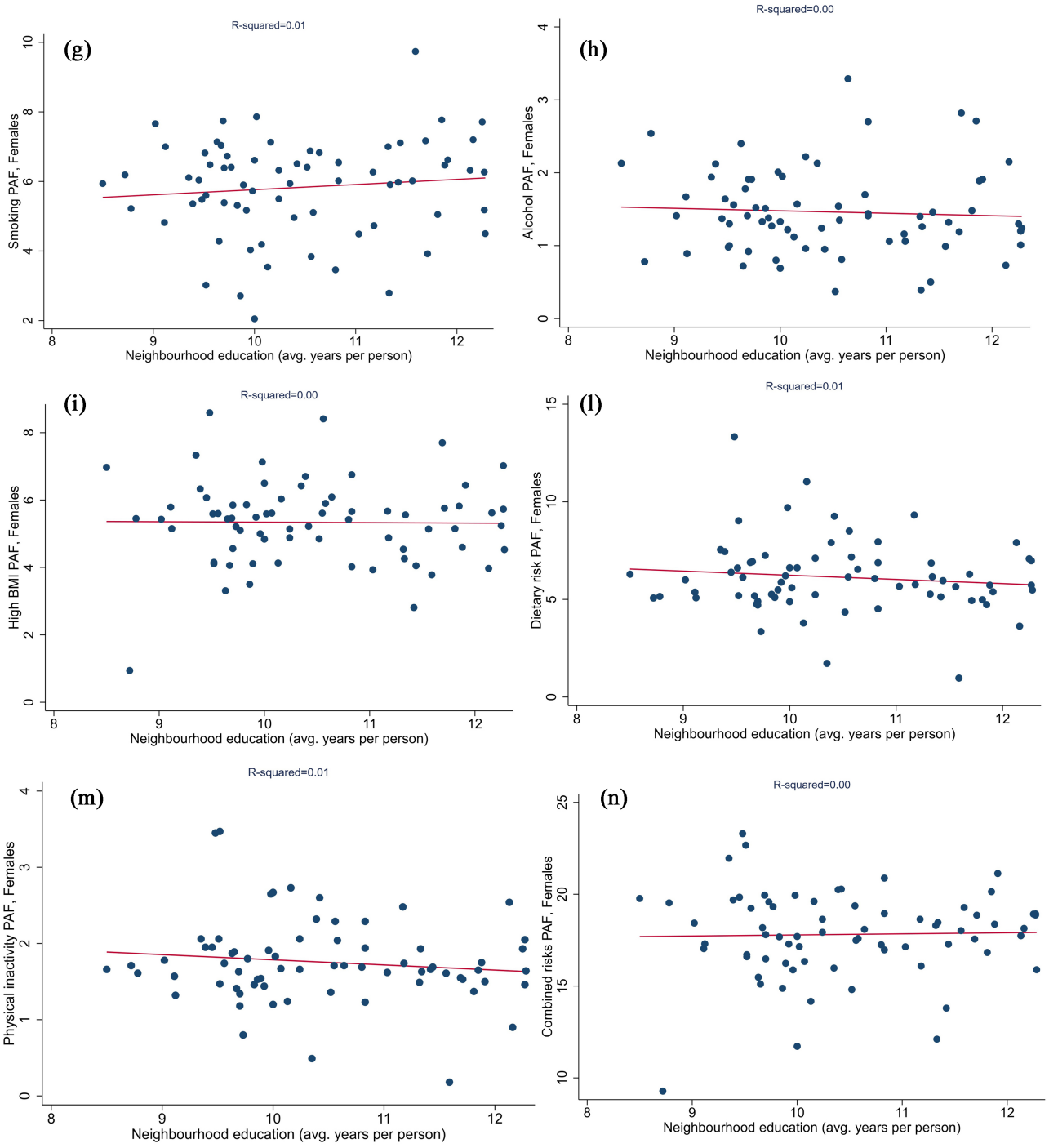


Figure A4. Bivariate correlations between neighbourhood-level education and PAFs in males and females.

*Note A*1.

The PAFs were calculated in the GBD study (GBD, 2015) using the following formula:

$$\mathrm{PAF}_{\mathrm{r}} = \frac{p_r\left(RR_r - 1\right)}{p_r\left(RR_r - 1\right) + 1}$$

where p_r is the prevalence of the risk factor r in the study population; RR_r is the

relative risk of death for the exposed to the risk factor r compared to the non-exposed.

*Note A*2*.*

The formula used to calculate the combined PAF is as follows:

$$\mathrm{PAF}_{\mathrm{combined}} = 1 - \prod_{r=1}^{5} \left(1 - \mathrm{PAF}_{\mathrm{r}}\right)$$

where PAF_r is the PAF relative to the risk factor r.