

Research Article

Age-Period-Cohort Analysis of Mesothelioma: Flat Incidence Trends for Males Entering US Workforce After 1972

Brent D. Kerger^{1*}, Nnaemeka U Odo², and Anne E Loccisano³¹Exponent Inc, Irvine CA, USA²Optum, Los Angeles, CA, USA³Exponent Inc, Alexandria, VA, USA

*Corresponding author

Brent D. Kerger, Exponent Inc, 15615 Alton Parkway, Suite 350, Irvine, CA 92618, USA

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Abstract

This study investigates the hypothesis that US government regulations on occupational asbestos exposures starting in 1972 caused a cohort inflection point after which mesothelioma incidence was flat for males subsequently entering the workforce. An age-period-cohort analysis of the SEER 8 cancer registries (1975-2019) was conducted using five-year intervals of age, calendar period, and birth cohort to evaluate US male incidence of all mesothelioma, as well as pleural, or peritoneal mesothelioma. Work cohort year was defined as birth cohort plus 18 years. Average percent change analysis was used to further evaluate period effects. Age- and period-adjusted work cohort rate ratios (RR) for all mesothelioma, and for pleural mesothelioma, were significantly elevated for work cohorts between 1920 and 1972 with subsequent decreased/flat trends. Similar age- and period-adjusted analysis of peritoneal mesothelioma showed non-significantly elevated work cohort RR between 1920 and 1954 with flat/not elevated incidence trends for work cohorts after 1954. Average percent change trend analyses showed statistically significant period effects on incidence rate trends for all mesothelioma and pleural mesothelioma, but not peritoneal mesothelioma; mesothelioma groups demonstrated significant longitudinal age trends. The cohort of US males first entering the workforce after 1972 shows no increased incidence of pleural mesothelioma. These trends may be related to the impacts of occupational asbestos regulations that took effect in 1972 in addition to plausible threshold dose-dependent risks from chrysotile asbestos which continued to be used through the 1990s. In contrast, US male workers first entering the workforce after the mid-1950s showed no increased risk of peritoneal mesothelioma which has been predominantly associated with amphibole asbestos exposures used in shipbuilding in the era of World War II. The flattening of work cohort mesothelioma rates after 1972 suggests the era of prominent risks from occupational asbestos exposure has ended; continuing incidence may be attributable to non-asbestos etiologies.

INTRODUCTION

The commonly considered etiology of malignant pleural mesothelioma is occupational asbestos exposure, although the disease can also arise due to exposures to erionite, non-commercial amphiboles, ionizing radiation, or aging, and from genetic predisposition or spontaneous occurrence [1,2]. In the past 3 decades, pleural mesothelioma incidence data from the SEER program revealed a peak male age-adjusted rate occurring in the early 1990s and a subsequent decline [3-5]. Age-period-cohort analysis revealed that this pleural mesothelioma trend involved the superposition of strong age-related and birth cohort-related trends that were most prominently impacted by large worker populations exposed to high dose amphiboles during shipbuilding in the World War II era [2].

In contrast to pleural mesothelioma, peritoneal mesothelioma is approximately ten-fold less prevalent among males than pleural mesothelioma, it is more specifically associated with high dose amphibole asbestos, and it often occurs among individuals without known asbestos exposures [3,6]. Peritoneal mesothelioma among US males has shown essentially flat incidence trends for the past 3 decades and occurs more frequently among females and at earlier onset for both sexes [5-7]. Peritoneal mesothelioma

risk is demonstrated to be a high cumulative dose phenomenon with excess risk following a threshold-dependent dose-response relationship and occurring predominantly among amphibole-exposed workers with asbestosis and very high lung fiber burdens typically over 100 f/cc*years [8-19]. The Occupational Safety and Health Act of 1970 was promulgated by US Congress in 1970 which formed the Occupational Safety and Health Administration and led to issuance of emergency temporary standards for occupational asbestos exposure in 1971 that became effective in 1972 (Title 29, Code of Federal Regulations). This study investigates the hypothesis that the US government regulations on occupational asbestos exposures starting in 1972 caused an inflection point after which mesothelioma incidence was flat for males subsequently entering the workforce. This hypothesis is investigated by conducting an age-period-cohort analysis of males within the SEER 8 cancer registries (1975-2019).

MATERIALS AND METHODS

The incidence data among males for all primary malignant mesothelioma (International Classification of Diseases for Oncology, 3rd edition, histology codes 9050-9055, all sites), and separately for only peritoneal mesothelioma (site codes C48.0 - C48.8), and pleural mesothelioma (site codes C38.4 and C38.8),

was obtained from the U.S. Surveillance, Epidemiology, and End Results (SEER) 8 population-based cancer registries from 1975 through 2019; certain comparisons also integrated the available data on SEER-12 and SEER-17, but only SEER-8 data provided the temporally congruous cancer incidence data required for this analysis between 1975 and 2019. Only male cancer incidence data were evaluated based on the predominantly male US workers in asbestos-exposed occupations historically. Data were extracted by 5-year age and calendar-year groups. Data were accessed using SEER*Stat software version 8.4.0.1 after execution of the SEER data use agreement, which includes compliance with ethical and privacy considerations and allows use of the cancer incidence data without separate requirements for study subject consent or Institutional Review Board approval.

The National Cancer Institute (NCI) web tool for age-period-cohort (APC) analysis was applied as described by Rosenberg et al., [20]. The NCI APC web tool enables analysis of net drift (annual percentage change in the expected age-adjusted rates over time), local drifts (annual percentage change in the expected age-specific rates over time), fitted temporal trends (expected rates over time in the reference age group, adjusted for cohort effects), cross-sectional age curve (expected age-specific rates in the reference calendar period, adjusted for cohort effects), longitudinal age curve (expected age-specific rates in the reference birth cohort, adjusted for period effects), period rate ratios (ratio of age-specific rates in each calendar period

relative to the reference period), and cohort rate ratios (ratio of age-specific rates in each birth cohort relative to the reference cohort). The NCI APC web tool also enables statistical testing of several null hypotheses related to the stability, log-linearity, and equality of observed trends. Default reference groups were used for comparisons, i.e., for the median calendar period and for the median birth cohorts. The work cohort was estimated by adding the age of typical full time work force entry among US males (age 18) to the birth cohort year, assuming that each individual first enters the workforce at 18 years of age.

DISCUSSION

Overall, the incidence of all mesothelioma and the predominant subset of pleural mesothelioma showed a work cohort peak in the 1940s and have declined since 1975 with average percent change estimates showing the most significant downward trend of all mesothelioma from 2000 to 2019 for SEER-17 data (Table 1). As expected, this trend is closely mirrored by the incidence trend of pleural mesothelioma, with a significant downward trend in the last two decades (-3.0; 95% CI: -3.6 to -2.4). A similar analysis of peritoneal mesothelioma incidence data (1975-2019) showed a comparatively nominal but statistically significant downward trend (-0.8; 95% CI: -1.5 to -0.1) for the temporally congruous data in SEER-8 (Table 1).

Table 2 presents the age-period-cohort analysis parameters segregated by all mesothelioma, pleural mesothelioma, and

Table 1: Summary of peak mesothelioma average percentage change trends.

| Cohort | Period | All mesothelioma | Pleural mesothelioma | Peritoneal mesothelioma |
|---------|-----------|------------------|----------------------|-------------------------|
| SEER-8 | 1975-2019 | -0.4 | -0.5 | -0.8* |
| SEER-12 | 1992-2019 | -2.1* | -2.3* | -0.8 |
| SEER-17 | 2000-2019 | -2.7* | -3.0* | -0.9 |

Percent changes were calculated using 1 year for each end point; average percent changes were calculated using weighted least squares method. * - Change is significantly different from zero ($p < 0.05$).

Table 2: Summary of age-period-cohort analysis for mesothelioma incidence in men across all, pleural and peritoneal mesothelioma cohorts.

| Age-Period-Cohort Analysis Parameters | All mesothelioma | Pleural mesothelioma | Peritoneal mesothelioma |
|--|----------------------|----------------------|-------------------------|
| Period effect: peak incidence | | | |
| Incidence years | 1979 - 1983 | 1979 - 1983 | 1979 - 1983 |
| Net drift % per year (95% CI) | -1.5% (-2.7 to -0.4) | -1.8% (-3.2 to -0.4) | -0.4% (-2.0 to 1.2) |
| p-value | 0.009 | 0.0108 | 0.6085 |
| Period RRs different from 1994 to 1998? | Yes | Yes | No |
| p-value | <0.0001 | <0.0001 | 0.5487 |
| Period deviation is non-linear? | Yes | Yes | No |
| p-value | 0.0001 | 0.0002 | 0.4575 |
| Cohort effect: peak incidence | | | |
| Birth cohort years | 1922 - 1927 | 1922 - 1927 | 1901 - 1906* |
| Local drifts equal net drift for all age groups? | Yes | Yes | No |
| p-value | <0.0001 | <0.0001 | 0.2076 |
| Cohort RRs different from referent? | Yes | Yes | Yes |

| | | | |
|---|----------------------|----------------------|--------------------|
| p-value | <0.0001 | <0.0001 | 0.0216 |
| Non-linear cohort deviation? | Yes | Yes | No |
| p-value | <0.0001 | <0.0001 | 0.1711 |
| Cross-sectional age trend (95% CI) | 11.3% (10.1 to 12.5) | 11.8% (10.5 to 13.0) | 7.4% (6.2 to 8.7) |
| Longitudinal age trend (95% CI) | 9.8% (8.2 to 11.3) | 9.9% (8.1% to 11.8) | 7.0% (5.0% to 9.1) |
| Longitudinal vs. cross-sectional RR trend | Negative | Negative | Negative/flat |
| Non-linear age deviation? | Yes | Yes | Yes |
| p-value | <0.0001 | <0.0001 | <0.0001 |

*Another non-statistically significant birth cohort incidence peak was noted between 1992 - 1997

peritoneal mesothelioma incidence data sets in SEER-8. Period effects showed their peak for all groups between 1979 and 1983. However, only incidence rates for all mesothelioma and pleural mesothelioma showed significant downward net drift (annual percentage change in expected age-adjusted rates over time), and in period rate ratios (the significant ratio of age-specific rates in each calendar period relative to the reference period [1994-1998]). All mesothelioma groups demonstrated significantly different cohort incidence rate ratios from the median referent group, with significant non-linear age deviation of incidence trends (Table 2).

Age-period-cohort analysis findings (which utilized SEER-12 data) for all mesothelioma in Figures 1 and 2 with error bars indicating 95% confidence intervals. The age-related rate of all mesothelioma by cross-sectional age adjusted for birth cohort and calendar year showed a statistically significant exponential fit between ages 40 and 80 (Figure 1). The work cohort rate ratio of all mesothelioma adjusted for age and calendar year showed significantly elevated values for US males entering the workforce between 1920 and 1970, and then became flat and not significantly elevated for six subsequent 5-year nodes of observation (Figure 2).

For the predominant subset of pleural mesothelioma, age-period-cohort analysis findings are similar to those for all mesothelioma as illustrated in Figures 3 and 4. The age-related rate of pleural mesothelioma by cross-sectional age adjusted for birth cohort and calendar year showed a statistically significant exponential fit between ages 40 and 80 (Figure 3). The work cohort rate ratio of pleural mesothelioma adjusted for age and calendar year showed significantly elevated values for US males entering the workforce between 1920 and 1970, and then became flat and not significantly elevated for six subsequent 5-year nodes of observation (Figure 4).

Peritoneal mesothelioma findings based on age-period-cohort analysis are distinctly different from those for pleural mesothelioma as illustrated in Figures 5 and 6. In contrast to the more congruent age-related rate trends for all mesothelioma and pleural mesothelioma, the age-related rate of peritoneal mesothelioma by cross-sectional age adjusted for birth cohort and calendar year appears to show different rates for males between age 40 and 55 when compared to rates observed between ages 55 and 80 (Figure 5). Similarly in contrast to findings for all mesothelioma and pleural mesothelioma, the work cohort rate ratio of peritoneal mesothelioma adjusted for age and calendar year showed inconsistently elevated values for

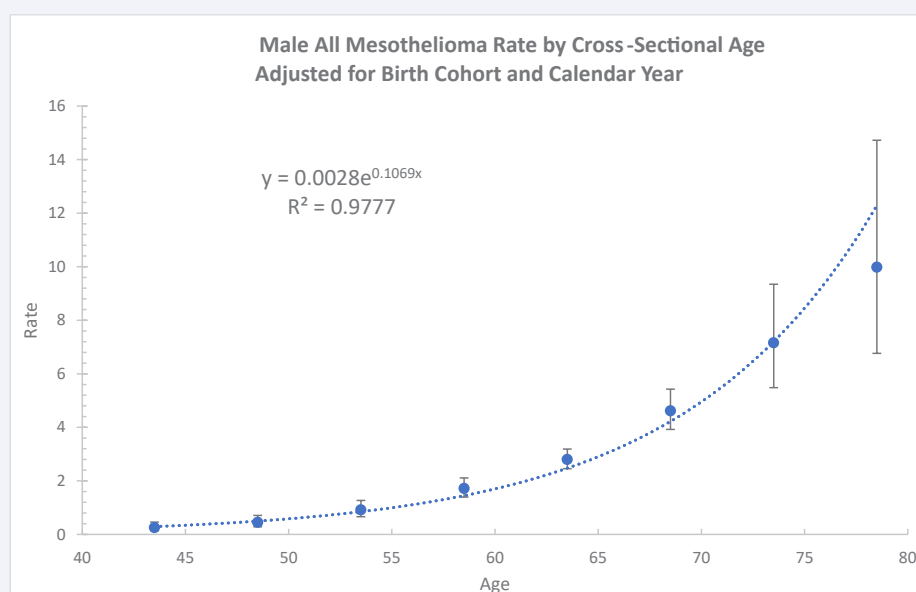


Figure 1

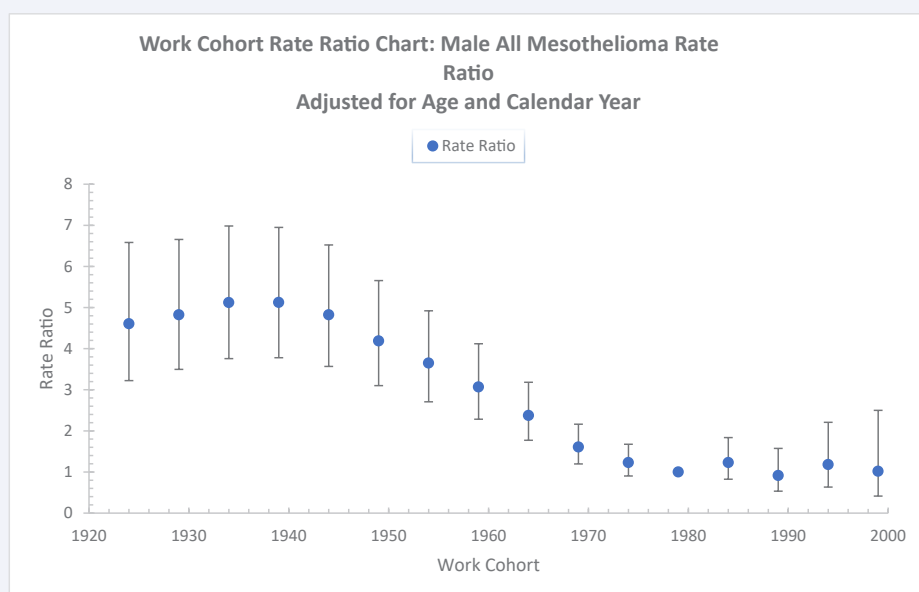


Figure 2

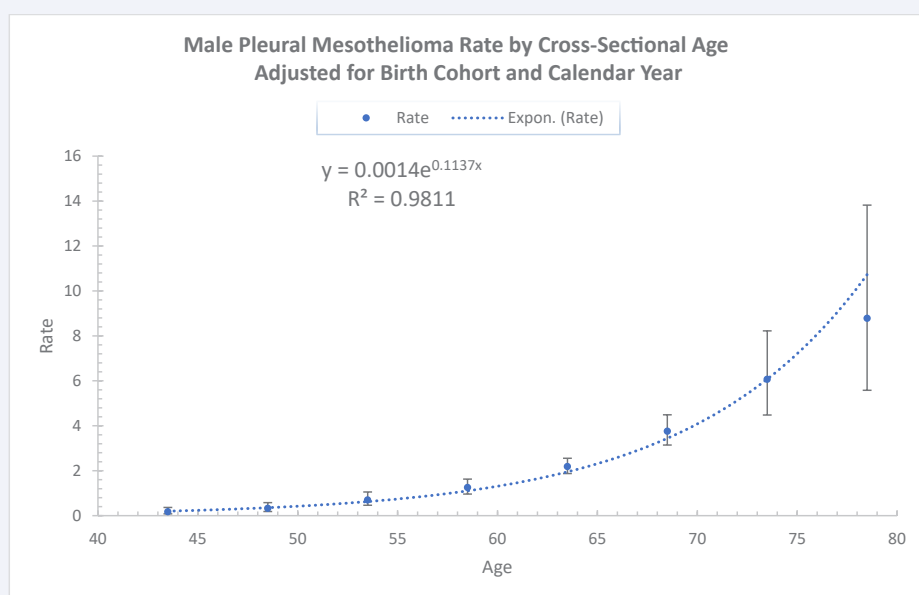


Figure 3

US males entering the workforce between 1920 and 1950, with apparently flat and not significantly elevated rate ratios for the ten subsequent 5-year nodes of observation for work cohorts after 1950 (Figure 6).

FINDINGS

The current study identified distinct age-, calendar period, and cohort-related effects on all mesothelioma, pleural, and peritoneal mesothelioma incidence trends among US males that are consistent with a prominent influence of high occupational exposures to amosite during the era of World War II. The findings demonstrated different trend patterns between peritoneal

mesothelioma incidence and the other two mesothelioma groups, including earlier flattening of peritoneal mesothelioma incidence rates starting in the mid-1950s. This earlier flattening of peritoneal mesothelioma rates may be attributable to non-asbestos etiology of peritoneal mesothelioma, particularly among work cohorts after 1954. Although mesothelioma incidence rates peaked between 1979-1983 for all three groups, only all mesothelioma and pleural mesothelioma showed robustly significant downward trends over the time period of the study (1975-2019). The flattening of all mesothelioma and pleural mesothelioma rates for males entering the US workforce after 1972 suggests the era of prominent risks from occupational

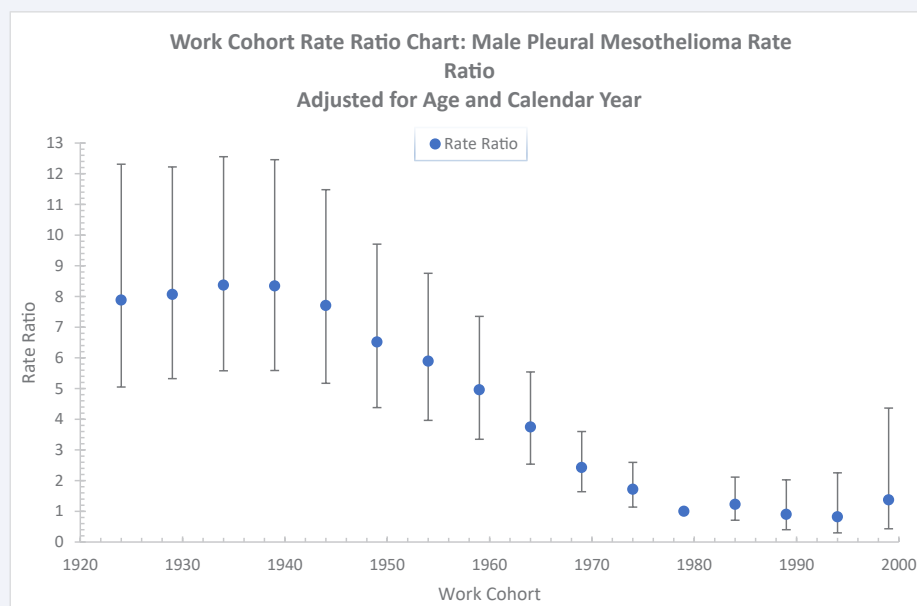


Figure 4

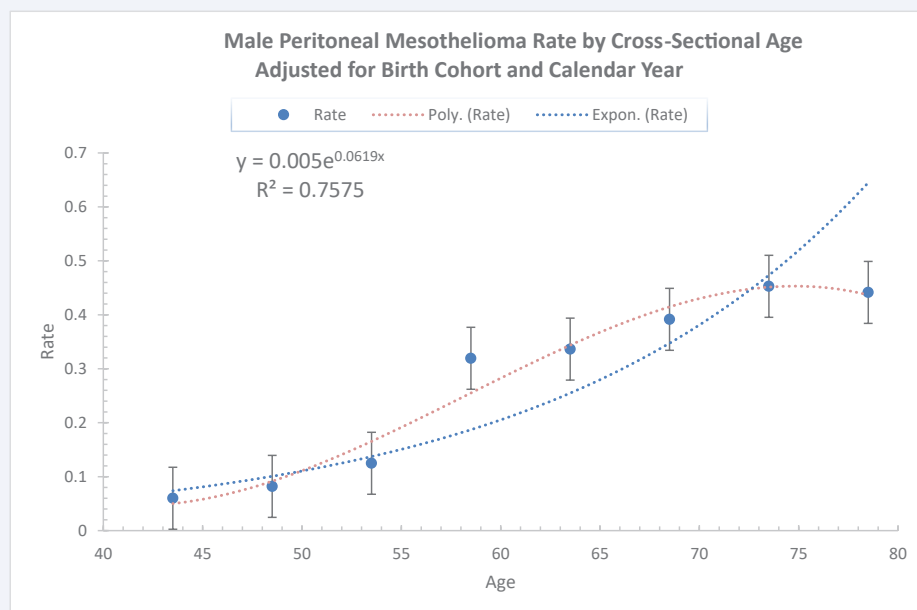


Figure 5

asbestos exposure has ended and that the continuing incidence may be attributable to non-asbestos etiologies.

Projections of occupational asbestos-related pleural mesothelioma (and background/non-asbestos cases) have been driven by assumptions of relatively high asbestos exposures to the large US workforce involved in shipbuilding and shipyard repairs in the World War II era [21-23]. A focus on this era is also supported by the more prominent use of amosite for ship insulation [24,4,25], because this amphibole form is a highly potent cause of mesothelioma relative to the chrysotile form that continued to be used in the US until the 1990s [26-28]. The timing

of a birth cohort's first entry into the workforce at age 18 can be simulated by adding 18 years to the birth cohort range. For example, adding 18 years to the male birth cohort range for peak PM incidence (1926-1932 for both age groups) leads to a calendar period (1944-1950) plausibly circumscribing the period of more intense post-World War II shipyard production and ship repair activities. Increasing recognition and control of asbestos hazards and associated declining amosite exposures [24,25], plausibly correspond with reduced pleural mesothelioma incidence among persons with first occupational exposure after the 1950s in US shipyards.

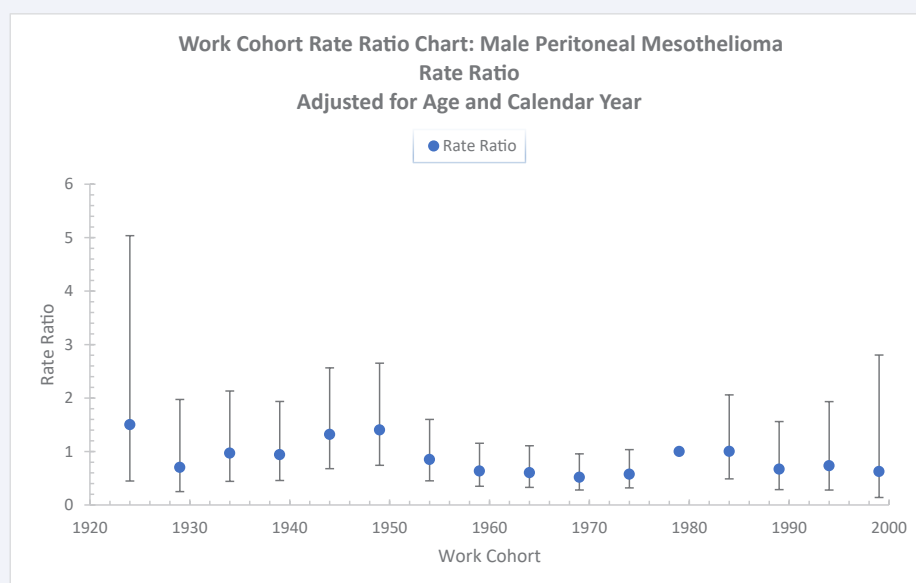


Figure 6

Nuances in the period and birth cohort trends in pleural mesothelioma incidence may also provide useful insights, particularly among males given their more robust data. First, a relatively monotonic decline in pleural mesothelioma incidence rate by calendar period (adjusted for birth cohort effects) is observed in age 0-74 males following the peak in 1978-1982 calendar years and a relatively monotonic decline is observed in this group following the birth cohort peak in 1928-1932 [2]. Adding 18 years to this birth cohort corresponds to an early workforce exposure period of 1946-1950 and an associated latency to peak pleural mesothelioma cohort rate ratio (in 1978-1982) of perhaps 28 to 36 years [2]. This latency range is consistent with other estimates for pleural mesothelioma caused by high occupational exposures to amphibole asbestos among mining and milling workers, shipyard workers, insulators, and pipefitters [8,29-31].

European researchers have reported shifting trends in PM incidence or mortality in the last two decades in Italy [32,33], France [34,35], Belgium [36], Sweden [37], the Netherlands [38], and the United Kingdom [39]. Studies examining age-specific trends are discussed further below. The extent to which these shifting PM trends are due to region-specific variation in occupational asbestos exposures and/or longevity-related factors is currently unknown. The attrition of pleural mesothelioma risk among younger age groups and the shift towards higher age-adjusted risks in older age groups as shown by Kerger [2], is apparent in studies from several European countries. Tan et al. [39] used Bayesian methods to predict peak (total) mesothelioma mortality occurring in 2016 in the UK, with a rapid decline thereafter. Birth cohorts after 1965 showed consistently low numbers of actual and projected mesothelioma deaths, and a steady linear rate of decline was noted for birth cohorts between 1955 and 1965 [39]. Jarvholm and Burdorf [37] reported that age-adjusted pleural mesothelioma mortality trends for all age groups in Sweden tended to mask a strong influence of age among

long-surviving individuals from more exposed birth cohorts (e.g., 1935 to 1949) that was offset by the rapidly decreasing rates among younger birth cohorts. Segura et al. [38] reported that an age-cohort model among men in the Netherlands showed the highest age-specific death rates among the oldest age group (age 75 to 84) and the highest relative risks for birth cohorts of 1938 to 1947. Segura et al. [38] noted that the strong increase in male pleural mesothelioma incidence between 1969 and 1998 may be affected by increasing diagnostic awareness of mesothelioma since the late 1970s.

Like previous analyses which showed that age-adjusted peritoneal mesothelioma rates for men exhibited no temporal trends from 1973 to 2005 [3], this study did not show any temporal trends using SEER-8 data from 1975 to 2019. Other authors recently reported analyses results limited to SEER-18 (2000 – 2018) data and reported that although specific histology showed significant incidence trends, the age-adjusted incidence of peritoneal mesothelioma was similarly stable over the period [40]. Multiple asbestos cohorts have also been investigated for peritoneal mesothelioma outcomes trends with demonstrated non-elevated, flat, or declining rates in populations in Norway [41], Sweden [42], Denmark [43], and mortality risk in Italy [44].

CONCLUSIONS, LIMITATIONS & RECOMMENDATIONS

The current study identifies distinct age- and cohort-related effects on all mesothelioma, pleural, and peritoneal mesothelioma incidence trends among US males that are consistent with a prominent influence of high occupational exposures to amosite during the era of World War II. These findings suggest that the cohort of US males first entering the workforce after 1972 show no increased incidence of mesothelioma (all, pleural, or peritoneal). This may be related to the impacts of occupational asbestos regulations starting in 1972 in addition to plausible threshold-dependent risks from chrysotile which continued to be used through the 1990s. In contrast, US males first entering the

workforce after 1954 showed an earlier flattening of peritoneal mesothelioma rates compared to pleural or all mesothelioma. The flattening of work cohort mesothelioma rates after 1972 suggests the era of prominent risks from occupational asbestos exposure has ended; continuing incidence may be attributable to non-asbestos etiologies.

This age-period-cohort analysis is limited by use of the less robust SEER-8 database, by the relative low incidence of mesothelioma generally, and more specifically, by the ten-fold lower incidence of peritoneal mesothelioma. However, the findings reported for all mesothelioma and pleural mesothelioma are reasonably robust for SEER-8 and attempts to include additional SEER registry data (e.g., SEER-12 or the currently available SEER-17) could introduce positive or negative bias that would be difficult to interpret. In addition, the SEER database does not include information on occupational exposures to asbestos or other carcinogens, making the interpretation of mesothelioma trends indirect and dependent on other information and data sources.

The authors recommend continued surveillance of the SEER database trends for mesothelioma to assess whether the trends observed in this study continue with the expanding robustness of observations for work cohorts after 1972. It is also recommended that non-asbestos sources be considered more diligently for continuing incidence of mesothelioma in the United States.

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Ethics Approval and Consent to Participate

Access to the SEER-12 database was obtained after execution of the SEER data use agreement at: <https://seer.cancer.gov/dataagreements/seer.pdf>. The data analyses and use of the SEER-12 database in this manuscript is in accordance with the data use agreement and does not require Institutional Review Board approval or other ethics approval or consent of the study subjects.

Consent for Publication

The cancer incidence data contained in this manuscript was analyzed and used in accordance with the SEER data use agreement and therefore does not require separate consent for publication.

Availability of Data and Materials

The SEER-12 cancer incidence data analyzed in this manuscript is publicly available but is subject to a signed data

use agreement which forbids sharing the underlying data files. The NCI webtool for Age-Period-Cohort analysis is also a publicly available program which other researchers may access and apply to the SEER-12 data (within the SEER data use agreement guidelines) to further analyze and/or validate the analyses presented here.

Author's Contributions

Two of the authors (BDK and AEL) designed the study, analyzed the scientific literature, interpreted the analytical findings of the age-period-cohort analysis performed by the epidemiologist author (NO), and drafted the manuscript. All three authors provided further revisions to complete the final manuscript and approved it for submission.

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