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Original Research Article

Histomorphometric Analysis of Elastic Fibre Remodelling in the Human Trachea and Main Bronchi During Ageing

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Abstract

Background: Elastic fibres are essential for maintaining the structural integrity, elasticity, and recoil of the trachea and main bronchi. Although age-related degeneration of elastin has been described in several connective tissues, quantitative information regarding age-associated changes in the central conducting airways remains limited. This study aimed to evaluate histomorphometric alterations in elastic fibres of the human trachea and main bronchi across different age groups.

Methods: A descriptive cross-sectional histomorphometric study was conducted on tissues obtained from 63 human cadavers. Tracheal and main bronchial specimens were processed using routine histological techniques and stained with Verhoeff–Van Gieson stain for elastic fibre visualization. Elastic fibre density, fibre thickness, fragmentation index, and degeneration score were quantified using digital image analysis and compared across six age groups. Regional differences between the trachea and bronchi were evaluated, while Pearson's correlation was used to determine the association between age and morphometric parameters.

Results: The mean age of the study population was 49.8 ± 17.2 years, with males constituting 66.7% of the specimens. Elastic fibre density declined significantly from 33.8 ± 2.5% in the 20–29-year group to 20.9 ± 2.0% in individuals aged ≥70 years ($F = 58.72$, $p<0.001$), while fibre thickness decreased from 2.81 ± 0.19 µm to 1.71 ± 0.14 µm ($F = 67.14$, $p<0.001$). Conversely, fragmentation index and degeneration score increased significantly with age (both $p<0.001$). Qualitative histological examination demonstrated progressive fragmentation, fibre waviness, loss of parallel orientation, and elastic fibre clumping in older age groups. No significant differences were observed between the trachea and main bronchi or between the right and left main bronchi (all $p>0.05$). Age showed strong negative correlations with elastic fibre density ($r = −0.857$) and fibre thickness ($r = −0.881$), and strong positive correlations with fragmentation index ($r = 0.912$) and degeneration score ($r = 0.894$) (all $p<0.001$).

Conclusion: Advancing age is associated with significant deterioration of elastic fibres within the human trachea and main bronchi, characterized by reduced fibre density and thickness together with increased fragmentation and structural degeneration. These findings provide quantitative baseline evidence of normal tracheobronchial ageing and improve understanding of extracellular matrix remodelling in the central airways, thereby serving as a valuable reference for future anatomical and respiratory research.

Keywords: Ageing; Elastic fibres; Trachea; Main bronchi; Verhoeff–Van Gieson stain.

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Introduction

The trachea and main bronchi form the proximal conducting airways of the respiratory system and play a crucial role in maintaining airway patency and facilitating efficient airflow between the upper respiratory tract and lungs [1]. Their structural integrity depends on a well-organized extracellular matrix comprising hyaline cartilage, smooth muscle, collagen, and elastic fibres [1]. Among these components, elastic fibres are essential for providing elasticity, resilience, and recoil, enabling the airways to withstand repetitive cycles of inspiration and expiration while preserving their shape and mechanical stability [1,2]. Elastic fibres are composed of an elastin core surrounded by

fibrillin-rich microfibrils and are among the most durable proteins in the human body [3]. However, their synthesis declines markedly after early adulthood, making them particularly susceptible to cumulative age-related damage [4]. Progressive fragmentation, loss of elasticity, oxidative stress, non-enzymatic glycation, and increased proteolytic activity contribute to the gradual deterioration of elastic fibres with advancing age [3,4]. These structural alterations reduce tissue compliance and impair the biomechanical properties of the airways, potentially contributing to age-associated decline in respiratory function [4].

Globally, population ageing is increasing rapidly, with individuals aged 60 years and above projected to account for nearly one-quarter of the world's population by 2050 [5]. Ageing is associated with physiological changes in the respiratory system, including reduced pulmonary elastic recoil, increased airway stiffness, diminished expiratory flow rates, and decreased ventilatory reserve, even in the absence of overt pulmonary disease [6]. While considerable research has focused on age-related changes in the lung parenchyma, comparatively few studies have investigated the histological alterations occurring within the central conducting airways, particularly the trachea and main bronchi [6].

Degeneration of elastic fibres in the tracheobronchial tree may compromise airway flexibility, reduce the efficiency of cough and mucociliary clearance, and increase susceptibility to respiratory complications in older adults [7]. Quantitative histomorphometric evaluation of elastic fibres can provide objective evidence of these structural changes and establish baseline age-related reference data for normal airway ageing [8]. However, literature describing age-dependent alterations in elastic fibre architecture of the human trachea and main bronchi remains limited [8]. Therefore, the present study was aimed to quantitatively assess age-related deterioration of elastic fibres in the human trachea and main bronchi, thereby enhancing the understanding of structural airway ageing and its potential implications for respiratory function.

Materials and Methods

Study Design and Setting: This descriptive cross-sectional histomorphometric study was conducted in the Department of Anatomy over a period of 24 months between June 2023 to May 2025 after obtaining approval from the Institutional Ethics Committee.

The study aimed to evaluate age-related changes in the elastic fibres of the human trachea and main bronchi using qualitative histological assessment and quantitative morphometric analysis. All procedures involving human tissues were performed in accordance with the ethical principles outlined in the Declaration of Helsinki and institutional guidelines for research involving cadaveric and autopsy specimens.

Study Specimens: The study included trachea and main bronchial specimens obtained from 63 human cadavers (126 tissue samples comprising the trachea, right main bronchus, and left main bronchus where applicable) during routine medicolegal autopsies and anatomical dissections. Cadavers with a documented history of chronic respiratory diseases such as chronic obstructive pulmonary disease, bronchial asthma, pulmonary fibrosis, bronchiectasis, tuberculosis, tracheal stenosis, or primary or metastatic malignancies involving the respiratory tract were excluded. Specimens showing gross evidence of trauma, decomposition, burns, congenital anomalies, previous airway surgery, or significant post-mortem tissue autolysis were also excluded to minimize factors that could alter the normal histological architecture.

Based on chronological age, specimens were categorized into six age groups: Group I (20–29 years), Group II (30–39 years), Group III (40–49 years), Group IV (50–59 years), Group V (60–69 years), and Group VI (≥70 years), with ten specimens included in each group. Age was confirmed using available medicolegal records or institutional documentation.

Tissue Collection and Histological Processing: Immediately following procurement, tissue segments measuring approximately 1.5–2.0 cm were excised from the mid-trachea, right main bronchus, and left main bronchus to ensure uniformity of sampling. The specimens were gently rinsed with normal saline to remove blood and debris before fixation in 10% neutral buffered formalin for 24–48 hours. Following fixation, tissues underwent routine dehydration in ascending grades of ethanol, clearing in xylene, and paraffin embedding. Serial sections of 4–5 µm thickness were prepared using a rotary microtome and mounted on poly-L-lysine-coated glass slides. Representative sections were stained with Verhoeff–Van Gieson (VVG) stain for specific visualization of elastic fibres, while adjacent sections were stained with hematoxylin and eosin (H&E) to evaluate the general histological architecture and exclude underlying pathological changes.

Histomorphometric Evaluation: Microscopic examination was performed using a research-grade light microscope equipped with a high-resolution digital camera. Histological images were captured under standardized illumination at ×100 and ×400 magnifications. Quantitative morphometric analysis was performed using ImageJ software (National

Institutes of Health, Bethesda, USA) after calibration with a stage micrometer. For each specimen, five non-overlapping randomly selected high-power fields from the submucosal and perichondrial regions were analysed, and the average value was considered for statistical analysis.

The primary morphometric parameters included elastic fibre density (percentage area occupied by elastic fibres), mean elastic fibre thickness (µm), elastic fibre fragmentation index (number of discontinuities per high-power field), and orientation of elastic fibres. Fibre density was calculated by threshold-based segmentation of VVG-stained sections, while fibre thickness was determined from calibrated linear measurements. Fragmentation was assessed by counting interruptions in continuous elastic fibres within each microscopic field, and fibre orientation was evaluated qualitatively as parallel, irregular, or disorganized with respect to the longitudinal axis of the airway.

Outcome Measures: The primary outcome measure was the quantitative assessment of age-related changes in elastic fibre morphology within the trachea and main bronchi. Secondary outcomes included comparison of elastic fibre density, thickness, and fragmentation among different age groups and between the trachea and right and left main bronchi. Histological evidence of degeneration, including fibre thinning, fragmentation, waviness, clumping, and loss of normal architectural arrangement, was also documented.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using the Shapiro–Wilk test.Differences in histomorphometric parameters (elastic fibre density, fibre thickness, fragmentation index, and degeneration score) among the six age groups were analysed using one-way analysis of variance (ANOVA). Comparisons of morphometric parameters between the trachea and main bronchi and between the right and left main bronchi were performed using Student's paired *t*-test. Qualitative histological features across age groups were compared using the Chi-square (χ^2) test. Pearson's correlation coefficient (r) was calculated to determine the strength and direction of the association between chronological age and quantitative histomorphometric parameters. All statistical tests were two-tailed, and a p value of <0.05 was considered statistically significant.

Results

A total of 63 human cadavers were included in the study. The age of the study population ranged from 20 years to over 70 years, with a mean age of 49.8 ± 17.2 years. The age distribution was nearly uniform across the six predefined age groups, with 10–11 specimens in each category. Male cadavers constituted two-thirds of the study population (42, 66.7%), while female cadavers accounted for 21 (33.3%), resulting in a male-to-female ratio of 2:1 (Table 1).

Table 1: Demographic characteristics of the study population included for histomorphometric evaluation of elastic fibres in the trachea and main bronchi (n = 63)

Variable	Frequency (%)/Mean ± SD
Age group (years)	
20–29	10 (15.9)
30–39	11 (17.5)
40–49	11 (17.5)
50–59	10 (15.9)
60–69	11 (17.5)
≥70	10 (15.9)
Mean age (years)	49.8 ± 17.2
Gender	
Male	42 (66.7)
Female	21 (33.3)

Histomorphometric analysis demonstrated a progressive age-related deterioration of elastic fibres. Mean elastic fibre density declined significantly from 33.8 ± 2.5% in the 20–29-year age group to 20.9 ± 2.0% among specimens aged ≥70 years ($F = 58.72$, $p <0.001$). Similarly, mean fibre thickness decreased steadily from 2.81 ± 0.19 µm to 1.71 ± 0.14 µm with advancing age ($F = 67.14$, $p <0.001$). Conversely, the fragmentation index increased significantly from 1.18 ± 0.39 to 5.36 ± 0.76 discontinuities per high-power field ($F = 89.36$, $p <0.001$), accompanied by a marked rise in the degeneration score from 0.42 ± 0.29 to 2.78 ± 0.32 ($F = 81.91$, $p <0.001$). These findings indicate progressive structural degeneration of elastic fibres with increasing age (Table 2).

Table 2: Comparison of histomorphometric parameters of elastic fibres across different age groups in the human trachea and main bronchi

Age group	Elastic fibre density (%)	Fibre thickness (µm)	Fragmentation index (No./HPF)	Degeneration score (0–3)
	Mean ± SD			
20–29 (n=10)	33.8 ± 2.5	2.81 ± 0.19	1.18 ± 0.39	0.42 ± 0.29
30–39 (n=11)	31.7 ± 2.3	2.63 ± 0.20	1.73 ± 0.46	0.71 ± 0.36
40–49 (n=11)	29.2 ± 2.4	2.39 ± 0.17	2.46 ± 0.53	1.18 ± 0.41
50–59 (n=10)	26.6 ± 2.2	2.15 ± 0.18	3.27 ± 0.64	1.72 ± 0.47
60–69 (n=11)	23.8 ± 2.1	1.91 ± 0.15	4.31 ± 0.71	2.19 ± 0.43
≥70 (n=10)	20.9 ± 2.0	1.71 ± 0.14	5.36 ± 0.76	2.78 ± 0.32
One-way ANOVA test	F = 58.72, p <0.001	F = 67.14, p <0.001	F = 89.36, p <0.001	F = 81.91, p <0.001

Elastic fibre density represents the percentage area occupied by elastic fibres. Fragmentation index denotes the average number of fibre discontinuities per high-power field (HPF). Degeneration score was graded on a semi-quantitative scale from 0 (no degeneration) to 3 (severe degeneration).

Comparison of morphometric parameters between the trachea and main bronchi revealed no statistically significant differences in elastic fibre density (27.95 ± 4.76% vs. 27.28 ± 4.58%; p = 0.414), fibre thickness (2.27 ± 0.41 µm vs. 2.22 ± 0.39 µm; p = 0.479), fragmentation index (3.08 ± 1.53 vs. 3.19 ± 1.47; p = 0.668), or degeneration score (1.52 ± 0.82 vs. 1.60 ± 0.79; p = 0.562).

Likewise, no significant differences were observed between the right and left main bronchi for any morphometric parameter, including elastic fibre density (p = 0.718), fibre thickness (p = 0.789), fragmentation index (p = 0.936), and degeneration score (p = 0.886), indicating comparable histological characteristics across the central conducting airways (Table 3).

Table 3: Comparison of histomorphometric parameters between the trachea and main bronchi and between the right and left main bronchi

Parameters	Elastic fibre density (%)	Fibre thickness (µm)	Fragmentation index (No./HPF)	Degeneration score (0–3)
	Mean ± SD			
Trachea (n=63)	27.95 ± 4.76	2.27 ± 0.41	3.08 ± 1.53	1.52 ± 0.82
Main bronchi (n=63)	27.28 ± 4.58	2.22 ± 0.39	3.19 ± 1.47	1.60 ± 0.79
Student's paired t-test*	0.82	0.71	-0.43	-0.58
p value	0.414	0.479	0.668	0.562
Right bronchus (n=63)	27.42 ± 4.60	2.21 ± 0.40	3.18 ± 1.46	1.61 ± 0.78
Left bronchus (n=63)	27.13 ± 4.54	2.23 ± 0.38	3.20 ± 1.49	1.59 ± 0.80
Student's paired t-test*	0.36	-0.27	-0.08	0.14
p value	0.718	0.789	0.936	0.886

Fragmentation index represents the number of fibre discontinuities per high-power field (HPF). Degeneration score ranges from 0 (no degeneration) to 3 (severe degeneration).

Qualitative histological evaluation demonstrated a significant age-dependent increase in degenerative changes of elastic fibres. Normal continuous elastic fibres were observed predominantly in younger specimens aged 20–39 years (85.7%) but declined markedly in the 40–59-year (38.1%) and ≥60-year (9.5%) groups (χ^2 = 28.47, p <0.001). In contrast, moderate and severe fragmentation were almost exclusively observed in older individuals, with severe fragmentation present only in the ≥60-year group (28.6%).

Additional degenerative features, including marked fibre waviness, loss of parallel fibre orientation, and elastic fibre clumping, increased progressively with age and were significantly more frequent among specimens aged ≥60 years (all p <0.001), indicating progressive disruption of the normal elastic fibre architecture (Table 4).

Table 4: Distribution of qualitative histological changes in elastic fibres according to age group

Histological feature	20–39 yrs (n=21)	40–59 yrs (n=21)	≥60 yrs (n=21)	Chi-square (χ^2) test	p value
	Frequency (%)				
Normal continuous fibres (n=28)	18 (85.7)	8 (38.1)	2 (9.5)	28.47	<0.001
Mild fragmentation (n=18)	3 (14.3)	10 (47.6)	5 (23.8)		
Moderate fragmentation (n=11)	0 (0.0)	3 (14.3)	8 (38.1)		
Severe fragmentation (n=6)	0 (0.0)	0 (0.0)	6 (28.6)		
Marked waviness (n=25)	1 (4.8)	8 (38.1)	16 (76.2)	24.96	<0.001
Loss of parallel orientation (n=19)	0 (0.0)	5 (23.8)	14 (66.7)	27.51	<0.001
Elastic fibre clumping (n=14)	0	3 (14.3)	11 (52.4)	22.63	<0.001

Histological changes were assessed on Verhoeff–Van Gieson-stained sections. Pearson's correlation analysis demonstrated a strong negative association between age and both elastic fibre density ($r = -0.857$, $p < 0.001$) and fibre thickness ($r = -0.881$, $p < 0.001$), indicating progressive reduction in the quantity and calibre of elastic fibres with advancing age. Conversely, age exhibited a strong positive correlation with the fragmentation index ($r = 0.912$, $p < 0.001$) and degeneration score ($r = 0.894$, $p < 0.001$), confirming that increasing age was associated with greater structural disruption and histological degeneration of elastic fibres (Table 5 and Figure 1).

Table 5: Pearson's correlation between age and histomorphometric parameters of elastic fibres

Variable	Correlation coefficient (r)	p value
Elastic fibre density	-0.857	<0.001
Fibre thickness	-0.881	<0.001
Fragmentation index	0.912	<0.001
Degeneration score	0.894	<0.001

Pearson's correlation coefficient (*r*) was used to determine the relationship between chronological age and quantitative histomorphometric parameters.

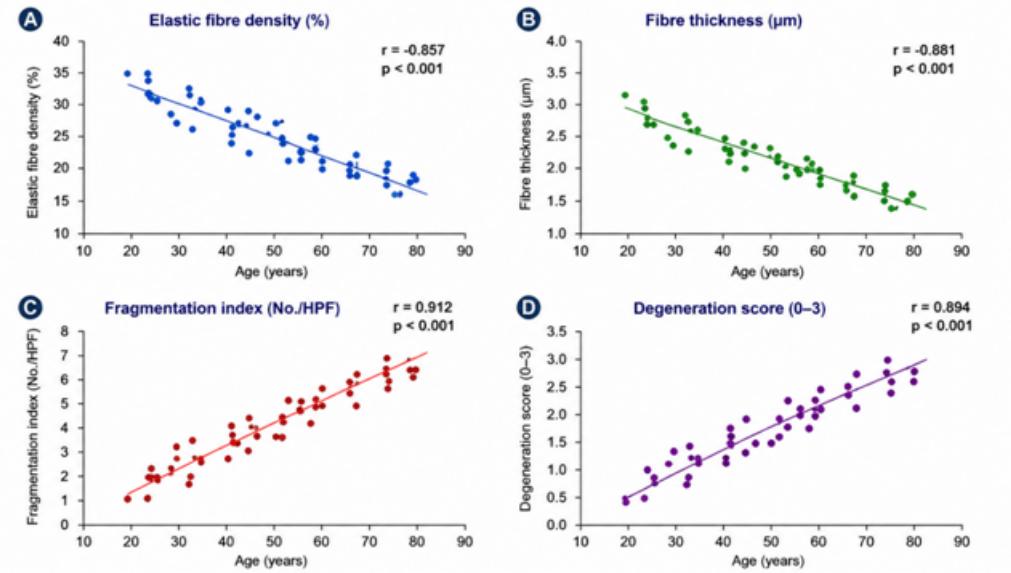

Figure 1: Correlation of chronological age with histomorphometric parameters of elastic fibres in the human trachea and main bronchi. Scatter plots with linear regression lines showing the association between age and (A) elastic fibre density, (B) fibre thickness, (C) fragmentation index, and (D) degeneration score. Pearson's correlation demonstrated significant negative correlations of age with elastic fibre density and fibre thickness, and significant positive correlations with fragmentation index and degeneration score (all $p < 0.001$). Each point represents one cadaver (n = 63)

Discussion

The present study quantitatively evaluated age-related changes in elastic fibres of the human trachea and main bronchi using histomorphometric analysis. The most notable finding of this study was the significant reduction in elastic fibre density and thickness across successive age groups. Elastic fibre density decreased from 33.8 ± 2.5% in

individuals aged 20–29 years to 20.9 ± 2.0% among those aged ≥70 years ($F = 58.72$, $p < 0.001$), while mean fibre thickness declined from 2.81 ± 0.19 µm to 1.71 ± 0.14 µm ($F = 67.14$, $p < 0.001$). These findings are consistent with previous studies by Chunder et al., and Sharma et al., demonstrating that elastin synthesis is largely confined to fetal life and early childhood, with minimal regeneration during adulthood [9,10]. Consequently, mature elastic fibres undergo cumulative damage from oxidative stress, non-enzymatic glycation, and proteolytic degradation by matrix metalloproteinases, resulting in progressive thinning and loss of elastic tissue [11,12,13]. Similar reductions in elastin content and disruption of elastic architecture with ageing have been reported in pulmonary tissues by Mrudula et al., while Schmelzer et al., highlighted elastic fibre degeneration as a hallmark of connective tissue ageing due to the limited capacity for adult elastogenesis [11,13].

A significant age-dependent increase in elastic fibre fragmentation was also observed, with the fragmentation index increasing from 1.18 ± 0.39 to 5.36 ± 0.76 discontinuities per high-power field ($F = 89.36$, $p < 0.001$). Parallelly, the degeneration score increased from 0.42 ± 0.29 to 2.78 ± 0.32 ($F = 81.91$, $p < 0.001$), indicating progressive structural disruption of the elastic framework. These findings are biologically plausible, as the airways are subjected to continuous cyclic mechanical stress throughout life. Repeated respiratory movements, together with age-related oxidative injury and chronic low-grade inflammation ("inflammaging"), promote elastin fragmentation and loss of fibre continuity [14,15,16]. Moreover, advanced glycation end products accumulate with age, increasing tissue stiffness while reducing elasticity and impairing repair mechanisms [17]. The qualitative observations in the present study support these mechanisms, as normal continuous elastic fibres predominated in younger individuals (85.7%) but progressively declined with age, whereas moderate-to-severe fragmentation, marked waviness, loss of parallel orientation and elastic fibre clumping became significantly more frequent in specimens aged ≥60 years (all $p < 0.001$). Comparable histological changes have been described by Sherratt et al., and Calabrò et al., who reported progressive elastin fragmentation and disorganization in ageing connective tissues [13,18].

Interestingly, comparison between the trachea and main bronchi, as well as between the right and left main bronchi, revealed no statistically significant differences in elastic fibre density, thickness, fragmentation index or degeneration score (all $p > 0.05$). This suggests that physiological ageing affects the elastic framework of the central conducting airways relatively uniformly rather than selectively involving a particular anatomical region [19]. Since the trachea and main bronchi experience similar respiratory pressures and repetitive mechanical loading, homogeneous age-related remodelling of their extracellular matrix is expected [19]. Although few studies such as Srinivasan et al., and Doni et al., have directly compared these airway segments, available anatomical evidence indicates a relatively uniform distribution of elastic fibres within the proximal conducting airways under normal conditions [20,21]. The present findings therefore extend existing knowledge by quantitatively demonstrating comparable ageing-related changes throughout the central airway [20,21].

Pearson's correlation analysis further strengthened the association between ageing and elastic fibre deterioration. Age showed strong negative correlations with elastic fibre density ($r = –0.857$) and fibre thickness ($r = –0.881$), while fragmentation index ($r = 0.912$) and degeneration score ($r = 0.894$) exhibited strong positive correlations (all $p < 0.001$). These robust correlations indicate that chronological age is the principal determinant of structural alterations in the tracheobronchial elastic network [22,23]. Similar age-dependent relationships between elastin degradation and declining tissue compliance have been reported in pulmonary and vascular connective tissues, supporting the validity of the present findings [24,25].

Limitations

The present study was limited by its cross-sectional design, which precluded longitudinal assessment of elastic fibre changes over time. Histomorphometric analysis was restricted to cadaveric specimens and did not include functional pulmonary measurements or molecular markers of extracellular matrix remodelling. Additionally, potential confounding factors such as smoking history, occupational exposure, and environmental pollutants could not be comprehensively evaluated because of limited clinical information available for all cadavers.

Conclusion

The present study demonstrates that ageing is associated with progressive deterioration of elastic fibres in the human trachea and main bronchi, characterized by significant reductions in fibre density and thickness together with increased fragmentation and histological degeneration. These structural alterations showed strong correlations with chronological age while remaining comparable across different regions of the central conducting airways. The findings highlight age-related extracellular matrix remodelling as an

important contributor to airway ageing and provide quantitative baseline data for distinguishing physiological ageing from pathological changes. This study enhances the anatomical understanding of tracheobronchial ageing and offers a foundation for future investigations integrating histological, molecular, and functional assessments of respiratory ageing.

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