Age-related odontometric changes of human teeth

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Objective. The number of older patients requiring restorative treatment are likely to increase due to improvements in oral health and increased longevity. However, aging odontometric data are lacking. The aim of this study was to determine possible changes in pulp cell density, pulp area, and dentinal thickness with age.
Study design. Incisors (50), canines (39), premolars (51), and molars (7) extracted from 60 patients aged between 10 and 59 years, were analyzed histomorphometrically for cell density (odontoblasts, subodontoblasts, and pulp core fibroblasts) and dentinal thickness.
Results. With increasing patient age, in both crown and root aspects of teeth, dentinal thickness increased ($P < .001$), while the density of odontoblasts ($P < .001$), subodontoblasts ($P = 0.001$), and pulp fibroblasts (crown, $P < .011$; root, $P = .0015$) decreased. The degree of age-related changes in teeth appeared to be asymmetrical, with decreases in the root being greater than in the crown. At all ages pulp cell densities, including odontoblasts, within the crown were greater than in the root ($P < .001$), even though the calculated rate of dentinal deposition was greatest in the root.
Conclusion. Decreases in pulp cell density may reduce pulp repair activity after restorative treatments, although increases in dentinal thickness may aid pulp protection. An understanding of these age-related changes will influence the provision of restorative and endodontic care and benefit older patients.
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The lifetime preservation and maintenance of functional teeth is a prominent aspiration of the dental profession. Recent improvements in oral health among many age groups as well as a decline in dental caries, particularly among children, suggests that more people will retain their natural teeth into old age than ever before.[1-5] In the United States, population projections suggest that the proportion of the population aged 65 years or older will nearly double between 2000 (12.6%) and 2030 (20.0%) and that the proportion of the population of those aged 85 years and older will increase substantially over the next 10 to 15 years.[6] This population trend, coupled with the evidence that people are retaining their teeth into old age,[7-9] suggests that there will be many more older adults with their natural teeth in years to come. A study of an elderly population found that both crown and root caries remain prevalent, and among those with natural teeth the use of dental services is high.[10] These factors place demands on the dental profession to provide an increasingly older patient population with appropriate restorative treatment. It is necessary to understand the precise nature of changes in functional tooth histology and repair activity with age, as these influence the diagnosis, planning, and provision of oral health care to fulfill these demands.

Age-related changes occur in teeth between approximately 10 weeks in utero to old age.[11] Lascassagne in 1889 was the first to characterize changes in fully formed teeth with aging[12]; however, the first scientific assessment was provided by Gustafson.[13] He characterized tooth aging based on a scale of the severity of attrition, gingival recession, transparency of root, root resorption, apposition of secondary cementum at the root apex, and increasing secondary dentinal thickness. Variations in these parameters with aging have provided a high degree of accuracy for estimating age and have been studied many times.[12,14,15] The pulp dentinal complex is capable of responding to a variety of stimuli over time. These stimuli can be physiological relating to the normal stresses to which a tooth would be exposed over a lifetime, but are also pathological due to caries, tooth surface loss, or restorative treatment.[16] Ultimately age-related changes in teeth are based on biological markers of age. Teeth reflect the biological or physiological age of the individual, and variations caused by genetic factors and chewing habits can influence tooth anatomy.[17]

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One of the most obvious features of aging is a reduction in size of the pulp chamber, caused by the continual secretion of dentinal matrix (physiological secondary dentinogenesis) by odontoblasts.[18] However, limited quantitative information exists on both the rate of physiological secondary dentinal formation and exact odontometric analysis of pulp chamber occlusion.[11-18] The rate of dentinal secretion by odontoblasts has been observed to vary according to the chronology and circumstances of its secretion, with some confusion about the rate of secondary dentinal deposition. The dentinal thickness has been calculated as increasing at a rate of approximately 0.5 μm per day and decreasing throughout life,[19] whereas the cervical pulp width of mixed human teeth was found to reduce by 2 mm over a mean patient age range of between 28 and 74 years,[18] giving an approximate rate of secondary deposition of 43 μm per year, or 0.119 μm per day. Details of pulp cell population responses to occlusion of the pulp chamber are similarly inconclusive. Quantitative studies in man have reported a reduction in total pulp cell numbers by 50% between the ages of 20 and 70 years.[20] Studies in the rat have found a 75% loss of pulp cells[21] and a 21% loss of odontoblasts in older rats in comparison with younger rats.[22,23] However, these reports have provided little information on changes in the different pulp cell populations with age.

Following restorative surgery, the repair capacity of the dentinal pulp complex is dependent on the density of odontoblasts,[24] or density of odontoblast-like progenitor cells following pulp exposure. These cells will secrete a tertiary dentinal matrix (sometimes subclassified as reactionary or reparative dentin on the basis of the cells responsible for its secretion) in a focal manner at the pulp-dentinal interface in response to the injurious stimuli. The repair capacity of the dentinal pulp complex also appears to be age dependent, and this may explain differences in the success of restorative treatments between patients.[25] These observations rationalize the need to study the effect of age-related changes on the dentinal pulp complex. Although it is generally accepted that changes occur in functioning teeth from the time of eruption to old age, there are conflicting views on the details of these changes.[19,26] One of the reasons for this conflict is that interpretation of findings has been based on study of sections without systematic consideration of age or the type of tooth from which the sections were made.[27] The objective of this study is to measure crown and root dentinal thickness according to tooth type and patient age. In these same tooth areas, the density of odontoblasts, cells of the subodontoblast layer, and fibroblasts will be measured to provide quantitative information on age-related changes in teeth.

MATERIAL AND METHODS

This study is based on the odontometric evaluation of sections of disease-free human teeth that are randomly selected from a large collection of 3000 human teeth extracted under local or general anesthetic.[28-30] The criteria for selecting teeth for inclusion in this study were only teeth available with full patient or parental consent with complete patient records. Before inclusion in this study, patient records were assessed and all histological sections of teeth were examined microscopically to remove the possibility of including teeth associated with periodontal or periapical disease or teeth with signs of caries, discoloration, erosion, abrasion, or excessive wear. Teeth were extracted from patients for orthodontic or prosthodontic reasons. Importantly, we included only functional teeth in this study. Teeth that were nonfunctional because of impaction were not included. Only histological sections showing the entire dentinal pulp tissue from crown apex to root tip were included. Once these exclusion criteria were applied, 147 specimens taken from 60 patients were available for analysis.

The 147 teeth consisted of incisors (50), canines (39), premolars (51), and molars (7) obtained from patients aged between 10 and 59 years. For the purposes of data analysis, patients were divided into 3 groups: 10 to 30 years (young patients), 31 to 50 (middle-aged patients), and 51 to 59 years (older patients). The apical third of the root was removed to aid pulp fixation with 10% formalin for 48 hours. Teeth were demineralized in 5% formic acid until radiographic evidence showed complete demineralization, followed by routine processing and embedding in paraffin wax for histological examination. Sections were routinely cut at 6 μm and stained with hematoxylin and eosin.

The lingual-labial longitudinal serial section of each specimen, exhibiting the greatest dentinal thickness and pulp area was used. All sections were analyzed blind on coded sections. Measurements were taken in the crown and root aspects of each tooth specimen, as previously described.[18] Briefly, teeth with curved roots were measured in the same way as straightened teeth to keep the protocol consistent between all teeth. All distance measurements from crown to root were measured in the central axis of teeth to take into account tooth curvature. Crown measurements were collected at lingual and labial positions measured to be 50% of the distance between the tooth cementoenamel junction, and the furthest crown pulp dentinal border (Fig 1). The root measurements were collected at lingual and labial positions measured to be 75% of the distance between the dentinoenamel margin, and the remaining root tip, equivalent to the apical mid root aspect (Fig 1). The dentinal thickness of crown and

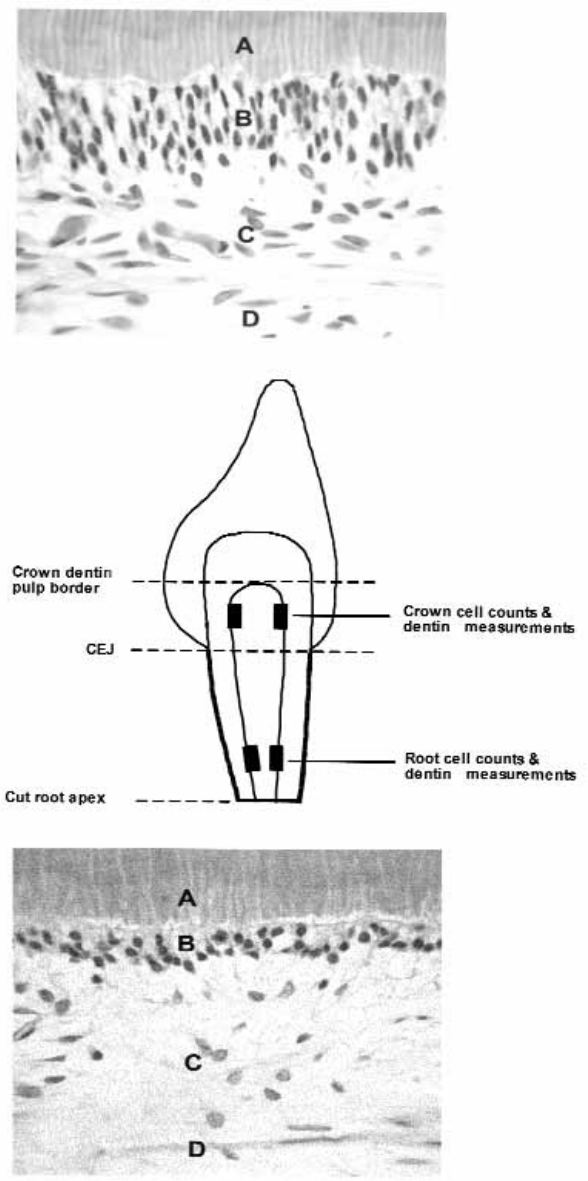

Fig 1. Photomicrographs of hematoxylin and eosin–stained sections showing cellular detail of areas used for cell counts in the crown [a] and root [c] (A = dentin, B = odontoblasts, C = cells of the subodontoblast layer and D = fibroblasts; original magnification × 320). The diagram (b) indicates the locations of these areas within a tooth.

root dentin was measured in each tooth specimen along the lingual-labial horizontal axis.

The peripheral dentinal lingual-labial horizontal width of each tooth was measured. From this measurement, the total horizontal pulpal width between the lingual and labial border was subtracted. This value was then divided by 2, because the 2 dentinal walls of teeth are included in this measure. This provided a measurement of the mean lingual-labial horizontal dentinal thickness of each tooth. Thickness measurements were collected by using an ocular micrometer grid at ×50 magnification under light microscopy. The ocular micrometer was also used at ×50 magnification to measure the length of the pulp dentinal border in each tooth.

At the same crown and root lingual and labial positions for measuring dentinal thickness, cell densities were counted as previously described,[24,25,31,32] by using a 10 × 10 mm ocular micrometer grid at a magnification of ×200. Briefly, morphologically intact odontoblast, subodontoblast, and fibroblast cell numbers were counted within ocular micrometer grid squares at ×200 magnification. Cell types were categorized on the basis of their morphology and position within the tooth pulp and only intact cells displaying a clearly identifiable nucleus were included. Odontoblasts (Fig 1) were identified as being the outermost stratum of palisaded, columnar cells, immediately adjacent to the predentin that displayed eosinophilic cytoplasm and a nucleus located in a basal polarized position. Cells of the subodontoblast layer (Fig 1) were identified as the cell stratum immediately below the odontoblast layer and comprised mainly of a cell rich zone of fibroblasts, with a distribution of a few assorted cell sizes and staining intensities. Fibroblasts (Fig 1) within the central pulp core were defined as cells showing a tapering bipolar spindle-shaped morphology with or without additional cell processes. Incorporated within this definition were cells showing the characteristics of both active (relatively abundant cytoplasm and large oval nucleus) and inactive (smaller nucleus and scant cytoplasm) fibroblasts. All pulp cells not conforming to these cell classifications, or being identified as systemic cell populations (eg, endothelial cells), were excluded from counts.

Cell densities were counted at the root and crown lingual and labial measurement positions (Fig 1) by aligning the ocular micrometer grid along each cell strata, parallel to the pulp dentinal border. Cell counting was performed in 5 alternate micrometer grid squares above and below each measurement position. This provided 10 cell counts for each of the 4 (crown-labial, crown-lingual, root-labial, root-lingual) measurement positions, a total of 40 cell counts for each cell type per tooth. The number of odontoblasts and subodontoblasts per ocular grid square were converted into cell numbers per unit length (mm) pulp dentinal border for presentation purposes. The number of fibroblasts per ocular grid square were converted to cell numbers per mm^2 of pulp core area for presentation purposes.

It was important to ensure that these values were not affected by collecting additional cell counts to test the reproducibility of the cell density values. It was found that stable cell density values were achieved by measuring a minimum pulp dentinal border length of

Table I. Odontometric changes in the crown (C) and root (R) aspect of different tooth types in patients of increasing age, expressed as a percentage of baseline value at age 10 to 30 years (number of teeth = 147)

		Age of patients (y)		
Variable	*Tooth type*	*10-30*	*31-50*	*51-59*
Number	Incisor	16	20	14
	Canine	11	15	13
	Premolar	22	21	8
	Molar	1	5	1
			% decrease from baseline (10-30)	
		Position	31-50	51-59
Odontoblasts	Incisor	C	5.5	11.7
		R	39.8	53
	Canine	C	0.7	18.3
		R	16.4	35.2
	Premolar	C	–4.4	15.5
		R	2.4	43.1
	Molar	C	–16.5	19
		R	13.9	45.9
Subodontoblasts	Incisor	C	9.6	29
		R	41.9	58.9
	Canine	C	17.7	33.4
		R	17.3	53.1
	Premolar	C	15.3	34.7
		R	33.2	62.2
	Molar	C	12.5	30.8
		R	39.6	67.9
Fibroblasts	Incisor	C	–0.2	30
		R	34.7	25.6
	Canine	C	18	35.1
		R	9.4	50.2
	Premolar	C	8.7	13.8
		R	11.6	44.8
	Molar	C	–9.3	–3.6
		R	12.5	68.7
Dentinal thickness	Incisor	C	–5.9	–15.5
		R	–12.9	–13.1
	Canine	C	16.5	–3.4
		R	5.3	–17.2
	Premolar	C	–6.5	–34.1
		R	–17.8	–39.5
	Molar	C	–6.2	–51.8
		R	0.6	–27.6

79.2 μm (× 4 ocular grid squares) or pulp area of 1176.2 μm^2 (× 3 ocular grid squares).

The raw data from all counts were examined by using 2-way linear analysis of variance (ANOVA) tests for multiple comparisons among the means at a confidence level of 95% (Statview software, SAS Inc.), followed by Scheffé post hoc ANOVA to compare means comparisons at a confidence level of 95%.

RESULTS

The changes taking place in teeth with aging, included increased dentinal thickness and decreased cellularity. Except for the incisor fibroblasts, the reduction in density of each pulp cell type was greatest in the root aspect of all tooth types. A summary of these aging changes between the ages of 10 to 30 years and 51 to 59 years is shown in Table 1.

Odontoblast density

In older patients aged 51 to 59 years, the density of crown odontoblasts for all teeth decreased by 15.6%, and the density of root odontoblasts by 40.6%, in comparison with younger patients aged 10 to 30 years (Fig 2).

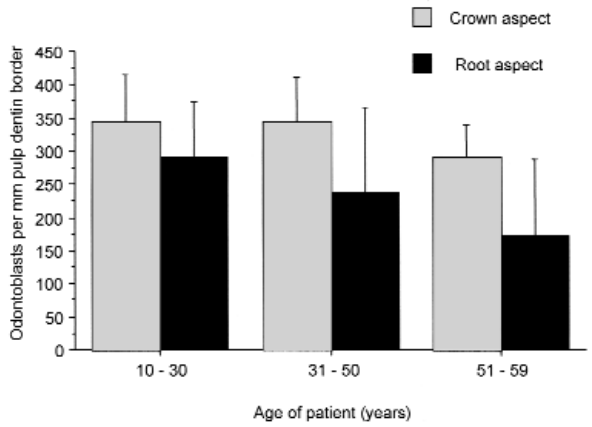

Fig 2. Reduction in odontoblast density in both crown and root aspects of teeth with increasing age (data for all teeth combined; *error bars* show standard deviations; number of teeth = 147).

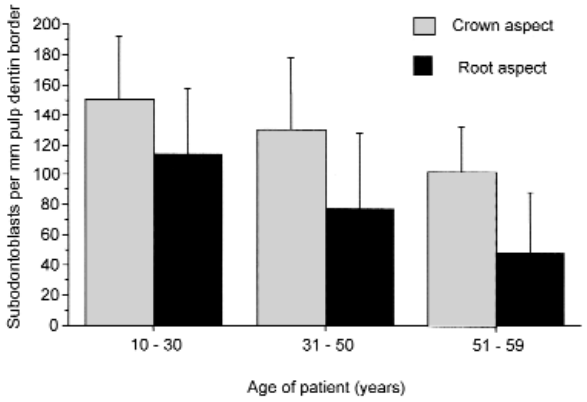

Fig 3. Reduction in subodontoblast density in both crown and root aspects of teeth with increasing age (data for all teeth combined; *error bars* show standard deviations; number of teeth = 147).

Furthermore, reductions in odontoblast density in both crown and root aspects of teeth were correlated to the age of the patient (ANOVA n = 147, $P < .001$). However, little difference could be observed between crown odontoblast density and patients aged 10 to 30 versus 31 to 50 years (Scheffé $P = .99$), although the density of crown and root odontoblasts was statistically different for comparisons of all other age groups (Scheffé $P < .05$). Irrespective of age group, odontoblast densities were always greater in the crown than the root (Fig 2; $P < .001$). The mean densities of odontoblasts in both the crown (316.0 - 350.4 cells/mm pulp dentinal border) and root (226.3 - 284.0 cells/mm pulp dentinal border) did not differ significantly between tooth types (Scheffé $P = .21$).

Subodontoblast density

The subodontoblast cell density distribution between crown and root, and its association with age, was similar to that found for odontoblasts. Thus, patient age correlated with the density of subodontoblasts in crown and root aspects of teeth (ANOVA n = 147, $P < .001$) and densities were always greater in the crown than root irrespective of age (Fig 3; $P < .001$). A comparison of subodontoblast density between younger (10-30 years old) and older (51-59 years old) patients, found that the density of these cells decreased by 32.1% and 57.4% in crown and root tooth aspects respectively. Although differences between the density of crown subodontoblasts in patients aged 10 to 30 and 31 to 50 years of age did not quite reach statistical significance (Scheffé $P < .06$), all other differences between crown and root subodontoblast density were statistically significant for all age comparisons (Scheffé $P < .05$). The density of subodontoblasts was least conserved in root aspects of the premolar teeth of older patients, and most conserved in crown aspects of incisor teeth (Table I). Between tooth types, the mean densities of subodontoblasts showed little variation in either crown (125.5–127.1 cells per mm pulp dentinal border) or root (69.4–89.5 cells per mm pulp dentinal border) aspects of the pulp (ANOVA $P = .6$).

Fibroblast density

The density of both crown and root fibroblasts in all teeth was found to be reduced significantly in older patients aged 50 to 59 years (by 26.9% in the crown and 41.3% in the root), in comparison with younger patients aged 10 to 30 years (ANOVA $P < .011$ and $P = .0015$ respectively; Fig 4). However, little difference could be observed between younger (aged 10-30 years) and middle-aged groups (aged 31-50 years), in the crown (Scheffé $P = .65$), or between older and middle-aged groups in the crown or root (Scheffé $P > .05$). As found for the other pulp cell populations, mean root fibroblast densities were significantly lower than those in the crown for all age groups ($P < .001$). Although the differences in fibroblast densities between tooth types (crown: 1048.0–1218.8 cells/mm^2; root: 835.9–1268.6 cell/mm^2) were larger than those seen for odontoblasts and subodontoblasts they were not significant (ANOVA $P < .21$).

Dentinal thickness

As expected, a greater dentinal thickness was observed in the teeth of older patients in comparison with teeth from younger patients (ANOVA $P < .001$). By using regression analysis for all teeth (n = 147) the

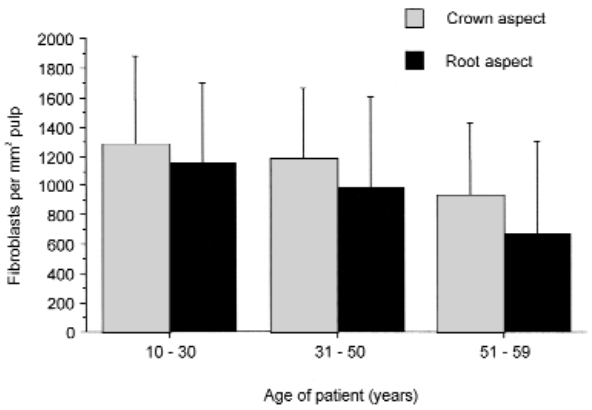

Fig 4. Reduction in fibroblast density in both crown and root aspects of teeth with increasing age (data for all teeth combined; *error bars* show standard deviations; number of teeth = 147).

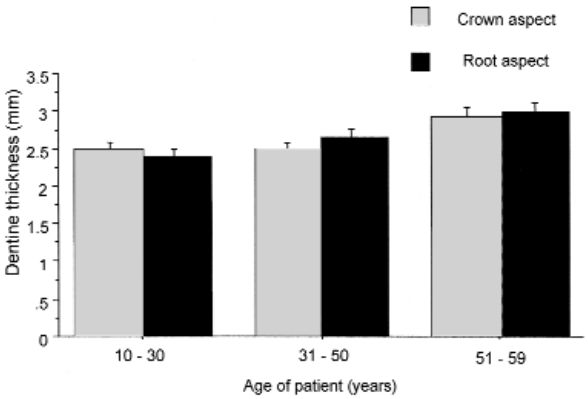

Fig 5. Increase in dentinal thickness in both crown and root aspects of teeth with increasing age (data for all teeth combined; *error bars* show standard deviations; number of teeth = 147).

mean rate of increasing dentinal thickness was found to be 6.5 μm per year (95% confidence interval, 2.5 to 10 μm) for the crown and 10 μm per year (95% confidence interval, 5.5 to 15 μm) for the root. The effect of continual dentinal deposition by odontoblasts was to increase the thickness of dentin by 0.45 mm (17.1%) and 0.60 mm (24.3%) in the crown and root aspects of teeth in older patients (Fig 5). However, age-related differences were observed between different tooth types, for example, little increasing dentinal thickness was detected in the crown aspect of canine teeth (+ 3.4%), while the crown dentin of incisor and premolar teeth displayed 15.5% and 34.1% increases, respectively, in older patients (Table 1). Irrespective of age, mean tooth dentinal thickness was observed to vary significantly across tooth types (ANOVA n = 147, $P < .001$; Table 2) with differences between all tooth types (Scheffé $P < .05$), except between canine and premolar teeth (Scheffé $P = .43$).

Gender effect

Patient gender has previously been associated with anatomical differences in teeth, including dentinal thickness. Analysis of the data according to patient sex revealed no statistically significant differences between any of the measured variables (ANOVA $P > .05$).

DISCUSSION

Age-related odontometric changes of teeth are important because these changes may influence pulpal vitality and repair responses. The likely postoperative responses of teeth have an impact on treatment planning decisions and, ultimately, on the success of restorative treatments. The greater numbers of odontoblasts, subodontoblasts, and fibroblasts in the crown aspect suggest it may be easier to preserve pulp vitality following crown trauma rather than root trauma. Although more investigation is required to confirm these observations, this information may be helpful for decision making on the appropriateness of restoration, pulp capping, pulpotomy, or tooth extraction. Attention to the dentinal thickness of teeth shown in Table 2, and the age-related changes in Table 1, may be helpful as a guide to estimate the remaining dentinal thickness during cavity preparation, and avoid the accidental creation of pulp exposure. Understanding the disadvantages of age-related changes such as reductions in the pulpal cell populations, and using the advantages such as increases in dentinal thickness, will benefit the provision of restorative care for the increasing numbers of older aged patients.

Aging of teeth has commonly been evaluated by using qualitative assessments of tissue alterations, such as increasing dentinal thickness,[27] narrowing of the pulp chamber,[33] pulp stone formation, dystrophic calcification,[26,34] and reduction of some pulp cell populations.[35] However, qualitative methods always contain an element of subjectivity.[36] At the molecular level, studies have observed age-related changes in the extracellular matrix,[37] nerve fibers,[38] and the cytoskeletal components of pulp cells.[39] However, these observations have a limited usefulness to the clinical situation. Ideally, quantitative assessments of age-related changes in teeth are required, which can provide accurate information on dentinal thickness, and pulp cell population density.

Table II. Non–age-related dentinal thickness (mm) in the crown and root aspects of different tooth types (± standard errors, number of teeth = 147)

		Dentinal thickness in mm (mean ± SE)		
Tooth type	*n*	*Crown*	*Root*	*Tooth average*
Incisor	50	1.08 ± 0.02	1.19 ± 0.03	1.1 ± 0.02
Canine	39	1.43 ± 0.06	1.42 ± 0.06	1.4 ± 0.04
Premolar	51	1.37 ± 0.04	1.30 ± 0.06	1.3 ± 0.04
Molar	7	1.57 ± 0.14	2.01 ± 0.19	1.8 ± 0.13

Accurate information on dentinal thickness can provide guidance to clinicians on the likely pulp reactions to cavity preparation forms and avoidance of pulp exposure, because the dentinal thickness beneath cavities has been found to be an important determinant of pulp reactions.[31,32]

Accurate information on odontoblast and other pulp cell densities can provide information on the dentinal repair capacity of the pulp tissue following restorative treatment. This is because odontoblast density is a critical factor mediating the scale of pulp reparative responses.[24,25] Consequently, this explains our need to evaluate the effect of aging using a sample of 147 teeth extracted from patients aged between 10 and 59 years of age. Incisor, canine, molar, and premolar types were evaluated individually and cumulatively to establish variations between tooth types. Odontoblasts, subodontoblasts, and fibroblasts were counted and the dentinal thickness measured in the crown and root aspects of teeth, to examine asymmetric age-related changes in teeth. ANOVA statistical methods were used to analyze the raw data, followed by Scheffé post hoc analysis tests.[40] These statistical procedures are reported to be among the most versatile and conservative of the multiple comparison tests.[41]

The continual secretion and mineralization of dentinal matrix by odontoblasts increased the mean crown dentinal thickness of all the teeth by 17.1%, while the mean root dentinal thickness was found to increase by 24.3% in older age patients (Fig 2). However, age-related differences were observed between different tooth types, for example, little increasing crown dentinal thickness was observed in canine teeth (+ 3.4%), while the crown dentinal of incisor teeth increased by 58.1% (Table 1). These odontometric observations confirm earlier reports of minimal secondary dentinal deposition in canine teeth,[27] and in comparison with other tooth types, greater age-related increases in molar secondary dentin.[18] The limited number of molar teeth of different ages available to us in this study have not allowed us to draw conclusions on the effects of aging on this tooth type. Information about the regulation of dentinal synthesis and secretion by odontoblasts is limited,[42] and the reason for the greater deposition of root-aspect dentin cannot be explained simply by factors such as odontoblast density, because odontoblast density is greater in the crown aspect of teeth (Fig 2). Clearly, increased root-aspect dentinal deposition must be explained by greater rate of dentinal secretion by the odontoblasts in this part of the tooth. Consequently, tooth dentinal thickness was found to increase by a total mean of 20 μm per year (10 μm per dentinal wall) in the root aspect, while tooth dentin was found to increase by a mean of 13 μm per year (6.5 μm per dentinal wall) in the crown aspect of all the teeth. The greater rate of secretion of dentin by odontoblasts in the root, in comparison with the crown, contrasts with the situation during root development and implies differences in the regulation of secretory activity of these cells through life. These findings of increased dentinal deposition in the root aspect of teeth, in comparison with the crown aspect of teeth, confirm observations of occlusion in the root canals of teeth with aging.[18,26] Diminution in size of the root canal has also been associated with gender, with males being more affected, and with calcification-related diseases such as arthritis, gout, kidney stones, gallstones, atherosclerosis and hypertension.[30] However, no significant difference was observed in dentinal thickness, according to patient sex in this study to support these claims (ANOVA $P > .05$).

Our calculations of dentinal secretion are considerably less than those suggested by published reports, which show great inter-study variation. For example, Morse[19] found a rate of human secondary dentinal deposition of approximately 182.5 μm per year whereas Solheim[18] obtained a value of approximately 43 μm per year. In monkeys, secondary dentin was found to average 0.8 μm per day (288 μm per year).[43] The rate of secondary dentinal secretion is sensitive to trauma and environmental effects.[30] Pulp dentinal injury caused by disease or cavity preparation and restoration events may cause tertiary dentinogenesis which is secreted by odontoblasts at more than 3 times the rate of secondary dentinal secretion.[29] Confusion between measurements of the rate of secondary and tertiary dentin, may provide an explanation for the disparities between studies. Clearly more large scale clinical investigations are necessary to determine a consistent rate for the secretion of physiological secondary dentin. Nevertheless this study has quantified the dentinal thickness of teeth at different ages, clinically referred to as pulp recession, and this may prove useful for determining the form of cavity preparation for certain restorative procedures. Interestingly

the greatest reduction in root pulp diameter was observed between the age groups of 10 to 30 and 31 to 50 years, suggesting that the rate of physiological secondary dentinal secretion is not constant throughout life.

The density of odontoblasts has implications for the reparative capacity of the pulp tissue to regenerate lost or damaged dentinal matrix.[24,25] Following dentinal injury by trauma, caries, or cavity preparation, the secretion of tertiary dentin is often necessary to protect the tooth pulp from infection and the chemical or cytotoxic effects of dental materials.[44,45] The 15.6% reduction in crown odontoblasts, and 40.6% of root odontoblasts in older patients (Fig 2) together with their decreased secretory activity, suggests that the reparative capacity of the pulp will be compromised in old age. The reduction in subodontoblast and fibroblast density (Figs 3 and 4) is more important following pulp exposure situations, and the loss of these cells will also compromise pulp reparative capacity. These observations imply that the tertiary dentinal secretory activity in the teeth of older age patients may be diminished or delayed in comparison with those of younger patients, as suggested previously.[25] However, the greater thickness of dentin and reduced pulp volume in elderly patients may compensate for compromised responses by allowing the preparation of slightly deeper cavity preparations before pulp reparative responses are necessary, or before a pulp exposure occurs.

The density of functional pulp cells is an important factor in pulp dentinal repair responses following caries, attrition, abrasion, erosion, tissue damage, and trauma[46] in addition to restorative surgery.[24,25,31,32,44,45] An obvious explanation for the reduction in pulp cell numbers and pulp cell density is that the pulp tissue undergoes remodelling in order to accommodate the narrowing of the pulp chamber. Further studies are required to investigate the mechanisms underlying these age-related changes which may be accomplished by apoptosis or programmed cell deletion.[47] Clearly, we still have much to learn of the biological control mechanisms responsible for cellular activity and survival throughout life. We have shown some of the complexity of the situation regarding age-related changes in teeth. The goal for scientists and clinicians must be to devise new treatment strategies to enable the increasing proportion of elderly patients to maintain healthy teeth throughout their lifetime.

This article is dedicated to the memory of Dr Harold R. Stanley, who died before publication.

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