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COMPARATIVE HISTOLOGICAL AND HISTOMORPHOMETRIC CHARACTERIZATION OF INFLAMED VERSUS NORMAL DENTAL FOLLICLE

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ABSTRACT

Objectives: This study aimed to characterize the histological and histomorphometric changes in inflamed dental follicle (DF) compared to normal DF using routine and specialized staining techniques.

Materials and methods: Twenty human DF tissue specimens (ten normal, ten inflamed) were sectioned. The samples were stained using Hematoxylin and Eosin (H&E) and picrosirius red to assess collagen fiber distribution. Histological features, including epithelial lining, connective tissue cellularity, collagen fiber density and organization, and interstitial edema, were evaluated. Quantitative analysis of collagen fiber area percentage, interstitial edema area percentage, and inflammatory cell number were performed; to ascertain statistical significance, the data underwent analysis of variance (ANOVA), and significant outcomes were further explored via post hoc tests.

Results: H&E staining of inflamed DF revealed increased cellularity with inflammatory cell infiltration and interstitial edema compared to normal DF, which showed a well-organized stratified epithelium and delicate connective tissue. Picrosirius red staining demonstrated a significantly denser and more disorganized collagen network in inflamed DF compared to the loosely arranged, delicate fibers in normal DF ($P < 0.001$). Quantitative analysis confirmed that the level of increase was statistically significant ($P < 0.001$) in collagen fiber area percentage, interstitial edema area percentage, and inflammatory cell number in the inflamed group.

Conclusion: Inflammation in the DF is associated with significant histological alterations, including increased inflammatory cell infiltration, interstitial edema, and increased collagen density and disorganization. These findings underscore the dynamic tissue response within the DF during inflammation and the possibility of odontogenic cyst formation.

KEYWORDS: Collagen; Dental Follicle; Extracellular Matrix; Edema; Inflammation

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INTRODUCTION

The intricate process of odontogenesis, the formation and development of teeth, has a critical dependence on the presence and proper functioning of the dental follicle (DF), a specialized mesenchymal tissue that intimately envelops the developing tooth germ [1]. This encompassing structure, also referred to as the dental sac, is not merely a passive covering but rather an active and dynamic participant in every stage of tooth development, beginning with the enamel organ's initial budding and the dental papilla's condensation extending to the tooth's final eruption within the oral cavity and the subsequent development of the supporting structures of the periodontium [2].

Originating from neural crest ectomesenchyme, DF orchestrates inductive interactions with the dental papilla, in conjunction with the enamel organ. During root formation, DF surrounds the Hertwig epithelial root sheath. (HERS), which induces root dentinogenesis[3]. Fragmentation of HERS forms the epithelial rests of Malassez (ERM) which permit the differentiation of DF mesenchymal cells into cementoblasts and periodontal ligament fibroblasts[4]. Signaling from the DF, HERS, and ERM coordinates root and periodontium development. This highlights the continuous reciprocal interactions between ectomesenchyme and epithelium throughout tooth formation.

The significance of DF extends beyond simply providing a physical boundary for the developing teeth. It serves as a critical signaling center, a hub of molecular communication that secretes a diverse array of extracellular matrix (ECM) components, growth factors, cytokines, and chemokines[5]. Signaling molecules from DF act on the enamel organ and dental papilla, controlling cell proliferation, differentiation, and movement [6]. This precise regulation dictates the developing tooth's form and structure[7].

Moreover, DF is critically important regarding tooth eruption as it mediates the resorption of overlying bone and connective tissue through the secretion of enzymes and signaling molecules by cells like osteoclasts and fibroblasts[8]. This active bone remodeling creates the necessary eruptive pathway[9]. DF's pivotal role in osteogenesis and alveolar bone remodeling during tooth eruption is intimately connected to the dynamic interplay between its cellular proliferation and the formation of its collagen-rich ECM. As a heterogeneous population harboring progenitor cells, DF cells exhibit active proliferation in specific zones and at critical developmental stages, enabling the expansion of cell populations necessary for both signaling and matrix production [1].

This proliferation is tightly regulated, allowing for the timely secretion of key osteogenic signaling molecules, such as bone morphogenetic proteins (BMPs), into the surrounding environment[10]. Simultaneously, these proliferating DF cells, particularly fibroblast-like cells, are responsible for synthesizing and organizing the collagen matrix, which provides structural support and a scaffold for cellular attachment and migration within the follicle[11].

The density and organization of this collagen network is determined by the extent of DF cell proliferation and the synthetic capabilities of the DF cells, which directly impact the follicle's ability to exert osteogenic and osteolytic effects on the adjacent bone[12]. Conversely, under certain conditions, inflammation of DF arises due to localized infections, traumatic events, or developmental anomalies associated with the adjacent tooth [13].

Notably, pericoronal infection initiates an inflammatory cascade characterized by the influx of immune cells and the secretion of pro-inflammatory cytokines [14]. These inflammatory mediators affect the delicate equilibrium between osteogenic and osteolytic activities within the follicle, which may predispose to the formation of odontogenic cysts[15]. Furthermore, persistent inflammation

within DF has been implicated in the pathogenesis of specific odontogenic neoplasms [16]. Consequently, the maintenance of DF's physiological homeostasis is critically important for ensuring optimal dental health and preventing the development of pathological conditions.

MATERIALS AND METHODS

Ethical approval

The Research Ethics Committee at the Faculty of Dentistry, Cairo University, granted ethical approval for this research protocol. Furthermore, all patients provided voluntary informed consent for the use of their tissue specimens in this study.

General histological examination

Twenty FFPE tissue specimens, comprising ten normal DF samples and ten inflamed DF samples, were utilized in this study. The protocol for preparing histological samples was consistent with the methods detailed by Bancroft and Stevens [17]. Briefly, following slicing to a thickness of 3-4 mm, DF tissues were fixed using 10% neutral buffered formalin. After fixation, the tissues were subjected to a graded ethanol dehydration process, followed by clearing in xylene and embedding in paraffin. Using a microtome, the paraffin blocks were cut into 4-6 μm thick sections. To facilitate the examination of overall tissue structure, these sections were stained with Hematoxylin and Eosin (H&E). For analysis, the H&E-stained sections were viewed under a Leica microscope (CH9435 Heerbrugg) from Leica Microsystems, Switzerland.

Histochemical examination (Picrosirius Red Stain)

Picrosirius Red stain (Sigma-Aldrich "Direct Red 80", Catalog # 36-554-8,6 St Louis, MO, USA) was used to stain DF sections, allowing for detailed visualization of collagen fibers, which appeared red. Non-collagenous proteins were concurrently stained a light-yellow color [18], facilitating histological estimation of collagen sedimentation.

Morphometric analysis

In H&E-stained sections, quantitative scoring was used to assess both the count of inflammatory cell infiltrations and the area of interstitial edema. This analysis was conducted per cross-sectional area with the aid of the Leica QWin DW3000 image analysis system (Leica Imaging Systems Ltd., Cambridge, England). Ten representative fields from each section in all groups were evaluated at 400× magnification using light microscopy viewed on a monitor.

The 'L.A.S. software v.4' image analysis system (Leica Microsystems, Cambridge, UK) was employed for the quantitative measurement of collagen fibers in Picrosirius Red stained DF sections (10 images per group, 400× objective lens). This system included a Leica microscope, a color video camera, a color monitor, and a Leica IBM personal computer with a hard disk, operated by Leica QWin 500 software. Results for area % were statistically summarized as mean ± standard deviation.

Statistical analysis

The Kolmogorov-Smirnov test was used to determine the normality of the data. As the majority of the data exhibited a normal distribution (parametric), intergroup relationships were analyzed using descriptive statistics, one-way analysis of variance (ANOVA), and post hoc tests. Statistical significance was considered to be present at $p < 0.05$. All statistical analyses were carried out using SPSS 26.0 (Statistical Package for the Social Sciences, SPSS, Inc., Chicago, IL, USA) for Windows.

RESULTS

Histological results

Histological examination of normal DF specimens revealed an epithelial lining enclosing connective tissue (CT). Higher magnification showed this lining to be a stratified epithelium,

characterized by a basal layer of cuboidal cells transitioning to superficial flattened cells. The adjacent CT exhibited fibroblasts, non-engorged blood vessels, and fine collagen fibers **(Fig. 1a, 1b)**.

Histological examination of inflamed DF specimens showed atrophic epithelial lining with epithelial fragmentation in some areas along with mesenchymal inflammation. Higher magnification showed congested blood vessels with thickened walls, inflammatory cells infiltration along with fibrotic areas and spongiosis **(Fig. 2a-c)**.

Histochemical results

In normal DF specimens, picrosirius red staining highlighted collagen fibers within CT. Closer microscopic examination revealed the presence of sparse, thick collagen bundles interspersed within a background of loosely arranged, delicate collagen fibers **(Fig. 3a, 3b)**.

In inflamed DF specimens, picrosirius red staining revealed a markedly different pattern of collagen fibers within CT. Closer microscopic examination demonstrated a more densely packed network of collagen fibers, often exhibiting a disorganized and irregular arrangement compared to the normal follicle. While some thicker bundles might still be present, they were frequently interwoven within a predominance of finer, more haphazardly oriented collagen fibers. The collagenous framework appeared more abundant and less delicate than that observed in normal dental follicle tissue **(Fig.4a,4b)**.

Morphometric results

A statistically significant difference ($P<0.001$) was observed between the normal and inflamed DF groups, as shown by ANOVA regarding the area percentage of collagen fibers, the area percentage of interstitial edema, and the number of inflammatory cells. The inflamed DF group showed a statistically significant increase ($P<0.001$) in all these parameters when compared to the normal DF group, according to post hoc testing **(Table 1) (Fig. 5)**.

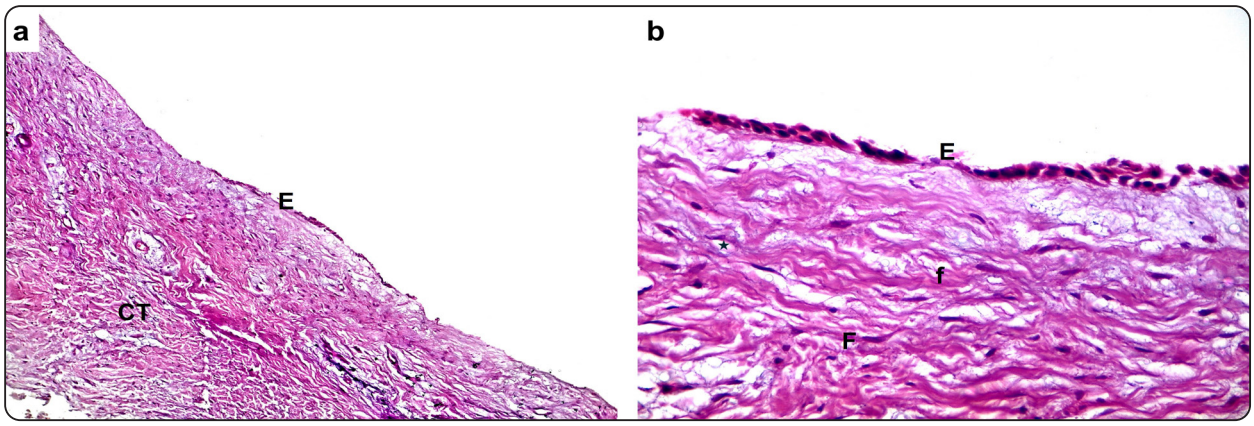

Fig. (1) A photomicrograph showing H&E-stained sections of normal DF revealing epithelial lining (E), surrounding CT (CT), fibroblasts (F), blood vessel (star), delicate collagen fibers (f). (Orig. Mag. (a) × 100; (b) × 400).

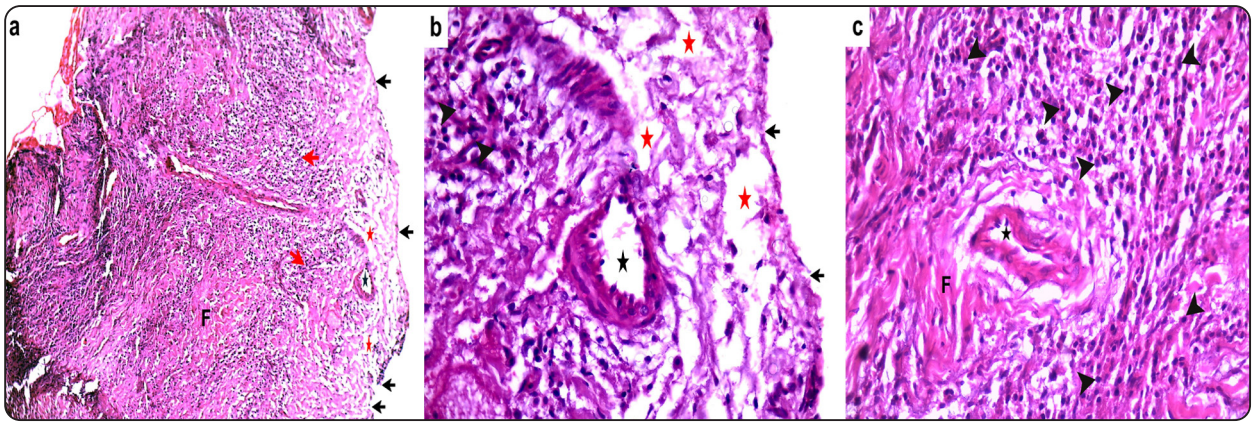

Fig. (2) A photomicrograph showing H&E-stained sections of inflamed DF revealing epithelial atrophy with epithelial fragmentation in some areas, atrophic epithelium (black arrows), mesenchymal inflammation (red arrows), congested blood vessels with thickened wall (black stars), spongiosis (red stars), fibrotic areas (F) & inflammatory cells (black arrowheads) (Orig. Mag. (a) × 100; (b, c) × 400).

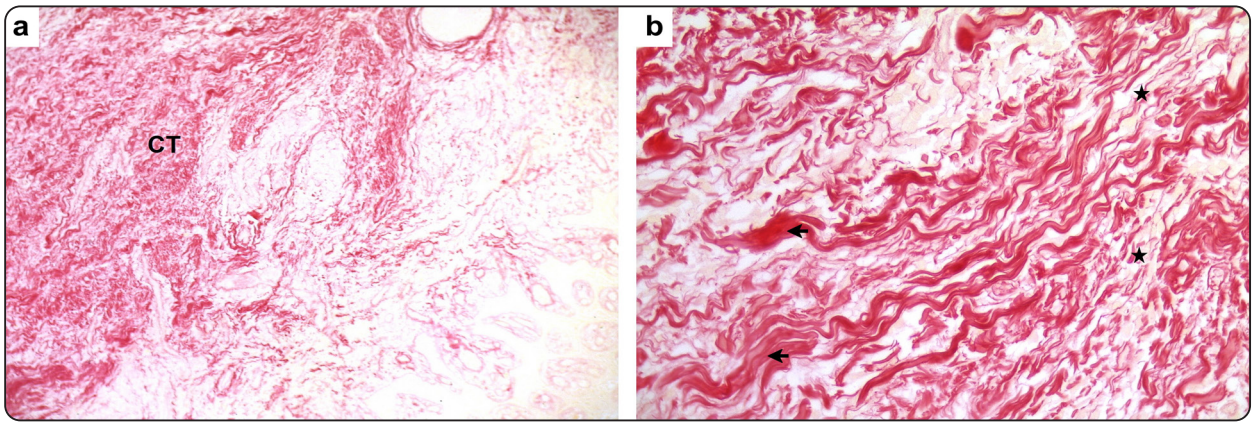

Fig. (3) A photomicrograph showing Picrosirus red stained sections of normal DF revealing CT (CT), thick collagen bundles (arrows) & loose delicate fibers (stars) (Orig. Mag. (a) × 100; (b) × 400).

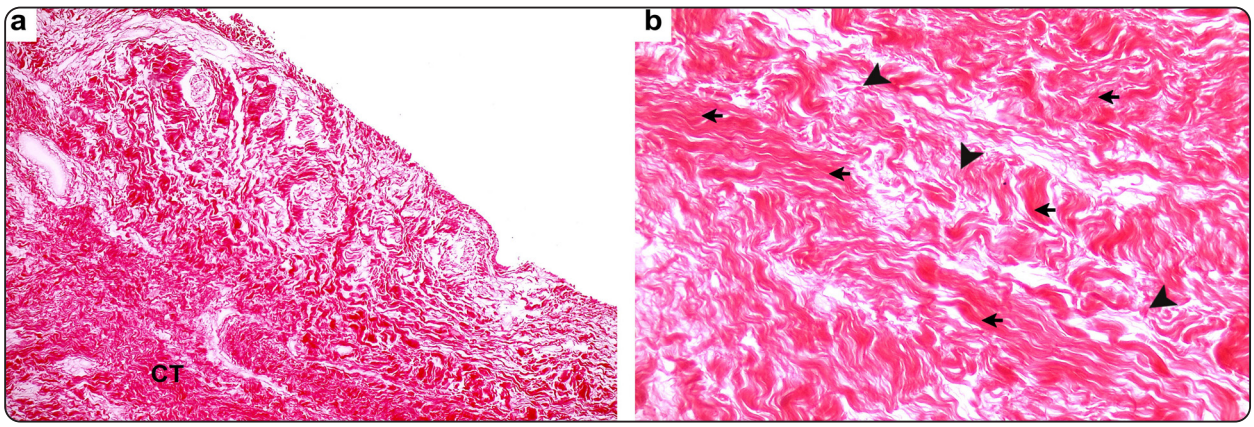

Fig. (4) A photomicrograph showing Picrosirus red stained sections of inflamed DF revealing CT (CT), thick collagen bundles (arrows) & irregular collagen bundles (black arrowheads) (Orig. Mag. (a) × 100; (b) × 400).

TABLE (1) Mean ± SD of area % of collagen fibers amount in Picrosirius Red stain as well as area % of interstitial edema and number of inflammatory cells in Hematoxylin and Eosin stain.

Groups / Parameters	Collagen Fibers (Area %)	Interstitial Edema (Area %)	Number of Inflammatory Cells
Normal DF	40.743 ± 3.087	5.372 ± 2.015	19.00 ± 5.963
Inflamed DF	71.753 ± 2.809 *	22.199 ± 2.721 *	495.80 ± 52.368 *
F- Value	552.043	246.971	818.365
P- Value	0.000	0.000	0.000

Values expressed as Mean ± SD, * Significant VS Normal. Superscript (*) indicates significant difference at p-value ≤ 0.001.

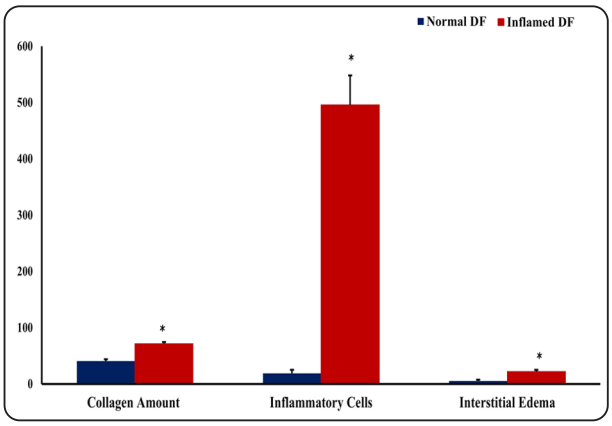

Fig. (5) A histogram showing mean ± SD of area % concerning collagen fibres amount as well as interstitial edema and number of inflammatory cells of normal and inflamed DF. Bars with superscript (*) indicates significant difference at p-value ≤ 0.001.

DISCUSSION

DF is a dynamic mesenchymal tissue critical for tooth development and eruption [19]. However, inflammatory stimuli can trigger significant alterations within DF, disrupting its cellular composition, ECM organization and subsequent odontogenic cyst development [20]. This study aimed to clarify these changes through a comparative histological, histochemical and histomorphometric analysis of normal and inflamed human dental follicles.

In the herein study, we assessed the structural characteristics of both normal and inflamed human dental follicles. In both groups, we examined the morphology of the epithelial lining and the connective tissue stroma. Specifically, we evaluated cellular composition, collagen fiber distribution using picrosirius red staining, and the presence of interstitial edema. This comparative approach aimed to clarify the alterations occurring within the DF in response to inflammation.

Our histological observations in the normal DF specimens aligned with the established histological descriptions reported by Haidry et al. [21]. On the contrary, the epithelial lining observed in inflamed DF in our study frequently exhibited atrophy, appearing thinner and sometimes discontinuous compared to the well-defined layers seen in normal follicles. This observation corroborated the findings of Esen et al. [22]. This finding suggests a potential disruption of the normal epithelial architecture in response to the inflammatory environment. Such atrophy could be attributed to the direct effects of inflammatory mediators, such as cytokines and enzymes, which can damage epithelial cells [23].

Furthermore, the altered microenvironment within the inflamed follicle, characterized by increased vascular congestion and cellular

infiltration, might compromise the nutritional supply and support necessary for maintaining a healthy epithelial lining [24]. This observation stands in contrast to the results of Li et al. [25] who observed epithelial hyperplasia or proliferation in inflamed follicles. This discrepancy underscores the complex and multifaceted nature of epithelial responses to inflammatory stimuli, which can vary depending on the specific stage and characteristics of the inflammatory process

Our histochemical analysis using picrosirius red staining revealed a distinct contrast in collagen fiber distribution between normal and inflamed dental follicles. In normal follicles, the connective tissue exhibited a delicate network of predominantly thin collagen fibers, with only sparse bundles of thicker fibers with parallel orientation. Those findings aligned with Naik et al. [26]. This arrangement likely contributes to the tissue's flexibility and supports the dynamic cellular interactions necessary for normal development [27].

Conversely, inflamed dental follicles displayed a notably different pattern, characterized by a substantial increase in collagen density and a more haphazard organization of fibers which also alignede with Naik's observations [26]. The presence of abundant, often thicker, and irregularly arranged collagen suggests an active process of collagen synthesis and remodeling in response to the inflammatory reaction [28]. This altered collagen architecture may compromise the normal tissue homeostasis and potentially influence subsequent pathological events within the follicle and consequent odontogenic cyst formation [26].

The quantitative histomorphometric analysis in our study provided conclusive evidence supporting qualitative histological observations. The highly significant increase in the area percentage occupied by collagen fibers within the inflamed dental follicles offers a precise measure of the enhanced collagen deposition observed with picrosirius red staining.

This substantial accumulation of collagen is likely a consequence of activated fibroblasts responding to the inflammatory insult by increasing their synthetic activity[29]. This altered collagen density and organization, as revealed by both the staining and the quantitative data, may have implications for the tissue's mechanical properties and could potentially influence the trajectory of any associated pathological development [30].

Furthermore, the statistically significant surge in the area percentage of interstitial edema in the inflamed group provides a quantifiable measure of the fluid extravasation characteristic of inflammation. This edema contributes to tissue swelling and can disrupt the normal spatial relationships between cells and matrix components [31]. The observed increase in interstitial edema within the inflamed dental follicles consistent with the established knowledge of the inflammatory response, in which vasodilation and elevated vascular permeability result in fluid extravasation into the surrounding tissues [32].

This fluid accumulation can contribute to tissue swelling and may further disrupt the local microenvironment, potentially affecting cellular function and matrix organization [33]. The presence of edema in the inflamed dental follicle suggests an active vascular response to the inflammatory stimuli, which could be triggered by various factors such as bacterial infection associated with pericoronitis [34] or mechanical pressure from an impacted tooth [35].

The concurrent and highly significant elevation in the inflammatory cell count directly corroborates the histological evidence of a robust inflammatory infiltrate[25]. These infiltrating cells are the primary drivers of the inflammatory process, releasing a cascade of mediators such as mast cell degranulation that contribute to tissue damage, matrix remodeling, and the recruitment of further immune cells[36]. The strong statistical significance across these parameters underscores the profound impact of inflammation on the microstructural composition of the dental follicle.

CONCLUSION

In conclusion, our study provides compelling histological and histochemical evidence of significant alterations within the dental follicle in response to inflammation. Compared to the well-organized epithelial lining and delicate collagenous stroma of normal dental follicles, inflamed follicles exhibited epithelial atrophy, a substantial increase and disorganization of collagen fibers, and a significant presence of interstitial edema and inflammatory cell infiltration.

These findings, further substantiated by quantitative histomorphometric analysis, underscore the dynamic and destructive remodeling processes that occur within the dental follicle during inflammation. These alterations in the tissue microenvironment may have implications for the clinical behavior of impacted teeth and the potential development of associated pathologies. Further research, potentially incorporating molecular analyses, is warranted to fully elucidate the mechanisms driving these changes and their long-term consequences.

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Conflict of interests

The authors have no conflict of interests to declare.

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