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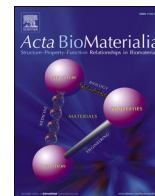
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Review article

Physiology of dentinogenesis and pathophysiology of dentinogenesis imperfecta: how does it affect dentin structure and biomechanics?



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ABSTRACT

Although the formation, structure, and biomechanical properties of dentin are well-studied, the impact of dentinogenesis imperfecta (DI) on dentin tissue structure and properties remains poorly understood. This perspective paper aims to summarize current knowledge on dentin and discuss the DI-related effects on dentin biological, structural and biomechanical properties. Macroscopically, DI is easily recognizable in clinics, characterized by a blue/gray appearance of the teeth and rapid pulp chamber obliteration after tooth eruption. Microscopically, the porous structure of healthy dentin is absent; tubules are scarce, and some areas are completely atubular. Clinically, the enamel of DI-affected teeth detaches quickly from the dentin, leading to rapid wear of the dentin under normal masticatory forces. The biomechanical properties of DI-affected dentin are poorly studied and challenging to compare with those of healthy dentin. Various factors, such as hydration, tissue heterogeneity, and the scale of analysis, significantly influence the measurement of these properties. An in-depth exploration of the micro- and nano-structure, as well as the biomechanical properties of dentin affected by DI, would provide a better understanding of the behavior of this diseased tissue in patients. This, in turn, would enable the adaptation of dental restoration treatments to meet the mechanical constraints specific to the oral environment.

Statement of significance: This manuscript presents a comprehensive perspective on dentin as a biological material. We detail the dentinogenesis processes that govern healthy dentin formation and its microstructural development. We link dentin's functional biomechanics to its hierarchical organization. The work highlights how Dentinogenesis Imperfecta disrupts dentin structure at multiple scales, resulting in altered mechanical properties. Importantly, it draws direct connections between biological alterations and functional consequences, offering critical insights into structure–function relationships in mineralized tissues. This perspective bridges developmental biology, genetic diseases, and a biomaterials-based approach.

1. Introduction

Dentin forms the core of teeth which surrounds the central pulp soft tissue. In the crown, it is located beneath enamel while covered by cementum in the root. It is a mineralized tissue formed by odontoblasts. These cells first secrete a non-mineralized matrix mainly composed of

type I collagen. This matrix is subsequently invaded by minerals notably hydroxyapatite (HAP) crystals. During the deposition of dentin, the odontoblast steps forward leaving in the dentin matrix an apical process embedded within a dentinal tubule [1] (Fig. 1). These cells process within their tubules would play a critical role in the transmission of physical or chemical stimuli to odontoblasts in connection with the

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dental pulp innervation [2,3].

The microstructure of dentin highly influences its mechanical properties. Due to the preferential orientation of tubules and collagen fibril network, the tissue displays an anisotropic behavior (Fig. 1). This is especially important for mechanical properties, as toughness is higher when cracks are propagating perpendicular to the fibrils i.e. along the tubules [4].

The most common genetic pathology affecting this tissue is Dentinogenesis Imperfecta (DI), affecting 1/6000-8000 individuals [5]. This pathology disturbs the dentin in the composition of extracellular matrix proteins, structure and mineralization [6–8]. These modifications result in structural alterations detectable macroscopically and on dental X-rays. Patients' teeth exhibit a blue-gray color and morphological appearance that are recognizable to clinicians. This condition also leads to a decrease in mechanical properties, as patients experience rapid tooth wear, ending in completely eroded crowns and ultimately premature tooth avulsion [9].

While the physiological mechanisms of dentin formation are reasonably defined, the pathophysiological mechanisms of DI are only partially understood. This work aims to summarize the different aspects of dentinogenesis from biology to biomechanics in healthy and DI dentin, identify gaps in the current knowledge on DI, and propose promising research perspectives/prospects.

2. Dentinogenesis

2.1. Healthy dentin

The development of the tooth starts with a sequence of interactions between ectodermal and mesenchymal tissues, playing a crucial role in morphogenesis and differentiation [10,11]. These interactions play a role in the cellular differentiation of odontoblasts, which are the cells involved in the synthesis, secretion, and reabsorption of matrix components of the dentin.

In the early stages of tooth development, cells located at the periphery of the dental papilla differentiate into pre-odontoblasts, which undergo their transformation into secretory polarized cells. At their apical pole, they actively secrete an extracellular matrix rich in type I collagen, non-collagenous proteins (NCP) and proteoglycans [12]. This matrix forms a non-mineralized tissue known as predentin. Simultaneously, as odontoblasts continue to deposit dentin matrix, they leave

behind cellular extensions which are embedded in the entire thickness of the dentin inside tubules [13] (Fig. 2). Consequently, the produced dentin is named intertubular dentin. Within the predentin matrix, calcium-phosphate minerals are deposited, initiating mineralization at the mineralization front. These ions then organize into hydroxyapatite (HA) crystals associated with collagen fibers, in a highly structured manner. The mineralization front progress centripetally, imparting its strength and stiffness to the dentin. In summary, dentin is a hierarchically organized nanostructured biological composite formed through matrix-mediated biomineralization [14], in which cellular components play a crucial role in controlling mineral deposition. After its formation, dentin is a non-vascularized tissue, including entrapped odontoblast process at the periphery of the pulp.

The organic matrix of both predentin and dentin is mainly composed of type I collagen (90 %), which essentially is a triple helix composed of two $\alpha 1$ chains (encoded by *COL1A1*) and one $\alpha 2$ chain (encoded by *COL1A2*). Non-collagenous proteins (NCPs) are also essential components of the dentin and predentin matrix (Table 1 – human dentin matrix protein composition).

This organic matrix serves as a scaffold for the tissue mineralization. Several studies have demonstrated that type I collagen creates a dynamic, three-dimensional scaffold that facilitates organized mineral deposition [15–18]. Present in predentin and dentin compartments, the NCPs also control the mineralization process. Being either inhibitors or activators in the crystallite initiation and growth, they are differentially distributed in the non-mineralized predentin and mineralized dentin compartments.

It appears that the mineralization of collagen occurs through pre-nucleation clusters formed by calcium and phosphate ions sequestered by NCPs. These clusters then self-assemble in relationship with the collagen fibers, notably inside the fibrils aligning within the gaps of the triple helices [19]. These non-collagenous and collagenous proteins are linked by specific covalent or non-covalent bonds, forming an architecture scaffold. The predentin matrix is organized around the secretory pole of the odontoblasts toward the cell process, which prefigures the microstructure of dentin [20,21].

Among the constituents of dentin, DSPP (dentin sialophosphoprotein) is a SIBLING that plays a crucial role in dentinogenesis [22]. The DSPP is a 940 amino acid polypeptide, which is cleaved into two NCPs: dentin sialoprotein (DSP), and dentin phosphoprotein (DPP) [23,24]. In porcine species, three cleavage products (DSP, DPP, and DGP (dentin

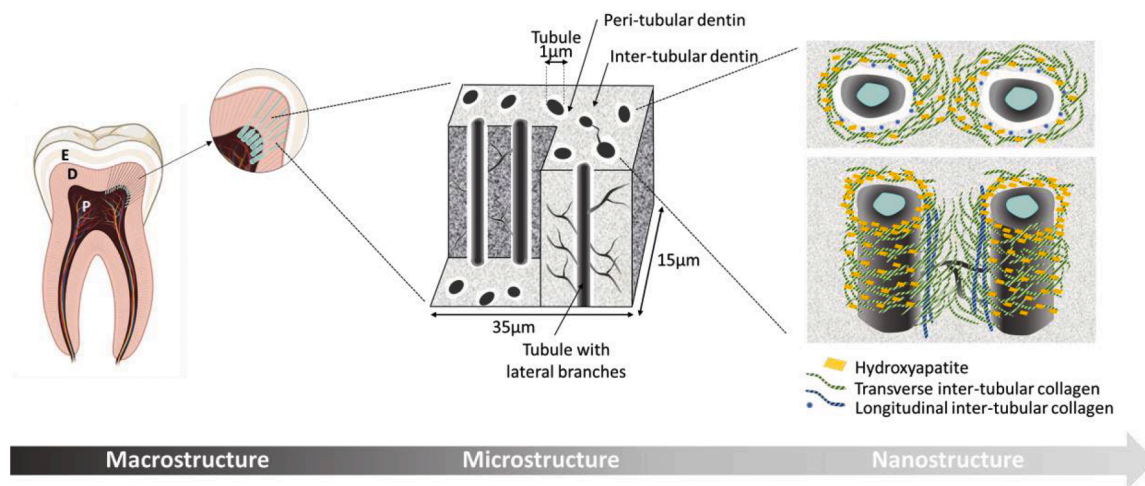


Fig. 1. Multiscale representation of the tooth from macro to nanostructure. Macroscopic view of the tooth, illustrating its main anatomical components (*E*=enamel, *D*=dentin, *P*=pulp); Microscopic view, showing tubules containing odontoblastic processes and the highly mineralized peritubular dentin, embedded in the inter-tubular dentin. Lateral branches connecting tubules can be observed; Nanoscale representation, depicting the interlocking between type I collagen fibers (in blue and green) and hydroxyapatite crystals (in yellow). Collagen fibers are organized in planes perpendicular to the tubule axis, except near the peritubular dentin, where their orientation is along tubules. Hydroxyapatite crystals are found around and within the collagen fibers. Inside the tubule, the odontoblastic process can be observed, with its cell body located within the dental pulp.

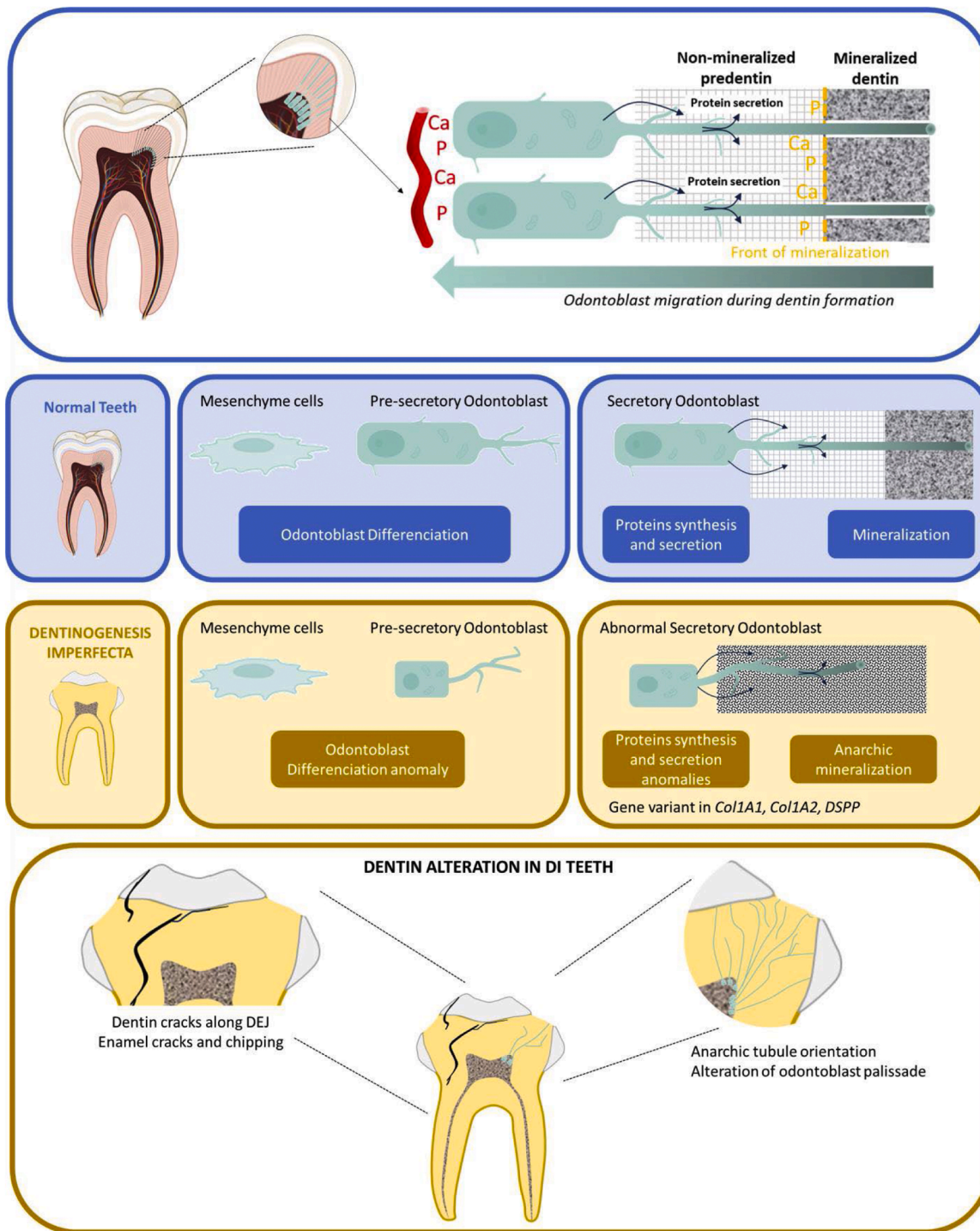


Fig. 2. Representation of physiological dentinogenesis process (blue part) and pathological one in DI (yellow part).

In physiological context, after odontoblast differentiation, cells become elongate and form a palisade. Odontoblasts secrete the non-mineralized dentin extra cellular matrix called predentin. As predentin is deposited, the odontoblastic body moves forward, leading to the elongation of the odontoblastic process, which remains anchored at the dentin enamel junction (DEJ). In a second phase, calcium and phosphate ions coming from blood vessels are released at the level of the odontoblastic process and organize into HA crystals around collagen fibers, resulting in mineralized dentin. A layer of predentin is always present near the odontoblastic body. In pathologic context of DI, the mesenchymal cell differentiates into abnormal odontoblast. Odontoblasts are cuboid and not well aligned in palisade. Alteration of proteins synthesis and dentin mineralization are observed.

DI tooth is characterized by enamel chipping, pulp calcification and dentin hypomineralization. Macroscopically, cracks are observed in the dentin, propagating into the enamel. The number of tubules is reduced and present anarchic orientation, with some being obstructed. The network of lateral branches is disorganized.

glycoprotein)) have been identified, but the presence of DGP in humans remains uncertain [25].

It appears that DSP is mainly involved in regulating the initiation of dentin mineralization. DPP is the most abundant non-collagenic

extracellular matrix component in dentin. It contains binding and affinity domains for HA crystals and Ca^{2+} ions, as well as for collagen fibers; DPP, also called phosphophoryn, is the mostly phosphorylated proteins in the vertebrates (>50 % of its molecular weight is due to its

Table 1
Composition of human dentin extracellular matrix [12,21].

	Family	Protein
Collagens	Collagens	Fibrillar collagens: type I collagen (90 % of matrix proteins), with 89 % of type I and 11 % of type I trimer
Phosphorylated NCP	SIBLINGS	DSPP (DSP and DPP), DMP1, BSP, OPN, MEPE
	SLRP	Decorin
Non-phosphorylated NCP	Enamel proteins	Amelogenin - Ameloblastin
	Other bone proteins	Osteocalcin, osteonectin
	Proteoglycans	CS/DS PGs: (decorin, biglycan) KS PGs: (lumican, fibromodulin, osteoadherin)
	Growth factors	FGF2 TGFβ1 BMPs ILFG1 - ILFG2 PDGF
	Serum-derived proteins	Albumin, α2-HA glycoprotein, Fetuin
Enzymes	Alkaline Phosphatase Acid phosphatase Serine proteases, Collagenases: MMP-1, -8, -13 Gelatinases A: MMP-2 Gelatinases B: MMP-9 Stromelysin 1: MMP3 Membrane-type MMP: MT1-MMP Enamelysin: MMP-20 ADAM - ADAMTS proteases Matrix phospholipids	
Lipoproteins		

NCP: Non-collagenous proteins; SIBLINGS: Small integrin-binding ligand N-linked glycoproteins; SLRP: Small Leucine Rich Proteoglycan; DSPP: dentin sialophosphoprotein; DPP: dentin phosphoprotein; DSP: dentin sialoprotein; DMP1: Dentin Matrix Protein 1; BSP: Bone Sialoprotein; OPN: Osteopontin; MEPE: Matrix Extracellular Phosphoglycoprotein; SLRP: Small Leucine-Rich Proteoglycans; CS/DS: Chondroitin Sulfate/Dermatan Sulfate; PGs: Proteoglycans; KS: Keratan Sulfate; FGF2: Fibroblast Growth Factor 2; TGFβ1: Transforming Growth Factor Beta 1; BMPs: Bone Morphogenetic Proteins; ILGFI - ILGFII: Insulin-like Growth Factor I & II; PDGF: Platelet-Derived Growth Factor; MMP: Matrix Metalloproteinases; ADAM: A Disintegrin And Metalloproteinase; ADAMTS: A Disintegrin And Metalloproteinase with Thrombospondin motifs.

phosphate groups). Secreted at the mineralization front, it acts as a transporter of phosphates and calcium to the collagen fibers, thus contributing to the maturation of mineralized dentin [26]. Additionally, its specific interaction with type I collagen promotes the structured organization of hydroxyapatite crystals, influencing their orientation and growth within the extracellular matrix. HA crystals are found in a carbonated form (carbonated-HA). Indeed, dentin can incorporate many elements into HA, with the most abundant being Mg, Zn, Sr, and Fe [27]. In extracellular fluids, the concentrations of calcium and phosphate are higher than those necessary for the spontaneous precipitation of carbonated-HA, but matrix proteins regulate ionic activity and prevent the uncontrolled formation of crystals [14].

To conclude, the extracellular matrix of dentin is primarily composed of collagen, with type I collagen being the dominant component. Other collagens, such as types III, V and VI contribute to fibril assembly and structural organization and could be found in smaller amounts. Additionally, NCPs represent about 10 % of the organic matrix but play crucial roles in mineral nucleation, crystal growth, hydroxyapatite crystallinity, and inhibition of amorphous mineral deposition. These NCPs, synthesized by odontoblasts, are distributed between and within collagen fibrils and accumulate along dentinal tubule walls, contributing to dentin mineralization [18,21,28–30].

Intertubular dentin (ITD) shows an important porosity due to its dense network of dentinal tubules with various diameters. This network

is composed by principal tubules giving rise to smaller lateral tubules branched between them. Tubules are commonly described as an elongated lumen surrounded by a highly mineralized dentin called peritubular dentin (PTD), more mineralized than the ITD. The PTD is in fact an evolutive tissue which is continuously secreted, explaining that its reported thickness in the literature varies [31].

PTD does not originate from the transformation of a PTD predentin into dentin, but rather from the adsorption along the lumen of the tubules of an amorphous matrix, also named lamina limitans which may be secreted by the odontoblastic processes within dentin tubules and/or originate from serum (dentinal fluid) [32]. This dentinal fluid, an ultrafiltrate of plasma composed mainly of water, ions, and proteins [33] flows within the tubules under pressure gradients and helps preserve the structural integrity of the dentin, especially under functional loading. This fluid plays a key role in maintaining dentin's hydration and contributes to its mechanical properties, particularly its viscoelastic, post-yield, and resistance to fracture properties.

Between the tubules, the ITD is a composite tissue made up essentially of collagen fibrils and hydroxyapatite crystals in roughly equal proportions by volume [34].

At the boundary between the enamel and dentin lies a wavy interface called the dentin-enamel junction (DEJ); this zone enhances the adhesion between enamel and dentin while improving the dissipation of mechanical stresses. Its scalloped structure strengthens the teeth by preventing crack propagation and absorbing masticatory forces [35,36]. Dentin porosity is heterogeneous throughout the dentin thickness with a gradient from enamel to pulp. Close to the enamel, a first layer called “mantle dentin” is commonly described as atubular. In fact, in this area, some branches are present with a small diameter (500 nm) [37]. The second layer, called “circumpulpal dentin” is composed of tubules measuring, at the periphery, around 1 μm in diameter [38] and linked to the branches of mantle dentin. Circumpulpal dentin shows a gradient in the tubule diameter from pulp to mantle dentin. Close to the pulp cavity, tubules are larger (to around 2.5 μm) and more abundant.

In addition, lateral canals, also called lateral branches, are connected to the main tubule. Their network also has a gradient in density, with more lateral canals close to the DEJ [37,38]. Gradients of “nanostructure” (what we call nanostructure here is the sub-tissue scale, corresponding to collagen fibril/HAP crystal imbrication) are also observed. Collagen fibrils, which initially (close to enamel) show no preferential orientation, gradually organize themselves in planes perpendicular to the tubule axis. In these planes, the fibrils appear to be isotropically organized [39].

Knowledge about micro- and nanostructure is well established, through various methods of investigation. Confocal laser scanning microscopy [38], X-ray ptychotomography (PXCT) [40] or FIB-SEM are probing 3D morphology at increasingly fine scales, which holds great promise for advancing our understanding of dentin tissue. The main

Table 2
Healthy dentin main structural characteristics.

Tissue scale	Volume ratio (%)	Diameter (μm)	Density (nb/mm ²)
Tubule lumen	1–22	0.8 - 2.5	10 000 – 70 000
Lateral branches	unknown	0.2 - 0.5	unknown
PTD	3–12	0.45 - 1.2	NA
ITD	60–96	NA	NA
Subtissue scale in ITD	Volume ratio (%)	Size (nm)	Density (nb/mm²)
Collagen fibrils	50	Diameter: 100 Periodic banding: 67	NA
HAP crystals	50	Length: 50–100 Width: 20–40 Thickness: 2–5	NA

NA: Not applicable ; tissue scale [38,67,107] sub-tissue scale [39,108,109].

figures characterizing healthy dentin are summarized in Table 2.

2.2. Dentinogenesis imperfecta

Dentin may be altered, and the most common genetic pathology is the dentinogenesis imperfecta (DI). DI is a rare disorder that affects the dentin of both primary and permanent teeth, with more severe phenotype in primary teeth [9]. This condition is usually described as a dentin hypomineralization. DI may be isolated due to variant of *DSPP* gene [41] or part of osteogenesis imperfecta (OI) [42]. OI is a heterogeneous skeletal dysplasia characterized by bone fragility and deformity with various degrees of severity [43]. Variants in type I collagen genes (*COL1A1* and *COL1A2*) are the most frequent cause of OI (85 %). Other OI rarer forms have been identified due to variants in genes encoding proteins involved in bone mineralization (*IFITM5*, *SERPINF1*), collagen modification (*CRTAP*, *LERPE1*, *PPIB*), collagen folding and crosslinking (*SERPINH1*, *FKBP10*, *BMP1*, *KDELR2*) or in osteoblast function and differentiation (*SP7*, *TMEM38B*, *WNT1*, *CREB3L1*, *SPARC*, *MBTPS2*) [44]. *TENT5A*, *MESD*, *CCDC134*, *P4HB*, *PLOD2* and *PLS3* are also reported to be associated with rare OI forms [42]. Depending on the gene, DI is not consistently observed in patients with OI. Certain pathogenic variants may not be associated with a dental phenotype, suggesting variable function during dentinogenesis. Conversely, the presence of DI may serve as an indicator of tissue-specific gene expression within the dental mesenchyme, implying a functional role of the corresponding gene during dentinogenesis.

Clinical phenotypes of DI vary from mild to severe forms (Fig. 3). The moderate form is the most frequent observed phenotype, followed by the mild form. The severe DI was only described in the Brandywine isolate [45].

Affected teeth appear with an opalescent bluish/brownish discoloration. The enamel-dentin junction is described as brittle, with enamel chipping, and exposing dentin in the oral cavity. At the microscopic level, cracks may be observed in the remaining enamel and in the dentin. Dentin exposed to oral milieu shows a rapid and severe abrasion despite

normal mastication. Radiologically, the appearance of teeth is pathognomonic, including significant cervical constriction with bulbous crowns, short and thin roots and pulp obliteration. Prior to tooth eruption, very large pulp chambers may be observed. Once tooth has erupted, a rapid pulp obliteration is observed, which can lead to the complete disappearance of the root canal and pulp chamber. Periapical abscesses may occur without any carious lesion [9]. One hypothesis proposed is the entrance/penetration of bacteria into dentin through the abnormal cracks.

While the clinical and macroscopic aspects are relatively well known, the underlying mechanical properties are not fully understood. Main microstructural characteristics of DI dentin (summarized in Table 3) is less described than normal dentin. At the microstructural level, the DEJ appears normal to less scalloping. Under DEJ, the mantle dentin shows the presence of branches as in a healthy condition [6]. By contrast, circumpulpal dentin is severely affected: an abnormal tubular network is reported [6,46–48], as well as “laminated mineralized dentin” and areas with large canals that have no counterpart in healthy dentin [6]. At the sub-tissular level, collagen fibrils exhibit the same banding length than in normal dentin (normal banding of collagen is known to be 67 nm), and a bimodal diameter distribution shows peaks around 30 nm and 60 nm [48]. If DI dentin appears to be less mineralized on average than healthy dentin [49–51], it however seems to exhibit varied mineralization, with areas more or less mineralized (striations, abnormal channels) [6]. Kerbel reported that the crystallites in DI dentin are normal in size but less numerous than in healthy dentin [50], and Kinney observed that the crystals in DI dentin are mainly extrafibrillar [52].

DI-dentin is classically described as being hypomineralized. While Sabel reported that the Ca/P ratio may be similar in DI and healthy dentin [53], Kerbel observed a tendency for an increased Ca/P ratio in DI-dentin, along with a loss of both Ca and P, and a significant decrease in Mg [50]. Sabel confirmed lower levels of C and Mg, along with higher levels of O and Na in DI-dentin compared to healthy dentin. The increase in Na may reflect a mineralization defect [54]. Finally, Park measured

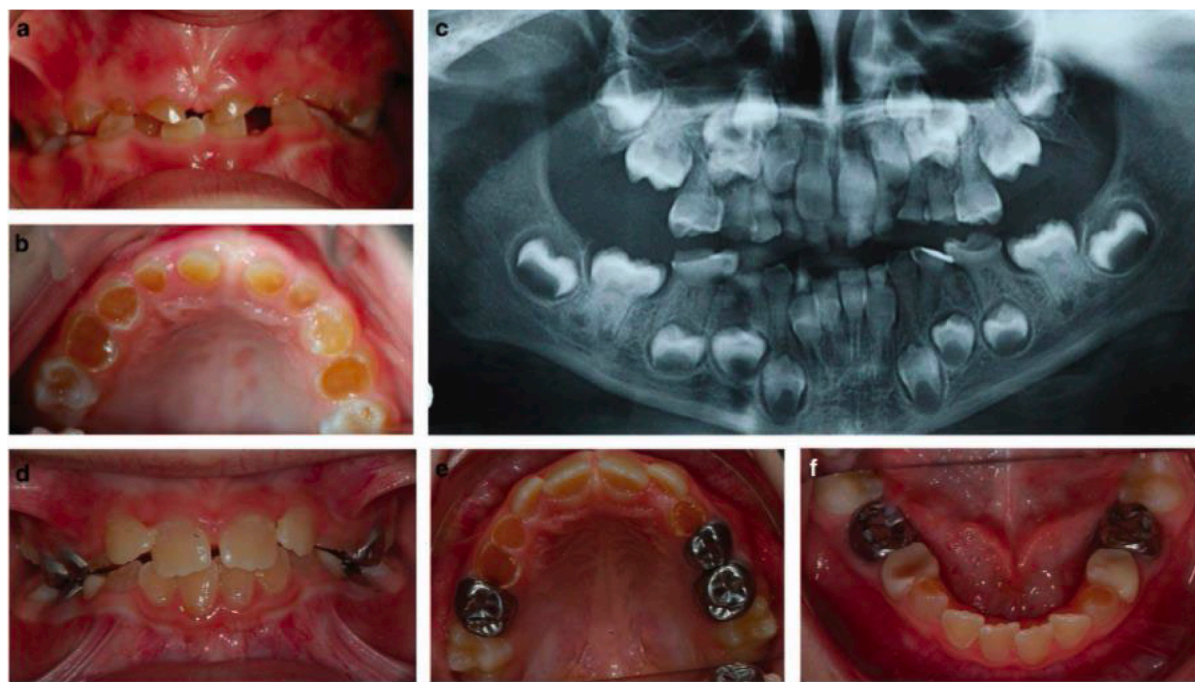


Fig. 3. Clinical phenotype of moderate dentinogenesis imperfecta in temporary teeth of 6-year-old boy (a-c) and permanent teeth at 8 years (d-f), primary teeth show brown opalescent discoloration, severe attrition, complete pulp obliteration of erupted teeth, bulbous crown, short root, periapical radiolucency without carie. Temporary teeth are more severely affected than permanent teeth. Reproduced with permission from [9].

Table 3
DI dentin main structural characteristics.

Tissue scale	Volume ratio (%)	Diameter (μm)	Density (nb/mm^2)
Tubule lumen	unknown	1.67 (lower than in healthy control dentin in *)	4000 (roughly 3 times lower than in the healthy control group) *
Lateral branches	unknown	unknown	unknown
PTD	unknown	unknown	NA
ITD	unknown	NA	NA
Subtissue scale in ITD	Volume ratio (%)	Size (nm)	Density (nb/mm^2)
Collagen fibrils	unknown	Diameter: 30 and 60 (bimodal distribution of diameter)* Periodic banding: 63 (same results as in healthy control group)*	NA
HAP crystals	unknown	Length: 100 Width: Thickness: 2 (reported to be identical to healthy dentin crystals in shape)**	NA

NA: Not applicable.

* data from [48] - on a limited amount of samples – teeth affected by DI associated with OI;

** [50].

that DI-dentin exhibits a lower overall mineral density [55]. Thus, DI dentin mineralization studies are contradictory. It is important to note that not all studies have been conducted at the same scale. However, the heterogeneity of DI-affected dentin may explain these different results. Studies at various scales, from the macroscopic level (whole tooth) to the nanoscopic level (interaction between collagen fibers and minerals), would provide a deeper understanding of microstructural disorders in DI dentin.

2.3. Mechanical properties of physiological and DI dentin

The mechanical properties of dentin are influenced by its intrinsic heterogeneity and anisotropy associated with its multiscale hierarchical structure and composition. As a result, mechanical properties depend on the structural orientation [56–64], and distribution [65] of various components in the tissue. In mineralized tissues, the elastic properties are mostly affected by the mineral content [66], while the post-yield properties are predominantly governed by collagen [67–69]. Fracture resistance, however, is related to both elastic and post-yield properties. In addition, dentin properties are also highly affected by its environment, in particular the hydration [56,58,70–72].

1. Elastic and hardness properties

Elastic properties in dentin describe dentin ability to withstand elastic (reversible) deformation and maintain structural integrity. These properties are mostly related to the mineral content and distribution.

The typical dentin Young's modulus is roughly 17 GPa at the macroscale level (see Table 4). Hardness is highly correlated with Young's modulus in dentin [73] and 0.7 GPa is a good assessment of the macroscale hardness of dentin. The yield stress of dentin is highly dependent on the type and direction of loading. Finally, 0.3 is generally considered to be a correct approximation of the macroscale Poisson's ratio for dentin.

It is known that peritubular dentin is more mineralized than intertubular dentin [74], and this anatomical difference influences

Table 4

Elastic and hardness properties of healthy human dentin [57,58,60,66,67,70–72,75,76,79,82,86,88,97,98,110–113].

	Dentin	Parallel to tubules	Perpendicular to tubules
E (GPa)	17 - [7.2 - 28.3]	16 - [8.6 - 23.2]	18 - [11.9 - 25.0]
σ_y (MPa)	130 - [22.4 - 223.5]	163.5	130 - [110.5 - 223.5]
H (GPa)	0.7 - [0.25 - 1]		
ν	0.3 - [-0.11 - 0.45]	0.3 - [0.29 - 0.3]	0.3 - [0.33 - 0.45]

E: Young's modulus; σ_y : Stress at proportional limit; H: hardness; ν : Poisson's ratio. In bold are values that can be considered as a good mean approximation, in brackets are the interval of all values found.

the mechanical properties of dentin at the microstructural level. Therefore, it has been demonstrated that human peritubular dentin is 30 % to 50 % stiffer than intertubular dentin [56,75]. Moreover, the hardness of intertubular dentin depends on the measurement position along the enamel-pulp direction: it is four times less hard near the pulp than near the DEJ [75]. Thus, depending on this position, peritubular dentin can be 2 to 16 times harder than intertubular dentin [63,75]. Regarding the influence of the dentin layer on the hardness, there was no influence of the position except for a drop in hardness values near the DEJ [76]. This drop in hardness and Young's modulus near the DEJ is explained by the presence of unmineralized interglobular dentin and increased tissue porosity due to the abundant branches [67,76,77]. In coronal dentin far from the DEJ the hardness of dentin drops when the tubular density increases which means that dentin is softer near the pulp [78]. All these observations on hardness may be applied to the study of Young's modulus since these properties are highly correlated in dentin with an essentially linear relationship [73]. Studying yield stress at the scale of intertubular and peritubular dentin becomes tough, and no such work has been conducted. Thus, yield stress values are only available for dentin at a higher scale. Collagen fibers are organized in planes perpendicular to the orientation of the tubules. Changes of orientation only lead to small anisotropy in elastic properties, with a local transverse isotropic behavior around tubules; thus, Young's modulus is 7 % higher in the direction orthogonal to the tubules (transverse direction, parallel to collagen fibers) than in the direction of tubules (longitudinal direction, perpendicular to collagen fibers) [58]. Moreover, this difference vanished in dried dentin [58]. Later studies reported a modulus 3 to 17 % higher in the transverse direction than in the longitudinal direction [57,59,79]. Conversely, some authors measured longitudinal modulus and hardness respectively 18 % and 11 % higher than the transverse ones [80]. The effect of tubule orientation on yield stress is not statistically significant [59, 80]. Finally, hydration also impacts the elastic properties of dentin. Dehydrated dentin is 15 to 30 % harder and stiffer [56,58,70,71]. Jameson measured no statistically significant difference in the yield strain between hydrated and dehydrated samples [71], but yield stress was 34 % higher on average in the dehydrated group. Rehydration restores almost completely the elastic properties of dentin [71,81].

2. Post-yield properties

Out of the elastic domain, the anisotropy of the dentinal tissue is stronger. Indeed, several studies report that the tensile, shear and flexural strengths of dentin is approximately 25 to 40 % higher when the crack propagates perpendicularly to tubules than when it

Table 5

Post-yield properties of healthy human dentin [63,66,70,71,82,108,114–117].

	Dentin	Parallel to tubules	Perpendicular to tubules
Ultimate strength (MPa)	150 - [33.9 - 326]	160 - [44.5 - 212]	130 - [48.7 - 210]
Strain to failure (%)	2 - [1 - 5.6]	2 % - [1.3 - 5.6]	

propagates parallelly to tubules [63,64,79,82–84]. In compression, this discrepancy is not as marked, approximately around 10 % [57] (Table 5).

Again, the mechanical properties of dentin vary depending on its anatomical location. Root dentin exhibits an ultimate flexural strength (UFS) 125 % higher than that of crown dentin, as determined by four-point flexural tests [85]. Furthermore, crown dentin itself presents internal heterogeneities, with the ultimate tensile strength of beams being up to 82 % higher in superficial dentin (near the dentino-enamel junction, DEJ) compared to deeper regions—26 % more than in middle dentin and 82 % more than in deep dentin [86]. This result shows the effect of the increase in peritubular dentin proportion with depth on the post-yield properties of dentin. DEJ should be studied separately since its structure is different from the rest of coronal dentin. In terms of mechanical strength, the ultimate tensile stress (UTS) of dentin from DEJ ($\sigma_f = 46.9 \pm 13.7$ MPa) is close from the UTS of mi-coronal dentin ($\sigma_f = 48.7 \pm 16.7$ MPa) [86].

As expected, hydration has a greater impact on post-yield properties than on elastic ones. A 26 % decrease in strain at failure has been observed, although this difference is not statistically significant [71].

3. Fracture and fatigue properties

The fracture properties of dentin have been well studied, and several values of toughness are summarized in Table 6. These values are highly dependent on the type of loading, its direction and on the used fracture mechanics theory.

It is important to note that toughness may designate different parameters. A toughness value is a critical value of a parameter above which crack propagation is triggered in the material. In linear elastic fracture mechanics (LEFM), the toughness is defined as the critical value of stress intensity factor (KI) or of energy release rate (GI). Several studies used this definition of toughness assuming that dentin behavior was mostly brittle with little non-linearity. However, some authors used Non-Linear Elastic Fracture Mechanics (NLEFM) to measure the toughness of dentin [72]. In this theory, toughness is defined as the critical value of the J-integral and should be equal to the critical value of GI from LEFM if samples behave as a perfectly brittle material. They demonstrated that toughness from NLEFM was 20 % higher than toughness from LEFM. This increase is associated to the consideration of post-yield properties in non-linear fracture mechanics which represent approximately 20 to 40 % of the toughness of dentin [59,60]. Therefore, using LEFM leads to significantly underestimating toughness and NLEFM should be preferred.

Dentin toughness is 40–45 % higher in the longitudinal direction

than in the transverse one, using tensile tests on notch-less prismatic samples [87]. Moreover, dentin toughness is 35–40 % higher when the crack propagates in the transverse direction rather than in the longitudinal one, although these results were obtained in elephants [61]. Several studies were conducted to compare the in-plane parallel (crack plane and direction of crack propagation both parallel to tubules) and the anti-plane parallel (crack plane parallel to tubules but direction of crack propagation perpendicular to them) but no significant differences were reported [59,61,63,87,88]. In terms of fatigue, the same kind of anisotropy was found with a threshold for crack propagation by fatigue higher in the direction parallel to tubules ($\Delta K_{th} = 1.03 \pm 0.1$ MPa $\cdot\sqrt{m}$) higher than in the direction perpendicular to tubules ($\Delta K_{th} = 0.83 \pm 0.1$ MPa $\cdot\sqrt{m}$) [62].

Heterogeneities of fracture properties at the scale of the peritubular cuffs are not easy to quantify. As a result, studies investigating heterogeneities of fracture properties are limited to studying the relationship between the distance to the pulpal chamber and toughness. Although data on root dentin in human teeth are lacking, a toughness 2.3 times higher in root dentin than in crown dentin has been measured in bovine incisors [89]. Data on variations in toughness within crown dentin are more abundant, with outer dentin being 54 % and 167 % tougher than middle and inner dentin, respectively [90]. This change is explained by the highest amount of brittle peritubular dentin closer to the pulp due to the increased tubular density. DEJ should be studied separately since its structure is different from the rest of coronal dentin. At last, the toughness of DEJ is $KIC = 2.45$ MPa $\cdot\sqrt{m}$ while the toughness of dentin 200 μm under the DEJ is $KIC = 2.2$ MPa $\cdot\sqrt{m}$ [91].

Finally, the effect of hydration on the fracture behavior is more pronounced than on the elastic behavior. Indeed, with a three-point flexure, the behavior of dehydrated dentin is almost exclusively elastic, making it significantly more brittle than the hydrated tissue [71]. This difference in behavior causes the toughness of dentin to decrease significantly when dehydrated, as shown in dentin from bovine incisors [72]. Since dentin is hydrated when under physiological conditions this difference is important to take into consideration when designing toughness measurements.

The biomechanical properties of dentin have therefore been extensively studied, with numerous tests that are not always comparable. These properties allow dentin to respond to the masticatory forces applied to it daily.

4. Effect of Dentinogenesis Imperfecta on mechanical properties

It is well-known that DI patients present enamel cracks, with enamel loss and exposure of the affected dentin [9,92]. Subsequently, abrasive forces lead to significant wear that can nearly erode the entire tooth, as illustrated in numerous case reports [93–96], resulting in a loss of coronal height and potentially causing the premature loss of the permanent tooth from the dental arch. In addition, the globular dental crown anatomy and the progressive pulpal obliteration observed in DI cases are likely to contribute to alterations in the tooth biomechanical properties. These macroscopic and clinical observations suggest a modification of the mechanical properties of dentin when it is affected by DI. Thus, clinically, DI is characterized by more numerous cracks in affected teeth. This means that fatigue and fracture properties are severely affected. However, due to the rarity of the pathology and the limited availability of samples, few tests have been conducted on this tissue. Here, we will synthesize the main findings on human dentin affected by DI regarding mechanical properties.

Hardness is the most extensively studied property of dentin in DI. In humans, studies consistently report a decrease in DI-affected dentin hardness compared to healthy dentin, regardless of whether DI is associated with a DSPP variant or OI. Ibrahim reported that the hardness of healthy dentin ($H = 560$ MPa) was significantly higher than that of DI-affected dentin, which measured $HI = 373 \pm 50$ MPa in cases associated

Table 6
Fracture and fatigue properties of healthy human dentin.

	Dentin	Parallel to tubules	Perpendicular to tubules
Fracture Toughness			
K_{IC} (MPa $\cdot m^{0.5}$)	2 - [1.1 - 4.8]	2.5 - [2.0 - 3.4]	1.5 - [1.1 - 1.6]
J_{IC} (J/m ²)	500 - [285 - 762]	500 - [285 - 762]	
Work of Fracture			
WoF (J/m ²)	500 - [254.9 - 900.0]	500 - [365.4 - 900.0]	250 - [254.9 - 270.0]
Stable crack growth parameters (R-curve)			
K_0 (MPa $\cdot m^{0.5}$)	2 - [1.1 - 2.7]	2 - [1.7 - 2.7]	1.2 - [1.1 - 1.3]
K_g (MPa $\cdot m^{0.5}/mm$)	1 - [0.2 - 2.2]	1 - [0.5 - 2.2]	0.5 - [0.2 - 1.1]
Fatigue Properties			
ΔK_{th} (MPa $\cdot m^{0.5}$)	1 - [0.6 - 1.2]	1 - [0.77 - 1.2]	0.6
m	25 - [23.11 - 26.7]	25 - [23.11 - 26.7]	24.16
C (mm/MPa $\cdot m^{0.5}$)	$[5.61 \times 10^{-10} - 1.58 \times 10^{-2}]$	$[5.61 \times 10^{-10} - 6.61 \times 10^{-5}]$	1.58×10^{-2}

Fracture Toughness [48,62,66,73,84,88,114] ; Work of fracture [78,82,87] ; Stable crack growth parameters [59,61,84] ; Fatigue properties [62,63,88,90].

with OI and HII = 370 ± 100 MPa when linked to a DSPP variant [86]. Similarly, microhardness values showed $H = 553 \pm 36$ MPa for healthy dentin, while DI dentin associated with a collagen disorder had a hardness of $H = 215 \pm 20$ MPa, representing a 2.57-fold reduction. In the same vein, Min reported that DI dentin linked to DSPP variants was 4.89 times softer [97,87], while Nutchoy observed a significantly lower hardness in DI associated with OI, with a reduction of approximately 2.5 times compared to healthy dentin [98].

The elastic modulus has also been investigated in a few studies, but the results remain contradictory. While DI dentin in mice appears to have a non-significant increase in Young's modulus [99], other studies report a decrease in Young's modulus in human DI dentin associated with a DSPP variant, with healthy dentin being approximately 1.5 times stiffer than the pathological one [100].

Beyond these works, data about the mechanical properties of dentin affected by DI remain scarce, highlighting the need for further studies. It is especially prejudicial since elastic and hardness properties are insufficient to understand the multiple fractures observed in pathological dentin. Thus, it is critical to investigate post-yield and fracture properties both at the macroscale and the scale of microstructural alterations in the tissue. Moreover, a significant variability in the results reported in the literature can be observed, which could be partially explained by the severity of DI, the localization of the test in dentin, the age of the patient, and the different mutations involved. To this day, genotype-phenotype correlations are unknown.

3. Conclusion and perspectives

Dentin is the central tissue of the tooth, providing most of its mechanical properties. Genes and proteins involved in its formation, and responsible for its final structure, are now quite well understood. However, while the genes associated with dentinogenesis imperfecta have been at least partially identified, the structural manifestations of their mutations are still not fully understood.

DI presents multiple abnormal clinical signs which vary in severity depending on patients. At clinical level, syndromic DI (associated with OI) and isolated DI (associated to DSPP variant) are similar. DI is classically described as an hypomineralization of the dentin. It is known that type I collagen is the major protein of dentin (90 %) and plays a structural and supportive role for carbonate-substituted HA crystals. Additionally, it is understood that the initiation of mineralization is mediated by phosphoproteins, particularly the DPP protein, which binds to and interacts with type I collagen. Mutations in the collagen genes, as well as in the DSPP gene, therefore, affect the formation and organization of these crystals. Moreover, depending on the type of mutation, collagen deficiencies can be either qualitative or quantitative [101]. However, no differences have been described yet depending on the type of the mutation, neither macroscopically nor microscopically. Finally, the dental phenotype(s) of rare OI forms remains unknown. It can be hypothesized that some genes exclusively involved in osteoblast differentiation may not affect odontoblasts and dentin, whereas genes encoding proteins associated with collagen (procollagen formation, post-translational modifications, etc.) could lead to DI-like defects. DI indeed leads to major alterations of the dentin microstructure: large atubular zones, mineralization heterogeneities and giant channels are reported. However, the number of reported cases is low, and the genes involved in the disorder are not known, leading to difficulties of interpretation of those defects. Moreover, the studied areas are also limited in terms of surface, and the tridimensional distribution of abnormalities is totally unknown. In conclusion, DI results in damages of microstructure and nanostructure that are not fully quantified and that change the biomechanical properties of the tissue, as shown at macroscopic level (changed of the color, the morphology and resistance of the DI teeth). Clinicians treating affected patients describe teeth of poor quality, more prone to fractures and wear. At the biomechanical level, the only investigated parameters were the macroscopic hardness and elastic properties. Despite a

noticeable dispersion of the results, the overall trend was a decrease in those properties in DI teeth which is consistent with the usual description of DI dentin as an hypomineralized tissue. However valuable, these conclusions present two major flaws. The first one is that, conducted at the macroscale, these experiments did not take the microstructural heterogeneities of the tissue into account. The second one is the absence of a quantification of toughness properties of the tissue which are necessary to understand the fractures observed clinically.

Current treatments include restorative technics [93,94,96,102], mainly pediatric crowns and resin-based composites for deciduous teeth, and prosthetic crowns for permanent teeth [93,95,102]. Integration of orthodontic treatments is often needed [93–96]. These procedures aim to enhance patients' esthetics, maintain the vertical dimension, and prevent cracks (and potential caries) from propagating and restoring the occlusion function. Although the use of restorative materials show promising results in mild forms of DI [93]. Dentin abnormalities and defects at the dentin–enamel junction may complicate bonding; therefore, sealed restorations are often preferable [103]. Patients with more severe disease frequently experience excessive wear and chipping [94, 102]. Then the cost and the repetitive procedures represent a major burden for the patients with severe forms.

Advances in the genetic diagnosis of rare diseases would give access to a better genotype-phenotype understanding and accurate characterization of gene-specific dental impairments. A better description and understanding of the underlying factors would allow for more specific treatment of our patients, adapting to the properties of the affected tooth, and tailoring the care and materials used accordingly. To achieve this, a better characterization of the samples from a clinical and genetic perspective, along with standardization of the morphometric and the biomechanical tests conducted, is necessary. The incoming morphological studies would benefit from microscale measurements allowing for a characterization at the scale of the disruption of the tissue's microstructure. A specific attention should be given to toughness tests which are mandatory to understand how DI causes affected teeth to fracture more easily. Ultrastructural studies with a specific emphasis on cells could also prove to be of paramount importance for enhancing our knowledge and comprehension of the disease.

In patients with OI caused by a collagen-related variant (*COL1A1*, *COL1A2*), bone tissue hyper-mineralization and accelerated mineralization kinetics are observed [104]. The mineral phase is also immature, with less well-organized crystals deposited within a defective collagen matrix [105]. This leads to increased bone fragility despite hyper-mineralization, as the tissue remains disorganized [106]. By analogy, similar mechanisms may be hypothesized in teeth affected by DI, where structural alterations could also be investigated across multiple scales. Indeed, large channels were already observed [6] on SEM, which do not correspond to any structure found in sound dentin, and additional ultrastructural changes at the level of mineral deposition could potentially be revealed by microCT, nanoCT, TEM or diffraction analyses.

Multidisciplinary teamwork, including dentists, geneticists, and biophysicists, is essential to design research efficiently using everybody's competences and speed the development of treatments up. Creating opportunities for researchers from different fields to share ideas could help advance the understanding of dentinogenesis imperfecta and improve the treatment of patients.

This article is dedicated to the memory of Prof. Muriel de la Dure-Molla, who passed away during the publication stages of this manuscript. Her human and scientific qualities were fundamental to complete this work.

CRediT authorship contribution statement

Margot Charlotte Riou: Writing – original draft, Methodology, Investigation. **Shangaya Touraivane:** Writing – original draft, Methodology, Investigation. **Ariane Berdal:** Writing – review & editing. **Valérie Cormier-Daire:** Writing – review & editing. **Benjamin PJ**

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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