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Enlightening Diagnosis and Differential Diagnosis of Dental Fluorosis—A Hidden Entity in a Crowd

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Abstract

Aim To provide diagnosis as well as differential diagnosis of dental fluorosis.

Background Dental fluorosis is a developmental defect of enamel, due to consecutive exposures of tooth during the formative stage of development to the higher concentration of fluoride, resulting in enamel with lesser mineral content and enhanced porosity. Several epidemiological indices have been utilized for diagnosis and assessment of dental fluorosis on the basis of clinical appearance. Fluorosis of the deciduous teeth occurs less commonly and is milder than that of permanent teeth.

Highlights The diagnostic difficulties are usually associated between fluorotic and nonfluoride opacities. A complete history of the clinical condition, teeth affected with specific areas, pattern of lesion, color and its method of detection are the few important diagnostic criteria for differentiating dental fluorosis from nonfluoride discolorations of the teeth.

Conclusion This review article has enlightened the diagnosis and differential diagnosis of dental fluorosis among various nonfluoride tooth discolorations. A correct diagnosis results in an appropriate and early management of dental fluorosis and plays an important role in oral epidemiology and public health.

Keywords
- dental fluorosis
- diagnosis
- differential diagnosis
- discoloration
- opacities

Introduction

Dental fluorosis is one the most frequent causes of intrinsic tooth discoloration.[1] It can be defined as "hypomineralization of the enamel, caused by the excess of fluoride in the environment surrounding the enamel during tooth formation."[2] It is a developmental defect of enamel, due to consecutive exposures of tooth during the formative stage of development to the higher concentration of fluoride, resulting in enamel with lesser mineral content and enhanced porosity. A daily fluoride intake of 0.05 to 0.7 mg F/kg/day or 1 parts per million (ppm) is the known recommended level. A daily intake of more than this safe level leads to an enhanced risk of dental fluorosis.[3]

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Fluorosis can be diagnosed through visual examination.[4] Numerous indices have been developed to diagnose, qualify, and quantify the clinical features of dental fluorosis.[2] Differentiation between fluorotic and nonfluorotic disturbances of tooth enamel is a prime necessity for evaluating an appropriate treatment plan. Dental fluorosis could be distinguished from other defects of the tooth enamel on the basis of enamel color, its distribution on the affected teeth, and the extent of unaffected enamel.

A dental surgeon should be able to differentially diagnose dental fluorosis among other similar nonfluoride tooth discolorations. Hence, this paper attempts to provide a review on diagnosis as well as differential diagnosis of dental fluorosis.

Diagnosis of Dental Fluorosis

Diagnosis of dental fluorosis is mainly through its appearance.[5] The diagnosis can be confirmed by reviewing the patient's medical history, with consideration given to systemic fluoride use.[4] The major known risk factors are high fluoride level in drinking water, infant milk formula reconstituted with fluoridated water, dentifrices and supplements. Knowledge of enamel fluoride content could assist in diagnosis of dental fluorosis. In normal enamel, the outer enamel contains much higher levels of fluoride, whereas decreasing level is seen in the inner enamel. Fluoride content of inner enamel is directly related to the availability of fluoride during the development of tooth, while the surface enamel is affected by posteruptive fluoride uptake.[5,6]

The diagnostic difficulties are usually associated with the assessment of mild dental fluorosis in primary teeth, owing to their thinner enamel and more whitish appearance than permanent teeth. Primary teeth reveal less dental fluorosis than their permanent successors. This is thought to be due to placental tissue performing as a barrier to fluoride in fetal blood, less fluoride consumption during the lactation period, or by smaller duration of enamel formation and maturation of deciduous teeth. An enhancement in the severity of dental fluorosis is observed from anterior to posterior teeth in both the dentition. The thickness of enamel verifies the severity of dental fluorosis.[5,7]

It was observed that later in the life enamel is completed, the greater is the severity of dental fluorosis. Enamel formation of cosmetically important teeth like permanent incisors and canines is normally completed by 7 years of age; thus, concern about development of dental fluorosis lies mainly in the initial 7 years of life.[5,7]

The Initial Signs of Fluoride Induced Enamel Changes Visible As[5,8]

1) Fine striae of accentuated perikymata evenly dispersed over the tooth surface and are simply viewed in tangential direction.
2) In more affected teeth, these fine lines turn broader and more prominent. With increase in severity, the irregular opaque areas merge until widespread areas appear chalky white.
3) Pitting may take place, either as tiny depressions, or as single or multiple, rounded holes representing a loss of outer most enamel surface.
4) In badly affected tooth, almost the complete enamel surface appears to be "corroded" and an extensive loss of enamel surface is seen.

Several epidemiological indices have been utilized for the diagnosis and assessment of dental fluorosis on the basis of clinical appearance. Dean in 1934 developed an index for determining the presence and severity of mottled enamel.[9] Dean was concerned about cosmetic deviations of the teeth; hence, he examined dental fluorosis under natural conditions, that is, on wet surfaces. In contrast, Thylstrup and Fejerskov were interested in the exact association between F exposure and the entire range of clinical as well as histological categories of dental fluorosis. It consists of 10 scores intended to characterize the degree of dental fluorosis involving buccal/lingual and occlusal surfaces.[9-12] The tooth surface index of fluorosis was given by Horowitz et al.[13] A separate score is provided for facial and lingual surface of anterior teeth and for occlusal, buccal and lingual surface of posterior teeth. It highlights cosmetic appearance by examining the teeth without drying them[9,11] (►**Tables 1** and **2**).[9-26]

Differential Diagnosis of Dental Fluorosis

Differentiation between fluoride and nonfluoride disturbances of dental enamel is a valuable diagnostic step toward an oral epidemiology and public health concern. Several investigators have suggested descriptive indices for classifying other similar nonfluoride enamel opacities (►**Table 1**). However, endemic prevalence, history of more than optimal fluoride ingestion in drinking water during tooth development, and its bilateral symmetrical pattern are considered as important differentiating features for diagnosing dental fluorosis (►**Fig. 1**)[6] (►**Tables 3–5**).[5,9,27-32]

Table 1 Various epidemiological indices for dental fluorosis[9]

S.No.	Indices	Year
1.	DI, Modified DI	1934, 1942
2.	Community index of fluorosis	1946
3.	Moller's index	1965
4.	TFI, modified criteria	1978, 1988
5.	TSIF	1984
6.	Fluorosis risk index	1990
7.	CFAI	1993
8.	Descriptive classification applied to quantify the enamel opacity	
	• Young's classification	1973
	• Al Alousi et al	1975
	• FDI index: DDE	1977
	• Murray and Shaw	1979

Abbreviations: CFAI, chronological fluorosis assessment index; DDE, developmental defects of enamel; DI, Dean's index; FDI, Fédération Dentaire Internationale; TFI, Thylstrup–Fejerskov index; TSIF, tooth surface index of fluorosis.

Table 2 Review of various studies using indices and newer methods for diagnosing dental fluorosis with their outcomes[10-26]

Author	Year	Indices/ newer method	Outcomes
Thylstrup and Fejerskov[12]	1978	TFI	TFI was valuable when biological effect of long-term exposure of young individuals to F^- is to be verified, whereas classical DI was not able to differentiate between dental fluorosis with 3.5, 6.0 and 21.0 ppm F^- in drinking water
Horowitz et al[13]	1984	TSIF	DI lacks sensitivity in areas with very high conc. of F^- in water; however, TSIF was sensitive enough for communities with different concentration of F^- levels and is easy to learn and less time consuming as well
Granath et al[14]	1985	DI and TFI	Scoring given by DI was not statistically significant between tablet and control group, whereas TF gave significant difference between these two groups and thus recommended for epidemiological surveys
Horowitz et al[15]	1986	TSIF	TSIF was able to distinguish between prevalence and severity of F^- in different group of communities with different conc. of F^- in drinking water
Burger et al[16]	1987	DI and TF	The two scoring systems shown almost similar prevalence but varied in the severity. TF is easier to use and will be selected for future studies
Clark et al[17]	1993	TSIF	Evaluated the aesthetic concern of children and their parents for dental fluorosis and found less children with aesthetic problems with TSIF "score 1," while children with TSIF "score 2–6" were more concerned about tooth color
Mabelya et al[10]	1994	DI and TF	TFI was able to present more dental fluorosis than DI in communities with low and moderate dental fluorosis; however, DI lacks accuracy to differentiate within low fluorosis score
Rozier et al[11]	1994	DI, TFI and TSIF	It was observed that TFI was more appropriate than other two indices for clinical and analytic epidemiological studies
Van Palenstein et al[18]	1997	TFI	In low-fluorosis communities, incisors and first molars are least affected; In high-fluorosis communities, increasing TF values from anterior to posterior teeth in both the arches, and proposed that later in the life enamel is completed, greater is the severity of dental fluorosis
Anya Vieira et al[19]	2005	VAS for dental fluorosis and TFI	Study showed better correlation between fluoride conc. and the VAS for dental fluorosis than between F^- conc. and the TFI. VAS can be valuable in dental fluorosis
Martins et al[20]	2009	Standardized digital photographic method	The photographic method offered a high specificity and positive predictive value, indicated its reproducibility and reliability for diagnosing dental fluorosis
Adelario et al[21]	2010	Simplified TFI	The simplified TFI proved appropriate for evaluating the prevalence of dental fluorosis in regions with high concentration of fluoride in drinking water/endemic fluorosis
Mohamed et al[22]	2010	DI and DDE	Comparatively little concordance was observed between DDE and DI in determining person prevalence of defects among children. DDE records F^- and non F^- defects, yet is relatively complex and time consuming
Pretty et al[23]	2012	A novel dual camera imaging system. DI and TFI	The novel dual camera imaging system using both polarized white light (PWL) and QLF imaging was suitable for determining enamel fluorosis in an epidemiological setting
Sudhir et al[24]	2012	TFI	The intraoral distribution of different degrees of dental fluorosis was associated with the completion of primary enamel formation in high fluoride communities with an exception of permanent first and second molars
Michael et al[25]	2012	TFI and QLF	Fluorescence imaging technique distinguished between fluoridated and non-fluoridated population and is useful for the assessment of dental fluorosis when used adjunctively with photographic scoring
Sabokseir et al[26]	2016	DI, TF and ECEL	The frequently used measures of fluorosis appear to overscore fluorosis. Use of ECEL grid method helps to differentiate between genuine fluorosis and fluorosis-resembling defects

Abbreviations: DDE, developmental defects of enamel; DI, Dean's index; EDEL, early childhood events life; QLF, quantitative light-induced fluorescence; TFI, Thylstrup–Fejerskov index; TSIF, tooth surface index of fluorosis; VAS, visual analog scale.

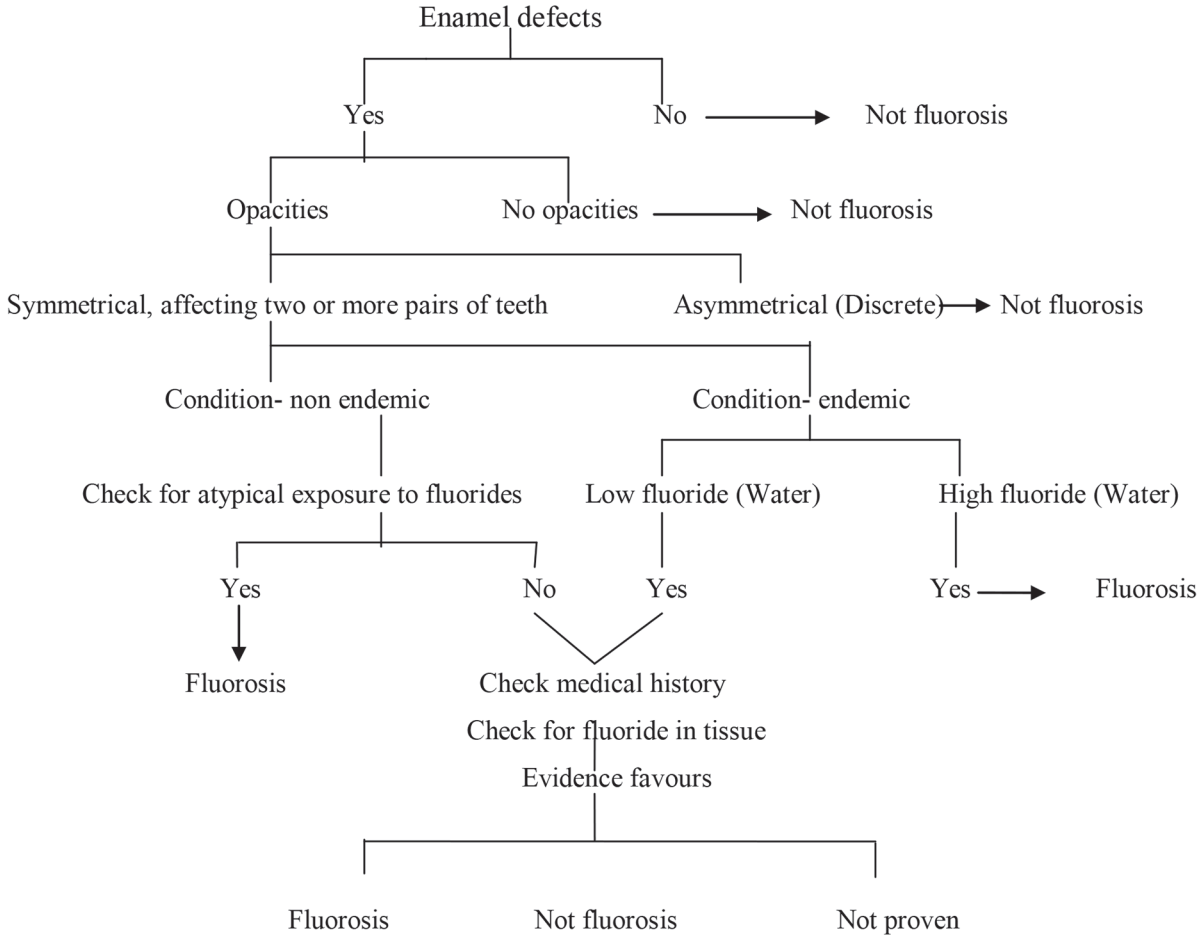

Fig. 1 Flow chart showing the sequence recommended for differential diagnosis of dental fluorosis.

Table 3 Difference between dental fluorosis and nonfluoride developmental defects and opacities[9,27-29]

Features	Dental fluorosis	Amelogenesis imperfecta	Enamel hypoplasia	Dentinogenesis imperfecta	Molar-incisor hypomineralization
Area affected	The entire tooth surfaces often enhanced or near tips of cusps/ incisal edges	Centered in smooth surface of limited extent Hypoplastic—buccal surface Hypomaturation—incisal or occlusal	Localized to the center of the smooth surface	The entire tooth surfaces	Loss of initially formed surface enamel after tooth eruption
Lesion	Show a horizontal striated pattern across the tooth. Bilateral symmetrical Qualitative defect—enamel has normal thickness, but changes in its translucency	Show vertical bands of white, opaque or translucent enamel surface and are round and oval in appearance. Qualitative/ quantitative	Round and oval in appearance. Occurs in the form of pits, grooves Quantitative defect—enamel is of reduced thickness	Affected teeth show distinct translucency and enamel frequently separates from underlying defected dentin	Soft, porous, brittle enamel resembling discolored chalk or old Dutch cheese Qualitative defect—enamel is of normal thickness with a smooth surface
History	High-fluoride level in drinking water in a community with water fluoridation, history of fluoride supplementation	Hereditary/familial pattern	If injury occurs during formative stage of enamel development Incomplete or defective formation of the organic enamel matrix of teeth	Hereditary/familial pattern. Autosomal dominant condition	Systemic alteration during maturation phase of enamel / idiopathic enamel defect with unknown etiology

(*Continued*)

Table 3 (*Continued*)

Features	Dental fluorosis	Amelogenesis imperfecta	Enamel hypoplasia	Dentinogenesis imperfecta	Molar-incisor hypomineralization
Demarcation	Diffuse distribution	Clearly differentiated from adjacent normal enamel.	Clearly differentiated from adjacent normal enamel	Clearly differentiated	Sharp demarcation between the affected and sound enamel
Color	Slightly more opaque than normal enamel; "paper white." Incisal edges, tips of cusps may have frosted appearance Does not show stain at the time of tooth eruption	Usually pigmented at the time of eruption; often creamy yellow to dark reddish orange	Brownish discoloration	Bluish to amber brownish	White to yellow or brown in color
Teeth affected	Teeth that calcify slowly (cuspids, bicuspids, second and third molars) Always on homologous teeth Extremely rare on deciduous teeth	Any tooth may be affected Frequent on labial surface of lower incisors Common in deciduous teeth	**Hereditary type**—Both the dentitions are affected **Environmental type**—Either dentition is involved Hutchinson's incisor and mulberry molars are seen (congenital syphilis). Incisors and Premolars (Turner's hypoplasia)	Both the dentitions are involved Deciduous teeth are more affected followed by permanent incisors, first, second and third molars are least affected	Both the dentitions are involved Usually affects permanent molars and incisors
Detection	Often invisible under strong light; easily detected by line of sight tangential to tooth crown	Line of sight perpendicular to tooth surface under strong light	Translucent or opaque	Affected teeth show distinct translucency	Alterations in the translucency of the enamel

Table 4 Difference between dental fluorosis and other tooth discoloration of systemic origin[9,27,30,31]

Features	Dental fluorosis	Tetracycline staining	Pulpal hemorrhage	Internal resorption	Congenital erythropoietic porphyria	Congenital hyperbilirubinemia and eythroblastosis fetalis	Alkaptonuria
Area affected	The entire tooth surfaces or near tips of cusps/ incisal edges	The entire tooth surfaces	The entire tooth surfaces. Anterior teeth are more prone	The entire tooth surfaces	The entire tooth surfaces	The entire tooth surfaces	The entire tooth surfaces
Color	Slightly more opaque than normal enamel; "paper white" to yellowish brown in severe cases	Bright yellow to dark brown	Bluish black	Crown display a pink discoloration (pink tooth of Mummery)	A marked red brown discoloration	Green discoloration	Blue black discoloration
History	High fluoride level in drinking water	Past drug history	History of trauma	Pulpal inflammation is usually caused by bacterial invasion	Medical history. An autosomal recessive disorder of porphyrin metabolism	Medical history	Medical history. An autosomal recessive metabolic disorder
Mechanism	Successive exposures to high concentration of fluoride during the formative stage of tooth development	Tetracycline and its homologous form complexes with calcium ions on the surface of hydroxyapatite crystals within bone and dental tissues	Hemolysis of the red blood cells releases the hem group to combine with the putrefying pulpal tissue to form black iron sulphide	Vascular resorptive process approaches the surface of the crown. Resorbed dentin is replaced by inflamed granulation tissue	Increased synthesis and excretion of porphyrins	Deposition of biliverdin (the breakdown product of the bilirubin) and may vary from yellow to green	Defect in the enzyme "homogentisate oxidase," homogentisate accumulates in tissues and blood

(*Continued*)

Table 4 (*Continued*)

Features	Dental fluorosis	Tetracycline staining	Pulpal hemorrhage	Internal resorption	Congenital erythropoietic porphyria	Congenital hyperbilirubinemia and eythroblastosis fetalis	Alkaptonuria
Critical time period and the teeth involved	Teeth that calcify slowly (cuspids, bicuspids, second and third molars). Always on homologous teeth. Extremely rare on deciduous teeth	Both the dentitions are involved. Avoided during pregnancy and in children up to 8 years of age	Both the dentitions are involved	Both the dentitions	Both the dentitions are involved. In deciduous teeth porphyrin is present in both enamel and dentin while in permanent teeth only dentin is affected	Mostly deciduous teeth are affected during the neonatal period Occasionally, the cusps of the permanent first molar may be affected	Both the dentitions
Detection	Often invisible under strong light; most easily detected by line of sight tangential to tooth crown	a bright yellow fluorescence under UV light	Visible bluish black discoloration.	Visible pink discoloration. On radiograph, a well circumscribed radiolucent enlargement of the pulp chamber is seen	A red fluorescence when exposed to a Wood's UV light	Sharp dividing line, separating green portions from normal colored portions	Bluish black discoloration

Table 5 Difference between dental fluorosis and dental caries[5,9,32]

Features	Dental fluorosis	White spot lesion (incipient caries)	Rampant caries
Area affected	The entire tooth surfaces or near tips of cusps/incisal edges	Smooth surfaces near the cervical margin or contact area at proximal surface	Entire tooth surfaces often involved
History	High fluoride level in drinking water or any history of fluoride supplementation	Multifactorial (diet, oral hygiene, etc.) Posteruptive etiology	Multifactorial (diet, oral hygiene) Improper feeding practice
Mechanism	Successive exposures to high concentration of fluoride during the formative stage of tooth development	Change in tooth enamel due to loss of the structure in the oral environment/ demineralization of tooth enamel	Demineralization of tooth enamel and progression of lesion to dentin and even pulp
Lesion	Show a horizontal striated pattern across the tooth. Bilaterally symmetrical	The subsurface porosity caused by demineralization gives a milky white appearance	Single tooth or even bilateral teeth involvement
Demarcation	Diffuse distribution over the surface of varying intensity	Visible on drying the tooth surface	Visible cavitated brown/black lesions.
Teeth involved	Teeth that calcify slowly (cuspids, bicuspids, second and third molars). The mandibular incisors are least affected. Always on homologous teeth. Extremely rare on deciduous teeth	Any tooth may be affected depending on the local attack of acid Both the dentitions are involved	Proximal surface of mandibular anterior teeth which are immune to decay are also involved Both the dentitions are involved
Detection	Often invisible under strong light; detected by line of sight tangential to tooth crown	Seen under fiberoptic light at an angle to the tooth surface	Visible cavitated brown /black lesions
Color	Slightly more opaque than normal enamel; "paper white," incisal edges, tips of cusps may have frosted appearance No staining at the time of tooth eruption	Usually pigmented at the time of eruption; often creamy yellow to dark reddish orange	Dark brown to blackish in appearance

Discussion

The adequate diagnosis of dental fluorosis necessitates examination of dry, clean tooth surfaces in the presence of good source of light. Its clinical appearance varies in intensity from hardly evident white striations to confluent pitting and staining. The fluorosed enamel is not discolored on eruption into the mouth; the discoloration develops eventually due to diffusion of exogenous ions into the porous enamel. It was observed that teeth develop later in the life are more susceptible for dental fluorosis.[3,7,15] Thylstrup and Thylstrup and Fejerskov noted a progressive increase in the severity from anterior to posterior teeth in maxilla as well as mandible.[7,12] Various indices have been employed in surveys to measure the presence and severity of enamel fluorosis. Dean's index (DI) due to its simplicity and extensive use over the years and historical importance provides a standard of comparison for other indices. However, it is unable to provide adequate information on the distribution of dental fluorosis within the dentition and particularly with its lowest score—the questionable score. Higher Dl scores are not sensitive enough to differentiate between the severe forms of dental fluorosis, while Thylstrup and Fejerskov index (TFI) was capable to assess dental fluorosis in areas with different levels of fluoride in drinking water (3.5, 6.0, and 21.0 ppm).[9-12] Owing to drying of tooth before scoring and appropriateness of TFI to identify very mild type of dental fluorosis, a higher prevalence of dental fluorosis was noted with TFI than with DI in the low and moderate fluorosis areas.[9-12] Moreover, it has been noted that tooth surface index of fluorosis (TSIF) diagnostic criteria are clearer and valuable in analytic studies.[10-11,15]

Numerous investigators have found DI to be less accurate and sensitive as compared with TFI and TSIF, as shown in ►**Table 2**, along with few newer diagnosing methods for dental fluorosis.[10-14] The acceptance of these indices depends on the ability of the examiner to discriminate fluoride-induced variations in the tooth enamel from those nonfluoride induced. Excess fluoride ingestion is commonly blamed for mottling; however, opacities and pigmentation of enamel can be due to several other causes and present clinical problems to the dental practitioner.[11,33] A complete history of the clinical condition, distribution on the affected tooth or within the dentition, pattern of lesion, color and its method of detection are the few important diagnostic criteria for differentiating dental fluorosis from nonfluoride discolorations of the teeth (►**Tables 3–5** and ►**Fig. 1**).

Conclusion

Dental fluorosis can be prevented and treated. Dental practitioners should not only have appropriate knowledge regarding etiology, occurrence and clinical appearance of dental fluorosis but also be able to differentiate it from other nonfluoride resembling defects.

Clinical Significance

This review article enlightened the detail clinical presentation of various fluoride and other resembling tooth discolorations. A correct diagnosis results in an appropriate management of dental fluorosis and plays an important role in public health concern.

Conflict of Interest

None declared.

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