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Fluoropolymers as low-surface-energy tooth coatings for oral care

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Abstract

A range of low-surface-energy fluoropolymers has been synthesised and their effectiveness as dental-care coatings for plaque, stain and erosion prevention has been evaluated using a series of oral care models employing pressed discs of calcium hydroxyapatite or sections of human teeth. Since the blocking of dentinal tubules is a key mechanistic strategy in the treatment of dentine hypersensitivity, the capability of these non-permanent fluoropolymer coatings to occlude the pore structure of human dentine and to reduce the outward flow of simulated dentinal fluid has also been investigated. Several of the fluoropolymer coatings have been found to inhibit bacterial adhesion but no correlation has been established between anti-adhesion efficacy and fluorine content or surface energy. All the fluoropolymers have been seen to reduce stain uptake by pellicle-coated HA discs, with homopolymers being considerably more effective than copolymers. Some fluoropolymer coatings have also been shown to inhibit the acid demineralisation of hydroxyapatite discs and to reduce dentine permeability. Coatings of the 2:1 copolymer of 1*H*,1*H*,2*H*,2*H*-perfluorodecyl acrylate and 2-hydroxyethyl acrylate are most promising, exhibiting significant anti-adhesion and anti-erosion efficacy and reducing dentine permeability to a level that is comparable with that achieved with the standard treatment employed in commercial anti-sensitivity formulations.
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Keywords: Fluoropolymer; Hydroxyapatite; Demineralisation; Bacterial adhesion; Dentine hypersensitivity

1. Introduction

A possible alternative to the use of antimicrobials to control dental plaque is to employ non-toxic polymers that can be deposited as protective coatings at the tooth surface (Marsh and Bradshaw, 1999; Tsibouklis et al., 1999; Van der Mei and White, 2002). Plaque-free tooth surfaces are coated with a pellicle (typically 1 μm in thickness) that is formed predominantly of salivary proteins and contains receptors that confer a degree of specificity towards primary bacterial colonisers (Rickard et al., 2003). Since the binding of individual bacteria relates to the number and nature of interactions between the microorganism and the addressed surface (Glantz, 1969; Quirynen et al., 1989; Weerkamp et al., 1989; Skvarla, 1993) the approach is rationalised in terms of the assumption that the application of a non-permanent polymeric thin film will screen such interactions and delay plaque development.

In this paper we consider the potential of fluoropolymers as components of dental care products by examining a series of materials with respect to their film formation characteristics onto calcium hydroxyapatite (PCHA) and dentine substrates. We also assess the usefulness of such films as a means of inhibiting microbial binding and the uptake of dietary stain by pellicle-coated PCHA discs. Additionally, we evaluate their efficacy as barriers to dietary acid-mediated tooth demineralisation, and consider their capability to reduce the permeability of human dentine to simulated dentinal fluid.

2. Experimental

2.1. Materials

Ceramic hydroxyapatite (HA) discs (9.5 mm diameter) were supplied by Clarkson Chromatography (USA). Earle's

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balanced salts, HA powder, MOPS–EDTA–sodium acetate buffer (MESA), and sodium dodecyl sulphate (SDS) were obtained from Sigma–Aldrich (Poole, UK). Vanadomolybdate reagent was purchased from VWR (Poole, UK). Octadec-1-ene–*co*-maleic acid disodium salt, PA18, was supplied by GlaxoSmithKline (Weybridge, UK). Bacteria were obtained as freeze dried cultures from the National Culture Collection (London, UK). [Methyl ^{3}H]-thymidine (37 MBq ml^{-1}) was purchased from Amersham Bioscience (Buckinghamshire, UK). Blood agar and brain heart infusion were sourced from Oxoid (Basingstoke, UK). Ultima Gold scintillation cocktail was obtained from Packard Bioscience (Buckinghamshire, UK). All other reagents were purchased from Fisher Scientific (Leicestershire, UK).

Codes: PPFDA—poly(perfluorodecyl acrylate); PPFDMA—poly(perfluorodecyl methacrylate); PNFBEA—poly(nonafluorobutylethyl acrylate); PHFBA—poly(heptafluorobutyl acrylate); PPFDDI—poly(perfluorodecyl diitaconate); PNFBEDI—poly(perfluorohexyl diitaconate); 3/1 A (M)—fraction indicates the co-monomer (fluorinated (meth)acrylate, 2-hydroxyethyl (meth)acrylate) molar feed ratio; "A" denotes acrylate, "M" methacrylate.

2.2. Polymer synthesis

Three classes of polymeric materials were considered, as illustrated in Fig. 1.

2.2.1. General method for the preparation of homopolymers

To 1*H*,1*H*, 2*H*,2*H*-perfluorodecyl acrylate (inhibitor-free; 15.0 g, 28.9 mmol) in perfluorodecalin (2.68 g; 5.80 mmol) was added SDS (750 mg) in deionised water (85 g). The resultant mixture was sonicated (0 °C, 40 min) to form an emulsion. This was transferred to a 5-necked reaction vessel equipped with an overhead stirrer, double-walled condenser, nitrogen inlet and dropping funnel and was degassed by nitrogen purge (2 h). Following the addition of potassium persulphate (150 mg in 2.5 ml of water), the mixture was heated (80 °C) and stirred (600 rpm) for 24 h to give a polymeric mini-emulsion, which was allowed to cool to room temperature under constant stirring. The polymer particles were separated by centrifugation (40,000 rpm), followed by the removal of SDS by repeated washes with water, and then by the Soxhlet extraction (petroleum ether 40–60 °C) of perfluorodecalin. The isolated polymer was dried to constant weight (40 °C, vacuum).

Polymeric mini-emulsions were also prepared from 1*H*,1*H*,2*H*,2*H*-perfluorodecyl methacrylate, from 1*H*,1*H*,2*H*,2*H*-perfluorohexyl acrylate and from 1*H*,1*H*-perfluorobutyl acrylate.

2.2.2. General method for the preparation of copolymers

Inhibitor-free 1*H*,1*H*,2*H*,2*H*-perfluoroalkyl acrylates (methacrylates) and 2-hydroxyethyl acrylate (methacrylate), in molar ratios in the range 5:1–1:5, were placed in a series of quickfit test tubes. Propan-2-ol was added to each tube (2 ml of freshly distilled propan-2-ol per 1 g of monomer) along with 1,1-azobiscyclohexanecarbonitrile (1 %, w/w of monomer; the

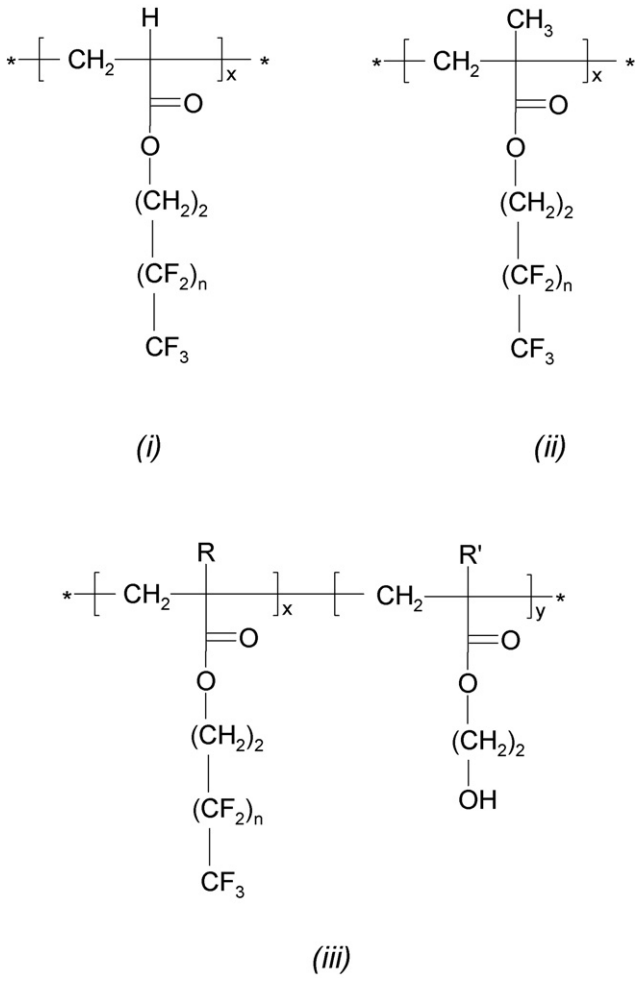

Fig. 1. Low-surface-energy polymers: (i) 1*H*,1*H*,2*H*,2*H*-perfluoroalkyl acrylates, (ii) 1*H*,1*H*,2*H*,2*H*-perfluoroalkyl methacrylates and (iii) 1*H*,1*H*,2*H*,2*H*-perfluoroalkyl acrylate (methacrylate)-*co*-2-hydroxyethyl acrylate (methacrylate); $n = 2,3,7$; R,R′ = H/CH$_3$.

effects of varying amounts of initiator were not investigated). After freeze–thaw degassing, the tubes were placed in an oil bath (70 °C, 72 h) and, following the removal of propan-2-ol by rotary evaporation, the isolated polymeric products were purified by Soxhlet extraction (methanol or diethyl ether) and dried *in vacuo* (0.5 mbar, 8 h, room temperature).

2.3. Pellicle formation

HA discs were cleaned by sonication in ethanol (15 min), rinsed several times with ethanol and dried (37 °C, 24 h). Parafilm-stimulated whole human saliva was collected in sterile centrifuge tubes maintained at 0 °C. To inhibit bacterial growth, the saliva was centrifuged (8000 rpm, 10 min, 4 °C), the supernatant was separated and sodium azide (NaN_3) was added to a final concentration of 0.04% (w/v). Saliva was stored at 5 °C and used within 30 min. Pellicle-coated hydroxyapatite (PCHA) discs were obtained by immersion of the cleaned discs in the prepared saliva (37 °C, 2 h), followed by rinsing in sterile deionised water (DI). The pellicle-coated discs were dehydrated (37 °C, 24 h) and stored in a dehumidified environment (4 °C) until

required. Prior to use, specimens were rehydrated (deionised water, DI, for 60 min).

2.4. Bacterial adhesion studies

Streptococcus mutans (NTCC 10449), *Streptococcus sanguinis* (NTCC 10904), and *Actinomyces naeslundii* (NTCC 10301) were grown on horse blood agar (37 °C, 48 h). Oral bacteria were harvested from fresh untreated human saliva (300 μl). Each of these cultures was added to sterile brain heart infusion (9 ml) and incubated (37 °C, 24 h, aerobic conditions).

To obtain radiolabelled stationary phase cultures, an aliquot (50 μl) of each bacterial sample was incubated (37 °C, 24 h) with a mixture of sterilised brain heart infusion broth (9 ml) and [methyl ^{3}H]-thymidine (50 μl; 37 MBq ml^{-1}). After centrifuging (sterile tubes, 8000 rpm), the supernatant was discarded and the pellet resuspended in sterile MESA buffer (10 ml, pH 7); this procedure was repeated three times. Each bacterial pellet was suspended in sterile MESA buffer (2 ml), the contents of all tubes were combined and, following dilution (150 μl of this bacterial suspension to 850 μl of MESA buffer) the absorbance (600 nm, 1 cm path length) was recorded. To obtain a stock bacterial suspension, further aliquots of MESA buffer were added to give an absorbance of 0.40, corresponding to a bacterial concentration of 10^{8} to 10^{9} cfu ml^{-1}.

For scintillation counting, universal bottles containing sterile MESA buffer (4 ml, 37 °C) were each inoculated with stock bacterial solution (300 μl). Polymer-treated HA discs and untreated control discs were placed into each bottle. After 24 h, the sample and control discs were removed and immersed in MESA buffer (5×) with gentle agitation to remove unbound bacteria. The discs were digested (48 h, occasional vortexing) in 0.40 M NaOH containing 1.0% (w/v) SDS. Aliquots (1.0 ml) of the digested bacterial suspension were mixed with Ultima Gold scintillation cocktail (20.0 ml) and liquid scintillation counting was performed using a Packard Tricarb T6100 scintillation counter.

2.5. Tooth staining

The staining solution consisted of a standardised filtered tea solution prepared in hot DI water. Treated and untreated PCHA discs were individually incubated (staining solution (100 ml), 37 °C) for either 2 h or 24 h, then rinsed (DI) and dried (37 °C, 24 h). HA staining was assessed from reflectance measurement using a Hunter LabScan XE Spectrophotometer (Hunter Associates Laboratory, Reston, VA) equipped with a Xenon flash lamp (filtered to approximately CIE D65 illumination). Six readings were recorded for each sample by rotating the disk 60° between each measurement. Chromametric data were obtained using the Commission Internationale de l'Eclairage (CIE) 1976 CIELAB convention recommendation. For this evaluation only L^* (lightness) values were compared.

2.6. Inhibition of acid demineralisation

HA-coated microtitre plates were prepared by suspending HA powder (2.0 g) in acetone (200 ml). Aliquots (60 μl) of the homogenous suspension were transferred into a 96-well polystyrene microtitre plate, which was placed on an orbital shaker (50 rpm, room temperature). After evaporation of the acetone, loosely bonded HA was removed by rinsing (DI, 5×); the dried, coated microplates were sealed until required.

Prior to use, the HA-coated plates were rehydrated (DI, 60 min). Aqueous suspensions of the polymers under test (1.0%, w/v; 200 μl) were added to each well and agitated (orbital shaker, 50 rpm, room temperature, 30 min). Aqueous sodium fluoride (663 μg ml^{-1}) and DI were employed, respectively as positive and negative controls. After exposure to the treatments, the plates were rinsed (5×, DI) and aqueous acetate buffer (0.10 M 200 μl, pH 4.0) was added to each well, as an erosive challenge. After further shaking (60 min), aliquots (50 μl) were transferred into new microplates containing vanadomolybdate reagent (50 μl per well). After a further 5 min, phosphate release was determined photometrically using a 96 well plate reader (450 nm, Wallac Victor 1420 Multilabel Counter).

2.7. Hydraulic conductance testing

Caries-free human third molars were sourced from patients who had given informed consent. Dentine discs were prepared by sectioning the tooth parallel to the occlusal surface, below the crown and above the root canal, and were stored in saline (0.90%). To remove the smear layer formed during cutting and to expose the dentinal tubules, each dentine disc was etched in aqueous EDTA (0.50 M, 2 min). Hydraulic conductance (Hc) experiments were performed using a Pashley flow apparatus (Pashley et al., 1996; Pashley and Matthews, 1993).

3. Results and discussion

Detailed characterisation data for the aqueous-media-processable fluoropolymers are presented elsewhere (Churchley et al., 2007).

3.1. Bacterial adhesion

The microbiological component of the present study focussed on the ability of fluoropolymer-coated surfaces to resist bacterial adhesion, which is the primary event in biofilm development. The strengths of adhesion between bacteria and polymer-coated surfaces or pellicle-coated hydroxyapatite have not been considered. The capability of the polymeric coatings to inhibit bacterial colonisation, Table 1, was investigated using two primary colonisers (*S. sanguinis*, *A. naeslundii*) and a major aetiological pathogen implicated in dental caries, *S. mutans* (Kolenbrander and London, 1993). To simulate *in vivo* conditions, the polymer films were also challenged with a mixed bacterial culture isolated from human saliva.

All polymer films suppressed the attachment of *A. naeslundii* markedly, but three of the homopolymers PPFDA, PPFDMA and PHFBA appeared to promote the adhesion of at least some of the microorganisms against which they were tested. With the exception of the 3:1 copolymer of 1*H*,1*H*,2*H*,2*H*-perfluorodecyl acrylate and 2-hydroxyethyl acrylate (3/1A), copolymers films

Table 1
Percentage reduction in numbers of adhered bacteria for a series of fluoropolymers samples coated onto PCHA discs and exposed to bacterial cultures for 24 h, relative to corresponding PCHA controls

Polymer	Reduction in bacterial adhesion (%)			
	Streptococcus mutans	*Streptococcus sanguinis*	*Actinomyces naeslundii*	Saliva culture
PPFDA	−91 (29)	nd	nd	−45 (44)
PPFDMA	28 (32)	−52 (34)	88 (7)	−55 (50)
PNFBEA	16 (45)	nd	70 (19)	6 (22)
PHFBA	14 (49)	nd	40 (40)	−19 (41)
3/1A	28 (21)	−4 (30)	61 (24)	−1 (31)
2/1A	58 (11)	27 (16)	74 (23)	27 (22)
1/4M	9 (32)	nd	68 (19)	39 (22)
1/5M	15 (33)	nd	nd	44 (23)
$2/1A_{HEMA}$	28 (19)	nd	37 (25)	14 (17)
$2/1A_{solution}$	76 (8)	64 (11)	80 (8)	70 (17)

S.D. = standard deviation; nd = not determined. A negative value implies that an increase in bacterial adhesion was observed.

exhibited some resistance to bacterial colonisation; films of the 2:1 copolymer of 1*H*,1*H*,2*H*,2*H*-perfluorodecyl acrylate and 2-hydroxyethyl acrylate (2/1A), especially those deposited from 1,1,2-trichlorotrifluoroethane solution, were found to be highly effective at inhibiting the attachment of bacteria.

Although the observed variations in bacterial adhesion are primarily related to the change in the nature of the addressed surface consequent to film deposition, both the quality of the deposited film and its surface roughness may also be of significance (Quirynen, 1994). For example, relatively smooth films of 2/1A deposited from organic solution exhibited superior performance than the rougher structures deposited from aqueous latex dispersions. Attempts to quantify surface roughness using the atomic force microscope in profilometry mode were unsuccessful, but scanning electron microscopy images showed fewer and less pronounced topographical features for solution-deposited coatings.

All aqueous fluoropolymer formulations contained amounts (0.18–0.5%, w/w) of the bactericidal surface-active agent SDS. Since this is readily desorbed from the surface of aqueous latex films (Tzitzinou et al., 1999), surface-related toxic effects are not expected to be of significance, but complications due to the adhesion of non-viable cells (Refgo et al., 1989; Chen et al., 1998; Bruinsma et al., 2001) may be of importance. In general, SDS-free and SDS-containing 2/1A formulations yielded films that inhibited colonisation to similar degrees, showing that the presence of this agent exerted little influence over the observed bacterial adhesion behaviour.

In electrolyte-containing aqueous environments for which the DLVO theory holds true, materials with very low-surface energies do not attract bacterial colonisers (Tsibouklis et al., 1999). However, under the imposed *in vitro* conditions, the coatings with the lowest surface energies did not appear to confer the best resistance to bacterial colonisation. The quality of the films formed and/or the ionic strength of the medium may account for this. The oral environment is even more complex on account of the dynamic interactions between bacteria and other colloidal species including salivary proteins (Skopek and Liljemark, 1994). Furthermore, polymeric thin films formed or placed in the oral cavity are themselves potential adsorptive surfaces. Although the above *in vitro* results can only be taken as a guide to potential *in vivo* performance, the data support the findings of Weerkamp et al. (1989), who reported that a fluoropolymer substrate (PTFE) placed *in situ* reduced plaque formation relative to a hydroxyapatite control.

3.2. Staining of pellicle-coated hydroxyapatite

Tooth colouration is affected by a combination of intrinsic and extrinsic factors, with the former normally reflecting changes in the structural composition or thickness of dental hard tissue (Addy and Watts, 2001). Intrinsic changes may be the result of metabolic diseases (e.g. alkaptonuria, congenital erythropoietic porphyria, congenital hyperbilirubinemia) or they may arise from the incorporation of exogenous chromogens into demineralised dental tissue (Addy and Watts, 2001). Extrinsic tooth staining results from the incorporation of chromogens (e.g. polyphenolic components of tea, coffee and red wine) into the salivary pellicle and, where present, plaque biofilms and calculus deposits (Joiner et al., 1995).

Differences in the degree of staining between sample and control specimens were compared using a one-way ANOVA with a post hoc Tukey's test. From the data obtained for the 2 h study (Table 2), the L^* values associated with the polymer-treated specimens were all directionally greater than that for the PCHA control, but statistical significance ($p < 0.05$) occurred only for PPFDA, PPFDMA and PPFDDI. It is possible that the large standard deviation characterising the control data may have masked the performance variations of samples within the series.

The data series from the 24 h stain study (Table 3) showed a similar pattern: PPFDA, PPFDMA, PNFBEA, PHFBA and 3/1A conferred statistically significant stain inhibition relative to the untreated control. Contrary to expectation, the highly fluorinated diitaconate polymers (Barbu et al., 2002) did not exhibit high levels of stain inhibition. With the exception of 1/5M films, the L^* values of polymers were larger than that of the control. The observed trends at both 2 h and 24 h indicate that a reduction in perfluorocarbon content impairs stain protection. By contrast, the presence of hydrophilic subunits reduces the polymers' ability to inhibit stain formation.

Table 2
Hydroxyapatite stain data after 2 h exposure to tea extract (averages of 20 readings, obtained by 90° rotations from each of five discs per type of treatment)

Polymer	L^* (S.D.)	ΔL
PCHA (pre-stain)	92.2 (0.2)	0
PCHA (post-stain)	70.5 (12.8)	21.7
PPFDA	89.2 (2.5)	3.0
PPFDMA	86.1 (2.0)	6.1
PNFBEA	86.0 (8.2)	6.2
PHFBA	84.5 (3.3)	7.7
PPFDDI	89.9 (1.2)	2.3
PNFBEDI	82.6 (4.2)	9.6
3/1A	74.9 (5.1)	17.3
2/1A	76.4 (4.1)	15.8
1/4M	74.0 (6.8)	18.2
1/5M	75.7 (5.2)	16.5

ΔL represents the difference in staining between each sample and the pellicle-coated HA control; S.D. = standard deviation.

Table 3
Hydroxyapatite stain data after 24 h exposure to tea extract (averages of 20 readings, obtained by 90° rotations from each of 5 discs per type of treatment)

Polymer	L^* (S.D.)	ΔL
PCHA (pre-stain)	92 (0)	0
PCHA (post-stain)	54 (9)	38
PPFDA	82 (1)	10
PPFDMA	78 (4)	14
PNFBEA	76 (4)	16
PHFBA	73 (8)	19
PPFDDI	66 (12)	26
PNFBEDI	62 (12)	30
3/1A	67 (3)	25
2/1A	64 (14)	28
1/4M	62 (10)	30
1/5M	53 (13)	39

ΔL represents the difference in staining between the sample and the pellicle-coated HA control relative to the pellicle-coated HA discs (pre-stain); S.D. = standard deviation.

3.3. Inhibition of acid demineralisation

In tooth erosion, dental hard tissues are lost through an acid-mediated chemical process that does not involve the active participation of oral microflora; a smooth surface results due to the wear of the demineralised surface (Lussi et al., 1993). As with tooth staining, both intrinsic and extrinsic factors have been implicated in dental erosion (Amaechi and Higham, 2005). In the absence of extreme aetiological factors, such as gastric reflux, oral care products rely on fluoride to enhance the resistance of enamel and dentine to acid attack. The most widely used fluoride sources are sodium fluoride, sodium monofluorophosphate, stannous fluoride and amine fluoride, which act by substituting fluoride for hydroxyl groups in the mineral lattice to form fluorohydroxyapatite (FHA) at the surface of the tooth, Eq. (1).

$$Ca_{10}(PO_4)_6(OH)_2 + xF^- \rightleftharpoons Ca_{10}(PO_4)_6(OH)_{2-x}F_x + xOH^- \quad (1)$$

As the solubility of FHA is an order of magnitude lower than that of HA, substitution of OH^- with F^- improves the resistance of the enamel to acid demineralisation (Diarra et al., 2003).

The current approach relies on a deposited protective film structure, rather than on the fluoride-induced surface-structural modification of dental tissue. Based on their performance in the staining and Hc models, the capability of four fluoropolymers to inhibit the acid demineralisation of HA was determined using a validated model that is widely used to screen anti-erosion actives: performance was compared with that of an aqueous fluoride solution (300 $\mu g\,ml^{-1}$), with deionised water serving as the negative control, Fig. 2. With the exception of PPFDA, all fluoropolymers tested inhibited acid demineralisation.

3.4. Hydraulic conductance

Within the oral environment, dentine may become exposed as a result of acid erosion, tooth abrasion, gingival recession or trauma. Dentine hypersensitivity is the transient pain that is induced following exposure to chemical, thermal, tactile or osmotic stimuli, which does not arise from any other dental

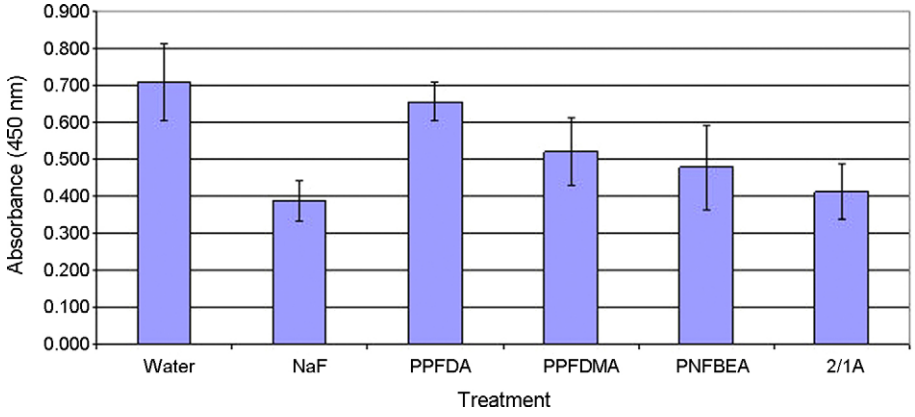

Fig. 2. Inhibition of HA demineralisation by selected fluoropolymers tested as aqueous dispersions (0.50%, w/v). The magnitude of A450 is directly related to the concentration of released phosphate determined using a vanadomolybdate reagent. DI and 300 $\mu g\,ml^{-1}$ sodium fluoride were included as the negative and positive controls, respectively.

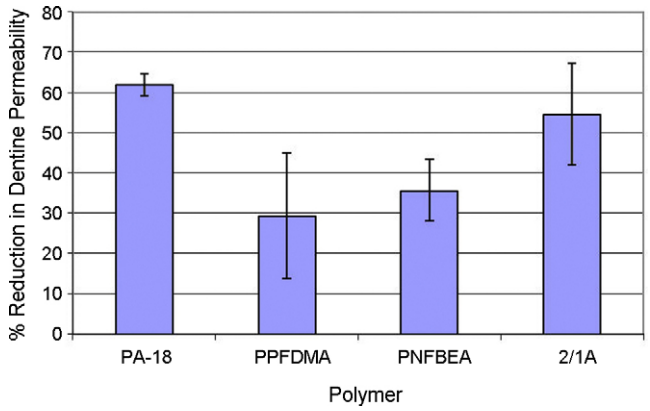

Fig. 3. Reduction in dentine permeability to simulated dentinal fluid following treatment of specimens with selected fluoropolymers, and with the PA-18 positive control.

defect or pathology. According to the widely accepted hydrodynamic theory, the pain response is elicited by activation of mechanoreceptors brought about by a rapid change in dentinal fluid flow (Dowell and Addy, 1983). Treatment may involve suppression of nerve excitability or occlusion of dentinal tubules (Collaert and Speelman, 1991), the latter being addressed in the present study.

The polymers PPFDMA, PNFBEA and 2/1A were selected for screening in the Hc model. After determining the baseline permeability of pellicle-coated dentine specimens to simulated dentinal fluid, aqueous dispersions (1.0%, w/w) of the polymers were applied by brushing and the changes in permeability were determined, Fig. 3. All polymers tested reduced dentine permeability to a statistically significant extent *versus* baseline ($n = 3$, $p < 0.05$). The PA-18 positive control gave the greatest reduction in dentine permeability (62%): its performance was significantly superior to that of PPFDMA or PNFBEA ($p < 0.05$) but comparable to that of 2/1A.

4. Conclusion

Aimed towards the development of non-permanent barrier coatings for dental care applications, several fluoropolymers have been prepared as aqueous dispersions, which have been used to deposit thin films onto calcium hydroxyapatite discs and onto sections of human teeth. The *in vitro* performance of coatings of the 2:1 copolymer of 1*H*,1*H*,2*H*,2*H*-perfluorodecyl acrylate and 2-hydroxyethyl acrylate was of particular note, conferring resistance to microbial adhesion, inhibiting erosive acid demineralisation and considerably reducing dentine permeability.

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