In Vitro Lipid Deposition on Hydrogel and Silicone Hydrogel Contact Lenses

Andrew D. Pucker, Mirunalni Thangavelu, and Jason J. Nichols

PURPOSE. To understand various soft contact lens materials’ ability to adsorb common tear lipids.

METHODS. Ten unworn polymers of nine types were individually soaked in 1.0 mL of 1.75 μg/mL cholesterol oleate or 1.0 mL of 0.5 μg/mL phosphatidylcholine solutions for 1 or 14 days. The adsorbed lipids were extracted with chloroform-methanol, which underwent assay quantification for cholesterol oleate and inorganic phosphate.

RESULTS. More phosphatidylcholine was extracted after 14 days than after 1 day in only lotrafilcon B, balafilcon A, and enfилкон A (all P < 0.005). After 1 day of incubation in phosphatidylcholine, 0.54 to 4.17 μg/lens phosphatidylcholine was recovered from the polymers, and after 14 days of incubation in phosphatidylcholine, 0.58 to 5.77 μg/lens phosphatidylcholine was recovered from the polymers. etafilcon A had significantly more cholesterol oleate at 1 day than at 14 days, and lotrafilcon A, lotrafilcon B, and balafilcon B had significantly more cholesterol oleate at 14 days than at 1 day (P < 0.005). After 1 day of incubation in cholesterol oleate, 0.14 to 4.80 μg/lens cholesterol oleate was recovered from the polymers. After 14 days of incubation in cholesterol oleate, 1.40 to 6.84 μg/lens cholesterol oleate was recovered from the polymers.

CONCLUSIONS. Hydrogel and most silicone hydrogels appear to adsorb lipids relatively quickly (i.e., within the first day). Although there is some variability in the amounts recovered across materials, it is uncertain whether these differences have any clinical significance. (Invest Ophthalmol Vis Sci. 2010;51:6334–6340) DOI:10.1167/iovs.10-5836

Contact lens wear is associated with changes to the ocular surface and tear film, sometimes contributing to dry eye during lens wear.1 Most studies indicate that the frequency and severity of dry eye symptoms are much more significant in contact lens wearers than in non–contact lens wearers.2 These symptoms could potentially be caused by the contact lens altering certain aspects of the structure or function, or both, of the tear film.3–6 As contact lens–related dry eye is considered “evaporative” in nature, the more obvious focus becomes the impact of contact lens wear on the lipid layer as the lipid layer retards evaporation.7–12 Consequently, contact lens wear can be associated with ocular discomfort, visual disturbances, and inflammation of the ocular surface that are often associated with an increase in tear film osmolarity.1

In light of this, there remains a dearth of information concerning the relation between the lipids of the tear film and the contact lens itself. This becomes a complicated issue given the array of contact lens polymer chemistries. Hydrogel contact lens materials are composed of various monomers cross-linked to form polymers, each containing different side groups intended to attract water to varying degrees, making them hydrophilic in nature. Since their advent, a great deal of work has been conducted to create materials with increased oxygen permeabilities given the oxygen needs of the cornea.13 In more recent years, the traditional hydrogel material was successfully combined with silicone moieties to create silicone hydrogel materials.13 The hydrogel technology combined with the hydrophobic silicones creates a polymer with more hydrophobic properties, which could theoretically decrease the material’s wettability or could attract certain components of the tear film (e.g., lipids). These issues were mitigated first by adding surface treatments such as a plasma coating and later by incorporating internal wetting agents.14

As discussed, one major issue under investigation is the interaction between tear film lipids and contact lens polymers. It is traditionally thought that the lipid layer can be further broken down into an inner polar layer and an outer nonpolar layer.15 The inner polar layer is thought to consist primarily of phospholipids such as phosphatidylcholine, phosphatidylethanolamine, and sphingomyelins.14,15 The outer nonpolar layer is thought to consist primarily of cholesterol and cholesterol esters, in addition to other lipids such as wax esters and fatty acids.14,15 When these layers are altered in structure or composition, increased evaporation and the common symptoms of dry eye may result.9,10,16–25 For instance, studies have shown that low levels of phospholipids in tears are associated with short tear breakup times and contact lens intolerance (phospholipid absorption to lens materials could potentially lead to a nonuniform, nonpolar lipid layer).26

Lipid deposition on contact lenses during normal wear was first described by Doughman27 in the mid 1970s. More recently, investigators have begun to explore lipid deposition or adsorption on traditional and silicone hydrogel materials. Jones et al.28,29 found that silicone hydrogel materials tended to adsorb more lipids than traditional hydrogel materials; however, this study compared only first-generation silicone hydrogel materials (and thus included only materials treated with coatings), and the methodologies have since been questioned. Zhao et al.30 found that the lens polymer is a prominent factor affecting lipid accumulation on silicone hydrogel contact lenses. Carney et al.31 more recently found there were material differences in cholesterol and phospholipid adsorption, whereby the non-surface–coated silicone hydrogels generally adsorbed more of each of these lipids than the surface-coated silicone hydrogels, and a traditional hydrogel adsorbed the least of the lipids.

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There is still much clinical uncertainty regarding the relation between contact lens polymers, the lipids of the tear film, and the clinical relevance of lipid adsorption to contact lenses. This is true given the continually emerging understanding about the lipids of the tear film themselves, emerging polymer technologies, and differing methodologies that might be considered in unraveling these questions. It is critical that additional research be conducted to yield consistency in findings to solidify our understanding between tear film lipids and contact lens materials and the physiological effect of this relationship. Thus, the purpose of this study was to determine the time course of adsorption and the pattern of absorption of polar and nonpolar lipids on commonly dispensed silicone hydrogel materials.

**Materials and Methods**

**Experimental Design**

Contact lenses used in this study included the following: etafilcon A hydrogel contact lenses (Acuvue 2; Vistakon, Inc., Jacksonville, FL), and balafilcon A (PureVision; Bausch & Lomb, Inc., Rochester, NY), lotrafilcon A (Night and Day; CIBA Vision, Inc., Duluth, GA), lotrafilcon B (O2; Optixx; CIBA Vision, Inc.), gafilcon A (Acuvue Advance; Vistakon, Inc.), narafilcon A (1-Day Acuvue TrueEye; Vistakon, Inc.), senofilcon A (Acuvue Oasys; Vistakon, Inc.), comfilcon A (Biofinity; CooperVision, Inc., Pleasanton, CA), and enfilcon A (Avaira; CooperVision, Inc.) silicone hydrogel contact lenses. All lenses were initially rinsed with water before use to remove any phosphate associated with the packaging solution. Ten individual lenses from each category described were soaked individually in 1 mL of 0.5 µg/mL phosphatidylcholine (P2772; Sigma, St. Louis, MO) for 14 days; the phosphatidylcholine solution was replaced each day. Chlороform was used to dilute the phosphatidylcholine-chloroform solution to the proper concentration. Incubation in this solution caused the contact lenses to increase in width and become more malleable. At the end of the 14-day period, the phosphatidylcholine solution was removed, the lenses were blotted (Kim Wipe; Kimberly-Clark, Dallas, TX), and the bound phosphatidylcholine was extracted. Phosphatidylcholine extraction was performed by adding 250 µL of 1:1 chloroform/methanol solvent for approximately 25 seconds, after which, approximately 100 µL solvent was recovered from the lens (some of the solvent was absorbed by the lens). The extract was stored in an open amber vial to lyophilize overnight. Individual extraction samples were then stored at −80°C until the samples were ready for analysis. The entire phosphatidylcholine segment of the experiment was repeated for an 8-hour (approximately 1 day of wear) incubation time, as described. These same 1- and 14-day incubation periods were repeated using a 1.75-µg/mL cholesterol oleate (C-9253; Sigma) solution, for brevity, the cholesterol oleate extraction and lyophilization procedure was performed as described by Pucker et al. 32

**Phospholipid Assay**

A stock phosphatidylcholine solution was diluted in chloroform, and standards were prepared (range, 0–5 µg) in duplicate in new HPLC grade, phosphate-free amber vials and left to dry overnight (for approximately 14 hours). To avoid phosphate contamination, freshly washed and autoclaved glassware was used for preparation of all solvents. The extracted phosphatidylcholine samples were removed from −80°C and were allowed to equilibrate to room temperature. The phosphatidylcholine and standard samples were hydrolyzed by the addition of 10 µL of 10 N sulfuric acid and 30 µL of 70% perchloric acid to each sample, followed by incubation in a heat block at 140°C until all the perchloric acid had evaporated. The hydrolyzed samples and standards were then allowed to cool to room temperature and were subsequently resuspended in 50 µL ultrapure water (care was taken to wash the sides of the tubes to ensure resuspension of all phosphate). Assay reagent was freshly prepared by adding 0.2% malachite green solution in water to 4.2% ammonium molybdate in 4 N HCl in a 3:1 ratio. The solution was stirred vigorously for 30 minutes, filtered, and kept from light. Then 100 µL of assay reagent was added to both the standards and the extracted (phosphatidylcholine) contact lens samples; this was then followed by the addition of 5 µL of 1.5% Tween 20. The standards and samples were incubated at room temperature for 20 minutes and then moved to a 96-well plate. The absorbance of each sample was read at 645 nm in a microplate reader (Infinite M200; Tecan, Männedorf, Switzerland). The average blank (0 µg) reading from the standard curve samples and untreated contact lens samples were subtracted from the average absorbance of each of the standard curve samples and treated samples in order remove any remaining background phosphate signal or other contaminants, and a standard curve was prepared. Linear regression was used to determine the concentration of each sample.

**Cholesterol Assay**

One liter of cholesterol assay reaction buffer (pH 7.0) and 1 L of 0.10 M phosphate-buffered saline (PBS; pH 7.0) were created. Peroxidase (P6782–5MG; Sigma), cholesterol oxidase (ICN-15067480; Fischer Scientific, Pittsburgh, PA), and cholesterol esterase (C3766–100UN; Sigma) were diluted in PBS to the appropriate concentrations. Next, a cholesterol oleate (C9253; Sigma) stock solution was prepared at a final concentration of 8.6 mM (5.6 mg/mL), as directed by the manufacturer, and was used to create a serially diluted cholesterol oleate standard curve. Samples and standards then underwent enzymatic quanitification, and their absorbance was subsequently read at 500 nm in a microtiter spectrophotometer (Infinite M200; Tecan). The blank (0 µg) reading from the standard curve was subtracted from the average absorbance of each of the standards and samples. Linear regression was then used to determine the concentration of each sample (again, reported in micrograms per lens).

**Statistical Analysis**

Descriptive statistics such as averages and standard deviations were used to describe the data. Wilcoxon signed-ranks tests were used to describe the temporal relation between the 1-day and 14-day time points for extracts from each contact lens material type for each lipid type. Type 1 errors for this number of comparisons were minimized by setting the significance level at 0.006. Kruskal-Wallis one-way analysis of variance was used to compare lipid extracts across contact lens material types within each specific time point. For significant material effects within a time point, post hoc testing was used to compare individual materials on a two-by-two basis for each lipid. The significance level was set at 0.05 (Z = ±1.96).

**Results**

**Phosphatidylcholine**

Significantly more phosphatidylcholine was extracted at the 14-day time point than at the 1-day time point for lotrafilcon B, balafilcon A, and enfilcon A (Z = −2.803; P < 0.005; Fig. 1). There were no other significant differences between the amount of phosphatidylcholine extracted at the 1-day and 14-day time points for all other materials studied (Fig. 1).

Figure 1 shows the following amounts (µg/lens) of phosphatidylcholine recovered from each material studied after 1-day of incubation: etafilcon A (1.26 ± 0.51), balafilcon A (0.60 ± 0.44), lotrafilcon A (4.17 ± 3.71), lotrafilcon B (1.09 ± 0.49), gafilcon A (1.87 ± 1.11), narafilcon A (0.54 ± 0.27), senofilcon A (0.78 ± 0.22), comfilcon A (1.87 ± 0.79), and enfilcon A (0.97 ± 0.82) (Kruskal-Wallis Z = 33.30; P < 0.001). As shown in Table 1, significantly more phosphatidylcholine was extracted from lotrafilcon A than from narafilcon A (P < 0.01) or balafilcon A (P < 0.05), and
significantly more phosphatidylcholine was extracted from galyfilcon A than from narafilcon A (P \textless 0.05). However, significantly less phosphatidylcholine was extracted from narafilcon A than from comfilcon A (P \textless 0.01) and from balafilcon A than from comfilcon A (P \textless 0.05). There were no other significant differences among the materials incubated in phosphatidylcholine solution for 1 day (Table 1).

Figure 1 also shows the following amounts (\mu g/lens) of phosphatidylcholine recovered from each of the materials studied after 14 days of incubation: etafilcon A (0.63 \pm 0.38), balafilcon A (2.62 \pm 1.11), lotrafilcon A (4.12 \pm 1.92), lotrafilcon B (5.22 \pm 2.03), galyfilcon A (1.47 \pm 0.57), narafilcon A (0.58 \pm 0.25), senofilcon A (1.14 \pm 0.39), comfilcon A (3.96 \pm 1.51), and enfilcon A (5.77 \pm 1.74) (Kruskal-Wallis = 69.74; P \textless 0.001). As shown in Table 2, significantly more phosphatidylcholine was extracted from lotrafilcon A than from etafilcon A or narafilcon A (P \textless 0.001). Similarly, significantly less phosphatidylcholine was extracted from etafilcon A than from lotrafilcon B, comfilcon A, or enfilcon A (P \textless 0.001). Finally, significantly less phosphatidylcholine was extracted from galyfilcon A than from lotrafilcon B (P \textless 0.05) or enfilcon A (P \textless 0.01), and significantly less phosphatidylcholine was extracted from senofilcon A than from lotrafilcon B (P \textless 0.01) or enfilcon A (P \textless 0.001). Significantly less phosphatidylcholine was extracted from narafilcon A than from lotrafilcon B (P \textless 0.001), balafilcon A (P \textless 0.05), comfilcon A (P \textless 0.01), or enfilcon A (P \textless 0.001). There were no other significant differences among the materials incubated in phosphatidylcholine solution for 14 days (Table 2).

**Table 1.** Kruskal-Wallis Test Comparison of Phosphatidylcholine Recovered by Organic Extraction from Each Material after 1 Day of Incubation in 0.50 \mu g/mL Phosphatidylcholine Solution

<table>
<thead>
<tr>
<th>Material</th>
<th>Etafilcon A</th>
<th>Balafilcon A</th>
<th>Lotrafilcon B</th>
<th>Galyfilcon A</th>
<th>Narafilcon A</th>
<th>Senofilcon A</th>
<th>Comfilcon A</th>
<th>Enfilcon A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etafilcon A</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Balafilcon A</td>
<td>—</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
<td>P \textless 0.05</td>
<td>NS</td>
<td>—</td>
<td>NS</td>
</tr>
<tr>
<td>Lotrafilcon A</td>
<td>NS</td>
<td>P \textless 0.05</td>
<td>NS</td>
<td>P \textless 0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Narafilcon A</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>—</td>
<td>P \textless 0.05</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Senofilcon A</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>P \textless 0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Comfilcon A</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
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<td>—</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS, not significant.
Figure 2 shows that significantly more cholesterol oleate was extracted from etafilcon A (Z = -2.803; P < 0.005) at the 1-day time point than at the 14-day time point. Significantly more cholesterol oleate was extracted from materials at the 14-day time point than at the 1-day time point for lotrafilcon A, lotrafilcon B, and balafilcon A (Z = -2.803; P < 0.005) (Fig. 2). There were no other significant differences in the amount of cholesterol oleate extracted between the 1-day and the 14-day time points for all materials studied in this experiment (Fig. 2).

Figure 2 also shows the following amounts (μg/lens) of cholesterol oleate recovered from each material studied after 1 day of incubation: etafilcon A (3.95 ± 0.78), balafilcon A (0.14 ± 0.13), lotrafilcon A (4.80 ± 1.00), lotrafilcon B (0.33 ± 0.15), galyfilcon A (4.31 ± 0.89), narafilcon A (0.64 ± 0.32), senofilcon A (4.76 ± 0.93), comfilcon A (0.16 ± 0.19), and enfilcon A (0.20 ± 0.15) (Kruskal-Wallis = 69.73; P < 0.001). As shown in Table 3, significantly more cholesterol oleate was extracted from lotrafilcon A than from lotrafilcon B (P < 0.01), balafilcon A (P < 0.001), comfilcon A (P < 0.001), or enfilcon A (P < 0.001). Similarly, significantly more cholesterol oleate was extracted from etafilcon A than from balafilcon A (P < 0.01), comfilcon A (P < 0.01), or enfilcon A (P < 0.05) (Table 3). Finally, significantly more cholesterol oleate was extracted from galyfilcon A than from lotrafilcon B (P < 0.01), balafilcon A (P < 0.001), comfilcon A (P < 0.01), or enfilcon A (P < 0.01), and significantly more cholesterol oleate was extracted from senofilcon A than from lotrafilcon B (P < 0.01), balafilcon A (P < 0.001),

### Table 2. Kruskal-Wallis Test Comparison of Phosphatidylcholine Recovered by Organic Extraction from Each Material after 14 Days of Incubation in 0.50 mg/mL Phosphatidylcholine Solution

<table>
<thead>
<tr>
<th>Material</th>
<th>Etafilcon A</th>
<th>Balafilcon A</th>
<th>Lotrafilcon B</th>
<th>Galyfilcon A</th>
<th>Narafilcon A</th>
<th>Senofilcon A</th>
<th>Comfilcon A</th>
<th>Enfilcon A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etafilcon A</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Balafilcon A</td>
<td>P &lt; 0.001</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
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<td>NS</td>
<td>P &lt; 0.001</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Lotrafilcon B</td>
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<td>—</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Galyfilcon A</td>
<td>NS</td>
<td>P &lt; 0.05</td>
<td>—</td>
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<td>NS</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Narafilcon A</td>
<td>P &lt; 0.05</td>
<td>P &lt; 0.001</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>P &lt; 0.001</td>
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<td></td>
</tr>
<tr>
<td>Senofilcon A</td>
<td>NS</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
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<tr>
<td>Comfilcon A</td>
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</tr>
</tbody>
</table>

**NS**, not significant.

**Cholesterol Oleate**

Figure 2 shows that significantly more cholesterol oleate was extracted from etafilcon A (Z = -2.803; P < 0.005) at the 1-day time point than at the 14-day time point. Significantly more cholesterol oleate was extracted from materials at the 14-day time point than at the 1-day time point for lotrafilcon A, lotrafilcon B, and balafilcon A (Z = -2.803; P < 0.005) (Fig. 2). There were no other significant differences in the amount of cholesterol oleate extracted between the 1-day and the 14-day time points for all materials studied in this experiment (Fig. 2).

Figure 2 also shows the following amounts (μg/lens) of cholesterol oleate recovered from each material studied after 1 day of incubation: etafilcon A (3.95 ± 0.78), balafilcon A (0.14 ± 0.13), lotrafilcon A (4.80 ± 1.00), lotrafilcon B (0.33 ± 0.15), galyfilcon A (4.31 ± 0.89), narafilcon A (0.64 ± 0.32), senofilcon A (4.76 ± 0.93), comfilcon A (0.16 ± 0.19), and enfilcon A (0.20 ± 0.15) (Kruskal-Wallis = 69.73; P < 0.001). As shown in Table 3, significantly more cholesterol oleate was extracted from lotrafilcon A than from lotrafilcon B (P < 0.01), balafilcon A (P < 0.001), comfilcon A (P < 0.001), or enfilcon A (P < 0.001). Similarly, significantly more cholesterol oleate was extracted from etafilcon A than from balafilcon A (P < 0.01), comfilcon A (P < 0.01), or enfilcon A (P < 0.05) (Table 3). Finally, significantly more cholesterol oleate was extracted from galyfilcon A than from lotrafilcon B (P < 0.01), balafilcon A (P < 0.001), comfilcon A (P < 0.01), or enfilcon A (P < 0.01), and significantly more cholesterol oleate was extracted from senofilcon A than from lotrafilcon B (P < 0.01), balafilcon A (P < 0.001),

![Figure 2](http://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/932969/) Average cholesterol oleate (microgram per lens) collected from various contact lens samples assigned to the 1-day and 14-day cholesterol oleate incubation groups. **Hatched bars**: hydrogel contact lens material.
TABLE 4. Kruskal-Wallis Test Comparison of Cholesterol Olate Recovered by Organic Extraction from Each Material after 1 Day of Incubation in 1.75 μg/mL Cholesterol Olate Solution

<table>
<thead>
<tr>
<th>Etafilcon A</th>
<th>Balafilcon A</th>
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<td>—</td>
<td>—</td>
<td>NS</td>
<td>NS</td>
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<td>P &lt; 0.05</td>
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<tr>
<td>NS</td>
<td>—</td>
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<tr>
<td>Galyfilcon A</td>
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</tr>
<tr>
<td>Narafilcon A</td>
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<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.001</td>
<td>NS</td>
</tr>
<tr>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>P &lt; 0.01</td>
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</tr>
<tr>
<td>Comfilcon A</td>
<td>P &lt; 0.001</td>
<td>P &lt; 0.01</td>
<td>—</td>
<td>—</td>
<td>NS</td>
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</tr>
</tbody>
</table>

NS, not significant.

comfilcon A (P < 0.001), or enofilcon A (P < 0.01) (Table 3). There were no other significant differences among the materials incubated in cholesterol olate solution for 1 day (Table 5).

Figure 2 shows the following amounts (μg/lens) of cholesterol olate recovered from each material studied after 14 days of incubation: etafilcon A (2.75 ± 0.14), balafilcon A (2.40 ± 1.35), lotrafilcon A (6.84 ± 1.35), lotrafilcon B (1.72 ± 0.79), galyfilcon A (4.59 ± 1.04), narafilcon A (3.19 ± 1.94), senofilcon A (4.98 ± 1.07), comfilcon A (1.67 ± 1.02), and enofilcon A (1.40 ± 1.22) (Kruskal-Wallis = 58.68; P < 0.0001). Table 4 shows that significantly more cholesterol olate was extracted from lotrafilcon A than from etafilcon A (P < 0.05), lotrafilcon B (P < 0.001), balafilcon A (P < 0.01), comfilcon A (P < 0.001), or senofilcon A (P < 0.001), significantly more cholesterol olate was extracted from galyfilcon A than from lotrafilcon B (P < 0.05), comfilcon A (P < 0.05), or enofilcon A (P < 0.01), and significantly more cholesterol olate was extracted from senofilcon A than from lotrafilcon B (P < 0.01), comfilcon A (P < 0.01), and enofilcon A (P < 0.01). There were no other significant differences in the amounts of cholesterol olate extracted from the materials studied (Table 4).

**DISCUSSION**

In considering phosphatidylcholine extracted from the materials used in this study, the only statistically significant time point differences, when comparing the materials at the 1-day and 14-day time points, were for lotrafilcon B, balafilcon A, and enofilcon A. Similarly, there were statistical differences in some of the materials compared at the 1-day and 14-day time points. The actual differences between most of the materials were small, with some exceptions. These differences were associated primarily with the lotrafilcon A (1 day and 14 days) and galyfilcon A (1 day) materials, which accumulated more phosphatidylcholine than some others, and with the balafilcon A (1 day), etafilcon A (14 day), galyfilcon A (1 day), senofilcon A (14 day), and narafilcon A (1 day and 14 day) materials, which accumulated less phosphatidylcholine than some others (Table 2). The differences between materials at each time point can likely be attributed to a complex interaction of the properties of the materials (surface treatment, water content, and material polarity).

The observation of phospholipids being extracted from hydrogel contact lenses has also been noted by others. In one study, subjects who wore polymacon (FDA group I) had 186 μg/ml phospholipid in their tears, with 2.1 μg/ml phospholipids extracted from their lenses. Subjects who wore etafilcon A (FDA group IV) had 162 μg/ml phospholipid in their tears, with 1.8 μg/ml of phospholipids extracted from their lenses. In a later study suggest that phospholipid deposition on the contact lenses alone may not explain the overall difference in the amount of phospholipids in the tear film. They went on to attribute this discrepancy to the fact that more phospholipase A2 was deposited on the etafilcon A lenses, which then decreased the amount of phospholipids in the tears. Although this may account for a small portion of the tear phospholipid decrease, it is unlikely that this was the major cause for the significant decrease in tear phospholipids because polymacon lens wearers had a similar though slightly smaller decrease in tear phospholipids but, simultaneously, no detectable phospholipase A2 or phospholipid deposit on the polymacon material. Our study used a phospholipid dop- ing solution, with no exposure of the contact lens materials to phospholipase A2 found in normal human tears. If phospholipase A2 were actively working at the contact lens surface in etafilcon A lenses (as suggested by Yamada et al.), one would have expected to have recovered a significantly smaller amount of phospholipid from that contact lens material. It is typically thought that many of the tear film proteins become denatured when they are deposited on contact lenses, rendering them nonfunctional.

Carney et al.31 also explored phospholipid deposition on some of the same materials used in this study. They and we used the same concentration of phospholipid; however,
Carney et al.31 used phosphatidylethanolamine, whereas we used phosphatidylcholine in the present study. They found that all materials in their study (lotrafilcon A, lotrafilcon B, galyfilcon A, etafilcon A, balafilcon A, and senofilcon A) had deposits of <0.5 μg/lens phosphatidylethanolamine after 1 day of incubation,31 but we found in our study a range from ~0.6 to 4 μg/lens after 1 day of incubation. After 14 days of incubation, Carney et al.31 found the following: lotrafilcon A (0.4 μg/lens), lotrafilcon B (0.9 μg/lens), galyfilcon A (5.3 μg/lens), senofilcon A (3.2 μg/lens), balafilcon A (1.7 μg/lens), and etafilcon A (0.1 μg/lens). We found the following differences after 14 days of incubation: lotrafilcon A (4.1 μg/lens), lotrafilcon B (5.22 μg/lens), galyfilcon A (1.5 μg/lens), senofilcon A (1.1 μg/lens), balafilcon A (2.6 μg/lens), and etafilcon A (1.8 μg/lens). Differences were observed between materials after 14-day time point, with the exception of lotrafilcon A and lotrafilcon B, which accumulated similar amounts of cholesterol (Fig. 2). As to with the phospholipids already mentioned, likely causes for these differences were the use of different methods, different nonpolar lipids (cholesterol vs. cholesterol oleate), and different labeling of lipids for quantification purposes.

In another related study, Iwata et al.38 used gas chromatography/mass spectrometry to quantify a cholesterol ester along with other nonpolar lipids associated with silicone hydrogel materials. They recovered <0.2 μg/lens of cholesterol palmitate (cholesterol ester) after 60 hours of incubation from all materials tested (galyfilcon A, lotrafilcon A, lotrafilcon B, balafilcon A, and etafilcon A). This contradicts the findings from our study. A possible explanation for this result is that Iwata et al.38 were attempting to quantify levels of cholesterol ester below what was stated to be their statistically relevant quantification limit of 2.1 μg/lens.36

Zhao et al.5 investigated another important aspect related to nonpolar lipid deposition. They focused on four care systems and their ability to remove cholesterol deposited on silicone hydrogel lenses.5 They found that the amount of cholesterol extracted from a worn lens was directly related to the lens material and the care system used. They also found that the least cholesterol was extracted from lotrafilcon B lenses and that the most cholesterol was extracted from balafilcon A lenses after the care systems were applied. Although it is difficult to make direct comparisons with our work because Zhao et al.5 analyzed cholesterol instead of cholesterol oleate and because their subjects wore their lenses for up to 30 days (thus making this an ex vivo study), Zhao et al.3 recovered a similar range of deposited cholesterol (0.1–8.2 μg/lens).

Finally, to the best of our knowledge only one other study addresses temporal lipid deposition on hydrogel and silicone hydrogel materials. Carney et al.31 found that for polar and nonpolar lipids, maximum lipid absorption was reached by the 12- to 14-day time point for galyfilcon A, whereas all other silicone hydrogels (lotrafilcon A, lotrafilcon B, senofilcon A, and balafilcon A) reached a maximum plateau of absorption by day 20.31 Our data suggest that some of these materials appear to become saturated with lipids relatively quickly (i.e., within the first day) with phosphatidylcholine (lotrafilcon A, etafilcon A, galyfilcon A, senofilcon A, narafilcon A, and comfilcon A) and cholesterol oleate (galyfilcon A, senofilcon A, narafilcon A, comfilcon A, and enfilcon A) but that some materials accumulate more phosphatidylcholine (lotrafilcon B, balafilcon A, and enfilcon A) and cholesterol oleate (lotrafilcon A, lotrafilcon B, and balafilcon A) over time (Figs. 1, 2). The etafilcon A (traditional hydrogel) material saturated with cholesterol oleate quickly, and the amount of cholesterol oleate decreased over time.

In conclusion, these data suggest that hydrogel and some silicone hydrogel materials tend to accumulate phospholipids and cholesterol quickly (1 day). The enfilcon A material appears to absorb phosphatidylcholine the best and the lotrafilcon A material appears to adsorb cholesterol oleate the best, whereas the narafilcon A material appears to adsorb phosphatidylcholine the least and the enfilcon A material appears to absorb cholesterol oleate the least. Additional studies should be conducted to elucidate why one material preferentially absorbs lipids over another.
References


