An earlier age of breast cancer diagnosis related to more frequent use of antiperspirants/deodorants and underarm shaving

K G McGrath

Breast cancer incidence suggests a lifestyle cause. A lifestyle factor used near the breast is the application of antiperspirants/deodorants accompanied by axillary shaving. A previous study did not support a link with breast cancer. If these habits have a role in breast cancer development, women using antiperspirants/deodorants and shaving their underarms frequently would be expected to have an earlier age of diagnosis than those doing so less often. An earlier age of diagnosis would also be expected in those starting to use deodorants and shaving at an earlier age. This is the first study to investigate the intensity of underarm exposure in a cohort of breast cancer survivors. Four hundred and thirty-seven females diagnosed with breast cancer were surveyed. Once grouped by their frequency of underarm hygiene habits, the mean age of diagnosis was the primary end point. Secondary end points included the overall frequency of these habits, and potential usage group confounding variables were evaluated. All statistical tests were two-sided. Frequency and earlier onset of antiperspirant/deodorant usage with underarm shaving were associated with an earlier age of breast cancer diagnosis. Combined habits are likely for this earlier age of diagnosis. In conclusion, underarm shaving with antiperspirant/deodorant use may play a role in breast cancer. It is not clear which of these components are involved. Reviewed literature insinuates absorption of aluminium salts facilitated by dermal barrier disruption. Case-controlled investigations are needed before alternative underarm hygiene habits are suggested.


Key words: Aluminium, antiperspirants, breast cancer, deodorants, lifestyle, underarm shaving

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questionnaire by mail. On the survey there was an optional confidential section for contact information for investigator interview use only. Data collected included patient demographics (age, racial origin, birth country, country of residence), extent of disease at diagnosis, age at menarche, number of live childbirths, fibrocystic breast disease history and family breast cancer history. Using a categorical scoring scale, other risk factors for breast cancer (dietary fat, alcohol intake, oestrogen usage, exercise), and frequency of antiperspirant/deodorant (A/D) use and underarm shaving (S) were recorded. The ages of onset of these underarm hygiene habits were recorded.

Respondents were instructed to check their product labels to properly record if they used antiperspirants, deodorants, or a combined product. This cohort was subdivided into usage groups. The primary end point was the mean age of breast cancer diagnosis compared between the usage groups. The usage groups are as follows: Maximum (Max), Middle (Mid), Minimum (Min), None (Non), and All. The criteria for each group are listed in Table 1. The mean age of breast cancer diagnosis was also compared between subjects starting these habits before the age of 16 and those subjects starting these habits at or after the age of 16. This start age was defined as the mean of the start age of antiperspirant use, deodorant use, and start age of underarm shaving. Secondary end points included the overall frequency of these habits and the effect of potential confounding variables.

**Statistical analysis**

Results from this study were aggregated for analysis, and Stat Works, Inc. (Carrboro, NC, USA) assisted the investigator in performing the data management procedures and statistical analysis, utilizing Version 8.1 of the SAS statistical software package. Categorical data are reported as numbers and percentages of subjects in each category, and continuous measures as number of subjects, means, standard deviations, standard errors of the mean, medians, minimum and maximum values, or confidence intervals as appropriate. For all pair-wise comparisons between the usage groups, the two-sample \( t \)-test was
used. While categorical outcome variables (e.g. exercise amount) were tested for between user groups, differences were tested using the Cochran Mantel Haenszel test with modified ridit scoring. All statistical tests were two-sided and all statistical testing were declared statistically significant if the calculated P-value is $\leq 0.050$.

Results
Of the 1344 questionnaires sent, 437 were returned. The general characteristics of the respondents are shown in Table 2. As presented in this table the age distribution of the respondents was not skewed. Not all respondents had usage group designation, due to missing or incomplete responses. Within all race respondents, the mean age at diagnosis of the Max group was 52.6 years ($n = 35$) compared with: the Mid group 58.6 years ($n = 120$), the Min group 64.9 years ($n = 50$), and the Non group 67.3 years ($n = 32$). The mean differences were: 6 years/ $P = 0.0121$, 12.3 years/$P < 0.0001$, 14.7 years/$P < 0.0001$, respectively. The mean age at diagnosis of the Min group was 2.4 years earlier than the Non group ($P = 0.4243$). Of Caucasian respondents the mean age at diagnosis of the Max group was 53 years ($n = 30$), compared with: the Mid group 59.7 years ($n = 105$), the Min group 65.6 years ($n = 44$) and to the Non group 75 years ($n = 20$). The differences were: 6.8 years/$P = 0.0104$, 12.5 years/$P < 0.0001$, 22 years/$P < 0.0001$, respectively. The mean age at diagnosis of the Min group was 9.5 years earlier than the Non group, $P = 0.0014$. The mean age at diagnosis of the USA-born Max group (all races, $n = 29$) was 16 years earlier than the USA-born Min group (all races, $n = 41$), $P < 0.0001$. Figure 2 summarizes the mean age at diagnosis of all four groups.

Table 3 presents the pair-wise comparisons of each usage group followed by the usage group’s summary statistics in Table 4. As presented in Figure 3, within the all-race Max group, those that began use at an age of $< 16$ ($n = 24$) had a mean age at diagnosis of 46.3 years, 19 years earlier than those starting at age $\geq 16$ with a mean age at diagnosis of 65.3 years ($n = 10$, 1 subject omitted A/D/S start age), $P < 0.0001$. Of All users (Max + Mid + Min), those that began these habits at age $< 16$ ($n = 193$, 12 omitted A/D/S start age) had a mean age of diagnosis of 57 years, 9.6 years earlier than those beginning at age $\geq 16$ with a mean age of diagnosis of 66.6 years ($n = 127$), $P < 0.0001$. The All group (Max + Mid + Min, $n = 205$), had a mean age of diagnosis of 59.1 years, 8.2 years earlier than the Non user group’s mean age of diagnosis of 67.3 years, ($n = 32$), $P = 0.0012$. There was no significant difference in the mean age of diagnosis of those who responded: ‘Did not to never/rarely shave but used A/Ds at any frequency’ ($n = 142$) compared with those who responded: ‘Did not to never/rarely used A/Ds but did shave at any frequency’ ($n = 62$), $P = 0.9201$. Those who used antiperspirants, but not deodorants, and shaved at any frequency ($n = 31$) had a mean age of diagnosis 3 years earlier than those who used deodorants, but not antiperspirants, and shaved at any frequency ($n = 63$), $P = 0.2688$ (Figure 4).
The age distribution of the 437 respondents was not skewed towards younger individuals. The mean age of breast cancer diagnosis was progressively lower proceeding from the Non to Max usage groups (Figure 2). In addition, beginning these habits at an earlier age was associated with a significantly earlier age of diagnosis (Figure 3). These results suggest that combined habits were necessary. Separately done, these habits were not associated with a significant earlier age of diagnosis (Figure 4).

There was no significant difference between each usage group's family history of breast cancer, mean age of menarche and number of live childbirths, dietary fat, oestrogen use, amount of exercise or hard liquor consumption \( (P > 0.05) \). However, the mean beer/wine consumption was not similar between the usage groups \( (P = 0.0113) \). The responses to the beer/wine 'none to rare' category were as follows: Non \( (n = 30, 94\%) \) > Min \( (n = 36, 77\%) \) > Max \( (n = 25, 71\%) \) > Mid \( (n = 75, 62\%) \).

Discussion

Circumstances involving a woman’s environment and lifestyle clearly play a role in the incidence of breast cancer. Environmental and lifestyle investigations have included diet (fat, alcohol, charred meat and fibre), exercise, body habitus, hormones, breastfeeding, reproductive history, smoking, radiation exposure, electromagnetic fields, viruses, and pesticides. None of these have fully explained such a common female cancer. Five to ten per cent of breast cancer has a genetic basis and over 50% of women with breast cancer have yet to be linked to a major risk factor (Carroll, 1975; Dupont and Page, 1985; Harris et al., 1992; Madigan et al., 1995; Collman et al., 1996; Coogan et al., 1996; Zheng et al., 1998; Holmes et al., 1999; Abeloff et al., 2000; Clemons and Goss, 2001). This study suggests that a woman’s underarm hygiene habits may provide such a link. The data from this study are consistent with the hypothesis that the degree of antiperspirant/deodorant usage and axillary shaving is associated with an earlier age of breast cancer diagnosis. The age distribution of the 437 respondents was not skewed towards younger individuals. The mean age of breast cancer diagnosis was progressively lower proceeding from the Non to Max usage groups (Figure 2). In addition, beginning these habits at an earlier age was associated with a significantly earlier age of diagnosis (Figure 3). These results suggest that combined habits were necessary. Separately done, these habits were not associated with a significant earlier age of diagnosis (Figure 4).

Ninety per cent of the United States population uses daily antiperspirants and deodorants (Laden, 1999), and in women, this use is frequently associated with underarm shaving. Figure 1 plots the annual incidence of

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Table 3 Pair-wise comparisons of usage groups

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Max versus Mid</th>
<th>Max versus Min</th>
<th>Max versus Non</th>
<th>Mid versus Non</th>
<th>Min versus Non</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean difference in age of diagnosis</td>
<td>-6.03 ( -10.73, –1.34)</td>
<td>-12.32 ( -18.05, –6.60)</td>
<td>-14.74 ( -21.44, –8.05)</td>
<td>-8.29 ( -10.38, –2.20)</td>
<td>-8.71 ( -13.63, –3.79)</td>
</tr>
<tr>
<td>95% Confidence interval</td>
<td>-12.22/-1.21</td>
<td>-18.38/-6.56</td>
<td>-21.23/-8.13</td>
<td>-10.48/-1.80</td>
<td>-13.59/-3.96</td>
</tr>
<tr>
<td>P-value (t-test)</td>
<td>0.0012</td>
<td>&lt; 0.0001</td>
<td>&lt; 0.0001</td>
<td>0.0028</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Fig. 3

Age-related differences in mean age of breast cancer diagnosis related to onset of underarm antiperspirant/deodorant use and shaving. All users: difference 9.62 years, \( P < 0.0001 \); Max users: difference 19.01 years, \( P < 0.0001 \). Note: Start age=mean start age of antiperspirant use, deodorant use and underarm shaving.

Table 4 Usage group’s summary statistics (all races/Caucasian only)

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Max</th>
<th>Mid</th>
<th>Min</th>
<th>Non</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age of diagnosis</td>
<td>52.8/53.03</td>
<td>58.63/59.71</td>
<td>64.92/65.57</td>
<td>67.34/75.05</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>13.34/13.95</td>
<td>12.08/11.95</td>
<td>12.86/11.06</td>
<td>14.10/9.28</td>
</tr>
<tr>
<td>Sample size</td>
<td>35/30</td>
<td>120/105</td>
<td>50/44</td>
<td>32/20</td>
</tr>
<tr>
<td>SE of mean</td>
<td>2.25/2.55</td>
<td>1.10/1.17</td>
<td>1.82/1.67</td>
<td>2.49/2.08</td>
</tr>
<tr>
<td>Lower 95% conf. limit</td>
<td>48.02/47.82</td>
<td>56.45/57.40</td>
<td>61.26/62.21</td>
<td>62.26/70.71</td>
</tr>
<tr>
<td>Upper 95% conf. limit</td>
<td>57.18/58.24</td>
<td>60.82/62.03</td>
<td>68.58/68.93</td>
<td>72.43/79.39</td>
</tr>
<tr>
<td>Minimum age</td>
<td>35/35</td>
<td>35/35</td>
<td>30/37</td>
<td>40/48</td>
</tr>
<tr>
<td>Median age</td>
<td>47/48.5</td>
<td>59/60</td>
<td>61/67</td>
<td>67.5/77.5</td>
</tr>
<tr>
<td>Maximum age</td>
<td>79/79</td>
<td>84/84</td>
<td>92/92</td>
<td>87/87</td>
</tr>
</tbody>
</table>
female breast cancer with the annual sales of antiperspirants/deodorants in the United States (Roush et al., 1987; Harris et al., 1992; SEER Cancer Incidence Public-Use Database, 2001; US Cosmetic and Toiletries Market, 2001). Often used in combination, antiperspirants (for dryness and malodour) and deodorants (for malodor) contain numerous components, including aluminium and other metal salts, antimicrobials, aliphatic alcohols and glycols, and fragrances (Maken et al., 1999; Benohanian, 2001). Adverse events from these topically applied products have included clothing damage, skin irritation/inflammation, contact dermatitis and granulomas. There has been no correlation to breast cancer (Montemarano et al., 1997; Laden, 1988; Maken et al., 1999; Jones, 2000; Scheman, 2000; Benohanian, 2001; Robb-Nicholson, 2001; Mirick et al., 2002).

The most consistently used components since the commercial introduction of antiperspirants in 1903 are aluminium salts to promote underarm dryness (Laden, 1999). Aluminium-based compounds persist in today’s antiperspirants. Daily transdermal exposure over long periods of time of metal-containing compounds in personal-care products has raised some health concerns but data are lacking regarding bioavailability (Hostynek et al., 1993). Intact human skin, dermal absorption of aluminium reveals only shallow epidermal penetration secondary to the metal’s avid formation of complexes with skin proteins (Hostynek et al., 1993). Experiments with adult mice, naked mouse pups and excised skin patches taken from adult mice have shown that the shaved skin was not a barrier to the absorption of topically applied aluminium salts (Anane et al., 1995). Transplacental passage of aluminium from pregnant mice to fetus organs occurred after maternal transcutaneous exposure to aluminium salts (Anane et al., 1997). Aluminium appeared in the milk of lactating rabbits following subcutaneous injections of aluminium lactate (Yokle and McNamara, 1985).

There are numerous animal and plant studies suggesting an adverse role of aluminium, especially when present in its active moiety (Al³⁺), affecting a number of biological processes, including cross-linking of DNA strands and enzyme system modification (DeBoni et al., 1980; Karlik et al., 1980; Wenk and Stemmer, 1982). Al³⁺ has an extremely high charge (3+) to ionic radius (0.05 nm) causing it to rapidly penetrate nuclear compartments, bind tightly to nucleic acids, ATP and heterochromatin, and inhibit or adversely affect DNA template activity, linker histones, and DNA and RNA polymerase enzyme systems (DeBoni et al., 1974, 1980; Crapser et al., 1980; Crapper McLachlan and DeBoni, 1977, 1980; Matsumoto and Morimura, 1980; Wen and Wisniewski, 1985; Wedrychowski et al., 1986). Associated with elevated concentrations of aluminium are a decreased rate of DNA synthesis, an increase in DNA replication errors, and an increase in the affinity of linker histones for DNA (Berlyne et al., 1972; Laussac and Commenges, 1983). These studies provide circumstantial evidence that Al³⁺ has oncogenic potential.

Experimental data indicate that both depth of penetration, which determines duration of antiperspirant effect, and relative antiperspirant efficacy are significantly dependent on the prevailing pH (size and charge). Thus, the prevailing pH is crucial and small pH changes within a relatively narrow range lead to formation of Al³⁺/H₂O/OH⁻ complexes of markedly different solubility and bioavailability. In aluminium hydroxide solutions, for example, concentrations of free Al³⁺ at pH 4.2 are 100–1000 times greater than at pH 6.2. Generally, increases in solution pH below 5.5 result in exponential increases in Al³⁺ concentrations (Kaehny et al., 1977; Martin, 1991; Borak and Wise, 1998). An analogy is acid rain releasing Al³⁺ from rocks and soil into lakes and streams (Dyearensen et al., 1987; Martin, 1991, 1994). Axillary sweat has a normal pH of 4.5–7.4, with increased sweating rates associated with higher pH values (Herrmann and Sulzberger, 1958; Jakubovic and Ackerman, 1985; Quinton et al., 1999). However, the lower pH of some aluminium salts (e.g. aluminium chloride pH < 4, aluminium chlorhydrate pH 4.38) and the sweat reduction that antiperspirants cause, along with microbial action on axillary apocrine sweat, could cumulatively contribute to an acidic underarm environment, ‘axillary acid rain’ (Herrmann and Sulzberger, 1958; Lansdown, 1995; McGee et al., 1998; Labows et al., 1999; Quinton et al., 1999). This may explain, at least in part, why world breast cancer incidence is lower in countries with different cultural habits or less disposable income for western axillary hygiene practices or in areas with less media exposure, as in some rural areas (Donegan et al., 1988; Cancer Facts and Figures, 1998; Laden, 1999).

Unique valveless and bi-directional lymphatic flow exists between the breast and axillae. Shared is a rich anastomoses, which could easily provide direct and chronic exposure of breast tissue to Al³⁺ or other applied axillary substances (Taylor, 1959; Moore and Dalley, 1999). This may also explain the most frequent tumour location occurring in the upper-outter breast (Lester and Cotran, 1999). If topical absorption occurs, normal plasma clearing of Al³⁺ by transferrin and citrate binding may be insufficient with daily exposure, especially over decades (Martin, 1997; Ohman and Martin, 1994).

Al³⁺ clearance through human breastfeeding has not been studied, yet the longer women breastfeed the more they are protected against breast cancer (Collaborative Group on Hormonal Factors in Breast Cancer, 2002).
USA breast cancer incidence by race/ethnicity versus body hair (Jakubovic and Ackerman, 1992; Dawber et al., 1998; Anon., 1998; Freinkel, 2000; SEER Cancer Incidence Public-Use Database, 2001; Collaborative Group on Hormonal Factors in Breast Cancer, 2002).

Fig. 5

Acknowledgements

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References


Breast cancer and underarm deodorants

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