Body-Site Distribution of Melanocytic Nevi in Young Australian Children

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Objective: To investigate the body-site distribution of melanocytic nevi (MN) with respect to habitually and intermittently sun-exposed surfaces.

Design: Cross-sectional survey of MN prevalence.

Setting: Townsville (19.16°S), Queensland, Australia.

Participants: Random sample of 506 1- to 6-year-old white children who were born and raised in Townsville (response, 87.6%).

Main Outcome Measures: Site-specific counts and densities (number per square meter) of MN.

Results: Densities of MN of all sizes were highest on the outer forearms, followed by the outer upper arms, neck, and face. The feet had the lowest density of MN. Densities of MN of 2 mm or greater were highest on the upper arms and trunk. Boys had higher densities of MN of all sizes on the neck than girls (P = .002). Girls had higher densities of MN of 2 mm or greater on the lower legs (P = .006) and thighs (P = .005) than boys. Habitually sun-exposed body sites had higher densities, particularly of small MN, than relatively sun-protected sites, and larger MN were most prevalent on the intermittently exposed skin of the trunk.

Conclusions: These children have higher total body and site-specific MN counts and densities than children from elsewhere, and their MN are distributed over the body in a way that implicates exposure to sunlight. As sun exposure in childhood and MN are risk factors for melanoma, intervention studies are required to determine if MN can be prevented.

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The number of melanocytic nevi (MN) is an important risk marker for cutaneous melanoma (CM).1 Although their causative role is uncertain, MN may be risk markers sharing similar causative factors with CM, or direct precursors for a substantial proportion of CMs.2,6 Epidemiological studies1-5 suggest that sun exposure is a common causative factor for both MN and CM. While total lifetime or intermittent sun exposure is difficult to estimate for adults, sun exposure during childhood or adolescence has been confirmed as a causative factor for CM,7-10 with MN counts in children increasing with the amount and intensity of sun exposure.3,11-13

Attempts have been made to determine whether MN and CM have similar body-site distributions,6,14-16 but have been inconclusive. Studies6,14,16 in temperate climates have shown that MN counts increase in the first 2 decades, peak in the third, and decline after the fourth decade. In contrast, incidence rates for CM are highest from age 50 years and older.17 Thus, the body-site distribution of MN in patients with CM may not reflect the site distribution relevant for the development of CM, as initiation of the disease may date back to childhood or adolescence.

This study provides baseline information on the body-site distribution of MN in young Australian children raised in an intense UV radiation environment. Analysis focuses on sun-exposed and sun-protected body sites in relation to models of the natural history of CM and MN16 and the body-site distribution of the latter in children.

Of the 506 children, 98.8% had MN. The 2 boys and 4 girls without MN were aged between 1 and 2 years. Forty-eight of 103 children between 1 and 2 years of age had MN of 2 mm or greater. Total MN counts, and counts of MN of 2 mm or greater, increased with age (P < .001, respectively).
SUBJECTS AND METHODS

In 1991, 516 one- to six-year-old children who were born and raised in Townsville (19.16°S), Australia, were included in a cross-sectional study of MN prevalence (response, 87.6%). All children had white parents, but 10 children who had 2 or more non-European grandparents were excluded from analysis. Further details of this sample are published elsewhere.5 The 506 children (259 boys, 247 girls) were examined for MN according to a standard international protocol.24 Thirty different body sites that differentiated between sun-exposed and less-exposed areas were distin-
guished. The scalp, buttocks, and genitals were not exam-
ined. Informed consent was given by the children’s moth-
ers, who also completed a standardized questionnaire regarding their child’s ancestry and sun exposure.

Malignant nevi were defined as brown- to black-
pigmented macules or papules of any size, darker in color than the surrounding skin, excluding lesions with the clinical characteristics of freckles, solar lentigines, or cafe au lait spots.25 As it is not possible to distinguish between MN and lentigo simplex clinically, and biopsy is not feasible in chil-
dren, lentigo simplex may have been misclassified as MN. Heavy freckling may lead to undercounting of MN that may be difficult to distinguish in an area of freckling. However, these caveats apply to all observational studies of MN.

Skin-colored palpable lesions with the morphologi-

cal features of compound or intradermal MN, halo nevi, nevi spilis, congenital nevuslike nevi, and blue nevi were counted separately and are included in total MN counts. Cafe au lait spots were recorded separately. The distribution of freckling was assessed on the face, arms, and shoul-
ders using a standardized semiquantitative scale.

Malignant nevi were measured with a clear plastic film imprinted with circles of 2-, 3-, 4-, and 5-mm diam-
eters attached to an illuminated (×3) magnifying glass. Les-
ions were measured with the skin unstretched and judged to be of a specific size if the lesion touched both sides of the circle. All children were examined by one of us (S.L.H.), who was previously trained in the recognition of MN by dermatologists.13 Replicate counts on 9% of the subjects showed 93% intraobserver reliability.

Height and weight of the children were measured to es-
timate the total surface area of the body.22 Proportions of to-
tal surface area attributed to the head, trunk, and upper and lower extremities were based on estimations by Boyd23 and modified according to results of Berkow24 and Lund and Brow-
der5 to allow further differentiation of body sites (Table 1). Surface proportions of the inner and outer upper arms and forearms, and the anterior and posterior thighs and lower legs were calculated under the assumption that each sub-
site covers 50% of the surface proportion of the upper arms, forearms, thighs, and lower legs. To compare the effects of exposure at different sites, it is necessary to compare numbers of MN per unit area (density). Malignant nevus den-
sities were calculated as follows:

\[
\text{Density/m}^2 = \frac{\text{No. of Nevi at Site}}{\text{Body Surface Area} \times \text{Proportion Body Surface at Site}}
\]

For total body MN densities, adjustments were made to account for the scalp, buttocks, and genitals, reducing the body surface area on average by 14.1% (Table 1).

Since total and site-specific MN counts and densities were positively skewed, median values and interquartile ranges are given. Sex differences were assessed with non-
parametric Wilcoxon-Mann-Whitney tests.26 Multiple lin-
ear regression analysis was performed with log-
transformed MN counts and densities to test for influence of age (in months) and sex. Differences in site-specific counts and densities of MN were assessed by nonparametric-
paired Wilcoxon and Friedman tests.26 Statistical analysis was performed using SPSS for Windows, Version 6.1.3 (SPSS Inc, Chicago, Ill). A significance level of .05 was assumed.

(Table 2) and there was no significant sex difference in either count.

Counts of MN of all sizes and MN of 2 mm or greater counted separately on the face, neck, trunk, upper arms, forearms, hands, feet, lower legs, and thighs increased steadily with age (P<.001, respectively) (Table 2). Across all ages, counts of MN of all sizes were highest on the trunk, followed by the upper arms, thighs, and forearms (over-
all test comparing body sites: P<.001, respectively, for MN of all sizes and ≥2 mm). Hands and feet had fewer MN than other sites, but only comprise a small proportion of the body’s total surface area. Boys had more MN of all sizes on the neck than girls (P<.05). In contrast, girls had more MN of 2 mm or greater on the lower legs (P<.05) and thighs (P<.01). These sex differences remained statisti-
cally significant after adjusting for age (P<.01, respec-
tively). More girls had MN of 2 mm or greater on the lower legs (43.3% vs 33.2%) and thighs (51.0% vs 36.7%) than boys. In total, 73 children had 82 MN of 5 mm or greater. Most large MN were on the trunk (35 [42.7%] of 82), arms (15 [18.3%] of 82, 8 on forearms and 7 on upper arms), and thighs (12 [14.6%] of 82).

Overall, densities of MN of all sizes were highest on the forearms, upper arms, neck, and face. The feet had the lowest MN densities (overall test, P<.001); (Table 3; Figure). The highest densities of MN of 2 mm or greater were found on the upper arms and trunk (overall test, P<.001). The results of MN density considered by age and sex reflect those for MN counts. That is, densities of MN of all sizes, and 2 mm or greater, increased significantly with age for all body sites (P<.001, respectively); densities of MN of all sizes on the neck were higher for boys than girls (P<.01, adjusted for age), particularly the back of the neck (P<.005); and densities of MN of 2 mm or greater on the lower legs and thighs were higher for girls than boys (P<.01, respectively, adjusted for age). Densities of MN of all sizes, and 2 mm or greater, were highest (among whole body sites) for the neck of 5- and 6-year-
olds. Only 1 of the 6-year-olds had no MN on the neck, 22.2% had no MN of 2 mm or greater on the neck, 92.6% had a neck-specific density of 100 or greater for MN of all sizes, and 38.9% presented with a neck-specific density of 100 or greater for MN of 2 mm or greater.

The median number of MN of all sizes on the inner upper arms as well as the inner forearms (median, 1, re-
spectively) was significantly lower than on the outer upper arms (median, 4) and outer forearms (median, 3) (P<.001, respectively). There was no significant differ-
ence between the anterior and posterior lower legs (median, 1; \( P = .09 \), respectively). However, the difference between the anterior (median, 3) and posterior (median, 1) thighs was highly significant \(( P < .001 \)).

Densities of MN of all sizes, and 2 mm or greater, were significantly lower on the anterior upper arms and inner forearms than on the outer arms and outer forearms \(( P < .001 \), respectively) and were significantly lower on the posterior lower legs and thighs than on the anterior lower legs and thighs (lower legs: all sizes, \( P < .05 \); \( \geq 2 \) mm, \( P < .01 \); thighs: all sizes, \( P < .001 \); \( \geq 2 \) mm, \( P < .001 \)) (Table 4). The density of MN of all sizes was also higher on the dorsal surface of the feet (median, 0; mean, 32.3) and hands (median, 47.2; mean, 65.0) compared with the soles of the feet (median, 0; mean, 1.3), and the palms of the hands (median, 0; mean, 5.3) \(( P < .001 \), respectively). Densities were highest on the outer forearms and outer upper arms, consistently for all ages (Table 4). One 6-year-old girl had 31 nevi on the outer upper arm and 24 nevi on the outer forearm, a surface equivalent to 6.7% of the total surface area at this age.

These children had extremely high total body MN counts, with the highest MN densities occurring on the outer forearms, outer upper arms, neck, and face, all of which are highly exposed to the sun. Children from Townsville have total body and site-specific MN counts and densities that are higher than those reported for children the same age or slightly older from other Australian states\(^{11,12,28-30}\) and abroad\(^{13,15,16,29-30}\) even though the prevalence of MN is similar at birth.\(^{31,32}\) This may be partly explained by geographic differences in ambient solar UV radiation and annual temperature variation, which influences the length of the season during which clothing offering little protection from the sun is worn. Using the DNA Action Spectrum of Setlow\(^2\) and monthly ratios for cloud cover,\(^34\) the calculated average annual total DNA-weighted UV radiation received in Townsville \((19^\circ \text{S}, 1.91 \times 10^6 \text{ J/m}^2)\) is higher than for other Australian cities (Perth, 32°S; 1.48 \times 10^6 \text{ J/m}^2; Sydney, 34°S; 1.1 \times 10^6 \text{ J/m}^2) where MN sur-

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**Table 1. Proportion of Surface Area of Different Body Sites for 1- to 6-Year-Old Children**

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Face†</th>
<th>Scalp</th>
<th>Neck</th>
<th>Genitals</th>
<th>Buttocks</th>
<th>Trunk</th>
<th>Forearms</th>
<th>Upper Arms</th>
<th>Hands</th>
<th>Feet</th>
<th>Lower Legs</th>
<th>Thighs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7.41</td>
<td>9.79</td>
<td>2.03</td>
<td>1.00</td>
<td>5.06</td>
<td>26.32</td>
<td>5.28</td>
<td>7.02</td>
<td>5.48</td>
<td>7.13</td>
<td>10.19</td>
<td>13.25</td>
</tr>
<tr>
<td>2</td>
<td>6.55</td>
<td>8.65</td>
<td>1.98</td>
<td>0.97</td>
<td>4.94</td>
<td>25.70</td>
<td>5.56</td>
<td>7.39</td>
<td>5.59</td>
<td>7.40</td>
<td>10.81</td>
<td>14.47</td>
</tr>
<tr>
<td>3</td>
<td>6.21</td>
<td>8.19</td>
<td>1.98</td>
<td>0.97</td>
<td>4.94</td>
<td>25.71</td>
<td>5.68</td>
<td>7.57</td>
<td>5.55</td>
<td>7.29</td>
<td>10.90</td>
<td>15.01</td>
</tr>
<tr>
<td>4</td>
<td>5.91</td>
<td>7.80</td>
<td>1.95</td>
<td>0.96</td>
<td>4.87</td>
<td>25.32</td>
<td>5.98</td>
<td>7.97</td>
<td>5.43</td>
<td>7.20</td>
<td>11.02</td>
<td>15.60</td>
</tr>
<tr>
<td>5</td>
<td>5.65</td>
<td>7.45</td>
<td>1.95</td>
<td>0.96</td>
<td>4.85</td>
<td>25.25</td>
<td>6.01</td>
<td>7.80</td>
<td>5.57</td>
<td>7.07</td>
<td>11.11</td>
<td>16.16</td>
</tr>
<tr>
<td>6</td>
<td>5.43</td>
<td>7.17</td>
<td>1.97</td>
<td>0.97</td>
<td>4.91</td>
<td>25.55</td>
<td>5.78</td>
<td>7.69</td>
<td>6.12</td>
<td>6.99</td>
<td>11.22</td>
<td>16.21</td>
</tr>
</tbody>
</table>

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**Table 2. Median Number of Melanocytic Nevi of All Sizes (First Row) and Median Number of Melanocytic Nevi \( \geq 2 \text{ mm} \) (Second Row) by Body Site and Age in Australian Children**

<table>
<thead>
<tr>
<th>Body Site†</th>
<th>1 y (n = 103)</th>
<th>2 y (n = 97)</th>
<th>3 y (n = 95)</th>
<th>4 y (n = 80)</th>
<th>5 y (n = 77)</th>
<th>6 y (n = 54)</th>
<th>Total (n = 506)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Face‡</td>
<td>1 (0-2)</td>
<td>2 (1-4)</td>
<td>3 (1-4)</td>
<td>5 (3-8)</td>
<td>5 (3-8)</td>
<td>5 (3-10)</td>
<td>3 (1-6)</td>
</tr>
<tr>
<td>Neck</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-1)</td>
<td>1 (0-2)</td>
<td>1 (0-2)</td>
<td>1 (0-3)</td>
<td>0 (0-1)</td>
</tr>
<tr>
<td>Trunk</td>
<td>1 (0-2)</td>
<td>3 (1-5)</td>
<td>8 (5-11)</td>
<td>12 (8-15)</td>
<td>16 (10-22.5)</td>
<td>20 (14-31.25)</td>
<td>7 (2-15)</td>
</tr>
<tr>
<td>Upper arms</td>
<td>1 (0-2)</td>
<td>3 (1-6)</td>
<td>6 (4-9)</td>
<td>8 (5-11)</td>
<td>10 (6-17)</td>
<td>12 (8-18.25)</td>
<td>5 (2-10)</td>
</tr>
<tr>
<td>Forearms</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-1)</td>
<td>1 (0-2)</td>
<td>2 (0-3.5)</td>
<td>2.5 (1-5.25)</td>
<td>1 (0-2)</td>
</tr>
<tr>
<td>Hands</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Thighs</td>
<td>1 (0-1)</td>
<td>2 (1-5)</td>
<td>5 (3-8)</td>
<td>6 (3.25-10)</td>
<td>8 (4-14)</td>
<td>10 (6-15.25)</td>
<td>4 (1-8)</td>
</tr>
<tr>
<td>Lower legs</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Feet</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Total body</td>
<td>6 (3-11)</td>
<td>19 (12-33)</td>
<td>35 (23-52)</td>
<td>51 (37-63)</td>
<td>61 (42-88)</td>
<td>73 (55-103)</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>

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*Reported by Boyd\(^2\) and modified according to Berkow\(^2\) and Lund and Browder.\(^3\) All values are percentages.
†Face including ears.
‡Face including ears.

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veys have been conducted\textsuperscript{13,27} (Aurel Moise, MSc, written communication, April 1997). Consequently, people mostly wear lightweight summer clothing that exposes the forearms, neck, and most or all of the upper arms and legs. As in children from Perth,\textsuperscript{27} the sites with the highest MN densities are those exposed to the sun in summer clothing and not protected by the structure of the human body, or its movement. The arms are often held so that the medial surface of the upper limb is protected by the trunk, while the lateral surface is exposed. When sitting, the posterior thighs and the soles of the feet are shielded from the sun, whereas the anterior thighs may be exposed (depending on clothing). In keeping with this pattern, we found more MN on the anterior than the posterior surface of the lower limbs that was caused by an excess of MN on the anterior thighs compared with the posterior thighs, rather than the lower legs where there was little difference in MN density between the anterior and posterior surface. Likewise, MN were less concentrated on the soles than on the dorsa of the feet. Also, boys had significantly more MN on the posterior neck than girls that may be because of girls having longer hair.

Apparent anomalies in nevus densities on the anterior and posterior lower legs may be attributable to these sites having equal opportunities for exposure when standing and perhaps even when sitting. The only markedly different opportunity for sun exposure occurs when lying down. However, sunbaking is a relatively rare pastime in Townsville, particularly for children. Melanocytic nevi were more concentrated on the dorsum than the palm, but as reported by others,\textsuperscript{27} there were fewer MN than expected on this heavily exposed surface. This deviation from the sun-exposure hypothesis is more difficult to explain. Site-specific CM rates in Queensland\textsuperscript{35} and other populations\textsuperscript{36,37} also show few cases at this site. Green and MacLennan\textsuperscript{35} suggest that this may be due to a protective factor or a differential susceptibility according to site. The fact that in Queensland the proportion of CMs with adjacent nevi at different body sites is not readily explained by site-specific MN densities\textsuperscript{19} suggests that MN are more likely to be risk markers for CM than obligatory precursors.

The model proposed by Armstrong\textsuperscript{18} suggests 3 pathways linking a normal melanocyte to CM: via a melanocytic nevus cell; via an MN; or by some other undefined mode. For the first 2 pathways, nevus cells are first-step

**Table 3. Median Densities of Melanocytic Nevi of All Sizes (First Row) and Median Densities of Melanocytic Nevi \( \geq 2 \text{mm} \) (Second Row) for Major Sites of the Body by Age\textsuperscript{*}**

<table>
<thead>
<tr>
<th>Body Site†</th>
<th>1 y (n = 103)</th>
<th>2 y (n = 97)</th>
<th>3 y (n = 95)</th>
<th>4 y (n = 80)</th>
<th>5 y (n = 77)</th>
<th>6 y (n = 54)</th>
<th>Total (N = 506)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Face‡</strong></td>
<td>25 (0-54)</td>
<td>54 (26-105)</td>
<td>67 (26-116)</td>
<td>108 (75-181)</td>
<td>117 (64-177)</td>
<td>118 (68-219)</td>
<td>74 (26-132)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-26)</td>
<td>24 (0-50)</td>
<td>23 (0-43)</td>
<td>24 (0-64)</td>
<td>0 (0-27)</td>
</tr>
<tr>
<td><strong>Neck</strong></td>
<td>0 (0-0)</td>
<td>83 (0-97)</td>
<td>84 (0-164)</td>
<td>158 (76-288)</td>
<td>200 (116-337)</td>
<td>269 (135-465)</td>
<td>93 (0-214)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-76)</td>
<td>0 (0-78)</td>
<td>63 (0-97)</td>
<td>65 (0-139)</td>
<td>0 (0-68)</td>
</tr>
<tr>
<td><strong>Trunk</strong></td>
<td>8 (0-14)</td>
<td>21 (7-35)</td>
<td>50 (30-72)</td>
<td>69 (46-85)</td>
<td>83 (55-117)</td>
<td>97 (69-136)</td>
<td>41 (14-80)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-7)</td>
<td>12 (5-23)</td>
<td>21 (0-34)</td>
<td>25 (10-42)</td>
<td>35 (20-58)</td>
<td>7 (0-26)</td>
</tr>
<tr>
<td><strong>Upper arms</strong></td>
<td>30 (0-59)</td>
<td>64 (25-126)</td>
<td>124 (73-193)</td>
<td>137 (90-189)</td>
<td>152 (90-173)</td>
<td>184 (136-277)</td>
<td>102 (45-174)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>19 (0-42)</td>
<td>20 (0-54)</td>
<td>32 (6-59)</td>
<td>40 (16-86)</td>
<td>14 (0-39)</td>
<td></td>
</tr>
<tr>
<td><strong>Forearms</strong></td>
<td>38 (0-70)</td>
<td>92 (33-169)</td>
<td>131 (77-202)</td>
<td>154 (100-240)</td>
<td>148 (100-234)</td>
<td>171 (121-245)</td>
<td>119 (42-190)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-31)</td>
<td>0 (0-33)</td>
<td>23 (0-46)</td>
<td>22 (0-46)</td>
<td>24 (0-64)</td>
<td>0 (0-34)</td>
</tr>
<tr>
<td><strong>Hands</strong></td>
<td>0 (0-33)</td>
<td>30 (0-35)</td>
<td>29 (0-75)</td>
<td>29 (0-76)</td>
<td>38 (9-11)</td>
<td>39 (20-65)</td>
<td>27 (0-57)</td>
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<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td></td>
</tr>
<tr>
<td><strong>Thighs</strong></td>
<td>15 (0-17)</td>
<td>27 (12-60)</td>
<td>49 (29-79)</td>
<td>56 (30-94)</td>
<td>61 (33-112)</td>
<td>73 (46-112)</td>
<td>40 (16-76)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-12)</td>
<td>0 (0-12)</td>
<td>9 (0-18)</td>
<td>8 (0-16)</td>
<td>7 (0-22)</td>
<td>0 (0-13)</td>
</tr>
<tr>
<td><strong>Lower legs</strong></td>
<td>0 (0-34)</td>
<td>40 (18-67)</td>
<td>51 (26-95)</td>
<td>50 (25-100)</td>
<td>60 (22-97)</td>
<td>64 (26-104)</td>
<td>39 (17-82)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-15)</td>
<td>0 (0-15)</td>
<td>0 (0-14)</td>
<td>11 (2-22)</td>
<td>4 (0-22)</td>
<td>0 (0-15)</td>
</tr>
<tr>
<td><strong>Feet</strong></td>
<td>0 (0-0)</td>
<td>0 (0-25)</td>
<td>18 (0-22)</td>
<td>19 (0-36)</td>
<td>18 (0-35)</td>
<td>17 (0-37)</td>
<td>0 (0-24)</td>
</tr>
<tr>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td></td>
</tr>
<tr>
<td><strong>Total body</strong></td>
<td>14 (7-28)</td>
<td>40 (24-61)</td>
<td>64 (43-101)</td>
<td>81 (60-100)</td>
<td>86 (59-131)</td>
<td>103 (75-138)</td>
<td>Not applicable</td>
</tr>
<tr>
<td>0 (0-5)</td>
<td>4 (2-10)</td>
<td>11 (5-20)</td>
<td>15 (10-26)</td>
<td>18 (10-30)</td>
<td>24 (14-42)</td>
<td>Not applicable</td>
<td></td>
</tr>
</tbody>
</table>

* Interquartile ranges are within parentheses.
† Nevus counts were not performed on buttocks, genital region, and scalp.
‡ Face including ears.
As we found that large (>3 mm) MN were concentrated on the intermittently exposed skin of the trunk and that small MN were concentrated on habitually exposed body sites, we agree with Richard and coworkers who suggest that intense episodic sun exposure, characteristic of intermittently exposed sites, causes small MN to grow. We propose that acute episodic exposure of unprotected skin may cause MN to grow in diameter, and that the degree of exposure for MN growth may be higher than the dose causing proliferation of melanocytes to form a nevus.

Table 4. Median Densities of Melanocytic Nevi of All Sizes (First Row) and Median Densities of Melanocytic Nevi >2 mm (Second Row) for Selected Subsites of the Body by Age*

<table>
<thead>
<tr>
<th>Body Site</th>
<th>1 y (n = 103)</th>
<th>2 y (n = 97)</th>
<th>3 y (n = 95)</th>
<th>4 y (n = 80)</th>
<th>5 y (n = 77)</th>
<th>6 y (n = 54)</th>
<th>Total (N = 506)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inner upper arm</td>
<td>0 (0-51)</td>
<td>0 (0-51)</td>
<td>44 (0-121)</td>
<td>70 (32-117)</td>
<td>91 (49-162)</td>
<td>104 (65-164)</td>
<td>48 (0-106)</td>
</tr>
<tr>
<td>Outer upper arm</td>
<td>57 (0-110)</td>
<td>95 (47-177)</td>
<td>165 (90-304)</td>
<td>196 (117-306)</td>
<td>189 (126-388)</td>
<td>258 (189-420)</td>
<td>141 (61-260)</td>
</tr>
<tr>
<td>Inner forearm</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Outer forearm</td>
<td>70 (0-68)</td>
<td>135 (60-293)</td>
<td>196 (108-319)</td>
<td>243 (137-364)</td>
<td>234 (128-347)</td>
<td>289 (158-392)</td>
<td>164 (70-306)</td>
</tr>
<tr>
<td>Anterior thigh</td>
<td>27 (0-32)</td>
<td>48 (22-96)</td>
<td>71 (39-126)</td>
<td>75 (51-133)</td>
<td>87 (48-143)</td>
<td>94 (48-155)</td>
<td>59 (24-105)</td>
</tr>
<tr>
<td>Posterior thigh</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Anterior lower leg</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Posterior lower leg</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
<td>0 (0-0)</td>
</tr>
</tbody>
</table>

* Interquartile ranges are within parentheses.

Mutations in a sequence thought to be triggered by exposure to UV radiation in a susceptible host. Our results provide support for Armstrong's pathway from melanocyte to MN, which is said to result from mutations caused by exposure to sunlight, followed by a period of clonal expansion as a result of the same. However, the model states that not all altered melanocytes (nevocytes) will undergo sufficient clonal expansion to produce a visible nevus before undergoing malignant transformation, and nevocytes can be destroyed by biological mechanisms or lose their ability to progress. Thus, the nevus cell pathway explains why precursor lesions cannot be identified for all melanomas.

As we found that large (>5 mm) and moderately large (>3 mm) MN were concentrated on the intermittently exposed skin of the trunk and that small MN were concentrated on habitually exposed body sites, we agree with Richard and coworkers who suggest that intense episodic sun exposure, characteristic of intermittently exposed sites, causes small MN to grow. We propose that acute episodic exposure of unprotected skin may cause MN to grow in diameter, and that the degree of exposure for MN growth may be higher than the dose causing proliferation of melanocytes to form a nevus. Intense sun exposure is more likely in environments with high ambient UV radiation and may explain why large MN are more concentrated on the trunk of children from Townsville than from elsewhere. Perhaps larger MN are less prevalent on habitually exposed sites because the melanocytes are protected by the thickening of the stratum corneum and/or deposition of melanin in children who can tan. It also is conceivable that a sufficient dose of UV radiation could be received by the melanocyte, through tanned skin, in individuals who spend a lot of time outdoors in environments with intense solar UV radiation.

The development of atypical features in a susceptible benign nevus may also be associated with an accumulation of mutations in response to sunlight exposure in a susceptible host or may be associated with a different site-dependent susceptibility of the melanocytes to dysplasia and/or malignant transformation as proposed by Green. The latter could be incorporated into the host’s response in the model of Armstrong to explain why atypical nevi are found primarily on the trunk of older fair-skinned children from Townsville and why a high proportion of nevus-associated melanomas are located on the back.

Despite a lack of congruence in all aspects of the anatomical distribution of CM in adults and MN in children, the similarities deserve consideration. In Queensland, MN and CM are common on several body sites that are always exposed to the sun. In 1987, the highest density of invasive CM occurred on the ears in men and the face in women, followed by the shoulders for both sexes. High densities of MN were found on the face (including ears) of children born in Townsville between 1985 and 1990, although the densities were not as high as they were for the forearms, upper arms, and neck. Nevertheless, the predilection of MN for the forearms of children is similar to the increase in incidence of CM at this site between 1979/1980 and 1987. Melanocytic nevi of all sizes were more prevalent on the neck of boys than girls. Similarly, over the same period, the incidence of CM on the neck doubled in males and declined significantly in females. Girls had more MN of 2 mm or greater on the lower limbs than boys. Significant increases were also seen for incidence of CM on the thighs of women. While we are forced to compare CM incidence in adults and MN prevalence in children, the similarities that exist may reflect similar sun-related behaviors.

Our study is unique because it focuses on young white children living in an intense solar UV radiation environment. Our results suggest that MN develop preferentially on maximally sun-exposed sites in young children and bear some resemblance to the site-specific incidence of CM in Queensland. As sun exposure in childhood and MN are risk factors for CM, the development of MN should be prevented as much as possible.
sible. Intervention studies are required to determine whether a significant proportion of MN can be prevented by reducing sun exposure in early childhood.

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