Acrylic nail curing UV lamps: High-intensity exposure warrants further research of skin cancer risk

To the Editor: Recent media headlines have highlighted safety concerns regarding skin cancer risk and ultraviolet (UV) lamps used to cure artificial nail acrylics. We also read with great interest a recently published article by Markova and Weinstock refuting this risk.3

Wanting to evaluate these assertions, we measured the UV exposure from different UV nail lamps used in nail salons using two different measurement methods. We used dosimeters (Biosense Viospor, Bornheim, Germany) that measure DNA damage caused by UV irradiation to viable spores in minimal erythema dose (MED) and joules/m². Additionally, we used a spectrometer (Ocean Optics USB2000, Dunedin, FL) calibrated for absolute UV irradiance for a snapshot comparison to solar radiation.

Following the manufacturer’s instructions for curing acrylic nails using UV light and the assumption that the nails would be refinished every 3 weeks, or 17 times per year, we exposed the dosimeters for an equivalent cumulative dose over 1 year. The Biosense dosimeters measured 0.6 MED/hour for phototype II skin. The curing time recommended by the manufacturers varied the MED from 0.06 to 0.09 per treatment. These exposures amounted to yearly cumulative totals of between 1.1 and 1.5 MEDs. The total energy was 285 and 386 J/m². The total J/m² was 15 and 22.5 per nail session (Table I). The International Commission on Non-Ionizing Radiation Protection set the exposure limit for outdoor workers and recreationalists at 30 J/m² for 8 hours.4 Although the cumulative MEDs are low, in less than 10 minutes, a person’s hands receive an energy dose equivalent to the day-long recommended limit for outdoor workers. UVA irradiation produces less cell cycle arrest than UVB; however, it produces DNA damage via oxidative stress and free radical formation, suggesting that UVA may be more mutagenic than UVB. Therefore, the amount and accumulation of DNA damage following short, higher intensity UVA exposures are unclear.

We confirmed the dose of UVA irradiation emitted by the UV lamps relative to UV Index = 6 sun exposure with the spectrometer. The UV lamps emitted 4.2 times more energy between 355 nm and 385 nm than the sun (Fig 1).

To our knowledge, nail lamp UVA exposure amounts have only been quantified once before, and we have replicated and extended the data showing UV exposure corresponding to MEDs from the nail lamps and in comparison to sunlight.3 The increased depth of penetration of UVA wavelengths of radiation is responsible for the majority of photoaging in human skin and long-term exposure to UV nail lamps may have the potential to increase both cancer risk and photoaging.

Future epidemiologic and molecular genetic studies are needed to evaluate the physiologic and carcinogenic effects of high-dose UVA irradiation to human skin. We recommend that people who choose this acrylic nail treatment apply full spectrum sun block to their hands 30 minutes before their appointment.

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Table I. Ultraviolet nail lamp measurements

<table>
<thead>
<tr>
<th>Lamp</th>
<th>Exposure time (min)</th>
<th>Total MED/yr</th>
<th>Total J/m²</th>
<th>MED/hour</th>
<th>Total MED/nail session</th>
<th>Total J/m²/nail session</th>
</tr>
</thead>
<tbody>
<tr>
<td>OPI lamp</td>
<td>150</td>
<td>1.5</td>
<td>386</td>
<td>0.62</td>
<td>0.09</td>
<td>22.5</td>
</tr>
<tr>
<td>CND lamp</td>
<td>108</td>
<td>1.1</td>
<td>285</td>
<td>0.63</td>
<td>0.06</td>
<td>15.0</td>
</tr>
</tbody>
</table>

*OPI Products, North Hollywood, CA.

1 CND, Creative Nail Design, Vista, CA.
Intralesional interferon alfa-2b for refractory, recurrent squamous cell carcinoma of the face

To the Editor: Aggressive recurrent squamous cell carcinoma (SCC) is a management challenge. Intralesional interferon-alfa has been reported as an effective treatment of primary SCC and basal cell carcinomas. It is also effective in decreasing tumor recurrence in patients with stage III and IV oropharyngeal carcinoma. Interferon, a proinflammatory cytokine, mediates its antitumor effect through immune system activation and apoptosis. Serology and genetic studies have been inconclusive in establishing human papillomavirus as a risk factor for cutaneous SCC. Although the virus may not be necessary for tumor maintenance, human papillomavirus infection may synergize with ultraviolet radiation to initiate tumorigenesis. Antiviral cytokine interferon alpha 2b may directly decrease human papillomavirus viral load and subsequent DNA mutations. Because of its anticancer and antiviral effects we explore its use as an adjuvant treatment in SCC refractory to conventional therapies.

We present 2 patients with recurring cutaneous SCC after surgery and radiation therapy who received intralesional interferon alfa-2b on an adjuvant basis.

Patient 1, a 71-year-old man, presented with a well-differentiated SCC on his right cheek previously treated with wide local excision. Within 1 year, recurrent SCC invaded the parotid gland. A parotidectomy and neck dissection were performed and lymph nodes were negative for tumor. Intraoperative brachytherapy was started. Eleven months after treatment, he developed a right facial palsy and recurrent SCC was diagnosed. He underwent a third wide local resection including mandibular condylectomy and mastoidectomy. Given the aggressiveness of the cancer additional treatment was pursued. Intralesional interferon alfa-2b, 2.0 million U, 3 times a week for 7 weeks for a total dose of 21.5 mU was administered. Headache and fever caused by the

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