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Benefit cost analysis of three skin cancer public education mass-media campaigns implemented in New South Wales, Australia

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Public education mass media campaigns are an important intervention for influencing behavior modifications. However, evidence on the effectiveness of such campaigns to encourage the population to reduce sun exposure is limited. This study investigates the benefits and costs of three skin cancer campaigns implemented in New South Wales from 2006–2013. This analysis uses Australian dollars (AUD) and 2010–11 as the currency and base year, respectively. Historical data on skin cancer were used to project skin cancer rates for the period 2006–2020. The expected number of skin cancer cases is derived by combining skin cancer rates, sunburn rates and relative risk of skin cancers due to sun exposure. Counterfactual estimates are based on sunburn exposure in the absence of the campaigns. Monetary values are attached to direct (treatment) and indirect (productivity) costs saved due to fewer skin cancer cases. Monetary benefits are compared with the cost of implementing the campaigns and are presented in the form of a benefit-cost ratio. Relative to the counterfactual (i.e., no campaigns) there are an estimated 13,174 fewer skin cancers and 112 averted deaths over the period 2006–2013. The net present value of these benefits is \$60.17 million and the campaign cost is \$15.63 million. The benefit cost ratio is 3.85, suggesting that for every \$1 invested a return of \$3.85 is achieved. Skin cancer public education mass media campaigns are a good investment given the likely extent to which they reduce the morbidity, mortality and economic burden of skin cancer.

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Molecular evidence that cutaneous melanomas arise through multiple causal pathways

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There is increasing epidemiologic and molecular evidence that cutaneous melanomas arise through multiple causal pathways. The purpose of this study was to explore the relationship between germline and somatic mutations in a population-based series of melanoma patients to reshape and refine the pathway model for melanoma. Melanomas collected from 414 Australian patients were analyzed for MC1R status and twenty-five common mutations using the MelaCarta Panel designed by Agena Bioscience. Detailed phenotypic and sun exposure data were systematically collected from all patients. Mutually exclusive BRAF-mutant and NRAS-mutant tumors occurred at frequencies of 39% and 9% respectively. There was no association between germline MC1R variants and somatic BRAF mutations in melanomas from this population. However, we did observe differences within BRAF-mutant melanoma, with BRAFV600E mutations associated with increasing nevus counts, contiguous neval remnants and a family history of melanoma while there was an inverse association between the numbers of solar keratoses and excised skin cancers and BRAF V600E mutational status. Older patients were more likely to have melanomas harboring BRAF V600K or NRAS mutations and cumulative sun exposure showed an interesting trend, with highest risks of BRAF V600E and V600K mutations associated with the middle categories of sun exposure, suggestive of an intermittent pattern of sun exposure. This study demonstrates marked differences in the associations between sun exposure, melanocyte susceptibility and host characteristics with a suite of melanoma mutations, strongly suggestive of different casual pathways to melanoma development.

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