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Research Article

Cancer Epidemiology, Biomarkers & Prevention

MC1R Variation in a New Mexico Population

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Abstract

Background: The Melanocortin 1 Receptor (*MC1R*) contributes to pigmentation, an important risk factor for developing melanoma. Evaluating SNPs in *MC1R* and association with race/ethnicity, skin type, and perceived cancer risk in a New Mexico (NM) population will elucidate the role of *MC1R* in a multicultural population.

Methods: We genotyped *MC1R* in 191 NMs attending a primary care clinic in Albuquerque. We obtained individuals' self-identified race/ethnicity, skin type, and perceived cancer risk. We defined genetic risk as carriage of any one or more of the nine most common SNPs in *MC1R*.

Results: We found that one *MC1R* SNP, R163Q (rs885479), was identified in 47.6% of self-identified Hispanics and 12.9% of non-Hispanic whites (NHW), making Hispanics at higher "genetic risk" (as defined by carrying one of the *MC1R* common variants). When we deleted R163Q from analyses, Hispanics were no longer at higher genetic risk (33.3%) compared with NHW (48.3%), consistent with melanoma rates, tanning ability, and lower perceived risk. Hispanics had a perceived risk significantly lower than NHW and a nonsignificant better tanning ability than NHW.

Conclusions: The R163Q variant in *MC1R* may not be a risk factor for melanoma among NM Hispanics. This suggestion points to the need to carefully interpret genetic risk factors among specific populations.

Impact: Genetic risk cannot be extrapolated from Northern European populations directly to non-European populations.

Introduction

In 2019, it is estimated that 96,480 new cases of invasive melanoma, the most deadly form of skin cancer, will be diagnosed in the United States, and 7,230 people are expected to die of the disease (1). The most recent data for the United States indicates there were approximately 6,623 cases of melanoma among Hispanics in 2015 (2). While there are reports of increasing incidence among Hispanics from California (2) and Florida (3), data from 2003 to 2012 show an overall 1.4% decline in the incidence of melanoma in this population (2) with a stable frequency of deeper lesions. Overall, the lifetime risk of getting melanoma is about 2.6% (1 in 38) for whites and 0.58% (1 in 172) for Hispanics (1). Although fewer Hispanics are diagnosed with melanoma than non-Hispanic whites (NHW), they are more often diagnosed at an advanced stage (4) and at a younger age (56 vs. 63; ref. 5). Hispanics are one of the fastest growing populations in the United States, further highlighting that understanding their risk for melanoma is an important public health issue.

The major risk factor for melanoma is pigmentation. Melanin, a major determinant of pigmentation important in skin, hair, and eye color (6), is primarily located on the surface of melanocytes. Individuals with less eumelanin, the darker pigment, and more pheomelanin, the lighter pigment, are at highest risk for cutaneous malignant melanoma. Individuals with more pheomelanin generally tan poorly and potentially perceive themselves at high risk, whereas those with more eumelanin tan more easily (6) and potentially perceive themselves to be at lower risk for melanoma.

The melanocortin 1 receptor (*MC1R*), a G-protein–coupled receptor, plays a major role in skin and hair pigmentation (7). *MC1R* is polymorphic, and some of these SNPs may alter the receptor's function (8). A number of SNPs have been associated with cutaneous melanoma, basal cell carcinoma, and squamous cell carcinoma risk (9, 10). Few studies have examined *MC1R* SNPs in U.S. Hispanic populations, where their frequency and impact are unknown, particularly in relation to phenotype.

New Mexico's population comprises 48% Hispanic (1.8% of all Hispanics in the United States, the largest Hispanic statewide population nationally), and has a unique mixture of individuals who identify as Spanish and/or recent mixed Native American and European ancestry (11, 12). New Mexico therefore provides a distinctive study population for characterizing *MC1R* variants.

This work aimed to determine whether presence of SNPs in the *MC1R* gene, defined as higher than average genetic risk for melanoma, are associated with self-identified race/ethnicity, skin

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type, and perceived cancer risk in a New Mexico (NM) population. A better understanding of genetic risk in the Hispanic population will guide the development of public health interventions to raise skin cancer awareness.

Materials and Methods

Data were collected as part of a randomized controlled trial (NCT03130569) examining interest, uptake, and outcomes associated with an offer of testing for *MC1R* gene variants associated with increased melanoma risk (10). Study enrollment methods have been described previously (13, 14). In brief, 600 participants were recruited from a primary care clinic in Albuquerque, New Mexico (Supplementary Table S1). They were randomized 5:1 to an intervention group which received an invitation to assess their genetic risk for melanoma using *MC1R* genotyping compared with a control group where the participants were not offered genetic assessment until after the follow-ups in the intervention group were complete ($n = 499$ in the intervention arm; $n = 101$ in the control arm). Participants in the intervention arm were balanced across self-reported Hispanic ($n = 242$) versus NHW ethnicity ($n = 220$; 36 reported "other" ethnicity; 1 did not report ethnicity). Participants in the control group were evenly distributed across self-reported Hispanic ($n = 44$) versus NHW ethnicity ($n = 44$;13 reported "other"). Each participant provided informed consent as approved by the University of New Mexico Health Sciences Center Institutional Review Board.

Baseline surveys were completed in-person and have been published. Measures used in this study included (i) phenotype (ability to tan; ref. 15 and history of sunburn), (ii) demographics (ethnicity, race, age, income, and education level), (iii) family and personal history of skin cancer, and (iv) perceived skin cancer risk compared with persons of the same age and sex. Participants in the intervention arm were given access to the study website with information about skin cancer prevention and genetic testing (232, or 46%, accessed the website and 166 of those sent saliva samples for genetic testing). The controls were offered access to the study website, and the potential for genetic testing, after the final follow-up assessment (25 sent saliva samples for genetic testing). Genetic risk was assigned on the basis of the nine most common and most-studied *MC1R* genotypes (10). These included V60L, D84E, V92M, R142H, R151C, I155T, R160W, R163Q, and D294H. The entire *MC1R* gene was sequenced, but only these genotypes were used to assess risk. If an individual had one or more of the nine SNPs, they were told that they had a "higher risk" variant. If a participant had none of the nine, then they were told that they were at "average" risk. Results from the genetic tests were sent by email or mail to participants. Two weeks after receiving their results, those in the intervention arm were contacted to complete a survey regarding their responses to receiving their results.

MC1R genotyping

Saliva samples were mailed to the University of New Mexico Molecular Epidemiology Laboratory. *MC1R* genotypes were described in Kanetsky and colleagues (16). Genomic DNA was isolated from buccal cells using a version of the QIAamp DNA Mini Kit protocol by the manufacturer (Qiagen, Inc.). Using standard PCR technique, an Eppendorf Mastercycler gradient thermocycler was used to amplify the entire 951-nucleotide *MC1R* coding region. All amplified products were directly sequenced on a 3730 Series Genetic Analyzer (Applied Biosystems) using BigDye Terminators (Applied Biosystems) according to the manufacturer's specifications. PCR primers consisted of a set of two oligonucleotides: 5′-GCCATGAGCACCAGCATAG-3′ and 5′-GACCACACAAATATCACCACCT-3′ and a set of four sequencing primers: 5′-TCGTCTTCAGCACGCTCTTC-3′; 5′-TTTAAGGCCAAAGCCCTGGT-3′; 5′-AACCTGCACTCACCCATG-TA-3′; and 5′-CTGCAGGTGATCACGTCAAT. *MC1R* chromatograms were read aided by Finchtv sequencing software version 1.5 (Geospiza Inc.). All *MC1R* genotypes were double entered into a customized Excel sheet and a RedCAP database. We used the *MC1R* consensus sequence (GenBank accession no. AF326275) nomenclature and definitions suggested by Pasquali and colleagues (10) to group *MC1R* variants by risk.

Univariate associations (OR) were evaluated for *MC1R* variants and self-reported race and ethnicity. Unconditional logistic regression was used to obtain adjusted estimates. Models were adjusted for age, sex, and family history of skin cancer. Both unadjusted and adjusted ORs and corresponding 95% confidence intervals (CI) are presented. Analyses were carried out in SAS 9.4 (SAS). We restricted analyses to Hispanics and NHW given the "other" category (Asian, American Indian or Alaskan Native, Native Hawaiian/Pacific Islander, African American, or other) that provided a sample for genotyping represented a small group ($n = 12$).

Results

Characteristics of those genotyped, on the basis of 63 Hispanic and 116 NHW individuals (159 from the intervention group and 20 from the control group who requested genetic testing, excluding "other" category $n = 12$) show that in this analysis Hispanics compared with NHW are more likely to be female, have less education beyond high school, have a lower income (borderline significant), and be of similar age (Table 1).

Genetic results comparing Hispanics and NHW showed carriage of several different variants. The variant R163Q (rs885479) was more common among Hispanic individuals and V92M (rs2228479) and R160W (rs1805008) were more common among NHW (Table 2).

Only 22.2% of Hispanics perceived themselves to be at increased risk of skin cancer; in contrast, 46.6% of NHW felt themselves to be at increased risk of skin cancer. On the basis of the genotyping of the nine *MC1R* variants, 63.5% of Hispanics and 56.4% of NHW are at increased genetic risk. When R163Q

Table 1. Comparison of key demographic characteristics between Hispanics and NHW who were genotyped ($n = 179$)

Variable	HW *n* (%)	NHW *n* (%)	OR (95% CI)	*P*
Gender				
Male	6 (9.5)	34 (29.3)		
Female	57 (90.5)	82 (70.7)	0.25 (0.10–0.64)	0.0003
Age				
Median (IQR)	54 (23)	56 (17)		0.28
Education				
Less than HS	14 (22.2)	7 (6.0)		
HS or greater	49 (71.8)	109 (94.9)	0.22 (0.09–0.39)	0.0003
Income				
<$50,000	41 (65.8)	22 (34.9)		
≥$50,000	58 (50)	58 (50)	0.54 (0.29–1.01)	0.06

NOTE: "Other" participants ($n = 12$) were excluded from analysis due to small sample size.

Abbreviations: HS, high school; HW, Hispanic white.

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Table 2. Comparison of *MC1R* genotype in Hispanic and NHW[a]

Variable	HW ($n = 63$)	NHW ($n = 116$)	OR (95% CI)	*P*
MC1R Genotype				
V60L	10 (15.9)	18 (15.5)	1.03 (0.44–2.38)	0.93
D84E	0	2 (1.7)	Not estimable	
V92M	1 (1.6)	12 (10.3)	0.14 (0.02–1.10)	0.06
R142H	0	2 (1.7)	Not estimable	
R151C	6 (9.5)	16 (13.8)	0.66 (0.24–1.78)	0.41
I155T	2 (3.2)	4 (3.5)	0.92 (0.16–5.16)	0.92
R160W	2 (3.2)	15 (12.9)	0.22 (0.49–0.99)	0.03
R163Q	30 (47.6)	15 (12.9)	6.12 (2.94–12.75)	<0.0001
D294H	1 (1.6)	1 (0.9)	1.86 (0.11–30.17)	0.66

Abbreviation: HW, Hispanic white.

[a]Includes those from control group who asked for genetic testing ($n = 25$) and those responding to the invitation for testing in the intervention group ($n = 166$). We excluded "other" ethnicity participants ($n = 12$) due to the small sample size.

was excluded from genetic risk assessment, the number of Hispanics with a higher risk variant was reduced by almost half to 33.3% compared with a small reduction to 48.3% among NHW (Table 3).

There was no significant difference in genetic risk, that is, between those with any *MC1R* variant compared with those with no variants, between Hispanics and NHW who reported a family history of skin cancer ($P = 1.00$; Supplementary Table S2). In NHW participants, there was a borderline association between family history and high risk genotypes (OR = 2.00; 95% CI, 0.93–4.30; $P = 0.08$; Supplementary Table S2).

Even after adjusting for family history of skin cancer, Hispanics still perceived themselves to be at a lower skin cancer risk than NHW ($P = 0.004$; Table 3). The majority of genetic risk in Hispanics was due to the contribution of R163Q (Table 3). In this sample, *MC1R* risk variants were associated neither with tanning ability ($P = 0.60$) nor with perceived risk ($P = 0.82$; Supplementary Table S2).

Discussion

Few studies have examined the frequency and impact of *MC1R* SNPs in the U.S. Hispanic population. *MC1R* risk variants have been considered major determinants of sun sensitivity, conferring a 2- to 3-fold increase in melanoma risk in the general population, including those who report increased ability to tan. Interestingly, *MC1R* variants predict melanoma risk in darker-skinned European populations more strongly than those with lighter skin (17). As Hispanics are a phenotypically diverse group with marked variations in tanning ability (17), one might expect relatively wide variation in *MC1R* SNPs.

A genome-wide association study of pigmentation SNPs in more than 6,000 subjects in Latin America found a very strong association of R163Q with Native American populations (17). As many Hispanics in New Mexico have approximately 24%–37% Native American ancestry, our results regarding R163Q are not surprising (18).

NM Hispanics may have a significant contribution of Native American genes (18), and as Native Americans have genetic ties to Northeast Asia (17) where R163Q does not appear to increase risk for melanoma (19), it is critical to continue to evaluate the role of R163Q in NM Hispanics in relationship to melanoma risk. Other studies have found similarly divergent associations for risk SNPs in populations looking at different diseases (e.g., 20). There have been no specific explanations proposed explaining why the particular SNP variant is not associated with melanoma risk in Native Americans. It is likely that pigmentary risk in relationship to melanoma will differ by population and that there are a variety of as yet unstudied interactions among pigmentary genes in Native Americans and Europeans to produce different risk profiles (21). Relationships among *MC1R* genotype, ethnicity/race, self-reported skin cancer, family history of skin cancer, and tannability all contribute to skin cancer risk and warrant further investigation in Hispanic populations.

Our study is the first to evaluate *MC1R* variants with self-identified ethnicity in a diverse NM population. Results indicate that when participants are categorized by self-reported ethnicity, the most common *MC1R* variant in Hispanics is R163Q compared with NHW who had increased risk with R151C and R160W. As the Hispanics in our study perceive their skin cancer risk to be lower, understanding how or whether the R163Q variants contribute to genetic risk for melanoma among NM Hispanics could inform public health initiatives. A relatively small sample size limits generalizability of our results; they should be investigated in a larger group of Hispanics and NHWs in NM. As the incidence rate of melanoma among NM Hispanics is low and steady, the role of *MC1R* may be more complex than originally thought. New Mexico is a unique setting to further evaluate the role of *MC1R* and other genetic factors in its multi-cultural population.

Table 3. Tanning ability, perceived risk, and genetic risk among Hispanics and NHW

Variable			Bivariate association		Multivariable association[a]	
Tanning ability[b]	Poor	Good	OR (95% CI)	*P*	OR (95% CI)	*P*
HW	16 (28.6)	40 (71.4)				
NHW	41 (38.3)	66 (61.7)	0.64 (0.37–1.30)	0.22	0.66 (0.32–1.39)	0.27
Perceived risk	High risk	Average risk				
HW	14 (22.2)	49 (77.8)				
NHW	54 (46.5)	62 (53.5)	0.32 (0.16–0.66)	0.0014	0.34 (0.16–0.70)	0.004
Genetic risk[c]	High risk	Average risk				
HW	40 (63.5)	23 (36.5)				
NHW	66 (56.4)	50 (48.1)	1.32 (0.70–2.78)	0.39	1.58 (0.80–3.13)	0.19
Genetic risk without R163Q	High risk	Average risk				
HW	23 (33.3)	42 (66.7)				
NHW	56 (48.3)	60 (51.8)	0.54 (0.28–1.01)	0.06	0.59 (0.30–1.16)	0.13

Abbreviation: HW, Hispanic white.

[a]Controlling for age, sex, and family history of skin cancer.

[b]Tanning ability was answered as "don't know" by 7 Hispanics and 9 NHW.

[c]Genetic risk is based on having any one *MC1R* variant (V60L, D84E, V92M, R142H, R151C, I155T, R160W, R163Q, and D294H).

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Disclosure of Potential Conflicts of Interest

D.B. Buller is a senior scientist at and has ownership interest (including patents) in Klein Buendel, Inc. No potential conflicts of interest were disclosed by the other authors.

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