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Socioeconomic and Tobacco Mediation of Ethnic Inequalities in Mortality over Time

Repeated Census-mortality Cohort Studies, 1981 to 2011

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Background: Racial/ethnic inequalities in mortality may be reducible by addressing socioeconomic factors and smoking. To our knowledge, this is the first study to estimate trends over multiple decades in (1) mediation of racial/ethnic inequalities in mortality (between Māori and Europeans in New Zealand) by socioeconomic factors, (2) additional mediation through smoking, and (3) inequalities had there never been smoking.

Methods: We estimated natural (1 and 2 above) and controlled mediation effects (3 above) in census-mortality cohorts for 1981–1984 (1.1 million people), 1996–1999 (1.5 million), and 2006–2011 (1.5 million) for 25- to 74-year-olds in New Zealand, using a weighting of regression predicted outcomes.

Results: Socioeconomic factors explained 46% of male inequalities in all three cohorts and made an increasing contribution over time among females from 30.4% (95% confidence interval = 18.1%, 42.7%) in 1981–1984 to 41.9% (36.0%, 48.0%). Including smoking with socioeconomic factors only modestly altered the percentage mediated for males, but more substantially increased it for females, for example, 7.7% (5.5%, 10.0%) in 2006–2011. A counterfactual scenario of having eradicated tobacco in the past (but unchanged socioeconomic distribution) lowered mortality for all sex-by-ethnic groups and resulted in a 12.2% (2.9%, 20.8%) and 21.2% (11.6%, 31.0%) reduction in the absolute mortality gap between Māori and Europeans in 2006–2011, for males and females, respectively.

Conclusions: Our study predicts that, in this high-income country, reducing socioeconomic disparities between ethnic groups would greatly reduce ethnic inequalities in mortality over the long run. Eradicating tobacco would notably reduce ethnic inequalities in absolute but not relative mortality.

Keywords: Causal mediation analysis; Ethnicity; Mediation; Mortality; Race; Socioeconomic factors; Tobacco

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Social inequalities in health are pervasive,[1] with inequalities by ethnicity or race being a common axis of inequality[2–4] with Māori mortality disadvantage in New Zealand (NZ) being one example that has been studied.[5–9] Explanations for ethnic inequalities in mortality are context-specific but typically involve colonization and/or racism as a driver of differential access to determinants of health such as material resources and educational opportunities, differential exposures to health risk factors (e.g., smoking, unhealthy diet), and differential access to and quality of health care.[3,10] Understanding these potential causes is an important step toward informing policy responses.[11]

Focusing on mortality inequalities in NZ for Māori (Indigenous people of NZ, 15% of population) versus Europeans [majority of population, excluding people self-identifying as Pacific or Asian, and a heterogeneous group of (descendants) of British, European, and other nation settlers since the 19th century], we have previously attempted to characterize and explain inequalities up to about the year 2000.[7,8] Inequalities widened in the 1980s to 1990s as there was accelerated decline in mortality among the European population after the peak of the cardiovascular disease epidemic. In contrast, the health of Māori suffered from being hit hard by neoliberal reforms that saw high and persistent unemployment rates disproportionately borne by Māori.[7] However, since the 1990s, life expectancy gaps decreased from a nearly 10-year gap in the 1990s to about 7 years by 2011.[5]

In the last decade, causal inference methods—notably counterfactual approaches premised on a potential outcome approach—have exposed the limitations of previous analytical

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approaches to mediation.[11–13] But these new methods have not, to our knowledge, been used to analyze how mediation of social group health inequalities change over time—for example, in national cohort studies.

Our study included three key methodologic advances. First, we accounted for the large relative variations in the association of smoking with mortality by ethnicity (eFigure 1; http://links.lww.com/EDE/B345, eTable 1; http://links.lww.com/EDE/B345 and eTable 2; http://links.lww.com/EDE/B345; namely exposure-mediator interaction; there was no substantive interaction of socioeconomic position with ethnicity).[14,15] Second, we avoided a sole reliance on standard multiplicative regression modeling and instead predicted the expected potential outcomes for all census respondents under varying scenarios, allowing an easy depiction of mortality risks and risk differences, instead of just relative risks. Third, the conceptual and analytical approach to the question "how much did smoking contribute to inequalities as we see them now?" (natural direct and indirect effects) differs from the question "how much would mortality (inequalities) change if we reduced smoking through some future policy?" (controlled direct effects).

Accordingly, this study considered the following research questions:

1. How much of the ethnic inequality in mortality in a high-income country (NZ) is mediated by socioeconomic factors (income, education, neighborhood deprivation, and labor force status)? Furthermore, does this change over time (1981–1984, 1996–1999, and 2006–2011)?
2. What is the incremental increase in mediation when including smoking over and above socioeconomic position, and does this change over time?
3. If, counter-to-fact, NZ had been tobacco free, how much less would current ethnic inequalities in mortality be?

Note that research question 2 is about quantifying the joint-mediated effect through socioeconomic position (SEP) and tobacco[13] and does not require alternative methods such as randomized intervention analogues that quantify mediation by tobacco alone. Research question 3 estimates controlled direct effects through counterfactual manipulation of the smoking variable.

METHODS

NZ Census-mortality Linked Cohort Data

Each of the 1981 and 1996 national censuses for NZ were linked at the individual level to 3 years of subsequent mortality data, and the 2006 census to 5 years of subsequent mortality data, creating three separate census-mortality cohorts. Records were anonymously and probabilistically linked using sex, age, date of birth, residential geocode, country of birth, and ethnicity as matching variables.[16,17] This resulted in 72%, 80%, and 81%, respectively, of eligible mortality records (i.e., 25 to 74 years of age at time of census) being linked. Of these links, approximately 98% were estimated to be true positive links (i.e., a decedent's record correctly linked to their previous census record).[18] To allow for incomplete linkage, each linked census-mortality record was weighted by the inverse probability of linkage in regression modeling. (See eAppendix; http://links.lww.com/EDE/B345 for details on linkage weights.) The record linkages have all been reviewed and approved by health ethics committees, although authority to do so is primarily through the Statistics Act (1975).

Each census included questions on ethnicity: any people recording Māori as one of their potentially multiple ethnicities were classified as Māori. Census respondents reporting no Māori, Pacific, or Asian identity were classified as Europeans.

A range of socioeconomic factors were recorded equivalently on the three censuses: education (post-school, school [i.e., at least one qualification in the final 3 years of secondary school] and nil-qualifications), household income (sum of all personal incomes in the household, equivalized for number of children and adults in the household (J. Jensen, unpublished, 1988) and log-transformed), and labor force status (employed, unemployed, not in the labor force). Additionally, each individual was assigned a neighborhood deprivation decile (NZDep; NZ Index of Deprivation) based on an index calculated at the small area level (typically around 100 people in each neighborhood), calculated separately for the 1996 and 2006 censuses.[19,20] NZDep is strongly associated with ethnicity and mortality and is probably more stable over time than income. NZDep was not calculated for the 1981 census and instead we back-coded 1996 NZDep scores to 1981 respondents, assuming neighborhoods had the same ranking by deprivation in 1981 as in 1996.

Table 1 presents frequencies and means of the above variables and shows strong associations of ethnicity with socioeconomic factors and smoking.

Conceptualization of Ethnicity, Structural Relationship of Variables, and Implications for a Potential Outcomes Approach Analysis

It is critical to have conceptual clarity about the ethnicity exposure. VanderWeele and Robinson[11] describe two perspectives for interpreting epidemiologic effect sizes for a race/ethnicity coefficient in regression modeling. First, a stronger perspective that (somehow) assumes aspects of race/ethnicity are considered manipulable, and second, a weaker (more realistic) perspective that interprets the race/ethnicity coefficient in the context of what covariates are in the model and particularly how the coefficient changes with addition of covariates.[11]

Two alternative conceptual models or directed acyclic graphs (DAGs), which could be applied in this study, are shown in Figure 1. In Figure 1A, adult self-reported ethnicity (as is available in the census data we use) is conceptualized as separable and independent from parental and intergenerational ethnicity and social factors, and history, as well as independent of early-life SEP. This is one possible conceptualization of VanderWeele and Robinson[11] stronger perspective whereby the

TABLE 1. Descriptive Data

	Males						Females					
	1981–1984		1996–1999		2006–2011		1981–1984		1996–1999		2006–2011	
	Māori	Europeans	Māori	Europeans	Māori	Europeans	Māori	Europeans	Māori	Europeans	Māori	Europeans
No. census respondents[a]	59,361	653,436	98,028	741,249	109,890	785,799	62,742	676,305	110,826	779,487	130,143	835,593
Eligible respondents included (%)[b]	39,423 (66.4%)	513,894 (78.6%)	74,826 (76.3%)	645,405 (87.1%)	79,224 (72.1%)	665,319 (84.7%)	41,070 (65.5%)	523,059 (77.3%)	82,128 (74.1%)	673,206 (86.4%)	92,772 (71.3%)	703,155 (84.2%)
Age group												
25–44 yrs	68.5%	52.4%	66.7%	49.8%	60.1%	44.1%	69.0%	52.2%	69.0%	50.4%	63.5%	45.7%
45–64 yrs	26.8%	36.2%	28.4%	37.1%	34.1%	43.6%	26.3%	34.9%	26.1%	35.8%	31.5%	42.2%
65–74 yrs	4.7%	11.4%	4.9%	13.2%	5.7%	12.3%	4.7%	13.0%	5.0%	13.8%	5.0%	12.0%
Education												
Nil	74.7%	50.1%	49.4%	30.0%	40.6%	21.5%	77.7%	58.6%	48.8%	32.5%	34.1%	21.2%
School	11.1%	17.0%	22.3%	24.4%	25.9%	26.9%	13.5%	19.0%	27.7%	32.1%	30.3%	34.0%
Post-School	14.1%	32.9%	28.3%	45.6%	33.5%	51.6%	8.8%	22.4%	23.5%	35.3%	35.6%	44.9%
Neighborhood deprivation												
Least deprived quintile	6.4%	22.3%	9.3%	26.0%	10.3%	26.8%	6.6%	22.2%	8.2%	25.6%	8.8%	26.5%
Quintile 2	11.1%	22.7%	12.9%	23.4%	13.7%	24.1%	10.7%	22.3%	11.5%	23.2%	12.3%	24.1%
Quintile 3	15.9%	21.3%	17.0%	21.2%	17.9%	21.1%	15.0%	21.2%	15.8%	21.3%	16.8%	21.4%
Quintile 4	23.2%	19.1%	23.6%	17.9%	23.8%	17.4%	22.4%	19.2%	23.5%	18.2%	24.1%	17.6%
Most deprived quintile	43.5%	14.5%	37.2%	11.6%	34.4%	10.6%	45.3%	15.1%	41.0%	11.7%	38.0%	10.4%
Labor force status												
Employed	85.7%	83.8%	70.1%	77.0%	78.8%	83.0%	46.0%	47.6%	52.8%	60.6%	66.0%	71.0%
Unemployed	5.0%	1.4%	9.0%	2.9%	4.6%	1.6%	1.6%	0.8%	8.3%	2.6%	5.7%	1.8%
Nonactive	9.3%	14.8%	20.9%	20.1%	16.6%	15.4%	52.4%	51.6%	38.9%	36.7%	28.3%	27.2%
Smoking												
Never	28.7%	36.7%	37.0%	47.0%	39.9%	52.3%	30.0%	56.4%	31.6%	56.0%	31.6%	57.0%
Ex	21.0%	29.9%	23.3%	30.5%	23.8%	28.6%	16.3%	16.5%	21.4%	23.7%	24.3%	25.7%
Current	50.3%	33.3%	39.7%	22.5%	36.3%	19.1%	53.7%	27.1%	47.0%	20.3%	44.1%	17.3%
Mean log household equivalized income	9.28	9.56	10.3	10.6	10.7	11.0	9.14	9.43	10.1	10.5	10.5	10.9
Deaths (raw numbers)	729	11,304	1,470	11,406	2,283	14,814	534	6,792	1,068	7,365	1,797	10,362
Deaths, weighted for linkage bias	1,287	15,303	2,178	14,022	3,294	18,183	903	9,285	1,536	8,751	2,304	12,045

[a]Usually resident population living in a private dwelling aged 25–74 years at census night.
[b]Most missing respondents were due to missing income (as any one adult in the household away from their usual residence on census night, or any one adult refusing to give their income, causes the household income to be missing).

causal effect corresponds to a manipulation of an identifiable specific component of race/ethnicity. Regarding Figure 1A, it is advantageous if one wants to compare mortality rates of self-identified Māori and Europeans, within strata of similar parental ethnicity and histories—but this makes little sense. First, in epidemiologic terms, there is a high risk of structural confounding (e.g., too few adult European adults with Māori parents to compare with Māori adults with Māori parents). Second, in theoretical terms, such conceptualization may inappropriately disembody people's ethnicity from its foundations.

The DAG in Figure 1B seems a better conceptualization. Here adults' self-identified ethnicity is incorporating ancestry and histories, closer to the real policy questions of "how large are ethnic inequalities in health?" and "what explains ethnic inequalities in health?" The corollary of this second DAG, though, is that we need to retain this conceptualization in interpretation[11]; we return to this in the Discussion.

Of note, sex and age are the only confounders of the association of ethnicity with mortality in Figure 1B. We treat socioeconomic position as a set of multiple mediators in analyses to answer the first research question. We further add smoking to answer the second research question. All analyses were undertaken in R 3.1.2 software (code provided in eAppendix; http://links.lww.com/EDE/B345).[21]

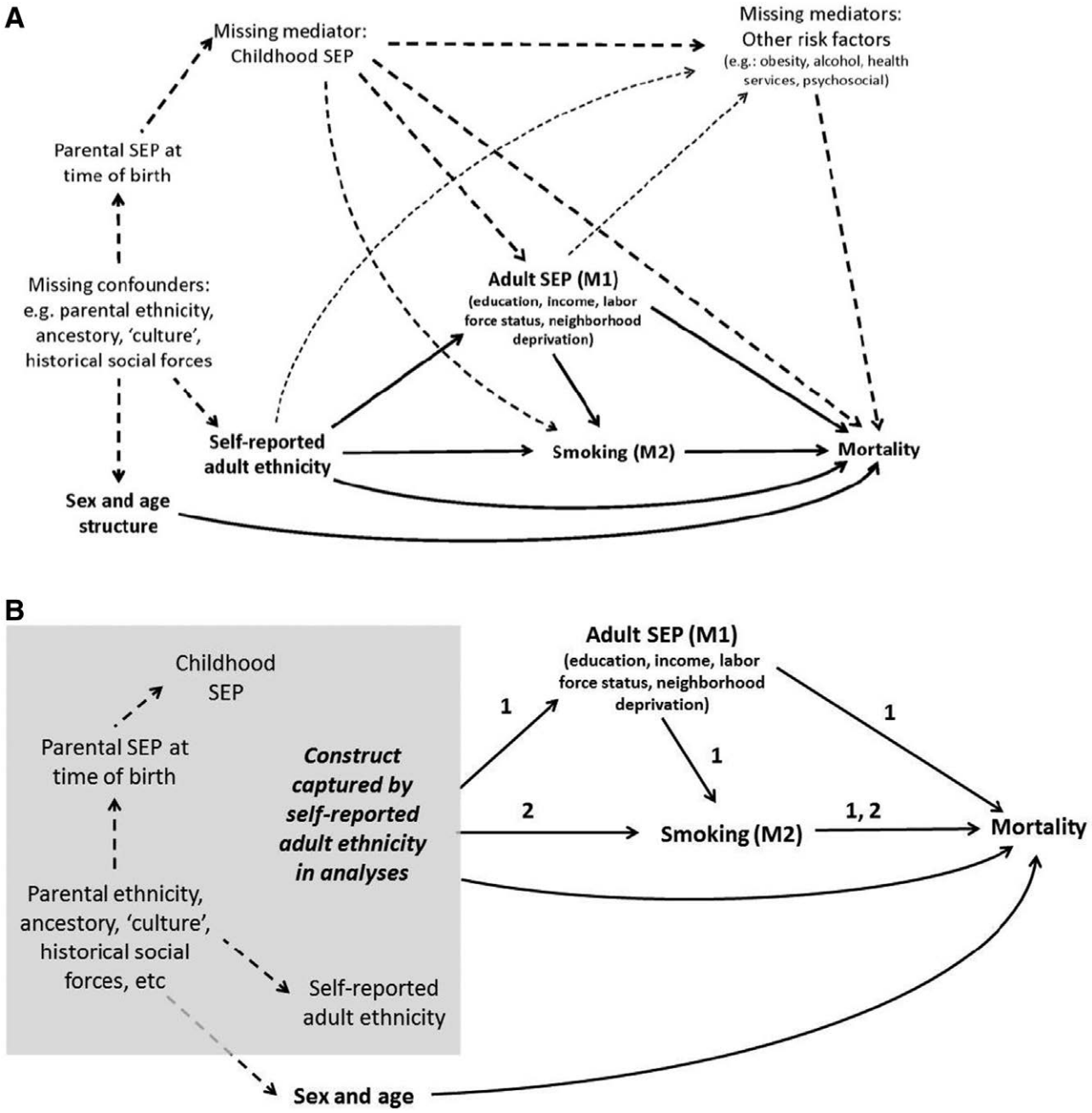

FIGURE 1. Posited DAG for the associations of ethnicity (exposure), mortality (outcome), SEP, and smoking risk factor mediators (M1 and M2). A, Fuller DAG conceptualizing self-reported adult ethnicity as separable from (conceptualized) confounding by parental and wider social factors and childhood SEP. B, Simplified DAG conceptualizing self-reported adult ethnicity as capturing intergenerational and early-life SEP (and more appropriate for this article). Bolded nodes/variables are those measured and adjusted for in this paper; bold solid arrows are those pathways directly assessed in this paper; bold dashed arrows are those unable to be directly assessed in this paper, and from the perspective of DAG A with self-identified adult ethnicity conceptualized as separable and independent of parental and other social factors could result in residual confounding of analyses in this article.

Causal Mediation Analyses

While we are adopting VanderWeele and Robinson[11] weaker perspective, we still use a natural (in)direct effects approach to causal mediation analysis premised on counterfactual manipulability of the mediators. The approach we describe below might be considered as a special case of randomized intervention analogues,[13] as we shift mediator distributions to those among the exposed or other counterfactuals.

Further, there are similarities with a counterfactual shift in mediators outline by Valeri et al,[22] with respect to cancer stage at diagnosis as a mediator of race inequalities in cancer survival. However, we will describe the method as a weighted sequential multiple mediator approach, producing natural effects and (with a universal setting of tobacco mediator levels to the same for both ethnic groups) a form of controlled mediational effect.

Research Questions 1 and 2: Mediation by (1) Socioeconomic Factors and (2) Socioeconomic Factors and Smoking

We selected a weighting for multiple mediators approach to estimate natural direct and indirect effects because of multiple mediators under consideration, a dichotomous outcome, and a dichotomous exposure (pp. 122–125 of Ref[12,23]). We calculated the risk of death for three mortality risks, called nested counterfactuals:

- $E[Y_{a*M*}]$, the expected mortality risk for the unexposed (a*, Europeans) with mediators at the values observed among Europeans (M*, socioeconomic factors and smoking);
- $E[Y_{aM}]$, the expected mortality risk for the exposed (a, Māori) with mediators at the values observed among Māori (M); and
- $E[Y_{aM*}]$, the expected mortality risk for Māori (a) with (counter-to-fact) mediators at the values expected had the person actually been European (M*).

We predicted the probability of these mortality risks for individual study participants ($E[Y_{aM}]$ for all Māori participants, and $E[Y_{a*M*}]$ and $E[Y_{aM*}]$ for all European participants (the latter explained further below) using a logistic regression model (linkage bias weighted) with mortality as the dependent variable, and exposure (ethnicity), confounders (sex and age), and mediators (socioeconomic factors ± smoking) as independent variables. As this is a predictive model, it should be rich with interactions (most notably the exposure with mediators, but also age with ethnicity and [for the smoking model] a three-way interaction of age, smoking, and ethnicity). The coefficients and standard errors for the 12 separate predictive models are shown in eTable 3; http://links.lww.com/EDE/B345.

$E[Y_{aM*}]$ required predicting what the mortality risk for each European census respondent would have been if (counter-to-fact) they were actually Māori by recoding all Europeans as Māori and using the logistic regression equations to predict an expected mortality risk.[24]

To calculate the age-adjusted values of $E[Y_{aM}]$, $E[Y_{a*M*}]$, and $E[Y_{aM*}]$, we adapted the weighting method published elsewhere (pp. 122–125 of Ref[12,23]) to give WHO World Standard age-standardized mortality risks for 25- to 74-year-olds combined, and 25- to 44-, 45- to 64-, and 65- to 74-year-olds, ensuring comparability (i.e., same age structure) across all six sex-by-census cohorts (see eAppendix; http://links.lww.com/EDE/B345: Weights section of supplementary material). These three mortality risks were estimated for 2,000 bootstrap iterations with replacement for each census-mortality cohort. Within each bootstrap iteration, the total effect is $E[Y_{aM}]-E[Y_{a*M*}]$ on the absolute scale, comprised of the natural direct effect (NDE; $E[Y_{aM*}]-E[Y_{a*M*}]$), and the natural indirect effect (NIE; $E[Y_{aM}]-E[Y_{aM*}]$). On a relative scale, the NDE is simply the ratio $E[Y_{aM*}]/E[Y_{a*M*}]$ and the NIE is the ratio $E[Y_{aM}]/E[Y_{aM*}]$. We report the median, 2.5th and 97.5th percentile values of each of these estimands.

Research Question 3: If New Zealand Were Tobacco Free

The approach of weighting for multiple mediators can also be used to estimate the mortality risk if (counter-to-fact) no one had smoked. Consider the multiple mediators split into two groupings: M1, the socioeconomic mediators; M2, the smoking mediator (preceded by M1 mediators). In a similar way to the estimation of $E[Y_{aM*}]$ above, one can also estimate the potential outcome (i.e., expected mortality risk) for each individual given their ethnicity (A) and socioeconomic factors (M1) by predicting the mortality risk from the logistic regression of mortality on ethnicity, confounder age, M1 and M2—but with M2 counter-to-fact set at never-smoker for all participants, for both exposed (Māori) and unexposed (Europeans). Using the same weights as above (Eq (2) and (3) for Europeans and Māori in the eAppendix; http://links.lww.com/EDE/B345), one can estimate the mortality risk as though the entire population had been never-smokers (but with no change in socioeconomic factors, which precede smoking in the DAG in Figure 1), and thence the controlled direct effects.

Sensitivity Analysis

Misclassification or mismeasurement of the mediators means we may have underestimated the extent of mediation through these variables.[25,26] Therefore, we undertook a sensitivity analysis about smoking misclassification on the 2006–2011 cohort data using expected value analyses and a simulation-extrapolation (SIMEX) approach.[27,28] We assumed our actual analyses were effected by a misclassification matrix of smoking that we have previously estimated.[29] We then made the misclassification 50%, 100%, 150%, and 200% greater, by probabilistically reassigning current, ex, and never-smokers to other categories in a Monte Carlo simulation. For example, if the actual estimated probability of true classification of current smokers as current smokers was 95%, then 50% greater misclassification corresponds to 92.5%, 100% greater misclassification to 90%, etc.

RESULTS

Between 1981–1984 and 2006–2011, mortality risks decreased by 34% and 38% for Māori males and females and by 50% and 43% for European males and females (Figure 2 and Table 2). Mortality risk differences comparing Māori to Europeans (i.e., the total effect) for males increased by 11% from 1981–1984 to 1996–1999 (from 679 to 756 per 100,000) then decreased by 22% in 2006–2011 (592 per 100,000). For

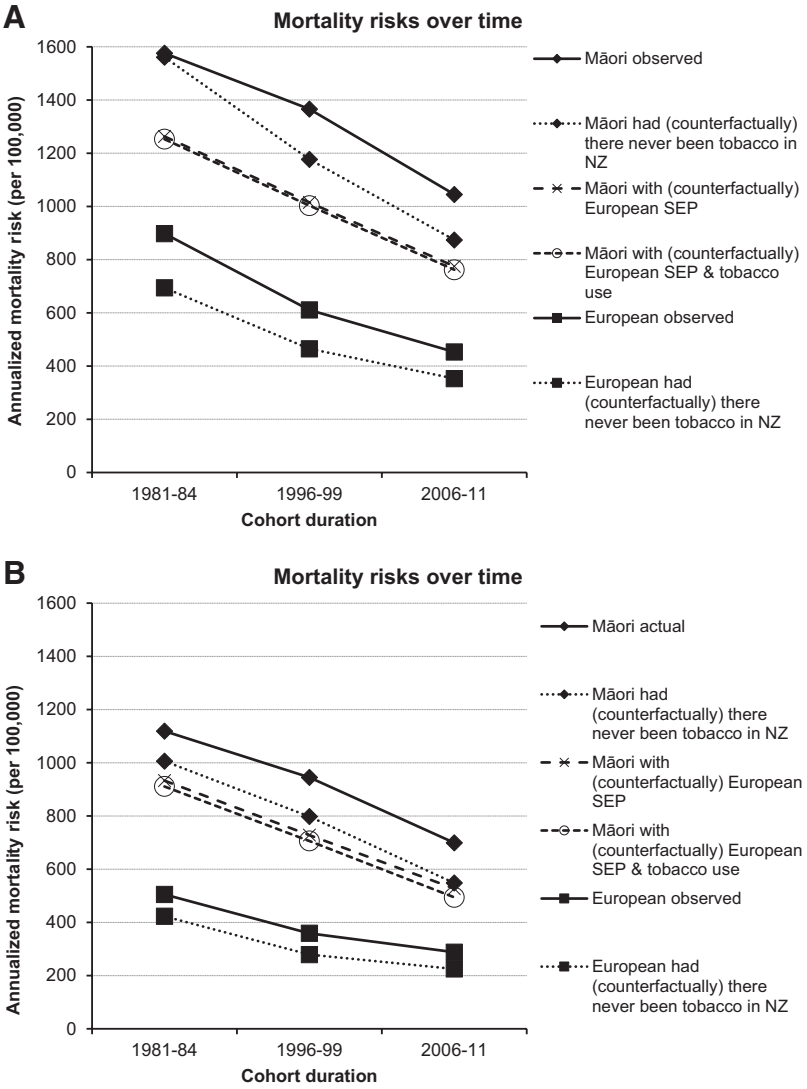

FIGURE 2. Observed and counterfactual mortality risks by ethnicity (Māori vs. Europeans) and time in the adult New Zealand population. A, Males. B, Females.

females these risk differences decreased monotonically from 614 per 100,000 in 1981–1984 to 410 per 100,000 in 2006–2011. On the relative scale, however, inequalities increased in males from a risk ratio of 1.76 in 1981–1984 to 2.31 in 2006–2011, and among females increased from 2.21 in 1981–1984 to 2.63 in 1996–1999, then decreased to 2.42 in 2006–2011.

Research Question 1: Mediation of the Ethnic mortality Association by Socioeconomic Factors

Approximately halfway between the actual Māori and European mortality risk lines in Figure 2 is the predicted Māori potential mortality risk had Māori (counterfactually) experienced European levels of socioeconomic factors (i.e., household income, neighborhood deprivation, education, and labor force status). The drop from actual Māori to this counterfactual or cross-world risk is the natural indirect effect (NIE); the part of ethnic inequality mediated by socioeconomic factors. In absolute terms, the NIEs were greatest in 1996–1999 for both males (352 per 100,000) and females (216 per 100,000; Table 2 and Figure 3). Expressed as the percentage of the ethnic inequality difference mediated by socioeconomic position, however, it was constant over time for males (46% for all three cohorts) and increasing over time among females (30.4% [95% confidence interval (CI)] = 18.1%, 42.7%), 37.1% (29.4%, 43.8%), and 41.9% (36.0%, 48.0%) by census cohort].

eTable 4; http://links.lww.com/EDE/B345 shows summary results by age. The pattern of constant percentage mediated by socioeconomic position among males, increasing among females, over time was apparent for 45- to 64-year-olds, and perhaps 25- to 44-year-olds. However, confidence intervals are wide for both 25- to 44- and 65- to 74-year-olds.

Research Question 2: Joint Mediation by Socioeconomic Factors and Smoking

The addition of smoking to the set of multiple socioeconomic mediators for males did not significantly increase the

TABLE 2. Mortality Risks, Risk Differences and Risk Ratios, and Other Causal Mediation Estimates for 25- to 74-year-olds (95% CIs in Brackets)

Population and Mediation	Parameter Estimates	Males			Females		
		1981–1984	1996–1999	2006–2011	1981–1984	1996–1999	2006–2011
Mortality risks (annualized, per 100,000)	Māori[a]	1,576 (1,459, 1,689)	1,366 (1,297, 1,437)	1,045 (1,003, 1,090)	1,119 (1,020, 1,215)	945 (889, 1,003)	699 (666, 730)
	Europeans (Euro)[a]	898 (882, 914)	611 (600, 623)	453 (445, 460)	505 (493, 517)	359 (351, 368)	288 (283, 294)
	Māori with Euro SEP (CW)	1,263 (1,123, 1,406)	1,015 (944, 1,088)	775 (733, 817)	933 (826, 1,044)	728 (670, 792)	527 (494, 562)
	Māori with Euro SEP + Tob (CW)	1,253 (1,115, 1,399)	1,003 (934, 1,076)	762 (720, 804)	911 (807, 1,023)	706 (648, 768)	494 (462, 528)
	Māori never smoking (counterfactual)	1,561 (1,362, 1,767)	1,177 (1,075, 1,286)	874 (811, 936)	1,006 (865, 1,155)	798 (717, 881)	548 (499, 599)
	Euro never smoking (counterfactual)	694 (667, 722)	465 (448, 481)	353 (343, 363)	423 (409, 438)	279 (269, 289)	225 (218, 231)
Risk Differences (annualized, per 100,000)	TE	679 (561, 792)	756 (686, 827)	592 (551, 639)	614 (516, 709)	586 (528, 646)	410 (378, 442)
	NDE (SEP mediation)	365 (223, 508)	404 (333, 477)	322 (281, 365)	427 (319, 538)	369 (312, 433)	238 (205, 273)
	NDE (SEP + Tob mediation)	356 (215, 502)	393 (323, 467)	310 (268, 352)	406 (299, 518)	347 (288, 409)	206 (173, 241)
	NIE (SEP mediation)	315 (214, 407)	352 (298, 406)	270 (237, 303)	187 (110, 262)	216 (171, 259)	172 (147, 197)
	NIE (SEP + Tob mediation)	317 (216, 418)	359 (306, 414)	282 (248, 315)	204 (128, 282)	238 (192, 281)	203 (178, 228)
	CDE (tobacco-free counterfactual)	867 (665, 1,074)	712 (609, 822)	520 (455, 581)	584 (442, 734)	519 (436, 602)	323 (274, 374)
% Mediated (natural effects)	% Mediated (SEP)	46.4 (31.4, 62.3)	46.6 (39.6, 53.6)	45.6 (40.3, 50.7)	30.4 (18.1, 42.7)	37.1 (29.4, 43.8)	41.9 (36.0, 48.0)
	% Mediated (SEP + Tob)	47.1 (31.6, 63.2)	47.8 (40.7, 54.6)	47.6 (42.4, 52.8)	33.5 (20.6, 46.3)	40.8 (33.3, 47.9)	49.7 (43.4, 55.7)
	Mediation change (SEP to SEP + Tob)	0.8 (−2.4, 4.0)	1.2 (0.4, 2.8)	2.0 (1.2, 2.8)	3.0 (1.7, 7.4)	3.7 (1.5, 5.8)	7.7 (5.5, 10.0)
Counterfactual changes had nobody smoked (controlled effects)	% Reduction in Māori risk[b]	0.5 (−10.2, 11.2)	13.6 (7.6, 19.9)	16.3 (11.2, 21.1)	9.7 (−0.8, 19.7)	15.5 (9.0, 22.0)	21.4 (15.8, 27.1)
	% Reduction in Euro risk[b]	22.6 (19.9, 25.3)	23.9 (21.7, 26.2)	21.9 (20.1, 23.6)	16.2 (14.1, 18.3)	22.2 (20.1, 24.2)	22.0 (20.3, 23.6)
	% Eradicated (1−CDE/TE)	−27.8 (−55.0, −2.22)	5.47 (−6.1, 17.3)	12.2 (2.9, 20.8)	4.8 (−14.3, 23.4)	11.5 (0.7, 22.2)	21.2 (11.6, 31.0)
Risk ratios	Total effect	1.76 (1.62, 1.88)	2.24 (2.12, 2.37)	2.31 (2.21, 2.42)	2.21 (2.02, 2.41)	2.63 (2.46, 2.81)	2.42 (2.31, 2.54)
	NDE (SEP mediation)	1.41 (1.25, 1.57)	1.66 (1.54, 1.78)	1.71 (1.62, 1.81)	1.85 (1.63, 2.07)	2.03 (1.86, 2.21)	1.83 (1.70, 1.95)
	NDE (SEP + Tob mediation)	1.40 (1.24, 1.56)	1.64 (1.53, 1.77)	1.68 (1.59, 1.78)	1.80 (1.59, 2.03)	1.97 (1.80, 2.15)	1.72 (1.60, 1.84)
	NIE (SEP mediation)	1.25 (1.16, 1.35)	1.35 (1.28, 1.41)	1.35 (1.30, 1.40)	1.20 (1.11, 1.30)	1.30 (1.22, 1.37)	1.33 (1.27, 1.39)
	NIE (SEP + Tob mediation)	1.25 (1.16, 1.36)	1.36 (1.29, 1.43)	1.37 (1.31, 1.42)	1.23 (1.13, 1.33)	1.34 (1.26, 1.42)	1.41 (1.35, 1.48)
	CDE (tobacco-free counterfactual)	2.25 (1.96, 2.56)	2.53 (2.30, 2.79)	2.47 (2.28, 2.66)	2.38 (2.03, 2.74)	2.86 (2.54, 3.18)	2.44 (2.22, 2.68)

[a]For consistency with cross-world estimates, the Māori and European mortality risks are also those predicted from the regression model.
[b]The percentage reduction in either the Māori of European mortality risk for the counterfactual of nobody ever having smoked, compared with the initial mortality risks in the first two rows of this table.
SEP = socio-economic position; CW=cross-world; Tob = tobacco; NDE = natural direct effects; NIE = natural indirect effects; CDE = controlled direct effects; effects; TE = total effect.

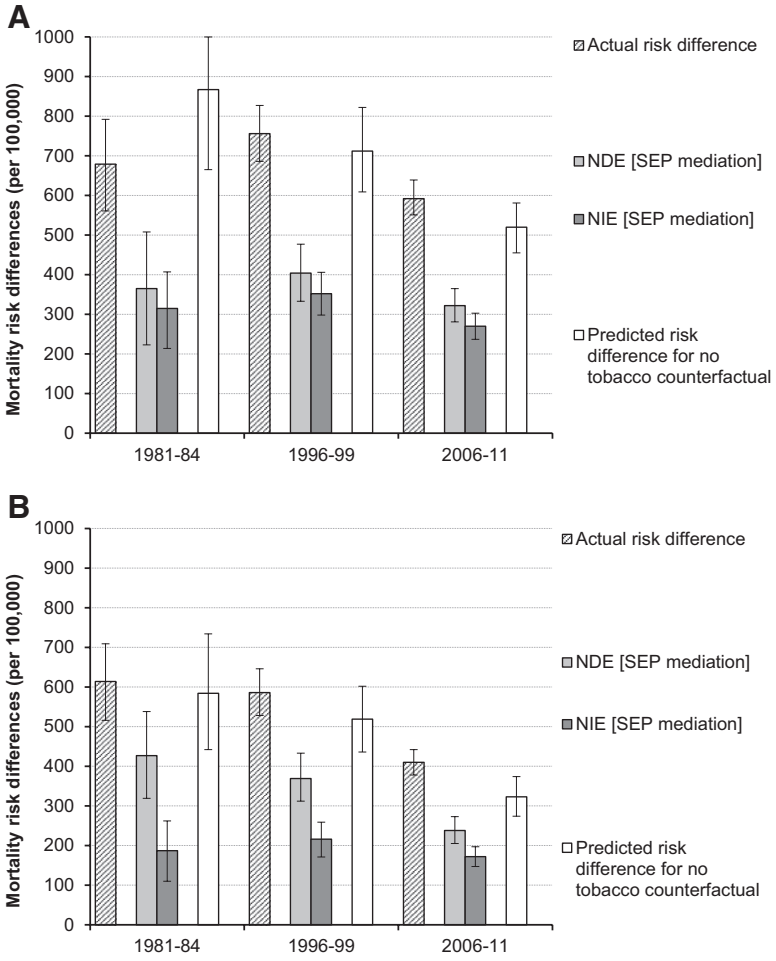

FIGURE 3. Total, natural direct effects (NDE) and natural indirect effects (NIE), and controlled direct effects on mortality risk difference scale for Māori versus Europeans (error bars are 95% confidence intervals). A, Males. B, Females.

percentage mediated in 1981–1984 [0.8% (95% CI = –2.4, 4.0); Table 2]. This modestly increased to 2.0% (1.2, 2.8) in 2006–2011. Among females, the addition of smoking made a larger incremental contribution to mediation of 3.0% points in 1981–1984 (1.7, 7.4), increasing to 7.7 percentage points in 2006–2011 (5.5, 10.0). That is, the ethnicity→smoking→mortality path, in contrast to the combined ethnicity→SEP→mortality and ethnicity→SEP→smoking→mortality paths, seems more important for females.

Research Question 3: If New Zealand Were Tobacco Free

Also shown in Figure 2 as dotted lines are the predicted age-standardized mortality risks for Māori and Europeans if (counter-to-fact) no one smoked tobacco but otherwise had identical SEP. With the exception of Māori in 1981–1984, both Māori and Europeans in all three census cohorts have substantial mortality reductions, ranging from 13.6% (Māori males 1986–1989) to 23.9% (European males 1986–1989; Table 2). Comparing the gap in absolute terms between these Māori and European counterfactual risks [controlled direct effect (CDE)] and the actual risks (total effect), the percentage of the gap eradicated is maximal in 2006–2011 at 12.2% (95% CI = 2.9%, 20.8%) for males and 21.2% (11.6%, 31.0%) for females (Table 2). While confidence intervals are wide about the percentage eradicated statistic, it monotonically increases over time for both males and females.

Sensitivity Analyses

Using SIMEX to correct for misclassification of tobacco consumption resulted in a 0.3% increase in the male 25 to 74-year-olds NIE (i.e., from 281.5 to 282.3 per 100,000) for SEP and smoking combined, and a 1.9% increase in this NIE for females. The SIMEX correction of the CDEs is more substantive (eFigure 2; http://links.lww.com/EDE/B345): the corrected CDEs are 510 and 310 per 100,000 for males and females, respectively, which is 2% and 4% less than the CDE estimates shown in Table 2. Consequently, the "percentage eradicated" increased from 12.2% to 13.9% for males with SIMEX correction, and from 21.2% to 24.4% for females.

DISCUSSION

This study has estimated that nearly half of the male mortality differences between Māori and Europeans in this high-income country were explained by four socioeconomic factors measured in adulthood (household income, labor force

status, neighborhood deprivation, and highest educational qualification). For women, the percentage of ethnic inequality mediated by adult socioeconomic position increased over time from about 30% to 42%.

The constant percentage mediated over time for males and increasing percentage mediated over time for females has sociologic face validity. That is, with increasing female participation in education post-World War II and the labor market from the 1980s, socioeconomic position among females is likely to have become more predictive of life chances, behaviors, and social outcomes, including health and mortality.

The interpretation and limitations of our study depend critically on the conceptualization of ethnicity and the assumed casual relationships of variables.[11] Our preferred conceptualization of the self-identified (in adulthood) ethnicity exposure recorded on the census is of capturing parental factors (including their ethnicity, social factors, socioeconomic position at birth, etc.), historical factors (e.g., colonization and its ongoing legacy impacts), and also the effects of early-life socioeconomic position. Under this conceptualization, the key limitation is—despite our study having rich socioeconomic data for the whole population traversing a period of 30 years—not having more measures of socioeconomic factors (e.g., assets) undertaken more accurately (e.g., annual income from tax records) over the adult life-course that would probably increase our estimate of mediation by socioeconomic position.[26] But for this incomplete measurement of adult socioeconomic position to generate bias in trends over time in the percent mediated would require this bias to vary over time—which seems unlikely. An alternative conceptualization is that self-identified ethnicity—despite being elicited in adulthood (within 5 years of death)—is actually a proxy for factors at birth, and therefore missing data on early-life socioeconomic position becomes an important confounder of the socioeconomic mediator associations with mortality. If our conceptualization of the target mediator remains adult socioeconomic position, then there is a bias to overestimating mediation by socioeconomic position as we did not isolate the adult paths (e.g., with randomized intervention analogues) separately from any effect of early-life socioeconomic position to adult mortality independent of adult socioeconomic position. In other words, there may be residual confounding of our target adult socioeconomic position measures by unmeasured early-life socioeconomic position. However, such residual confounding is also probably unlikely to vary over time and bias interpretation of trends. Third and finally, one may conceptualize ethnicity as that individual's ethnicity separable from parental characteristics (including their ethnicity) and historical antecedents, one possible realization of what VanderWeele and Robinson[11] call stronger interpretations of race/ethnicity. While we suspect that this is a conceptualization of limited usefulness (we know of no researchers or policy-makers interested in policies to reduce ethnic inequalities so conceived), analyses about this conceptualization would require richer intergenerational longitudinal data than available in this study.

The smoking mediation results are different between the natural and controlled effects approaches. Under natural effects, using multiple mediators where we add smoking to socioeconomic factors already modeled, the marginal extra mediation of ethnic inequalities is modest as socioeconomic factors themselves are causes of behaviors such as smoking, meaning that the ethnicity→socioeconomic position→smoking→mortality path is already captured by including socioeconomic position. The addition of smoking to the set of multiple mediators only captures the additional and specific ethnicity→smoking→mortality path not involving socioeconomic position. Interestingly, and also with sociologic face validity, this marginal mediation by smoking was greater for females. This is because Māori females have particularly high smoking rates, dating back to colonization when Māori females adopted smoking just as much as Māori males through to the 1900s where, using 1976 census data and demographic methods, Easton[30] (1995) showed that Māori women had notably higher smoking rates than non-Māori females in cohorts born back to 1900. Our finding that the marginal mediation from smoking increased over time for females is probably due to steeper rates of reduction in smoking rates by European women, compared with Māori women.

The controlled direct effects analyses for smoking are quite different in interpretation. Here one does not alter the distribution of socioeconomic factors by ethnicity, but rather counterfactually set the smoking variable as though it had always been "never." We found strong decreases in mortality for all sex-by-ethnic groups (Figure 2 and Table 2; ranging from 13.6% (Māori males 1981–1984) to 23.9% (European males 1981–1984, roughly equivalent to the population attributable risk % for smoking adjusted for confounding by socioeconomic position). Moreover, on an absolute scale at least, ethnic mortality inequalities would have been considerably less. For example, in 2006–2011 in a tobacco-free world, ethnic inequality would have been 12.2% (95% CI = 2.9%, 20.8%) less for males and 21.2% (11.6%, 31.0%) less for females (Table 2). However, there are limitations with our never smoking counterfactual. First, we are assuming exchangeability of mortality risks by smoking status in the outcome model: while we controlled for many socioeconomic factors, incomplete adjustment (e.g., missing early socioeconomic position if it has a notable association with mortality independent of adult socioeconomic position) will mean that mortality reductions under the counterfactual are likely overestimated. Second, and acting in the opposite direction, we quantified that misclassification of smoking may underestimate the controlled direct effect (eFigure 2; http://links.lww.com/EDE/B345). Third, had no one ever smoked, or never smoked since (say) World War II, NZ would be a different place given the importance of smoking in the economy, societal context, and survivorship effects. However, this stretch in

counterfactual thinking is also shared by other social epidemiology studies quantifying controlled direct effects.

Nevertheless, it is safe to conclude that there are notable decreases in ethnic inequalities for a counterfactual of tobacco smoking having never occurred in NZ, and this concurs with simulation modeling into the future of a tobacco-free world.[31] We argue this counterfactual is a plausible scenario to consider inasmuch as a growing number of countries (including NZ) have national tobacco-free goals and policies,[32,33] and alternative nicotine sources are gaining in the market (e.g., electronic cigarettes). Our study underlines the potential importance of this tobacco-free policy for reducing ethnic inequalities on an absolute scale but also discloses that relative inequalities may not change much (the controlled direct effect as a risk ratio is little different from the total effect risk ratios in 2006–2011; Table 2). Ideally, policy-makers should consider interventions that reduce both absolute and relative inequalities, but reducing inequalities on an absolute scale is still desirable[34] and may be the policy priority in terms of reducing inequalities.

Studies of race differences in mortality in the United States have found that socioeconomic position is an important mediator of Black:White inequalities.[35,36] But owing to lower smoking rates among Blacks compared with Whites up to the 1990s, hypothetically equalizing smoking rates between race groups would actually widen inequalities in the 1990 to 2006 period,[35] highlighting how mediation can be context and time specific. We have previously analyzed mediation for 45 to 74-year-olds in NZ for the 1981–1984 and 1996–1999 cohorts, using traditional methods of Poisson regression (comparing Māori and European rate ratios before and after adjusting for socioeconomic factors) and direct standardization (standardizing European mortality rates to Māori age and smoking distributions).[8] Our current study updates (e.g., including the new 2006–2011 cohort) and improves this previous analysis in several ways. The weighted multiple mediators approach allows an easier depiction of socioeconomic mediation on an absolute as well as relative scale, and by using an outcome model with all possible exposure (ethnicity) mediator (socioeconomic position) interactions we find greater percentage mediation in this current study. While our previous study quantified smoking mediation on an absolute scale using direct standardization, it did not allow for ethnicity-induced-smoking-mortality confounding by socioeconomic factors and did not permit a differentiation of natural compared with controlled direct effects. Compared with other previous studies on mediation of ethnic/race inequalities in mortality (cited above[35,36] and other studies examining slightly different research questions[37–40]), our study has many advantages: large cohort sizes, multiple measures of socioeconomic factors, inclusion of smoking questions from the census, repeated cohorts covering 30 years allowing an examination of trends over time in mediation, and the application of contemporary causal mediation methods.

There are two main implications of these new findings. First, reducing socioeconomic disparities between ethnic groups will tend to reduce ethnic inequalities in mortality over the long run. Second, making countries tobacco free may make a substantial contribution to decreasing ethnic mortality inequalities on an absolute scale—but perhaps not on a relative scale. These two implications are likely to be generalizable, qualitatively at least, to other high-income countries with ethnic inequalities in mortality and accompanying ethnic differences in socioeconomic position and smoking similar to those in NZ.

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