

Mortality Risk of High BMI in Life Insurance Applicants and the US Population

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Objectives.—This study seeks to quantify the mortality effect of high levels of body mass index (BMI) on life insurance applicants and participants in the National Health and Nutrition Examination Survey (NHANES) in univariate models and in successive models controlling for BMI-related diseases and conditions.

Background.—It is well established that a high BMI is associated with increased all-cause and cardiovascular mortality; however, the quantitative effect of controlling for related diseases and conditions is not well understood.

Methods.—Data were collected from over 7 million life insurance applicants submitting samples to Clinical Reference Laboratories (CRL) and 23,486 NHANES participants with available BMI and mortality status. Cox models were utilized, treating BMI as both a continuous predictor and as a categorical variable within various age and sex groups. Six Cox models were constructed in each age-sex-data group: a univariate model controlled only for age, then 5 more successively controlling for disease status (hypertension, diabetes, and heart disease), liver function tests, blood pressure/renal function, and finally hemoglobin A1c.

Results.—Overall, the effect of high BMI on mortality hazard was highest in the univariate model, and lower with successively controlled models. In the life insurance data, the residual effect of BMI in the final models was still significant above a BMI of about 35. In the NHANES data, the effect remained significant only above a BMI of about 40. In the continuous models, the hazard of BMI was persistently significant for both sexes in the CRL models, only for men in the final NHANES model.

Conclusion.—Based on this study, the effect of high BMI on mortality is significantly blunted when accounting for diseases and conditions that are associated with high BMI.

Body mass index (BMI), defined as weight (kg) divided by the square of height (m) is a commonly used measure of body composition and is frequently used as a measure of overweight and obesity. Elevated BMI has been shown, in numerous studies, to increase the risk of various adverse health outcomes,

including diabetes, heart disease, stroke, various cancers, and all-cause mortality.¹⁻³ However, in the life insurance industry, many of these other health outcomes are already considered during underwriting, leaving the question of how the BMI itself affects mortality risk. This study seeks to determine the

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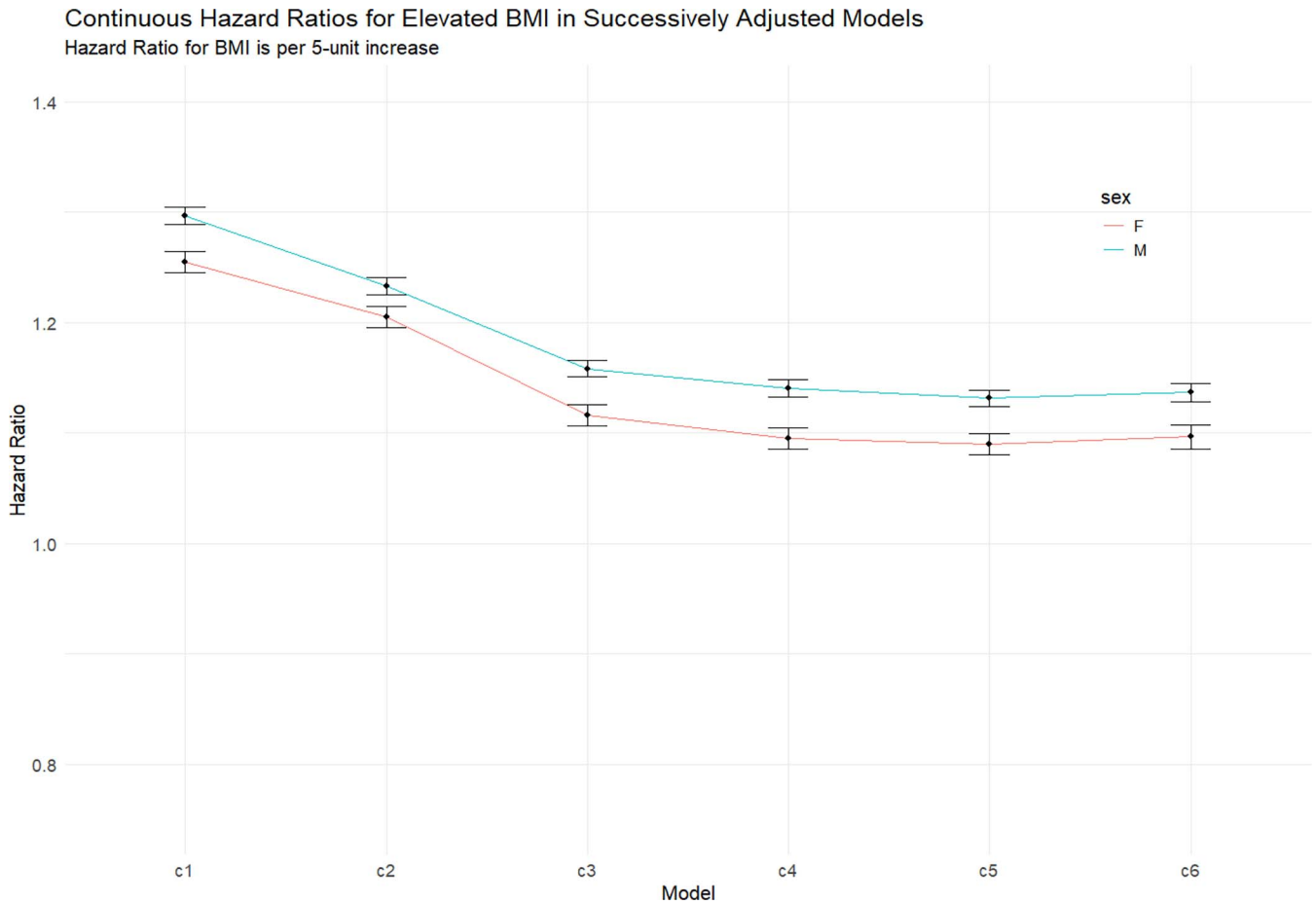


Figure 1. CRL Data, Continuous BMI.

residual mortality risk associated with elevated BMI once these other health effects have been considered.

METHODS

This study makes use of 2 different data sets. The first is from Clinical Reference Laboratories, Inc, and includes laboratory and biometric variables collected from life insurance applicants at the time of underwriting. Variables include age, sex, height, weight, BMI, laboratory tests (lipids, liver function tests, renal function tests, hemoglobin A1c and others), and answers to simple yes/no questions about the presence of diabetes, hypertension and heart disease. Vital status is determined by a third-party death audit, which includes outcomes from the Social Security Death Master File as well as data

from obituary listings, state databases, and other sources. The second data set is from the National Health and Nutrition Examination Survey (NHANES),⁴ which includes a representative sample of the US population. This data set includes similar variables, including height, weight, and BMI, as well as a variety of laboratory tests. Vital status assessment is determined via the paired mortality file, which is based on data from the National Death Index, a comprehensive source of vital status information.

Exclusion criteria included smoking (defined as answering “yes” to questions about current tobacco use or testing positive for urine cotinine at a level of 200 ng/dl), pregnancy (defined as answering “yes” to a pregnancy question or having a predicted probability of pregnancy > 0.5 on the basis of a previously determined logistic regression model), testing

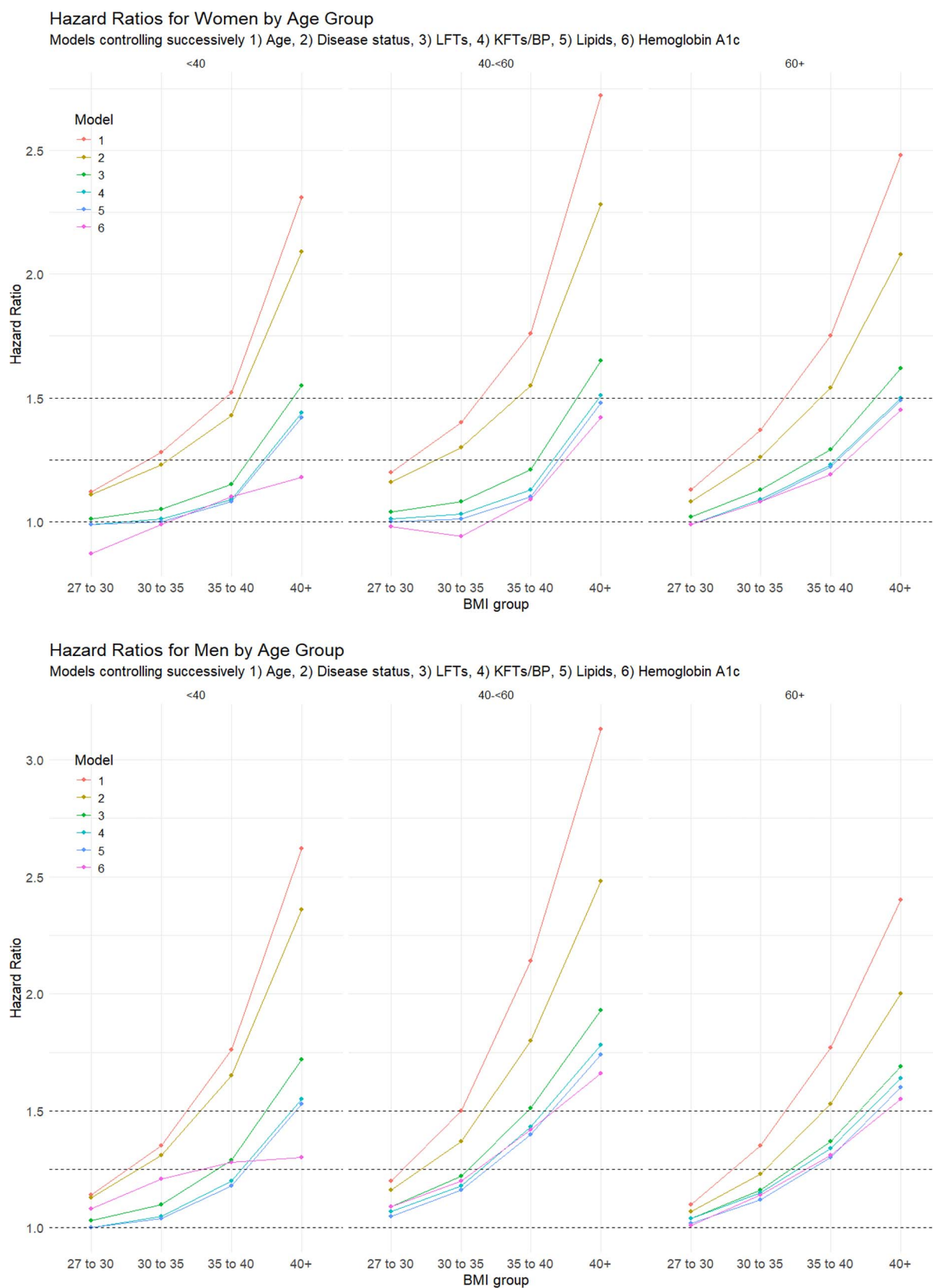


Figure 2. CRL Data, Categorical BMI.

Table 1. Basic Characteristics of the CRL Study Population

	Female			Male		
	Age <40	Age 40-59	Age 60+	Age <40	Age 40-59	Age 60+
n (millions)	1.22	1.25	0.38	1.78	1.99	0.59
Deaths	7263	18470	21423	14926	46518	49351
BMI kg/m ²	28 (5.5)	28.6 (5.4)	28.8 (5.1)	28.2 (4.5)	28.8 (4.4)	28.9 (4.3)
Systolic BP (mmHg)	111.2 (10.2)	116.8 (11.6)	123.1 (12.1)	118.3 (9.7)	121.6 (10.8)	125.6 (11.5)
Diastolic BP (mmHg)	70.9 (7.8)	73.9 (8.1)	75 (7.7)	74.9 (7.5)	77 (7.7)	76.6 (7.6)
Total cholesterol:HDL ratio	3.2 (0.9)	3.4 (1)	3.4 (1)	4.1 (1.3)	4.2 (1.3)	3.8 (1.1)
Triglycerides	107.4 (65.3)	125.1 (76.6)	135.6 (73.7)	146 (106.5)	156.8 (109.9)	141.1 (85.9)
AST (U/ml)	19.9 (11.9)	21.5 (11.3)	22.8 (11)	25.7 (15.6)	25.5 (12.7)	24.5 (11.1)
ALT (U/ml)	18.4 (14.8)	20.6 (14.7)	20.5 (12.8)	31.1 (21.6)	29.9 (17.5)	25 (14.7)
GGT (U/ml)	18.8 (20.5)	24.9 (31.6)	26.8 (33.3)	31.1 (31.6)	36.3 (43.6)	32.7 (41.9)
Albumin (g/dl)	4.4 (0.3)	4.4 (0.3)	4.4 (0.3)	4.7 (0.3)	4.6 (0.3)	4.4 (0.3)
Alkaline Phosphatase (mg/dl)	67.5 (22.9)	73.2 (24.7)	79.5 (24.5)	70.3 (19.6)	70.5 (20.4)	71.9 (22.6)
Hemoglobin A1c (%)	5.5 (0.8)	5.8 (1)	6 (1)	5.5 (0.8)	5.9 (1.1)	6.1 (1)
Serum Creatinine (mg/dl)	0.8 (0.2)	0.8 (0.2)	0.9 (0.2)	1 (0.2)	1 (0.2)	1.1 (0.3)
Hypertension (%)	3.8	18.3	44.1	4.8	20.9	44.8
Diabetes (%)	2.0	5.0	11.5	1.3	5.6	13.0
Heart Disease (%)	0.2	0.7	2.8	0.3	1.5	6.7

All quantitative variables presented as mean (sd). Qualitative variables presented as within-group percentage.

positive for hepatitis B, hepatitis C or HIV, having a missing value for cholesterol (a marker of whether blood testing was done), or having a BMI outside the study range of 22 to 80. Note that BMIs below 22 were excluded due to the previously observed inverse relationship between BMI and mortality in this range. The initial CRL data set included over 15 million applicants. After exclusions, there were approximately 7.2 million subjects, 39.6% female, mean(sd) age 44.9(12.9), with a mean follow-up time of 9.0 years, during which 157,951 deaths occurred.

In the NHANES data, participants of all ages were invited through a complex multistage probability sampling technique. This included oversampling of Mexican Americans, African Americans, low-income whites, and older people (aged 60 years and up) to create a representative sample of the general US population. Demographic data such as age and sex were collected through a questionnaire. Laboratory tests were also done in their survey which included cotinine level, lipids, liver function

tests, renal function tests, hemoglobin A1c, and other biomarkers. Body measurements such as BMI and blood pressure were measured by trained health technicians. Diabetic status was determined by yes/no question. For consistency between NHANES and CRL variables, the presence of hypertension and heart disease were derived from multiple variables. Hypertension status was determined by the average of all blood pressure readings and the participant's answer in the questionnaire ("Have you ever been told that you had high blood pressure?"). Heart disease status was determined by combining multiple yes/no questions: Have you ever been told that you had congestive heart failure, coronary heart disease, angina/angina pectoris, heart attack, or stroke? Similar exclusion criteria were also applied to the NHANES data. The initial data set included 70,190 participants. After the exclusion and removal of missing data, there were 23,486 participants, 48% female, mean (sd) age 52.8 (16.9). The mean follow-up time was 7.6 years, during which 2,735 deaths occurred.

Table 2. Basic Characteristics of the NHANES Study Population

	Female		Male	
	Age <60	Age 60+	Age <60	Age 60+
n	6,927	4,501	7,461	4,597
Deaths	173	969	261	1,332
BMI kg/m ²	31.9 (7.6)	30.8 (6.3)	30.1 (6.0)	29.5 (5.2)
Systolic BP (mmHg)	118.7 (16.1)	135.5 (20.9)	123.2 (14.7)	132.6 (18.8)
Diastolic BP (mmHg)	71.2 (11.3)	65.9 (14.8)	74.1 (12.1)	68.3 (13.9)
Total cholesterol:HDL ratio	3.9 (1.3)	3.8 (1.2)	4.7 (1.7)	4.1 (1.3)
Triglycerides*	120.3 (116.6)	131.72 (73.0)	154.9 (136.4)	132.8 (105.9)
AST (U/ml)	24.3 (18.1)	25.1 (10.0)	28.7 (14.1)	26.3 (11.8)
ALT (U/ml)	22.5 (19.3)	20.6 (11.3)	32.4 (21.7)	23.5 (17.0)
GGT (U/ml)	27.1 (40.7)	27.8 (33.1)	38.1 (50.9)	32.0 (39.6)
Albumin (g/dl)	4.1 (0.3)	4.1 (0.3)	4.4 (0.3)	4.2 (0.3)
Alkaline Phosphatase (mg/dl)	71.7 (24.6)	78.0 (25.5)	71.2 (21.3)	73.2 (29.1)
Hemoglobin A1c (%)	5.8 (1.1)	6.2 (1.1)	5.8 (1.1)	6.2 (1.2)
Serum Creatinine (mg/dl)	0.8 (0.2)	1.0 (0.5)	1.1 (0.3)	1.2 (0.5)
Hypertension (%)	31.5	75.3	33.4	69.8
Diabetes (%)	9.5	24.2	8.7	26.9
Heart Disease (%)	4.8	20.4	4.9	29.9

All quantitative variables presented as mean (sd). Qualitative variables presented as within-group percentage.

* Triglycerides samples are less than the n shown due to missing data.

To analyze the effect of BMI, each data set was split by sex and age group (<40, 40-59, and 60+ years). For each of these groups, 6 successive models were fit. The first included only age and BMI (adjusted so that the hazard ratio result is per 5-unit increase in BMI), the second adds disease question variables (hypertension, diabetes, and heart disease), the third adds in liver function studies (AST, ALT, GGT, alkaline phosphatase, and albumin), the fourth adds in renal function (creatinine level) and systolic blood pressure, the fifth adds in lipids (cholesterol:HDL ratio), and the final model adds in hemoglobin A1c. For each of these, the measure of interest is the hazard ratio for a 5-unit increase in BMI. The models were fit using Cox proportional hazards regression. For each group, models were constructed treating BMI as a continuous variable, and as a categorical variable with 5 categories (22-26.9, 27-29.9, 30-34.9, 35-39.9, and 40+). Note that when variables were heavily skewed (AST, GGT, alkaline phosphatase, creatinine, cholesterol:HDL ratio,

and hemoglobin A1c), a log transformation was applied, and when univariate testing had demonstrated a non-linear relationship between a given variable and mortality (age, AST, and creatinine), restricted cubic splines with 4 knots were used to model the relationship.

Models were constructed using R version 4.4.3⁵ and packages including tidyverse, rms, Hmisc, survey, survminer, dplyr, haven, and survival.

RESULTS

In the CRL data, with continuous models, the hazard ratio associated with a 5-unit increase in BMI was 1.30 for men and 1.25 for women in the initial model controlled only for age (Table 3). By the fourth model, the hazard ratio reached a level that was not significantly different than the next 2 models (Figure 1). This reflects a certain “saturation effect” which may indicate that this final hazard ratio (1.14 for men and 1.10 for

Table 3. Categorical Models, CRL data

					HR vs BMI of 22-27			
Sex	Age	Model	n	Deaths	27 to 30	30 to 35	35 to 40	40+
M	<40	1	1654473	13512	1.14*	1.35*	1.76*	2.62*
		2	1654473	13512	1.13*	1.31*	1.65*	2.36*
		3	1552769	12967	1.03	1.10*	1.29*	1.72*
		4	1552740	12967	1.00	1.05	1.20*	1.55*
		5	1552740	12967	1.00	1.04	1.18*	1.53*
		6	432440	1957	1.08	1.21*	1.28*	1.30*
	40-<60	1	2033022	44575	1.20*	1.50*	2.14*	3.13*
		2	2033022	44575	1.16*	1.37*	1.80*	2.48*
		3	2015340	43981	1.09*	1.22*	1.51*	1.93*
		4	2015290	43981	1.07*	1.18*	1.43*	1.78*
		5	2015290	43981	1.05*	1.16*	1.40*	1.74*
		6	924175	17408	1.09*	1.20*	1.42*	1.66*
	60+	1	673384	52708	1.10*	1.35*	1.77*	2.40*
		2	673384	52708	1.07*	1.23*	1.53*	2.00*
		3	667673	52021	1.04*	1.16*	1.37*	1.69*
		4	667655	52018	1.04	1.15*	1.34*	1.64*
		5	667655	52018	1.02	1.12*	1.30*	1.60*
		6	393628	26908	1.01	1.14*	1.31*	1.55*
F	<40	1	1146080	6641	1.12*	1.28*	1.52*	2.31*
		2	1146080	6641	1.11	1.23*	1.43*	2.09*
		3	1070491	6366	1.01	1.05	1.15	1.55*
		4	1070461	6365	0.99	1.01	1.09	1.44*
		5	1070461	6365	0.99	1.00	1.08	1.42*
		6	297167	852	0.87	0.99	1.1	1.18
	40-<60	1	1283824	17893	1.20*	1.40*	1.76*	2.72*
		2	1283824	17893	1.16*	1.30*	1.55*	2.28*
		3	1271402	17611	1.04	1.08*	1.21*	1.65*
		4	1271371	17611	1.01	1.03	1.13*	1.51*
		5	1271371	17611	1.00	1.01	1.10*	1.48*
		6	532284	5698	0.98	0.94	1.09	1.42*
	60+	1	426561	22622	1.13*	1.37*	1.75*	2.48*
		2	426561	22622	1.08*	1.26*	1.54*	2.08*
		3	422882	22320	1.02	1.13*	1.29*	1.62*
		4	422859	22320	0.99	1.09*	1.23*	1.50*
		5	422859	22320	0.99	1.08*	1.22*	1.49*
		6	245707	10693	0.99	1.08*	1.19*	1.45*
Continuous Models, CRL data								
Sex	Model	n	Deaths	HR				
M	1	4250084	110795	1.30*				
	2	4250084	110795	1.23*				
	3	4126813	108969	1.16*				
	4	4126719	108966	1.14*				
	5	4126719	108966	1.13*				
	6	1703970	46273	1.14*				

Table 3. Continued

Continuous Models, CRL data

Sex	Model	n	Deaths	HR
F	1	2809309	47156	1.25*
	2	2809309	47156	1.20*
	3	2718478	46297	1.12*
	4	2718395	46296	1.09*
	5	2718395	46296	1.09*
	6	1057915	17243	1.10*

HR = Hazard Ratio per 5-unit increase in BMI.

*p < 0.001.

women) is the final residual effect of BMI. In both sexes, the largest change in the BMI hazard ratio occurred in the 3rd model, after adding liver function tests.

In categorical models of the CRL data, the effect of various levels of BMI was significant at the 0.001 level in most age/sex categories. However, a BMI of 27-30 was non-significant in models 3-6 in young men, models 4-6 in older men, models 2-6 in young women, models 3-6 in middle-aged women, and models 3-6 in older women. In young women, only the hazard ratio associated with the highest BMI category was significant in models 3-5, and no category was significant in the final model - though this may be due to a marked reduction in the number of deaths due to missing hemoglobin A1c data (Table 3). Figure 3 illustrates the diminishment of the BMI hazard ratios in the categorical models along with the clustering of the hazard ratios in models 3 through 6.

The NHANES data were divided into 2 age groups (<60 and ≥60 years), unlike the CRL data, which used three age groups (<40, 40-59, and ≥60 years). This was due to the lower number of deaths among the younger individuals in NHANES.

Using NHANES data with continuous models, the hazard ratios for both continuous and categorical were lower than those in CRL. For men, the hazard ratio was 1.22 and it was 1.16 for women in the

continuous model controlled only for age (Table 4). The confidence interval was larger in the NHANES model (Figure 3) compared to CRL model (Figure 1), reflecting higher uncertainty in the estimated hazard ratios.

By Model 3, the hazard ratios in NHANES reached a plateau, with no further significant changes observed through Models 4 to 6 (Figure 3). The final hazard ratios were 1.10 for men and 0.98 for women, and they represent the residual association between BMI and mortality after full adjustment of the covariates. While CRL showed the largest change at Model 3, NHANES demonstrated comparable drops at Models 2 and 3, after adding disease question variables and liver function tests, respectively.

Compared to CRL, NHANES results showed limited significance at the 0.001 level. Table 4 shows the hazard ratios and their significance, both in continuous and categorical models. The continuous models hazard ratios were only significant (p < 0.001) for the first few models (Models 1-2 for men, and Model 2 for women). Models 4-6 were significant at p < 0.05 for males, but not in females.

In categorical models, high significance (p < 0.001) appeared only in younger men with BMI 40+ in Model 1 and in older men with BMI 40+ in Model 1-2. In females, the hazard ratios were only significant at 0.001 in the older age group, for BMI of 40+ in Model

Table 4. Categorical Models, NHANES data

Sex	Age	Model	n	Deaths	HR vs. BMI of 22-27			
					27 to 30	30 to 35	35 to 40	40+
M	<60	1	7461	261	1.16	1.41	2.05*	2.92**
		2	7461	261	1.09	1.21	1.58	2.02*
		3	7416	258	1.05	1.02	1.25	1.53
		4	7165	250	1.03	1.05	1.22	1.49
		5	7165	250	1.02	1.04	1.21	1.49
		6	7157	250	1.02	1.04	1.22	1.49
	60+	1	4597	1332	0.95	1.03	1.54*	2.56**
		2	4597	1332	0.92	0.96	1.38*	2.09**
		3	4560	1322	0.93	0.92	1.25	1.53
		4	4443	1273	0.95	0.97	1.33	1.70*
		5	4443	1273	0.95	0.97	1.34*	1.70*
		6	4439	1273	0.95	0.98	1.34*	1.70*
F	<60	1	6927	173	0.83	0.87	1.66	2.04
		2	6927	173	0.8	0.69	1.19	1.22
		3	6880	171	0.74	0.55*	0.83	0.82
		4	6576	157	0.71	0.53*	0.84	0.73
		5	6576	157	0.64	0.47*	0.76	0.67
		6	6566	157	0.65	0.47*	0.76	0.67
	60+	1	4501	969	0.84*	0.96	1.16	1.98**
		2	4501	969	0.77*	0.83*	0.94	1.58*
		3	4466	956	0.73*	0.76*	0.76	1.15
		4	4306	912	0.72*	0.74**	0.75	1.14
		5	4306	912	0.71**	0.73**	0.75	1.13
		6	4297	909	0.71**	0.73**	0.75	1.14

Continuous Models, NHANES data

Sex	Model	n	Deaths	HR
M	1	12058	1593	1.22**
	2	12058	1593	1.16**
	3	11976	1580	1.08*
	4	11608	1523	1.10*
	5	11608	1523	1.10*
	6	11596	1523	1.10*
F	1	11428	1142	1.16**
	2	11428	1142	1.09*
	3	11346	1127	1.00
	4	10882	1069	0.98
	5	10882	1069	0.98
	6	10863	1066	0.98

HR = Hazard Ratio per 5-unit increase in BMI.

* $p < 0.05$.** $p < 0.001$.

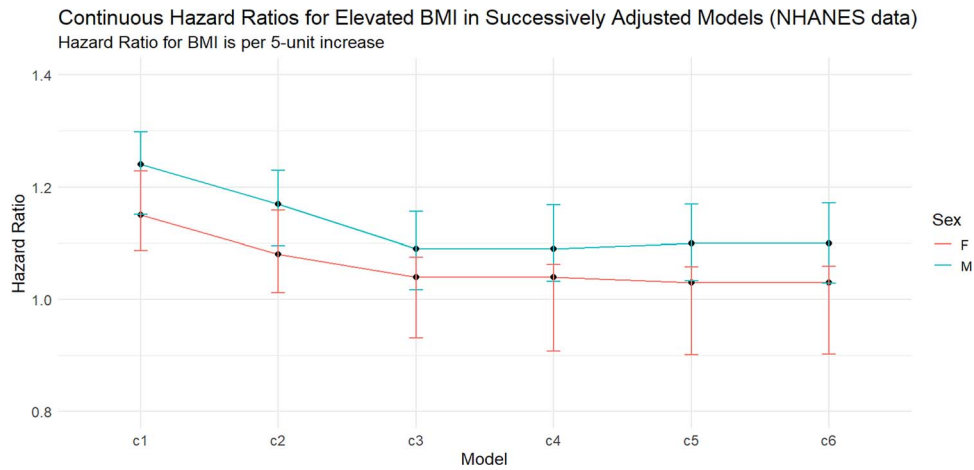


Figure 3. NHANES Data, Continuous BMI.

1, BMI of 27-30 in Models 5-6, and BMI of 30-35 in Models 4-6.

Figure 4 illustrates the hazard ratios for men and women in NHANES, revealing a slightly different pattern compared to CRL, particularly among females under age 60.

DISCUSSION

Overall, there was a good compatibility between the insurance population (CRL) and

the general US population. However, the results from NHANES are generally lower and lack significance compared to the CRL data.

The lack of statistical significance from the NHANES models likely reflects the smaller sample sizes within BMI categories and the overall lower number of deaths in the NHANES dataset. In Figure 4, the pattern of younger females' hazard ratio is likely influenced by the limited number of deaths in this

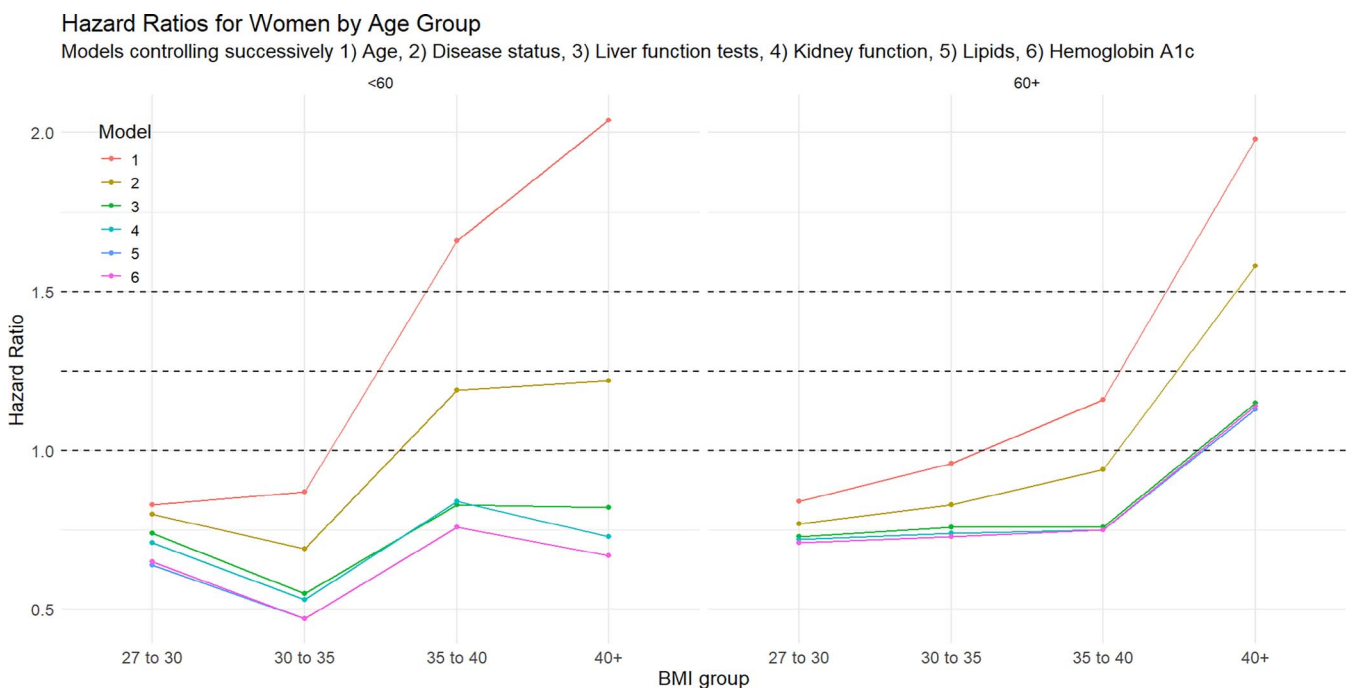


Figure 4. NHANES Data, Categorical BMI.

subgroup ($n = 173$; see Table 2), which may contribute to greater variability and less stable estimates.

Other studies have demonstrated a similar mediation effect of the BMI–mortality connection. Indeed, some have shown a mortality benefit of being overweight or obese when other disease states are present and accounted for. This is termed the “obesity paradox.” Osadnik et al studied consecutive primary care patients in the LIPIDOGram program in Poland⁵ and found that the mortality effect of elevated BMI was reduced after the inclusion of sex, education, diabetes, hypertension, and dyslipidemia in multivariable adjusted models. Indeed the “overweight” category (BMI 25–29.9) had the lowest mortality hazard. Afzal et al., in a large study of the Danish population,⁶ showed that in models controlled for cholesterol, activity, income, and smoking, the lowest risk BMI was 27.0 in the most recent (2003–2013 cohort), and that this risk nadir had moved upward substantially since the earliest cohort (1976–1978), when it was 23.7. The present study does show a similar effect in the NHANES data, with the 27–30 BMI group and, less commonly, the 30–35 BMI group having a lower risk than the reference, normal weight, group in some models. This effect was largely not present in the CRL data, and this may have to do with the screening effect of the underwriting process and other differences between the two populations.

This study demonstrates that BMI is just one part in a cluster of factors identifying mortality risk. The relationship between BMI and mortality is multifactorial, and the expression of that risk is demonstrated in the other criteria of the metabolic syndrome (also known as Syndrome X). Dyslipidemia, hypertension, metabolic dysfunction-associated steatotic liver disease (MASLD), coronary disease and type 2 diabetes are all recognized to be associated with mortality and high BMI. Thus, it is not surprising that, as variables related to these

disorders are added to Cox models examining the relationship between BMI and mortality, that the coefficients would shrink. It is perhaps surprising that the CRL-based models converged such that only individuals in the highest BMI category (40+) had hazard ratios around 1.5 (a bit higher for middle-aged men). For the insurance industry, these findings suggest that, when there is a thorough assessment of other risk factors, the mortality risk of BMI is substantially blunted but still elevated to a significant degree in the heaviest applicants. So, higher ratings for applicants with high BMI are often warranted, though consideration should be given to lowering the additional debits when ratings are already being given for diseases or conditions that are, in effect, the manifestation of the risks associated with high BMI.

CONCLUSION

The mortality effect of high BMI is substantially mediated by the inclusion of variables related to the diseases and conditions promoted by overweight and obesity.

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