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Changes in adult BMI and Waist circumference are associated with increased risk of Advanced Colorectal Neoplasia

Wambui G. Gathirua-Mwangi, PhD^{1,2}, Patrick Monahan, PhD³, Yiqing Song, MD, ScD¹, Terrell W Zollinger, DrPH¹, Victoria L. Champion, PhD², Timothy E Stump, MA³, and Thomas F. Imperiale, MD⁴

¹Department of Epidemiology, Richard M. Fairbanks School of Public Health, Indiana University, Indianapolis, Indiana

²Center for Nursing Research, School of Nursing, Indiana University, Indianapolis, Indiana

³Department of Biostatistics, Indiana University School of Medicine, Indianapolis, Indiana

⁴Indiana University Medical Center, Department of Medicine, Regenstrief Institute, Inc., and Center of Innovation, Roudebush Veterans Affairs Medical Center, Indianapolis, IN

Abstract

Background—Waist circumference (WC) is a stronger predictor of colon cancer (CRC) risk than body mass index (BMI). However, how well change of either WC or BMI predicts risk of advanced colorectal neoplasia (AN) is unclear.

Aims—To determine the relationship between change in BMI and WC from early adulthood to later age and the risk of AN, and which change measure is a stronger predictor.

Methods—In 4,500 adults, ages 50–80, with no previous neoplasia and undergoing screening colonoscopy, BMI and WC at age 21 and at time of screening were reported. Changes in BMI and WC were defined using universal risk cutoffs. Known CRC risk factors were controlled in the logistic models.

Results—Overall, model statistics showed WC change (omnibus-test $\chi^2=10.15$, 2 DF, *p*value=0.006) was a statistically stronger predictor of AN than BMI change (omnibus-test $\chi^2=5.66$, 5 DF, *p*-value=0.34). Independent of BMI change, participants who increased WC (OR=1.44; 95% CI 1.05-1.96) or maintained a high-risk WC (OR=2.50; 95% CI 1.38-4.53) at age 21 and at screening had an increased risk of AN compared to those with a low-risk WC. Study participants who were obese at age 21 and at screening had an increased risk of AN (OR=1.87; 95% CI 1.08-3.23) compared to those who maintained a healthy BMI. Maintaining an overweight BMI or increasing BMI was not associated with AN.

Conclusions—Maintaining an unhealthy BMI and WC throughout adult life may increase risk AN. WC change may be a better predictor of AN than BMI change.

Conflict of interest statement: The authors declare no potential conflicts of interest.

Corresponding author: Wambui G Gathirua-Mwangi, PhD, MPH, IU School of Nursing, 600 Barnhill Dr. NU 317C, Indianapolis, IN 46202, Phone: 317-274-1569, ggathiru@iu.edu.

Keywords

Obesity; BMI change; waist circumference change; colorectal neoplasia; cancer; precancerous polyps

INTRODUCTION

Colorectal cancer is a global public health problem [1], the third most common cancer and the third leading cause of cancer death in both men and women in the U.S. [1, 2]. In 2016, the American Cancer Society estimated 134,490 new cases and 49,190 colorectal cancer (CRC) deaths would occur [2]. Obesity is an established risk factor for CRC in both men and women [3–5], with a stronger link reported in men [6–8]. Epidemiological data suggests that 30% to 70% increased risk of CRC can be attributed to obesity [9].

Both body mass index (BMI) and waist circumference (WC) have been associated with increased risk of CRC and other chronic disease [10, 11]. However, WC is a better predictor of CRC than BMI [10], in part because WC measures visceral fat, which has been linked to increased risk of chronic diseases [10], After complete growth in height, often by age 21 [12], any weight gain in adulthood results mostly in visceral fat accumulation [13, 14], increasing an individual's BMI and WC. Increase in adult weight gain is associated with an increased risk of CRC [15, 16] and the risk stronger in men [15, 17]. Although a few studies have assessed the association of weight gain and risk of precancerous colorectal polyps [18, 19] and CRC [20–26], few studies have assessed WC change and risk of CRC [27, 28], and to our knowledge none on advanced colorectal neoplasia (AN), the composite of colorectal cancer and advanced precancerous polyps. An increase or maintaining high risk adiposity measures over time may be a better indicator of risk for AN than cross-sectional values at specific points in time.

Therefore, we sought to determine the association of changes in BMI and WC from early (age 21) to later adulthood (time of screening) and the risk of AN, and which of the two dynamic measures was a better predictor of the risk of AN. As an exploratory aim we compared the dynamic measures of BMI and WC to their static measures at age 21 and at time of screening in predicting risk of AN.

METHODS

This study was conducted at Indiana University Medical Center in Indianapolis and was approved by the institutional review boards at Indiana University and the Richard L. Roudebush Veterans Affairs Medical Center, Indianapolis, Indiana.

Study population

The parent study methods have been discussed in detail elsewhere [29]. The study was initiated to assess the factors associated with the risk for AN. Study participants were eligible for the study if they were aged 50 to 80 years and were undergoing first-time colonoscopy screening. Participants were initially recruited from two large corporations that provided screening colonoscopy for their employees, retirees, and their dependents. Due to

saturation of screening colonoscopy through these company-based programs, additional recruitment was sought from Indianapolis Gastroenterology and Hepatology, the Wishard Memorial Hospital, Roudebush Veterans Affairs Medical Center, affiliate hospitals of Indiana University Medical Center; and from Margaret Mary Community Hospital in Batesville, IN. Participants were excluded if they had previous colorectal cancer or adenomatous polyps, inflammatory bowel disease, or familial or non-familial polyposis syndrome. Persons with a previous sigmoidoscopy or diagnostic colonoscopy were not excluded unless it had been performed within the last 5 or 10 years, respectively.

Eligible subjects who were already scheduled for screening colonoscopy received a letter of introduction describing the study along with a 12-page, 50-item self- administered questionnaire and a 72-inch tape measure. Participants received a follow- up call to clarify eligibility and answer questions about the study. The study questionnaire gathered data on a variety of factors: demographic variables, family history of colorectal cancer, personal medical history (including previous lower endoscopic procedure findings and non-endoscopic screening test results), lifestyle habits (diet, exercise, cigarette smoking, alcohol use), medication use (particularly aspirin, non-steroidal anti-inflammatory drugs, and post-menopausal hormone replacement therapy), and anthropometric measures.

Adiposity measures

Participants were asked about their weight, height, and WC history. The weight history question was 1) "When you were age 21, what was your approximate weight and approximate waist circumference?" The participants were also asked to estimate their current weight (without shoes) and their waist size. In addition, a tape measure and instructions were provided in the package for the participants to accurately record their WC by measuring the smallest part, above the navel, body naturally erect, and abdomen neither drawn in nor protruded. On the day of the colonoscopy, nursing personnel at each site recorded physical measures (height, weight, waist and hip circumference). There were strong positive correlations between measured and self-reported WC (rho=0.899, p=<.0001) as well as between measured and self-reported BMI (rho=0.967, p=<.0001).

BMI was calculated as a ratio of weight and height squared (kg/m²) and grouped into three categories: normal (<25.00), overweight (25.00-29.99), and obese (30.00) using the World Health Organization's criteria [30]. In calculating BMI change, BMI at age 21 was the baseline BMI and was compared with BMI at Time 2 (current, i.e., time of screening). BMI changes were defined in 9 specific categories within three broad areas [31]: **A. Maintained BMI**: 1) *Stable-Normal*: having normal BMI at both time points; 2) *Stable-Overweight*. being overweight at both time points; and 3) *Stable-Obese*: being obese at both time points. **B. Increased BMI** from age 21 to Time 2: 4) *Normal to Overweight*: BMI increased from normal to overweight, 5) *Normal to Obese*: BMI increased from normal to obese; and 6) *Overweight to Obese*: BMI increased from overweight to obese to *Normal* from age 21 to Time 2.

Self-reported WC at age 21 and WC measured at screening (Time 2) were categorized into two risk groups using recommended international sex specific cutoffs: low risk (females <35

inches and males <40 inches) and high risk (35 inches for females and 40 inches for males) [32]. WC change was categorized as follows: 1) *Stable-low risk*: having a low risk WC at age 21 and screening; 2) *High-low risk*: having a high risk WC at age 21 but low risk at screening; 3) *Low-high risk*: having a low risk WC at age 21 and high risk WC at screening and 4) *Stable-high risk*: having a high risk WC at both time points.

Outcome Ascertainment

Colonoscopy and pathology reports were reviewed and coded by trained personnel who were blinded to survey information. Results of the colonoscopies were coded based on the most advanced histological findings. Advanced precancerous polyps were defined as an adenoma 1 cm or one with villous histology or high-grade dysplasia.

Statistical Analysis

Descriptive statistics comparing the characteristics of those with and without AN were performed. Pearson chi-square tests and two-sided t-tests were performed to compare the distributions and means of covariates and exposures of interest (BMI and WC) by AN status. Multiple logistic regression analysis was used to estimate the risk of AN based on changes in BMI and changes in WC. Three separate adjusted models were assessed: 1) BMI change as the risk factor; 2) WC change as the risk factor; and 3) both BMI change and WC change as the risk factors. Very few participants reduced their BMI (n=11) or reduced their WC (n=26), therefore, these individual cases were excluded from the analytical dataset due to the low statistical power of detecting a true effect. The exclusions of these two categories led to fewer BMI change and WC change categories. Within two broad BMI change categories (Maintaining and Increasing BMI), six specific risk categories were used in the models: 1) stable-normal, 2) stable-overweight, 3) stable-obese; increase from 4) normal to obese, 5) normal to overweight and 6) overweight to obese were used in the models with stable-normal as the reference category. For WC change, three risk categories were used, 1) stable-low, 2) low-high and 3) stable-high risk, with stable-low risk WC as the reference category.

Several known factors for CRC were included in the logistic regression model: age, race, gender, education, smoking, NSAID use, physical activity, alcohol intake, family history, red meat intake, vegetable intake, and estrogen use in women [33]. These factors were assessed as confounding variables by comparing the crude and adjusted OR of the BMI and WC.

To assess which adiposity measure (BMI change or WC change) was statistically significantly better at predicting risk of AN, we assessed the model statistics when each adiposity change variable was in the model alone (with covariates) and also when both BMI change and WC change were simultaneously considered in the model. Since BMI and WC are related, we assessed collinearity of BMI change and WC change. The collinearity diagnostics did not indicate that the correlations (rho=0.39, p-value <.0001) of the two measures were affecting the conclusions drawn from the analysis. The model statistics of interest were Akaike Information Criterion (AIC), c-statistic, the Type 3 (i.e., adjusted for other variables in the model) omnibus likelihood ratio test for the variable of interest, and the Hosmer and Lemeshow goodness-of-fit test [34]. Models with lower AIC, higher c-statistic, and lower p-value were considered better models statistically [34].

All statistical analyses were performed using SAS for WINDOWS software, version 9.4. All tests were two-sided and p-values of <0.05 were considered statistically significant.

RESULTS

Descriptive characteristics of the participants are summarized in Table 1. The mean age of the participants was 57.3 (\pm 6.8) years; 52% were women. Most of the study participants were non-Hispanic white and had high levels of education. Those with AN were more likely to be, males, to report a higher intake of red meat, and lower rates of vegetable intake as well as higher rates of, alcohol use, cigarette smoking, physical inactivity and a family history of colorectal cancer.

Table 2 shows the results of logistic regression analyses assessing the relationship between change in BMI and change in WC on the risk of AN. Being obese at age 21 and at the current age (stable-obese), compared to stable-normal, was associated with increased risk of AN (OR=1.87). Neither increasing BMI nor stable-overweight between age 21 and current age was associated with an increased risk of AN. All BMI change categories were not significant when WC change was included in the model. Regarding WC, when WC change was assessed alone in the model, only stable-high risk WC was significantly associated with a two-fold increased risk of AN (OR=2.16). However, when BMI change was controlled for in the model, an increase in WC from low to high risk (OR=1.44) and a stronger association of stable-high risk WC (OR=2.50) were associated with an increased risk of AN.

Model statistics that summarize the overall impact of including WC change alone, BMI change alone, and both variables of interest, on the overall model are presented in Table 3. All models (adjusted for confounders) were similar in discrimination and goodness-of-fit. As shown by the Hosmer and Lemeshow goodness of fit measures [34], the data fit well in predicting risk of AN. Furthermore, as indicated by the c-statistic, all models had a high (76%) and comparable ability to discriminate those with versus without AN. For all models, the likelihood ratio tests for significance of the overall model were significant, indicating that the set of covariates in the model, along with BMI and/or WC, were a strong set of predictors of AN. As expected, the base model (with covariates only) had the largest AIC. The lower AIC for the other models indicates that the model. The omnibus test for the variables of interest showed that WC change (*p-value=.004*) and not BMI change (*p-value=. 11*) predicted risk of AN. And when both variables were included in the model, only the omnibus test of WC change was significant (*p=.006*); BMI change was not significant (*p=.* 34)

In our exploratory aim, assessing whether dynamic or static measures were better at predicting risk of AN, we found that those models with dynamic measures of BMI or WC compared to those with static measures did not differ substantially in discriminating those with versus those without AN (Table 4). For BMI, the static measure of BMI at age 21 (omnibus-test χ^2 =9.53, 2 DF, *p*-value=0.009, obese vs. normal OR=1.91, 95% CI 1.22-3.00, overweight vs. normal OR=1.27, 95% CI 0.96-1.67), but not current BMI (omnibus-test χ^2 =0.22, 2 DF, *p*-value=0.89; obese vs. normal OR=1.07, 95% CI 0.79-1.45; overweight vs.

normal OR=1.02, 95% CI 0.76 vs. 1.37), was better than dynamic BMI (*p=0.11*) at predicting risk of AN. All WC measures, dynamic and static, significantly predicted risk of AN. Interestingly, WC at age 21 (omnibus-test χ^2 =7.53, 2 DF, *p-value*=0.006; high vs. low risk OR=1.85, 95% CI 1.19-2.86) was a stronger predictor of risk for AN than current WC (omnibus-test χ^2 = 4.60, 2 DF, *p-value*=0.03; high vs low risk OR=1.29, 95% CI 1.02-1.63).

DISCUSSION

In this study, we observed a positive association of increases in WC from early to later adulthood with risk of AN. Maintaining an obese status and a high risk WC over time were also associated with increased risk of AN. Overall, WC change appeared to be a stronger predictor of AN compared to BMI change. To our knowledge, no other study has examined change using risk categories, and none has examined the association of BMI change and WC change with the risk of AN.

Among the few studies conducted on the association between weight change and AN, weight gain was found to be associated with increased risk of AN; this is similar to the findings of our study. Our study differed from other studies since we assessed change using obesity risk categories while other studies assessed weight gain as an absolute value in kilograms or pounds. Weight gain from age 18 [18] or 10 years prior to screening [19] were associated with a two-fold increased risk of colon adenomas. We conducted a sensitivity analysis using similar methods and actual weight difference, but did not find any association with risk for AN (OR= 1.0; 95% CI 0.70-1.36 Quartile 4 vs. Quartile 1), data not shown. Also, since AN is the combination of colorectal cancer (CRC) and advanced precancerous polyps, we reviewed studies assessing weight gain and CRC and found conflicting results for the association of weight gain and CRC. In some studies, weight increase has been associated with increased risk for colorectal cancer [24, 26, 35] while others have shown increased risk in men but not women [21–23].

Waist circumference is considered a reliable surrogate of visceral obesity because it is more closely related to obesity-associated cardio metabolic disorders [10, 36]. Few studies have assessed WC change and risk of CRC. One study found increase in WC to be positively associated with CRC risk in men but not women [28], and another found no association [27]. To our knowledge, this may be the first study to examine the association of WC change and risk of AN, the composite of advanced precancerous polyps and colorectal cancer. Our findings indicate that participants who at age 21 and later at screening had WC equal to or larger than the recommended maximum value (women 35 inches and men 40 inches) had an increased risk of AN. In addition, those whose WC increased from early to late adulthood had an increased risk of AN, but only when their change in BMI was controlled in the model. Overall, WC measured as a dynamic or static value was found to be a better predictor of risk for AN compared to BMI measures. This is consistent with other studies that have shown WC to be a better predictor of diabetes [37] and cancer [10]. Even when BMI change was accounted for, WC change emerged as an independent predictor of AN. These results indicate that perhaps WC change provides a unique prediction of AN separate from the characteristics that WC and BMI share in common.

The findings of this study are strengthened by the concurrent assessment of both WC and BMI in relation to risk of AN. In adults, WC is a better predictor of obesity-related health risk than BMI [38], however, a combination of both factors is a better estimate of health risk than either factor alone [39]. This is because health risk increases from normal weight through obese BMI categories, but within each BMI category, those with higher WC values have a greater health risk than those with normal WC values [40]. Although we did not create a single variable that combined both WC and BMI, we adjusted for the effect of the other in the regression models to better understand the unique contribution of each adiposity measure on the risk of AN. Additional strengths of the study include the large sample size (n=4,500) and weight history assessment. The findings of this study may be generalizable to the non-Hispanic white population who were a majority in the study. Although a majority of the participants were non-Hispanic white, the obesity rates in our study are comparable to the national age-adjusted obesity rates (35.9% vs 34.9%) [41].

Study limitations are those inherent in an observational study. There was the possibility of intentional and unintentional errors for self-reported WC as well as height and weight used to calculate BMI. However, self-reported and measured weights have previously been reported to be highly correlated [42, 43]. The associations were modeled based on selfreported historical WC, weight and height, which may lead to a recall misclassification of WC and BMI. However, it is likely that the same amount of misclassification error (nondifferential) occurred in those with and without AN and perhaps attenuated the results. Also, we were unable to assess the impact of BMI decrease on risk of AN because the number of subjects were too small. Nonetheless, we adjusted several of the known colorectal cancer risk factors in an attempt to isolate the specific impact of adiposity measures on AN. Past studies have revealed there may be gender differences in the association of BMI, WC and the risk of CRC [15, 17], although we adjusted for the effect of gender in all our regression models we did not assess the relationship of WC Change, BMI Change and risk of AN for men and women strata. Finally, our assessment was based on neoplasia diagnosis rather than incidence. Neoplasia may have developed a considerable amount of time before the diagnosis, and this may lead to errors in estimating the period at risk. Due to these limitations, causal relationships cannot be drawn from this study.

In conclusion, our results support previous findings that adiposity is a risk factor for advanced neoplasia of the colon and rectum, with WC change being an independent and stronger predictor of AN compared to BMI change. The results emphasize the importance of maintaining a healthy BMI and WC throughout adult life which may prevent AN. Although the effect on risk for AN associated with BMI and WC dynamic measures did not differ substantially from that of their static values, dynamic measures may be useful in identifying and stratifying risk for AN. Weight gain, expressed in terms of movement between BMI or WC categories may be more practical and useful in clinical practice than absolute weight gain, however, this should be determined in subsequent studies. Health care providers may use the findings as a prevention strategy for colorectal cancer when counseling patients, in line with the American Society of Clinical Oncology's prioritization of educating providers and patients on the role of energy balance as a strategy to reduce the impact of obesity on cancer [44]. Long-term prospective studies are needed to validate our findings and to explore the association of changes in BMI and WC and the risk of AN. Finally, in our exploratory

aim, both BMI and WC at age 21 significantly predicted risk of AN, an aim worth further exploration. These preliminary results support emerging evidence that early life adiposity affects the risk of colorectal cancer many decades later [45].

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics of Study Subjects by Advanced Neoplasia Status

	5 5	5	1	
	Advanced Neoplasia (n=410)	No-Advanced Neoplasia (n=4,090)		p value
	Mea	an (SD)	t value	
Age (year)	61.4 (9.0)	56.9 (6.3)	568.7	<.0001
Pack years	20.8 (26.1)	8.7 (16.6)	36.5	<.0001
Vegetable intake-weekly	15.1 (8.2)	15.9 (7.5)	140.0	0.05
Red meat intake-weekly	5.1 (3.0)	4.1 (2.5)	111.3	<.0001
	n	(%)	X ² [DF]	
Gender				
Male	250 (61.0)	1,928 (47.1)	28.6 [1]	<.0001
Female	160 (39.0)	2,162 (52.9)		
Race				
Non-Hispanic White	358 (87.3)	3884 (95.0)	76.8 [2]	<.0001
Non-Hispanic Black	44 (10.7)	106 (2.6)		
Other	8 (1.9)	100 (2.4)		
Education				
High School Education	152 (37.1)	1060 (26.0)	47.8 [3]	<.0001
Trade/Vocational School	62 (15.1)	401 (9.8)		
College Education	141 (34.4)	1682 (41.2)		
Postgraduate	55 (13.4)	940 (23.0)		
*Alcohol Use				
No problem drinking	323 (78.8)	3492 (85.4)	12.9 [1]	0.0003
Problem drinking	87 (21.2)	595 (14.6)		
[†] Aspirin-NSAID intake				
Low	258 (62.9)	2649 (64.8)	6.6 [2]	0.04
Medium	46 (11.2)	581 (14.2)		
High	106 (25.9)	860 (21.0)		
Estrogen (Females)				
No	37 (23.1)	918 (42.3)	23.3 [1]	<.0001
Yes	123 (76.9)	1238 (57.4)		
Exercise				
0-<2 hrs./week	239 (58.3)	1743 (42.6)	32.6 [2]	<.0001
2 to <4hrs./week	153 (37.3)	2054 (50.2)		
>4 hrs./week	9 (2.2)	102 (2.5)		
Family History of Colored	ctal Cancer			
Yes	55 (13.4)	372 (9.1)	8.1 [1]	0.004
No	355 (86.6)	3718 (90.0)		

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* Problem drinking is defined as having 11 or 6 drinks/week for males and females respectively.

 † Aspirin-NSAID intake is defined as, Low 1/month for 1–5 years or 1-/month for 6–10 years; Medium 1–7/month for 11–20 years or 2–5/week for 6–10 years or 1–7/month for 20yrs; and High 2 per week for 10 years or daily for 6+ years.

Table 2

Association of BMI Change, Waist Circumference Change and risk of Advanced Neoplasia

			Change from Age 21 to Current	
	Distr	ibution	Models a [*]	Model b [*]
	2.000	(%)	WC change and BMI change in separate models	WC change and BMI change in model together
	AN	No AN		
BMI Change				
Stable-Normal BMI	81 (21.0)	1130 (28.1)	Reference	Reference
Stable-Overweight	37 (9.6)	246 (6.1)	1.54 (0.97–2.45)	1.34 (0.83–2.16)
Stable-Obese	23 (6.0)	132 (3.3)	1.87 (1.08–3.23)	1.01 (0.51–1.99)
Normal to Obese	80 (20.7)	707 (17.6)	1.14 (0.80–1.62)	0.86 (0.57-1.32)
Normal to Overweight	113 (29.3)	1328 (33.1)	1.00 (0.72–1.38)	0.89 (0.64–1.25)
Overweight to Obese	52 (13.5)	473 (11.8)	1.04 (0.69–1.57)	0.74 (0.46–1.19)
Waist Circumference change				
Stable-Low risk	199 (49.6)	2408 (59.7)	Reference	Reference
Low-High risk	175 (43.6)	1442 (35.8)	1.23 (0.97–1.57)	1.44 (1.05–1.96)
Stable-High risk	27 (6.7)	182 (4.5)	2.16 (1.35-3.46)	2.50 (1.38-4.53)

The table shows results of several analyses conducted to assess the association of individual and combined measurements and risk of advanced colorectal neoplasia. All logistic regression models were adjusted for age (continuous), race (non-Hispanic white, non-Hispanic black, Other), gender (male vs female), and education (high school, trade/vocational, college education and postgraduate), family history of colon cancer (yes/no), smoking (pack years), exercise, alcohol use (yes/no), red meat intake (daily), vegetable intake (daily), use of aspirin or other NSAIDs and estrogen use (yes/no).

Bolded Odds ratios and 95% CI indicate estimates that are statistically significant.

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Table 3

Model statistics of changes in Body Mass Index and Waist circumference in predicting risk of Advanced Colorectal Neoplasia

					Likeliho ov	hood Ratio Te overall mode	io Test for ode	Likelihood Ratio Test for Type 3 Omnibus Likelihood Ratio Test of Hosmer and Lemeshow overall mode Variable of Interest Goodness-of-Fit Test	lihood R Interest	atio Test of	Hosmer Goodi	r and L ness-of-	losmer and Lemeshow Goodness-of-Fit Test
		Z *	AIC	C-stat	* N AIC C-stat χ^2 DF p-value	DF	p-value	χ^{2}	DF	DF p-value χ^2 DF p-value	x ²	DF	p-value
M0:	M0: Base model-covariates only	4245	2306	0.757	4245 2306 0.757 370.37 21 <.0001	21	<.0001				1.91	~	0.98
M1:	M1: M0 + BMI Change	4227	4227 2215	0.757	344.24 26	26	<.0001	9.08	5	0.11	8.10	8	0.42
M2:	M2: M0 + Waist Change	4185	2260 0.762	0.762	368.12 23	23	<.0001	10.98	7	0.0041	2.88	8	0.94
M3:	M3: M0 + BMI Change + Waist Change 4112	4112	0010			00	1000	BMI change: 5.66	3	0.34		c	
			0617	0./01	1000.> 82 6C.04C 10/.0 0612	87	1000.>	WC change: 10.15 2	7	0.0062	c/.0	ø	0C.U

ege education, and graduate MU = age (years), general, nace (non-ruspane wine, non-ruspane ones), provident nace (non-ruspane) and one of aspirin (other NSAIDs and estrogen use education), family history of colon cancer, smoking (pack years), exercise (low, moderate, high), alcohol use (yes or no), red meat intake, vegetable intake, use of aspirin (other NSAIDs and estrogen use (yes/no).

* Because sample sizes are different for each model, the significance of the variable of interest is indicated by the omnibus test and cannot be computed by the chi-square difference test between the overall model chi-square values of the base model and the model of interest (e.g., M1, M2, M3); the overall model chi-square is displayed simply to convey the overall significance of each model. Author Manuscript

Table 4

Model statistics comparing dynamic and static measures of Body Mass Index (BMI) and waist circumference in predicting risk of advanced colorectal neoplasia

					Likelihood Ratio Test for overall model	ou wan		Test of	e 3 Omnibus Likelihood R: Test of Variable of Interest	9110	Good	Goodness-of-Fit T	Hosmer and Lemeshow Goodness-of-Fit T
		2 *	AIC	C-stat χ^2	x ²	DF	p-value	x²	DF	p-value	x ²	DF	p-value
M0:	M0: Base model-covariates only 4245 2306	4245	2306	0.757	370.37	21	<.0001				1.91	8	0.98
BMI	BMI Measures												
M1:	M1: M0 + BMI Change	4227	2215	0.757	344.24	26	<.0001	9.08	5	0.11	8.10	×	0.42
M2:	M2: M0 + BMI Current	4235	2289	0.758	362.15	23	<.0001	0.22	2	0.89	1.81	×	0.98
M3:	M3: $M0 + BMI$ at age 21	4228	2274	0.760	371.29	23	<.0001	9.53	2	0.0085	8.05	×	0.43
MC]	WC Measures												
M4:	M4: M0 + Waist Change	4185	2260	0.762	368.12	23	<.0001	10.98	2	0.0041	2.88	×	0.94
M5:	M5: M0 + Waist Current	4232	2281	0.760	367.25	22	<.0001	4.60	1	0.03	4.03	×	0.85
M6:	M6: $M0 + Waist at age 21$	4216	4216 2279	0.759	370.37	22	<.0001	7.53	-	0.0061	10.41	×	0.24

* Because sample sizes are different for each model, the significance of the variable of interest is indicated by the omnibus test and cannot be computed by the chi-square difference test between the overall model chi-square values of the base model and the model of interest (e.g., M1, M2, M3); the overall model chi-square is displayed simply to convey the overall significance of each model. (yes/no).