Home Cooked Meals With Whole, Plant Foods, and the Protection Against Central Adiposity

Nicholas V. Neuwald, Arlette C. Perry, FACSM, Wesley N. Smith
Laboratory of Clinical and Applied Physiology, Coral Gables, FL

ABSTRACT

Background: It is well-established that central adiposity (CA) is associated with the risk of chronic disease. Diets high in processed foods and low in whole, fibrous plant foods, are often cited as risk factors for the development of CA and its adverse metabolic sequelae. Much of the processed foods consumed consist of pre-prepared foods and foods eaten outside of the home. Purpose: To examine whether the consumption of processed, ready-to-eat (PRE) or restaurant meals that lack whole, plant-based foods was positively associated with CA in male and female adults. Methods: A total of 2,703 adults (1,521 females and 1,182 males), from a HealthSnap wellness assessment platform used in physicians' offices across the country, self-reported their frequency of consuming PRE foods or restaurant meals versus whole, plant foods. CA was based upon a waist-to-hip ratio of ≥ 0.95 and ≥ 0.85 for males and females, respectively. To identify the association between PRE and CA, a chi-squared analysis (χ^2) was performed across quintiles of PRE by CA, and an odds ratio (OR) was calculated. Results: A significant association between PRE and CA (χ^2 [4, n = 2703] = 48.27, p < 0.001) was observed. These associations remained significant regardless of gender. The OR for CA among patients in the top 20%, *Q5,* was compared to the lowest 20%, *Q1,* for PRE. The OR of a patient having CA in *Q5* for PRE was 275% higher than *Q1* (OR: 2.75, 95% CI: 1.95-3.89, p < 0.001). Conclusion: A strong positive association exists between dietary consumption of PRE and CA. This supports the consumption of more home-cooked meals with whole, plant foods over PRE in the clinical setting to protect against CA and its adverse health consequences.

INTRODUCTION

- Central adiposity has been associated with chronic diseases in various populations independent of body mass, smoking, activity level, alcohol intake, and gender.[1-4]
- The cause of the increased risk associated with central fat is likely the result of chronic low-grade inflammation and insulin resistance,[5] increases in hypertension,[6] and dyslipemia.[7]
- Waist to hip ratio is a superior measure of all-cause mortality, heart disease, and diabetes compared with BMI.[8-9]
- Americans consume roughly 36% of their total calories from eating outside the home[10] and 58% of their total calories comes from ultra-processed foods.[11]
- Higher ultra-processed food consumption is associated with weight gain and particularly increases in abdominal adiposity.[12-14]

PURPOSE

To examine whether the consumption of processed, ready-to-eat (PRE) or restaurant meals that lack whole, plant-based foods was positively associated with CA in male and female adults.

METHODS

Participants: 2,703 adults (1,521 females and 1,182 males), from a HealthSnap wellness assessment platform used in physicians' offices across the country. The assessment was designed to be fast, non-invasive incorporating anthropometric measurements, movement screens, and questionnaires to provide patients' wellness insights regarding exercise and nutrition.

Physical Measures: To obtain waist-to-hip ratio, each patient had waist circumference measured between the the 12th rib and iliac crest. Hip measurement was taken over the greater trochanters and widest segment of the torso. Patients were being categorized by having android-pattern, central adiposity if the waist-to-hip ratio was ≥ 0.95 for males or ≥ 0.95 for females .

Questionnaire: Patients were asked to report how often they consumed home-cooked meals using whole foods versus consuming foods at restaurants or in pre-prepared packages. Points were given for more frequent consumption of home-cooked meals using whole foods, and scored responsed were separated into quintiles.

Statistical Analysis: A chi squared analysis was used to determine significant associations between PRE and CA. An odds ratio was calculated within data quintiles to quantify strength of association between PRE and CA. significance was accepted at *p<.05. All data analysis was performed using JMP statistical software.

RESULTS

Figure 1. Mosaic Graph (Fat Patterning versus Sodium-Potassium Ratio Type)

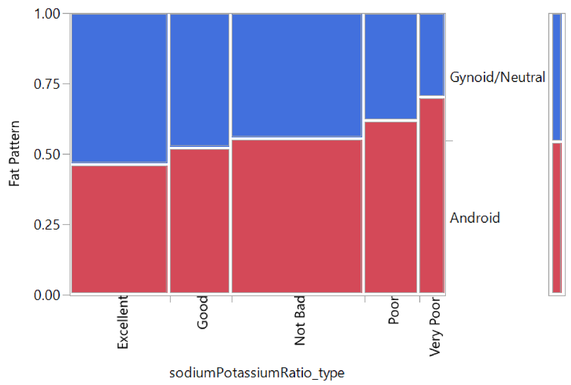

Figure 1. Graphical representation of contingency table (Table 1). Vertical length of each rectangle is proportional to the proportions of the Y variable in each level of the X variable.

RESULTS

Table 1. Contigency Table

sodiumPotassiumRatio_type	Count / Total % / Col % / Row %	Android	Gynoid/ Neutral	Total
Excellent		331	384	715
		12.25	14.21	26.45
		22.46	31.24	
		46.29	53.71	
Good		235	214	449
		8.69	7.92	16.61
		15.94	17.41	
		52.34	47.66	
Not Bad		530	423	953
		19.61	15.65	35.26
		35.96	34.42	
		55.61	44.39	
Poor		245	152	397
		9.06	5.62	14.69
		16.62	12.37	
		61.71	38.29	
Very Poor		133	56	189
		4.92	2.07	6.99
		9.02	4.56	
		70.37	29.63	
Total		1474	1229	2703
		54.53	45.47	

Table 1. Contingency table displaying frequency distribution of subjects' fat patterning versus ratio of sodium-to-potassium separated into quintiles.

Table 2. Chi Squared Analysis

N	DF	-LogLike	RSquare (U)
2703	4	24.503845	0.0132

Test	ChiSquare	Prob>ChiSq
Likelihood Ratio	49.008	<.0001*
Pearson	48.271	<.0001*

Table 2. Table showing number of subjects (N), degrees of freedom (DF), and results of chi squared analysis.

RESULTS

Table 3. Odds Ratio (Q5 versus Q1)

Odds ratio	2.7553
95 % CI:	1.9512 to 3.8908
z statistic	5.756
Significance level	P < 0.0001

Table 3. Table displaying results for odds ratio, 95% confidence interval, and z statistic for Q5 versus Q1.

DISCUSSION

- Central adiposity is a closer indicator of chronic disease risk than either BMI or total body fat percentage.
- Much of the obesity research fat patterning is often either overlooked or assumed to have a strictly genetic and hormonal etiology.
- This study found a strong positive association between dietary consumption of processed foods and central adiposity.
- The 20% of subjects with the diet highest in processed foods had 275% higher odds ratio than the 20% of subjects with lowest processed diet.
- This study highlights the impact of a diet high in processed food and the risk of having high central adiposity.
- While purely correlational, this research can help support the development of intervention studies to investigate the dietary mechanism of fat patterning.
- Studies like these may empower physicians, dietitians, and patients to switch from a reactive to a preventative outlook on obesity and other chronic diseases. Cancer, diabetes, Alzheimer's, heart disease, are to a considerable extent, all connected through nutrition.

IMPLICATIONS

The consumption of more home-cooked meals with whole, plant foods over processed foods in the clinical setting may protect against central adiposity and its adverse health consequences.

REFERENCES

1. Donahue, R., Bloom, E., Abbott, R., Reed, D., & Yano, K. (1987). CENTRAL OBESITY AND CORONARY HEART DISEASE IN MEN. *The Lancet*, *329*(8537), 821-824. doi:10.1016/s0140-6736(87)91605-9
2. Rexrode, K. (1998). Abdominal Adiposity and Coronary Heart Disease in Women. *JAMA*, *280*(21), 1843. doi:10.1001/jama.280.21.1843
3. Prineas, R., Folsom, A., & Kaye, S. (1993). Central adiposity and increased risk of coronary artery disease mortality in older women. *Annals Of Epidemiology*, *3*(1), 35-41. doi:10.1016/1047-2797(93)90007-q
4. Diaz, M., Jimenez, S., Garcia, R., Bonet, M., & Wong, I. (2009). Overweight, obesity, central adiposity and associated chronic diseases in Cuban Adults. *MEDICC Review*, *11*(4), 23-28. Retrieved from https://www.medigraphic.com/cgi-bin/new/resument.cgi?IDARTICULO=62412
5. Puder, J., Varga, S., Kraenzlin, M., De Geyter, C., Keller, U., & Müller, B. (2005). Central Fat Excess in Polycystic Ovary Syndrome: Relation to Low-Grade Inflammation and Insulin Resistance. *The Journal Of Clinical Endocrinology & Metabolism*, *90*(11), 6014-6021. doi:10.1210/jc.2005-1002
6. PARRINELLO, G., SCAGLIONE, R., PINTO, A., CORRAO, S., CECALA, M., & DISILVESTRE, G. et al. (1996). Central obesity and hypertensionThe role of plasma endothelin. *American Journal Of Hypertension*, *9*(12), 1186-1191. doi:10.1016/s0895-7061(96)00259-2
7. Pi-Sunyer, F. (2004). The Epidemiology of Central Fat Distribution in Relation to Disease. *Nutrition Reviews*, *62*, S120-S126. doi:10.1111/j.1753-4887.2004.tb00081.x
8. Welborn, T., & Dhaliwal, S. (2007). Preferred clinical measures of central obesity for predicting mortality. *European Journal Of Clinical Nutrition*, *61*(12), 1373-1379. doi:10.1038/sj.ejcn.1602656
9. Wang, Y., Rimm, E., Stampfer, M., Willett, W., & Hu, F. (2005). Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *The American Journal Of Clinical Nutrition*, *81*(3), 555-563. doi:10.1093/ajcn/81.3.555
10. Lachat, C., Nago, E., Verstraeten, R., Roberfroid, D., Van Camp, J., & Kolsteren, P. (2011). Eating out of home and its association with dietary intake: a systematic review of the evidence. *Obesity Reviews*, *13*(4), 329-346. doi:10.1111/j.1467-789x.2011.00953.x
11. Martínez Steele, E., Baraldi, L., Louzada, M., Moubarac, J., Mozaffarian, D., & Monteiro, C. (2016). Ultra-processed foods and added sugars in the US diet: evidence from a nationally representative cross-sectional study. *BMJ Open*, *6*(3), e009892. doi:10.1136/bmjopen-2015-009892
12. Halkjær, J., Tjønneland, A., Overvad, K., & Sørensen, T. (2009). Dietary Predictors of 5-Year Changes in Waist Circumference. *Journal Of The American Dietetic Association*, *109*(8), 1356-1366. doi:10.1016/j.jada.2009.05.015
13. Juul, F., Martinez-Steele, E., Parekh, N., Monteiro, C., & Chang, V. (2018). Ultra-processed food consumption and excess weight among US adults. *British Journal Of Nutrition*, *120*(1), 90-100. doi:10.1017/s0007114518001046
14. Bahadoran, Z., Mirmiran, P., & Azizi, F. (2015). Fast Food Pattern and Cardiometabolic Disorders: A Review of Current Studies. *Health Promotion Perspectives*, *5*(4), 231-240. doi:10.15171/hpp.2015.028