

## Increased Risk of Cardiovascular Disease in Obese Female University Students with Binge Eating Episodes

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### ABSTRACT

Since binge eating is known to be related to increased risk of body weight fluctuations, it may be associated with increased health risks. This study was conducted to investigate the risks of binge eating on the cardiovascular system of female university students in Korea. Sixty-five female university students who were interested in a weight control program were recruited from a university campus. After personal interviews were conducted using a semi-structured questionnaire, 36 individuals were classified as binge eaters and 29 as non-binge eaters according to modified criteria of the proposed DSM-IV by the American Psychiatric Association (APA). All subjects participated in anthropometric and clinical measurements to evaluate the level of obesity and cardiovascular risks. Binge eating subjects showed no significant differences in obesity index compared to non-binge eating subjects. However, they had a higher level of low-density lipoprotein (LDL) cholesterol and a higher atherogenic index. They also showed close correlations with general obesity and cardiovascular risk factors. Body mass index (BMI) was the main explanatory index related to cardiovascular risk factors according to the stepwise regression analysis. Furthermore, obese binge subjects had higher levels than non-obese binge subjects or non-binge subjects for total cholesterol, LDL-cholesterol, triacylglycerols, atherogenic index, and systolic blood pressure. The findings strongly suggest that obese young women having binge eating episodes might display a greater risk for cardiovascular disease than that shown for obese non-binge eating women. (*J Community Nutrition* 1(1) : 33~38, 1999)

**KEY WORDS :** binge eating · cardiovascular risk factors · body mass index · obesity.

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### Introduction

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Binge eating has recently received considerable attention in eating disorder literature. Binge eaters typically consume large amounts of food in controlled time periods and exhibit a lack of control over eating during binges (American Psychiatric Association 1994). Binge eating is frequently associated with self disgust, depression, and severe guilt after overeating. In America, community-based prevalence estimates are 6.3% for binge eating, and 1% to 5 % for binge eating disorder (Spitzer et al. 1993). Prevalence estimates of binge eating from the college population range 7% to 79% based on 16 studies (Fairburn & Baglin 1990). Approximately 30 % of obese subjects treated in hospital-based weight loss programs reported serious difficulties with binge eating or compulsive overeating (Spitzer et al. 1992 ; Spitzer et al. 1993).

Several studies have demonstrated that binge eating individuals differ from those who do not binge eat in several clinically relevant ways. Binge eaters have been associated with increased preoccupation with food and calories, increased preoccupation with weight (Wilson et al. 1993), heightened body dissatisfaction and greater drive for thinness (Marcus et al. 1990). They have also been found to report earlier onset of dieting (de Zwaan et al. 1994 ; Spitzer et al. 1993), earlier onset of obesity (Brody et al. 1994 ; Spitzer et al. 1993), and a history of a larger weight loss resulting from diet (Brody et al. 1994).

Furthermore, individuals with the most severe binge/purge behavior have been reported to become metabolically more efficient in terms of their resting metabolic rate (Bennett et al. 1989), and therefore may be at higher risk for body weight gain (Ravussin et al. 1988). Binge eaters have been reported to have weight fluctuations exceeding 10 kg of body weight (Marcus et al. 1988 ; Spitzer et al. 1993). Therefore it has been associated with increased cardiovascular risk (Lissner et al. 1991).

Lots of young Korean women, even though normal-weight, are dissatisfied with their weight, and want to

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be thinner(Rhu & Yoon 1998 ; Won 1998). Some have reported weight loss behavior, such as fasting, reducing calories, cutting down on fats and sugars, and increasing exercise(Rhu & Yoon 1998). Since they didn't use extreme weight control strategies, such as vomiting, diet pills, laxatives, and diuretics(Rhu & Yoon 1998), such preoccupation with weight could induce repetitive dieting behavior, and consequently become an important risk factor in developing eating disorders(Grigg et al. 1996). Intermittent dieting may contribute to obesity by decreasing basal energy needs(Steen et al. 1988), and thereby increasing cardiovascular risks.

In Korea, some research has been done in this area, but the reports have focused on dieting pattern, weight control and eating behavior (Won 1998). Very little research has been done on the eating disorders associated with cardiovascular risk factors. The following research was proposed to aid in identifying the impacts of eating disorders on the blood cardiovascular risk factors in non-clinical female young adults.

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## Subjects and Methods

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### 1. Subjects

This research was completed by a cross-sectional study. Eighty-four female university students were initially recruited through advertisements circulated on the campus of the University of Suwon, Korea. Applicants were students who were interested in enrolling in a weight control program, and were asked to volunteer for a study investigating the relations between diet and obesity.

Each applicant was personally interviewed using a semi-structured questionnaire to detect the presence of binge eating. The subjects were regarded as binge eaters, when they reported frequent episodes of binge eating in the last six months, according to the proposed Diagnostic and Statistical Manual of Mental Disorders criteria(DSM-IV)(American Psychiatric Association 1994). Most binge subjects reported binge episode approximately once or twice a month. Therefore the criteria of frequency of binge eating by DSM-IV which designated 'more than twice a week' was disregarded. Among the applicants, 19 persons(2 from binge group and 17 from nonbinge group) failed to become subjects, because they didn't come for blood collection. Sixty-five subjects were finally selected with 35 individuals classified as binge eaters and 29 as

non-binge eaters. Each subject was included only if she reported good health with no known medical conditions or current medications.

### 2. Anthropometric and clinical measurements

After an interview, demographic data was documented and anthropometric measurements were done. Height and weight were measured with the subject in light clothing without shoes. Waist circumference(WC) was taken as the minimum circumference between the umbilicus and xiphoid process, and hip circumference was measured as the maximum circumference around the buttocks posteriorly and the symphysis pubis anteriorly. Triceps, biceps, subscapular, suprailiac, midaxillary, abdomen, thigh, and medial calf skinfold thickness were taken using a caliper(Lange) by the method of Lee & Nieman(1996). A tetra-polar impedance analyzer(BIA, Gilwoo Korea) was used to measure body electrical resistance and to derive and estimate total body fat percentage. All anthropometric data were measured in duplicate, and the values were averaged.

Subjects underwent blood sampling after an overnight fast. After sitting for at least 5 min, blood pressure was measured in the right arm by a registered nurse using a standard mercury sphygmomanometer. Blood was taken for biochemical measurements. Plasma glucose level was measured with a commercial kit(Youngdong Pharmaceutical Co., Korea), immediately after separation of plasma by centrifugation at 3000rpm at 4°C for 10minutes. Plasma total cholesterol and triacylglycerols were analyzed by photometry using a commercial kit(Youngdong Pharmaceutical Co., Korea). HDL-cholesterol was analyzed by the same commercial kit, following the precipitation of LDLs and very low density lipoproteins(VLDLs), using the modified heparin-manganese methods(Warnick & Albers 1977). The LDL-cholesterol concentrations were calculated by subtracting the HDL- and VLDL-cholesterol concentrations from the total plasma cholesterol levels. The VLDL-cholesterol was estimated by the formula of Friedewald et al.(1972), which assumes that the concentration of VLDL-cholesterol approximates one-fifth of the plasma triacylglycerol concentration.

### 3. Statistical analysis

Statistical analysis was performed using the SAS software (version 6.12). All data are expressed as mean  $\pm$  standard error. Wilcoxon rank sum tests were used to compare

the mean value of the binge eaters and non-binge eaters. Pearson correlation coefficients were calculated to examine the associations of anthropometric data with cardiovascular risk factors. The stepwise multiple regression analysis was used to examine the variance in cardiovascular risk factor explained independently by body mass index (BMI, kg/m<sup>2</sup>), waist to hip ratio(WHR) and waist circumference(WC). One of the cardiovascular risk factors was used as the dependent variable and BMI, WHR and WC were used as independent variables. Kruskal-Wallis k-sample tests were used to examine the differences of cardiovascular risk factors among four groups of subjects divided by binge eating episodes and BMI. If significant differences were found, the Duncan's multiple range test was then used to determine the differences between groups.

## Results and Discussion

Of the subjects, 69.4% of binge subjects and 34.5% of non-binge subjects experienced weight control, but no one experienced self-induced vomiting nor use of diet pills(Table 1). Moreover their frequency of binge eating episodes was much less than the criteria of DSM-IV. Eighty-nine percent of binge subjects reported binge cat-

ing less than once per week. Therefore, our binge subjects were subclinical conditioned for binge eating disorder or bulimia nervosa. All binge subjects experienced episodes of eating a definitely large amount of foods within any 2-hour periods, and a sense of lack of control during those episodes during the previous 6 months. They also showed high percentages of rapid eating(88.9%), eating until feeling uncomfortably full(94.4%), feeling depressed after overeating(94.4%). But they showed similar relatively low proportions about 'eating alone during episodes'( 5.6%), and 'feeling guilty after overeating'(19.4%).

There were no significant differences of anthropometric variables between the subjects with binge eating episodes and the subjects without binge eating episodes(Table 2). Although binge eating has been reported to be closely associated with obesity(Brody et al. 1994 ; Spitzer et al. 1993), our subjects with binge eating episodes were not significantly different from non-binge subjects in general obesity and fat distribution.

From comparisons of cardiovascular risk factors, LDL-cholesterol level and atherogenic index of the binge subjects were significantly higher than those of non-binge

**Table 1.** Proportions of subjects endorsing episodes of engaging in binge eating related behaviors

Variables	Binge eating	Non-binge eating
Ever controlled weight	25( 69.4%)	10(34.5%)
Ever self-induced vomiting	0( 0.0%)	0( 0.0%)
Ever used diet pills	0( 0.0%)	0( 0.0%)
Ever had episodes of binge eating last 6 months	36(100.0%)	12(41.4%)
A sense of lack of control during episodes	36(100.0%)	8(27.6%)
During episodes, eating much more rapidly than usual	32( 88.9%)	3(10.3%)
During episodes, eating until feeling uncomfortably full	34( 94.4%)	7(24.1%)
Eating large amount of food when not feeling physically hungry	20( 55.6%)	3(10.3%)
During episodes, eating alone due to embarrassment	2( 5.6%)	0( 0.0%)
Feeling disgusted after overeating	13( 36.1%)	4(13.8%)
Feeling depressed after overeating	34( 94.4%)	20(68.9%)
Feeling very guilty after overeating	7( 19.4%)	2( 6.9%)
Binge eating caused marked distress	33( 91.7%)	11(37.9%)
Binge eating, at least twice a week for 6 months	4( 11.1%)	0( 0.0%)

**Table 2.** Anthropometric analysis of the subjects

Variables	Binge eating	Non-binge eating
Age(yr)	19.6 ±0.5	19.2 ±0.9
Height(cm)	159.6 ±1.0	157.8 ±1.0
Weight(kg)	57.1 ±1.4	54.3 ±1.4
BMI(kg/m <sup>2</sup> )	22.4 ±0.5	21.9 ±0.6
PIBW	106.4 ±2.3	104.3 ±3.1
%Fat	25.6 ±0.7	24.5 ±0.7
WHR	0.75±0.01	0.72±0.01
Waist circumference(cm)	69.9 ±1.1	67.1 ±1.6
Hip circumference(cm)	93.0 ±1.1	92.5 ±1.2
Mid-arm circumference(cm)	26.8 ±0.4	26.5 ±0.6
Thigh circumference(cm)	52.4 ±0.7	51.8 ±0.9
Mid-calf circumference(cm)	36.9 ±0.5	35.9 ±2.8
Triceps skinfold thickness(mm)	21.0 ±1.2	23.7 ±1.6
Biceps skinfold thickness(mm)	13.2 ±1.2	15.6 ±1.5
Subscapular skinfold thickness(mm)	24.9 ±1.5	28.9 ±1.7
Suprailiac skinfold thickness(mm)	15.8 ±1.4	13.6 ±1.0
Abdominal skinfold thickness(mm)	24.3 ±1.4	23.4 ±1.7
Mid axillary skinfold thickness(mm)	25.6 ±1.8	23.9 ±1.8
Thigh skinfold thickness(mm)	36.9 ±2.0	34.1 ±2.5
Medial calf skinfold thickness(mm)	34.5 ±2.2	32.0 ±2.8

Values are mean±SEM

BMI : Body Mass Index, PIBW : Percent Ideal Body Weight

WHR : Waist to Hip Ratio

All variables of binge subjects are not significantly different from those of non-binge subjects by Wilcoxon rank sum test

**Table 3.** Comparisons of cardiovascular risk factors of subjects

Variables	Binge eating	Non-binge eating
Total cholesterol(mg/dl)	160.0 ± 5.3	145.7 ± 5.2 <sup>NS</sup>
HDL-cholesterol(mg/dl)	50.3 ± 1.4	53.4 ± 1.8 <sup>NS</sup>
LDL-cholesterol(mg/dl)	87.6 ± 5.6	71.1 ± 4.9*
Triacylglycerol(mg/dl)	110.7 ± 3.1	106.2 ± 3.2 <sup>NS</sup>
Atherogenic index	2.30 ± 0.16	1.81 ± 0.13*
Systolic blood pressure(mmHg)	118.2 ± 2.3	117.1 ± 3.0 <sup>NS</sup>
Diastolic blood pressure(mmHg)	76.4 ± 1.3	78.5 ± 3.2 <sup>NS</sup>
Glucose(mg/dl)	90.5 ± 1.8	89.1 ± 1.3 <sup>NS</sup>

Values are mean ± SEM

NS : not significantly different

\*p&lt;0.05 significantly different from the value of binge eating subjects by Wilcoxon rank sum test

subjects(Table 3). Because cardiovascular diseases are closely related to general obesity and abdominal obesity, the relationships between obesity and cardiovascular risk factors were examined. BMI was positively correlated with total cholesterol, LDL-cholesterol, triacylglycerols, atherogenic index, and systolic blood pressure--and was negatively correlated with HDL-cholesterol levels in binge eating subjects(Table 4). Body fat percentage showed a positive correlation with systolic blood pressure, and a negative correlation with HDL-cholesterol, while WHR and WC, which are used to assess central adiposity, were less likely to correlate with cardiovascular risk factors. In non-binge eating subjects, obesity indexes were weakly correlated with cardiovascular risk factors--compared to binge eating subjects. Therefore BMI in non-binge subjects was positively correlated with systolic and diastolic blood pressure, and body fat percent with total cholesterol and LDL-cholesterol.

Although there are reports suggesting that intra-abdominal fat deposition may constitute a greater cardiovascular risk than general obesity(Peirís et al. 1989 ; Pouliot et al. 1994 ; Richelsen & Pedersen 1995), most researchers still report that both general and central obesity are important cardiovascular risks(Ding et al. 1993 ; Galanis et al. 1995). Our results are not consistent with such previous reports. From our data, general obesity(represented by BMI and body fat %), more strongly contributed to cardiovascular risks, compared to central obesity, represented by WHR and WC in young women(Table 4). Furthermore, we observed a stronger correlation between general obesity and cardiovascular risks in subjects with binge eating episodes compared to non-binge subjects.

To elucidate explanations for specific obesity index on

**Table 4.** Correlation coefficients of body mass index(BMI), percent body fat, waist to hip ratio(WHR), and waist circumference(WC) with cardiovascular risk factors

	BMI	% Fat	WHR	WC
Binge Eating subjects				
Total cholesterol	.372*	.126	.180	.265
HDL-cholesterol	-.414*	-.398*	.090	-.270
LDL-cholesterol	.396*	.193	.136	.279
Triacylglycerol	.562***	.124	.129	.373*
Atherogenic index	.526**	.330	.088	.366*
Systolic blood pressure	.610***	.359*	.298	.630***
Diastolic blood pressure	.088	.099	-.054	.056
Non-Binge Eating subjects				
Total cholesterol	.149	.393*	-.031	.087
HDL-cholesterol	-.289	-.017	-.340	-.274
LDL-cholesterol	.226	.375*	.058	.159
Triacylglycerol	.299	.354	.206	.264
Atherogenic index	.293	.279	.185	.240
Systolic blood pressure	.533**	.327	.156	.350
Diastolic blood pressure	.408*	.184	.069	.234

\*p&lt;.05, \*\*p&lt;.01, \*\*\*p&lt;.001 by Pearson correlation

BMI : Body Mass Index, kg/m<sup>2</sup>

cardiovascular risk factors, a stepwise multiple regression analysis was performed on the subjects as a whole and in each binge/non-binge subgroup(Table 5). In these analyses BMI, WHR and WC were included as independent variables. A significance level of p<.10 was used for entry into the model. BMI was the main explanatory variable related to plasma cardiovascular risk factors, whereas WHR and WC were not significantly related to any of the examined cardiovascular risk factors, except systolic blood pressure in binge subjects(R<sup>2</sup>=.385). BMI was used as an index of overall adiposity since it was generally correlated with risk factors more strongly than bioimpedence-determined body fat content.

To determine the relations of obesity and binge eating to the cardiovascular risks, we divided the subjects into 4 groups according to the binge episode and BMI 24. The cut-off point of 24 for the high BMI group was chosen because upper quartile level of BMI in whole subjects was 24, and because the population consisted of 56% women aged <25 y where the recommended BMI is in the range of 19 to 24(Bray 1990). From our results, obese-binge subjects had a higher level of total cholesterol, LDL-cholesterol, triacylglycerols, higher atherogenic index, and higher systolic blood pressure, than non-obese binge subjects or non-binge subjects(Table 6). Therefore this result suggests that obese young women with binge

eating episodes experience greater a risk of cardiovascular disease, compared to non-binge obese women.

There are some problems regarding generalization of results, pertaining to the subject sample size, socioeconomic level and reliance on self-reports for assessment of

**Table 5.** The variances in cardiovascular risk factors explained by BMI, WHR<sup>1)</sup> and waist circumference<sup>1)</sup> using Stepwise Multiple Regression Analysis

Dependent variable	BMI		
	R <sup>2</sup>	β	P
All			
Total cholesterol	0.084	2.883	0.024
HDL-cholesterol	0.145	-1.086	0.002
LDL-cholesterol	0.111	3.380	0.009
Triacyl glycerol	0.258	2.948	0.000
Atherogenic index	0.198	0.127	0.000
Systolic blood pressure	0.338	2.810	0.000
Diastolic blood pressure	0.067	0.813	0.045
Binge Eating subjects			
Total cholesterol	0.124a	3.950	0.045
HDL-cholesterol	0.153a	-1.630	0.004
LDL-cholesterol	0.136a	4.298	0.035
Triacylglycerol	0.352a	7.128	0.000
Atherogenic index	0.254a	0.368	0.003
Systolic blood pressure	-	-	-
Diastolic blood pressure	-	-	-
Non-Binge Eating subjects			
Total cholesterol	-	-	-
HDL-cholesterol	0.142	-1.016	0.053
LDL-cholesterol	-	-	-
Triacylglycerol	0.147	1.887	0.049
Atherogenic index	0.155	0.075	0.042
Systolic blood pressure	0.287	2.597	0.004
Diastolic blood pressure	0.174	1.459	0.030

1) The variance explained WHR and waist circumference was not presented in table due to lack of statistical significance

binge eating. All our subjects were non-clinical university students and were very interested in taking an active role to control their body weight. The sample was not a random or representative one, thus results on the prevalence and correlates of binge eating may not be completely generalizable. However, the results improve understanding regarding the relationships between eating disorders, obesity, and blood cardiovascular risk factors. While recent reports have contributed significantly to our understanding of the correlation between obese binge eating and cardiovascular risks, this area of research warrants further investigation. Specifically, we could use studies designed to directly assess the temporal relationship between onset of binge eating, dieting, and obesity, as well as predictive investigation of cardiovascular risk. Population based large sample research is needed to better understand the relation between obese binge eaters and cardiovascular risks.

## Conclusion

Even though young female binge eating subjects showed no significant differences in obesity index compared to non-binge eating subjects, they had a higher level of LDL-cholesterol and a higher atherogenic index. There were also close correlations between general obesity and cardiovascular risk factors in binge subjects. In binge subjects, BMI was the main explanatory index related to cardiovascular risk factors. Furthermore, obese binge subjects had a higher level of total cholesterol, LDL-cholesterol, triacylglycerols, a higher atherogenic index, and higher systolic blood pressure, than did non-obese binge subjects or non-binge subjects. In conclusion, obese young women

**Table 6.** Comparisons of cardiovascular risk factors among four groups

	Binge eating		Non-binge eating		P-value <sup>1)</sup>
	BMI ≥ 24	BMI < 24	BMI ≥ 24	BMI < 24	
	n=8	n=27	n=7	n=22	
Total cholesterol(mg/dl)	188.6 ± 7.7 <sup>a</sup>	151.5 ± 5.6 <sup>b</sup>	148.3 ± 11.1 <sup>b</sup>	144.9 ± 6.1 <sup>b</sup>	p<0.01
HDL-cholesterol(mg/dl)	115.8 ± 7.9 <sup>1</sup>	79.2 ± 6.0 <sup>b</sup>	77.7 ± 10.0 <sup>b</sup>	69.0 ± 5.8 <sup>b</sup>	p<0.01
LDL-cholesterol(mg/dl)	47.6 ± 9.6	51.0 ± 1.5	48.2 ± 1.5	55.0 ± 2.3	NS
Triacylglycerol(mg/dl)	126.1 ± 10.5 <sup>a</sup>	106.1 ± 2.0 <sup>b</sup>	112.0 ± 6.2 <sup>b</sup>	104.4 ± 3.7 <sup>b</sup>	p<0.05
Atherogenic index	3.11 ± 0.33 <sup>a</sup>	2.05 ± 0.16 <sup>b</sup>	2.09 ± 0.23 <sup>b</sup>	1.72 ± 0.15 <sup>b</sup>	p<0.01
Systolic blood pressure(mmHg)	129.0 ± 4.9 <sup>a</sup>	115.0 ± 2.4 <sup>b</sup>	129.9 ± 2.8 <sup>a</sup>	113.0 ± 3.5 <sup>b</sup>	p<0.01
Diastolic blood pressure(mmHg)	77.1 ± 2.7	76.2 ± 1.6	84.4 ± 2.9	76.6 ± 2.6	NS

Values are mean ± SEM

Results in the same row with different superscript are significantly different by the Duncan's multiple range test

1) Statistical significance by Kruskal-Wallis k-sample tests

with binge eating episodes might reveal a greater risk for cardiovascular disease, compared to obese non-binge women. There are some limitations regarding the generalization of results, pertaining to the subject sample size and reliance on self-reports. Therefore further research is needed to better understand the increased cardiovascular risk for obese binge eaters.

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