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C-Reactive Protein is Linked to Lower Cognitive Performance In Overweight and Obese Women

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Abstract

Our objective was to ascertain the nature of the associations between C-reactive protein (CRP) and cognition, and to examine how they are affected by gender and obesity. We evaluated 62 females and 63 males between 42 and 82 years of age. There were 20 lean females with a body mass index (BMI) of < 25 kg/m^2 and 42 were overweight or obese, with BMIs ≥ 25 kg/m^2. There were 14 lean males and 49 with BMIs ≥ 25 kg/m^2. CRP was associated with cognitive tests of frontal lobe function only among females and these associations were driven by the overweight/obese female group. No associations between CRP and cognition were found among males. Obesity-associated inflammation appears to be more pronounced in females and is associated with cognitive dysfunction, particularly of frontal lobe tasks.

Keywords

CRP; cognition; gender; overweight; obesity

INTRODUCTION

C-reactive protein (CRP) is an acute phase reactant produced by the liver in response to inflammation within the body. Low-grade elevations in serum CRP have been found to be independently predictive of coronary heart disease and have been implicated in the development of other conditions, such as type 2 diabetes (T2DM) and the metabolic syndrome[1, 2, 3]. Serum CRP levels less than 1 mg/l are associated with a low risk, levels between 1 and 3 mg/l confer an average risk, and those between 3 and 10 mg/l are associated with increased risk for cardiovascular disease (CVD)[4]. Roughly 33% of the population in the US has levels between 3 and 10 mg/l, and are thus considered at increased risk for CVD[5]. A wide variety of conditions have been found to elicit transient elevations in CRP. However, chronic low-grade elevations, accompanied by features of the metabolic syndrome[6], may be indicative of a constitutive inflammatory process, which may help explain why these low-grade elevations are linked to increased cardiovascular risk[1].

CRP, Obesity and Gender

Obesity, as defined by a body mass index (BMI) of ≥ 30 kg/m^2, may be accompanied by a state of chronic low-grade inflammation and CRP levels are often elevated in both men and

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women with BMIs in the overweight (≥ 25 kg/m^2) and obese range[7]. While CRP elevations are associated with the metabolic syndrome[8], they are particularly related to abdominal obesity[9, 10], and this association is more prominent among females[11, 12, 13]. Most studies have found that CRP levels are generally higher in females,[14, 15, 16, 17] possibly mediated by estrogen[18], a contention that is supported by the fact that hormone replacement therapy is also associated with increased levels of CRP[18]. However, it is also possible that higher CRP levels in women may reflect sex-related differences in the inflammatory response to obesity[19].

CRP, Metabolic Dysregulation, and Cognition

Obesity is frequently associated with metabolic syndrome, insulin resistance, and type 2 diabetes[20, 21]. It has been well established that type 2 diabetes mellitus (T2DM) and insulin resistance short of T2DM are associated with cognitive deficits[22, 23, 24, 25, 26]. Our group has demonstrated that both declarative memory deficits and hippocampal volume reductions are associated with impairments in glucose control[27, 28].

There is an expanding body of literature linking cognitive deficits with obesity, independent of metabolic dysregulation. Gunstad and colleagues found that a BMI greater than 25 was associated with executive dysfunction in a sample of 408 adult males and females[29]. A Korean study, utilizing the ethnic-specific criteria for obesity and waist circumference developed for Asian populations[30], found that among adults 65 and older, poorer cognition (as reflected by K-MMSE score) was associated with being overweight or obese and particularly with the presence of abdominal obesity[31]. The Framingham Heart Study reported gender-specific results describing adverse obesity effects on cognition only among men[32]. Significant associations between waist circumference, hypertension and cognition were found in healthy older adults[33]. The metabolic syndrome, which is comprised of several risk factors including abdominal obesity[34], has been associated with cognitive problems[35].

There is a growing literature assessing the associations between CRP and cognitive performance. For example, Yaffe and colleagues[36] found that elevated serum levels of CRP were associated with cognitive decline in individuals with the metabolic syndrome. Several studies have shown that CRP predicts cognition longitudinally. For example, Komulainen, et al,[37] found that in elderly women higher baseline CRP levels were predictive of poorer memory 12 years later. A meta-analysis of six studies looking at CRP and cognition concluded that higher levels of CRP were predictive of cognitive decline and dementia[38]. However, most of these studies utilized somewhat insensitive tools for the assessment of cognition, most frequently the Mini Mental State Examination (MMSE). The goal of the present study was to ascertain the associations between CRP and cognitive performance employing a comprehensive neuropsychological assessment. In addition, we were particularly interested in assessing how gender and obesity affect those associations.

METHODS

Participants

We evaluated 125 participants consisting of consecutive cases that were part of a study assessing the relationship between peripheral glucose regulation and brain function. All were community-residing individuals living independently. Subjects were either referred by collaborating endocrinologists, responded to advertisements, or were participating in longitudinal studies of normal aging. Participants were between 42 and 82 years of age and had a minimum of a high school education. All study subjects were functioning in the cognitively normal range, as indicated by scores on the Mini-Mental State Exam, and were

free of current or historical evidence of significant neurological, medical (other than type 2 diabetes, dyslipidemia, or hypertension), or psychiatric disease. Participants gave informed written consent and were compensated for their participation. The study was approved by the NYU Institutional Board of Research Associates. Subsets of 46 and 60 subjects, respectively, were previously included in two other reports on related topics[27, 39]. One female subject was excluded due to CRP values exceeding 10 mg/l, which may have been due to an acute infection.

Participants with elevated BMI—Participants qualified for inclusion in this group if they met general population standard criteria[40], namely if they had a BMI greater than or equal to 25 kg/m^2. As anticipated from national statistics, some two thirds of our study participants (91/125), were overweight or obese. Thirty-six of the 91 overweight or obese subjects (40%) had a diagnosis of T2DM and none were being treated with insulin or insulin secretagogues. Thirty-eight (42%) met criteria for hypertension and 33 (36%) were prescribed statins for dyslipidemia.

Normal BMI participants—Participants qualified for inclusion in this group if they had a BMI between 18 and 24.9 kg/m^2. Thirty four of our 125 participants (27%) were in the normal BMI group. Two of the 34 (6%) had a diagnosis of type 2 diabetes, 6 (18%) met criteria for hypertension and 4 (12%) were prescribed statins for dyslipidemia.

Evaluations

All subjects underwent an assessment consisting of a complete medical evaluation, including physical and neurological examinations, as well as neuropsychological and psychiatric evaluations. All subjects had fasting blood tests that included complete cell count, chemistries, liver profile, metabolic profile including lipids, glucose and insulin, and CRP.

Physical Examination

Blood pressure and definition of hypertension—Blood pressure was measured twice during one of the standardized visits, with readings performed at 08:30 h, 30 minutes after the participants arrived, and again at 13:40 h, after the procedure was complete. These readings were then averaged. Hypertension has been linked to both inflammation and cognitive dysfunction. Thus, hypertension was defined based on recommendations of the National Cholesterol Education Program (NCEP) guidelines, namely: (1) systolic value greater than or equal to 130 mm Hg, or (2) diastolic value greater than or equal to 85 mm Hg, or (3) use of anti-hypertensive medication.

C-Reactive Protein (CRP)—We assessed plasma levels of c-reactive protein (CRP) after an overnight fast. CRP levels were measured in our medical center clinical laboratory using an enzymatic immunoassay (Vitros CRP slide, Ortho Clinical Diagnostics).

Neuropsychological and Psychiatric assessment

All cognitive assessments are routinely used standardized neuropsychological tests described in detail elsewhere[41]. Briefly, declarative memory was assessed with the California Verbal Learning Test (CVLT), and subtests from the Wechsler Memory Scale–Revised (WMS-R). Working memory was evaluated using the Digit Span Backwards and Visual Memory Span Backwards from the WMS-R. Executive Function was measured with the computer-administered Tower of London (Colorado Assessment Tests). Attention was assessed with subtests from the Wechsler Memory Scale – Revised (WMS-R). General Intellectual Functioning was assessed with the Shipley Institute of Living Scale scores

which were used to estimate WAIS-R full scale IQ scores. Lastly, depressive symptoms were assessed with the Hamilton Depression Rating Scale[42].

Statistical Analyses

Differences between groups were analyzed using student's t-tests and analyses of variance (ANOVA) or covariance (ANCOVA) when appropriate. When assessing associations between different variables, correlations or partial correlations were used. A number of recent studies have found that statin medications are effective at lowering CRP levels[43,44,45], so, we controlled for statin use in some of our analyses. Because type 2 diabetes has been linked to cognitive dysfunction, we controlled for this diagnosis in the cognitive analyses where merited. Age has been associated with CRP levels[46]. Partial correlations, controlling for age and statin use, were used to ascertain associations between CRP and other biological variables. Education has been found to be associated with cognitive performance[47]. Therefore, partial correlations were also used to examine the associations between CRP and cognitive variables, controlling for age, education, diabetes, and statin use. To further assess the relationship between CRP and the cognitive variables that appeared to be associated with CRP in the correlation analyses, linear regression models were used. These models were utilized so as to assess the explanatory power of CRP after accounting for the appropriate potential confounds. Data were analyzed using SPSS for Windows, version 12.0 (SPSS Inc., Chicago, Ill., USA).

RESULTS

Demographic variables and group descriptors by gender

The demographic variables and group descriptors, listed separately for men and women, are summarized in Table 1.

Given that our main interest was to ascertain whether the associations between CRP and cognition differed by gender and by weight category, we next created descriptions of the sample separately for men and women by BMI groups. As can be seen in Table 2a, we had 20 lean females (32%) with a BMI of 24.9 kg/m^2 or less, and 42 females (68%) with a BMI of 25 kg/m^2 or greater. As expected, there were significant group differences for HbA1c, HDL, fibrinogen, CRP, and waist circumference between the female BMI groups.

The male subjects are presented by BMI group in Table 2b. We had 14 lean males (22%) and 49 (78%) in the overweight or obese category. We found significant differences in HDL, CRP, and waist size, but interestingly, HbA1c did not distinguish the male BMI groups.

Associations between CRP and Clinical Variables

Across all subjects we found positive associations between CRP and waist size (r = .42, p < .001) and HbA1c (r = .34, p < .001). A weak (although statistically significant) negative association existed between CRP and HDL (r = −.24, p = .010), but no significant correlation was detected between CRP and triglycerides (r = .12, p = .189). ANOVA detected no differences in CRP levels between hypertensives and non-hypertensives (F = 1.219, p = .272).

When we looked at these associations separately in men and women, we found that there were some distinct gender differences in the association of CRP with clinical variables.

Among males, CRP was positively associated with waist size (r = .478, p = .000) and HbA1c (r = .419, p = .001), and tended to be negatively associated with HDL (r = −.232, p

= .077). Among females, CRP was positively correlated with waist size (r = .421, p = .001) and triglycerides (r = .26, p = .045) and negatively correlated with HDL (r = −.413, p = .001). Interestingly, HbA1c was not significantly associated with CRP among our female group (r = .197, p = .132). No associations between hypertension and CRP were found for either the entire sample (F = 1.219, p =.272) or individual gender groups (males: F = .742, p = .393, females: F = 1.403, p = .241)

Cognitive differences between weight groups

When looking at both males and females together, no significant differences in cognition were found between the normal BMI and overweight/obese groups after adjusting for age, education, statin and diabetes. There were no cognitive differences by BMI group even when we did not adjust for the presence of type 2 diabetes.

We found significant differences in cognition between males and females. As shown in Table 4, males scored significantly poorer (had more excess moves) than females on the Tower of London, a measure of executive function. However, the most marked gender differences were on tests of declarative memory. Scores on the CVLT, a list-learning task, and all of the memory indexes on the Wechsler Memory Scale-Revised (WMS-R) showed that males scored significantly lower than females. This is consistent with the fact that our males as a group had significantly worse peripheral glucose regulation, a factor that has been linked to decreased memory performance. There were no gender differences on the Attention/Concentration Index of the WMS-R.

No associations were found between CRP and any of the cognitive variables among males. Because the distribution of CRP values within our male group were skewed, we performed non-parametric analyses to confirm our negative results among the males. In addition, we ran a linear regression analysis using all subjects and found a significant Gender and Gender x CRP interaction in explaining cognition (Partial Eta Squared = .10 and .04 respectively). This interaction was being driven by the females, whose CRP values were much more normally distributed.

It is known that, in general, HDL levels are higher among females, and there is evidence that higher HDL levels may be neuroprotective[48]. It is also known that CRP levels are higher in both females and among people who are overweight or obese. Given the above-mentioned cognitive differences by gender and the fact that females have both more protective (HDL) and risk factors (higher CRP levels), we chose to examine the effects of CRP on cognition more closely within our female group, paying particular attention to how BMI group affected those associations.

Age, education, and diabetes are all known to be associated with cognition. There are also links between CRP and statin use. Consequently, to ascertain the associations between CRP and cognition within each BMI group, we utilized partial correlations and controlled for age, education, diabetes, and statin use. Significant associations between CRP and cognition were only found among overweight/obese females: WAIS estimated full-scale IQ (r = −.386, p = .015), Tower of London Excess Moves (r = .509, p = .003), figural memory subtest of the WMS-R (r = −.463, p = .004) and Shipley total raw score (r = −.384, p = .016). Trends were detected in two tests of working memory: digit span backward (r = −.286, p = .081) and the visual memory span backward (r = −.313, p = .055) subtests of the WMS-R.

Contribution of CRP to explaining cognition (Linear Regressions)—Because our weight groups differed in peripheral glucose control, which we know plays a role in cognition, and also differed in other factors involved in the metabolic syndrome, we ran regression analyses to further examine the relationship between CRP and the cognitive tests

that appeared to be associated with CRP in our female group. After accounting for age, education, and statin use (3.4% of the variance), CRP was entered stepwise in a block with other clinical variables (HDL, triglycerides, hypertension (y/n), waist circumference, and HbA1c) for the entire female group (*n*=62). The only variables that emerged as related to scores on the Tower of London were CRP and hypertension. We then followed this up with hierarchical regression models using the Tower of London excess moves score as the dependent variable. After age, education, and statin use (3.4% of the variance), hypertension explained an additional 4.5 %, and CRP level when entered last independently explained 16.5% of variance.

When these regression analyses were restricted to the overweight/obese females, after accounting for age, education, and statin use (4% of variance), hypertension explains an additional 6% of the variance, and CRP level, when entered last, independently explained nearly 32% of the variance. Please note that when WAIS Full-Scale IQ was used as the dependent variable, although explaining less of the variance, similar significant results were obtained for CRP levels. Please refer to Table 6.

DISCUSSION

The objective of this study was to ascertain the associations between CRP and cognition, and to examine how those associations are affected by gender and obesity. Based on literature demonstrating associations between elevated CRP and impaired cognitive performance[36,49,50], we hypothesized that CRP levels would be inversely associated with cognitive performance. Furthermore, we sought to examine whether the associations between CRP and cognition would vary by gender or BMI.

Obesity is associated with an inflammatory state[51]. We therefore predicted that CRP, an inflammatory marker, would be elevated in overweight/obese subjects. Consistent with prior findings[9], CRP was significantly elevated in our overweight/obese subjects and was positively associated with waist circumference in both our males and females. We found that CRP was positively associated with HbA1C in males, but not in females. Florez et al similarly found that CRP was associated with insulin resistance only in males[9], perhaps suggesting that obesity-related impairments in insulin function, which were more pronounced among our male study participants, may vary by gender in its associated pro-inflammatory response. CRP in females was positively associated with triglycerides and negatively associated with HDL, which is consistent with previous findings of CRP's positive association with the metabolic syndrome[8]. Females naturally have higher levels of HDL[52], and given the inverse associations between HDL and CRP in females, this may help explain the protective role of HDL in cardiovascular health and perhaps brain health. Therefore, this may help explain why the females with lower HDL and larger waist circumference, who also have higher levels of CRP, may have more cognitive deficits.

We found that CRP levels were higher in females than in males, as has been established in other studies[14,53]. Of interest, women are more prone to autoimmune diseases such as Lupus and Rheumatoid Arthritis[54,55], which are characterized by inflammation, elevated CRP[56,57], and accelerated rates of microvascular disease and atherosclerosis[58,59]. Elevated CRP in these women is also associated with cognitive impairments. In one recent study, Shucard and co-investigators documented that elevated CRP in female lupus patients correlated with deficits in executive cognitive function, possibly due to microvascular compromise[60]. It has been documented that obesity is associated with low-grade systemic inflammation in both men and women. Given that women generally have higher constitutive CRP levels than men and are also more prone to inflammatory diseases[61], this may help explain why women have a stronger inflammatory response to obesity[11].

When we compared cognition by gender, the most notable finding was that males had lower performance in tasks of declarative memory compared to our females, which was not surprising considering our males had poorer glycemic control and declarative memory performance has been specifically associated with impairments in glucose metabolism[27].

We found that there was a significant negative association between CRP and cognition, but only for females, and the overweight/obese subjects were driving this relationship. Given that this group has the highest CRP levels, this led us to speculate that chronic low grade inflammation may be affecting brain health as manifested by cognitive performance. In our regression, CRP explained (after accounting for age, education, statin use, and hypertension) 16.5% of the variance in a test of executive function in all female subjects and 32% of the variance when only overweight/obese women were examined. In these overweight/obese women, the associations with elevated CRP were concentrated in tasks that reflect frontal lobe function.

CRP is a well-established risk factor for cardiovascular disease and endothelial dysfunction[62,63]. Data suggests that CRP may promote atherothrombosis[64] and attenuate nitric oxide production, thereby inhibiting angiogenesis[65]. This has important implications given that the frontal lobes have less collateral circulation than the rest of the brain and may therefore would be more susceptible to microvascular disease, as suggested by data in vascular dementia or hypertension, two conditions associated with frontal lobe dysfunction[66–68]. There is some support for this premise from direct *in vivo* brain assessments. For example, the Rotterdam Scan Study documented that higher CRP levels were associated with the presence and progression of white matter lesions, which are caused by small-vessel disease[69].

It has been suggested that CRP concentrates at sites of cell damage, possibly functioning as a ligand for leukocytes in order to assist in the removal of cell debris[70]. Acute increases in CRP are an important and beneficial part of the body's response to injury and disease[71], but chronic constitutive low grade elevations may be detrimental.

In sum, we find that the consequences of obesity differ for men and women. In males, obesity has a stronger association with glucose dysregulation and declarative memory deficits, while overweight and obese women appear to be more susceptible to inflammation and accompanying cardiovascular compromise. However, a larger percentage of men than women in our sample were taking statin medications, and this may have contributed to our results. Nevertheless, our findings may have important clinical implications for treating overweight and obese women, who may benefit from the anti-inflammatory properties of statins, which, given their higher HDL levels and consequently better cholesterol ratios, may less frequently be prescribed.

One limitation of this study was that the study groups were relatively small. We had a particularly small lean male group (n=14) compared to our overweight and obese male group (n=49). Future studies with larger samples are needed to replicate these findings and determine if the results we find here are indeed specific to females. Further research may be warranted examining whether treatment options including weight loss methods or medications with anti-inflammatory properties reduce the CRP and cognitive deficits observed here among overweight and obese women. It would also be interesting to measure other inflammatory markers (interleukin-6, homocysteine, etc.) to determine whether they have significant associations to cognition and whether those associations may be modified by gender and obesity.

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Table 1

Description of the population (mean ± SD)

	Females n = 62	Males n = 63
Age (yr)	61.75 ± 8.77	62.06 ± 8.05
Education (yr)	15.82 ± 2.30	16.07 ± 2.06
Body Mass Index (kg/m^2)	27.30 ± 5.36	28.68 ± 5.77
Waist Size (in)	92.98 ± 13.05	104.27 ± 12.26
HbA1c (%)	5.83 ± .91	6.40 ± 1.87
Fasting Glucose (mg/dl)	93.81 ± 27.05	107.21 ± 46.23
Fasting Insulin (μIU/ml)†	7.35 ± 4.15	8.74 ± 4.46
HDL (mg/dl)	61.56 ± 17.22	45.62 ± 10.55
Triglycerides (mg/dl)	104.98 ± 59.09	125.36 ± 75.34
LDL (mg/dl)‡	119.96 ± 34.72	104.67 ± 32.71
Fibrinogen (mg/dl)	351.74 ± 96.31	348.65 ± 112.63
CRP (mg/l)	2.24 ± 1.95	1.84 ± 2.03
Hamilton Depression Score	2.25 ± 2.65	2.36 ± 3.16
Hypertension	20 (32.3%)	24 (38.1%)
Type 2 Diabetes	17 (27.4%)	21 (33.3%)
Statin Treatment	12 (19.4%)	25 (39.7%)

† Diabetics excluded.

‡ Those on statin treatment excluded.

Table 2a

Description of the Females by BMI group (mean ± SD)

FEMALES	Normal BMI n = 20	Overwt/Obese n = 42
Age (yr)	60.54 ± 9.54	62.33 ± 8.44
Education (yr)	16.30 ± 1.63	15.60 ± 2.55
Body Mass Index (kg/m^2)**	21.97 ± 2.02	29.83 ± 4.52
Waist Size (in)**	80.96 ± 9.02	98.85 ± 10.45
HbA1c (%)**	5.33 ± .25	6.08 ± 1.01
HDL (mg/dl)**	72.10 ± 13.83	56.55 ± 16.51
Fibrinogen (mg/dl)**	310.85 ± 55.64	371.21 ± 105.65
CRP (mg/l)**	1.08 ± 1.53	2.80 ± 1.90
Hypertension**	2 (10%)	18 (42.9%)
Type 2 Diabetes**	0 (0%)	17 (40.5%)
Statin Treatment**	1 (5%)	11 (26.2%)

** $p < 0.05$

Table 2b

Description of the Males by BMI group (mean ± SD)

MALES	Normal BMI n = 14	Overwt/Obese n = 49
Age	64.64 ± 7.96	61.33 ± 8.01
Education (yr)	16.71 ± 2.40	15.89 ± 1.94
Body Mass Index (kg/m^2)**	23.03 ± 1.03	30.29 ± 5.54
Waist Size (in)**	92.40 ± 5.49	107.66 ± 11.54
HbA1c (%)	5.89 ± 1.65	6.55 ± 1.92
HDL (mg/dl)**	55.00 ± 9.37	42.83 ± 9.25
Fibrinogen	322.43 ± 87.07	356.14 ± 118.63
CRP**	.84 ± .83	2.13 ± 2.19
Hypertension	4 (28.6%)	20 (40.8%)
Diabetes**	2 (14.3%)	19 (38.8%)
Statin Treatment*	3 (21.4%)	22 (44.9%)

** $p < 0.05$

Table 3

Associations between CRP and clinical variables controlling for age and statin use.

	CRP	
	Males	**Females**
	n=57	**n=57**
Waist Circumference	**.478****	**.421****
HDL	−.232	**−.413****
HbA1c	**.419****	.197

Pearson correlation coefficients.

** $p < 0.05$

Table 4

Differences in cognition by gender controlling for age (except on WMS-R indices which are already age adjusted and education

	Males	Females	p
WAIS Full-Scale IQ	112.41 ± 12.70	113.08 ± 11.23	ns
London Excess Moves	10.5 ± 6.86	7.21 ± 6.05	.013
Digit Span Backward	7.67 ± 2.56	7.59 ± 2.55	ns
Vis Mem Span Backward	7.34 ± 1.61	7.42 ± 2.08	ns
Shipley Total	61.98 ± 11.80	62.71 ± 10.25	ns
CVLT Trials 1–5	54.30 ± 9.69	62.90 ± 10.12	<.001
CVLT Short Delay Free Recall	10.71 ± 3.63	13.20 ± 3.32	<.001
CVLT Long Delay Free Recall	11.15 ± 3.52	12.98 ± 3.22	<.001
WMSR - General Memory Index	116.85 ± 16.25	123.80 ± 13.65	.013
WMSR – Attn/Concentration Index	109.05 ± 15.09	107.20 ± 17.84	ns
WMSR – Verbal Memory Index	113.03 ± 16.52	120.31 ± 13.56	.008
WMSR – Visual Memory Index	116.15 ± 15.18	122.81 ± 13.66	.013
WMSR – Delayed Memory Index	118.54 ± 17.20	124.25 ± 16.23	.064

Table 5

Associations between CRP and cognition among females by BMI group adjusted for age, education, statin use and diabetes.

	Females	
	Normal BMI	**Ovrwt/Obese**
	n=15	**n=37**
WAIS-R Estimated IQ	0.292	**−0.353****
Tower of London Excess Moves	−.352	**0.497****
WMS-R Figural Memory	0.196	**−0.436****
WMS-R Digit Span Backwards	0.209	−0.247*
WMS-R Visual Mem Span Backwards	0.401	−0.317*
Shipley Total Raw Score	0.240	**−0.350****

Pearson correlation coefficients.

** $p < 0.05$;

* $p < 0.10$

Table 6

Contribution of CRP to cognitive variables (hierarchical regressions)

All Females	Age/Edu/Statin			HTN			CRP		
	Ξ	ΔR^2	p	Ξ	ΔR^2	p	Ξ	ΔR^2	p
London Excess Moves	−.092/.143/.359	.034	.670	−.317	.045	.152	.430	.165	.004
WAIS Full-Scale IQ	.278/.153/−.014	.063	.475	−.277	.090	.055	−.370	.108	.028

Overweight/Obese Females	Age/Edu/Statin			HTN			CRP		
	β	ΔR^2	p	β	ΔR^2	p	β	ΔR^2	p
London Excess Moves	−.240/.225/.477	.039	.746	−.356	.060	.176	.652	.318	.001
WAIS Full-Scale IQ	.351/.143/.021	.130	.043	−.253	.074	.025	−.247	.056	.043