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Decreases in BMI After Cardiac Rehabilitation Predict Improved Cognitive Function in Older Adults with Heart Failure

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To the Editor

Heart failure (HF) increases risk for Alzheimer's disease [1] and mild forms of cognitive impairments [2]. Cognitive impairment in HF may be modifiable through structured exercise and/or participation in cardiac rehabilitation (CR) [3]. The mechanisms for this phenomenon are unclear, but may involve weight loss. Obesity contributes to cognitive impairment in HF [4] and CR has been linked with body mass index (BMI) reductions in this population, in addition to improvements in health factors that influence cognition (e.g., physical fitness) [3]. We investigated the impact of BMI changes on cognition among older adults with HF 12-weeks after CR and again 12-months later. Additional analyses examined physical fitness as a possible mechanism between weight loss and post-CR cognitive gains.

50 HF patients (66.72 (±7.94) years of age; 26.0% female; 90.0% Caucasian) were recruited from an National Institute of Health study examining the impact of CR on cognition in HF. HF participants were enrolled in a 12-week Phase II CR program and were between the ages

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of 50-85 years, English speaking, and had a diagnosis of New York Heart Association class II, III, or IV. Potential participants were excluded for severe neurological or psychiatric conditions. The sample had an average left ventricular ejection fraction of 38.8 (±11.5).

The Institutional Review Board approved study procedures and participants provided written informed consent. At baseline, participants performed the 2-minute step test (2MST) to assess physical fitness levels and were administered a neuropsychological battery. Height and weight were measured. These procedures were repeated 12-weeks and 12-months later.

The sample had an average BMI of 28.49 (±6.02), placing them in the overweight classification, and repeated measures analysis of variance showed no significant BMI changes over time ($p = 0.57$). Repeated measures showed attention/executive function improved from baseline to 12-weeks ($F(1, 49) = 5.90$, $p = 0.01$) and to 12-months ($F(1,49) = 8.42$, $p = 0.01$). Memory improved from baseline to 12-months ($F(1, 49) = 5.93$, $p = 0.02$) and between the 12-week and 12-month time points ($F(1, 49) = 12.02$, $p < 0.01$). There was no time effect for language ($p = 0.14$).

Table 1 shows regression analyses examining BMI and cognitive changes over time. Decreases in BMI predicted improved memory 12-weeks later ($p = 0.02$), as well as improved attention/executive function ($p = 0.052$) at the 12-month follow-up. On average, for every one unit decrease in BMI there was a 5.7% improvement in memory 12-weeks later. 12-week BMI explained an additional 7% of the variability in memory increases over this time period. Likewise, on average, for every one unit BMI decrease there was a 1.4% improvement in attention/executive function 12-months later. 12-month BMI explained an additional 1% of the variability in attention/executive function increases at 12-months.

We examined whether the beneficial effects of BMI on physical fitness may contribute to improved cognition post-CR. 2MST performance improved across the three time points ($F(2, 48) = 3.16$, $p = 0.05$). Regression analyses controlling for baseline BMI and baseline 2MST revealed decreases in BMI predicted better 2MST performance 12-weeks later ($\Delta F(1, 46) = 14.34$, $p < 0.001$). Increased 12-week 2MST performance correlated with better 12-week attention/executive function ($r(48) = 0.40$, $p < 0.01$).

CR was associated with cognitive improvements and this effect may be associated with decreases in BMI. Improved fitness may partially explain the benefits of decreased BMI on cognitive improvements in HF. Reduced physical fitness is a classic symptom of HF that is exacerbated by obesity [4]. CR improves fitness in HF [3], which may promote neurocognitive outcomes through increased cerebral perfusion [5, 6]. In addition, the current study controlled for disease severity and obesity is linked with cognitive dysfunction in healthy individuals [7], suggesting weight loss from CR may benefit cognition through unique mechanisms linked with adiposity such as reduced adipokine concentrations. Future work is needed to clarify CR-related cognitive gains and whether CR can ultimately reduce dementia risk in HF.

This study is limited in several ways. BMI remained stable across the time points and it is likely that a subgroup of individuals that exhibited the largest gains in health factors such as physical fitness are driving the current findings or even subtle declines in BMI may benefit cognitive function. Future work that utilizes objective assessments of weight status and longer follow-up periods is needed to elucidate the current findings. Future work among larger and demographically diverse samples of HF patients that also employs a healthy control group will increase the external validity of our findings.

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Table 1

Predictive Validity of BMI on Cognitive Function 12-Weeks and 12-Months After Cardiac Rehabilitation

	12-Week Memory		12-Month Attention/EF	
	b	**SE b**	**b**	**SE b**
Block 1				
Ejection fraction	-0.07	0.09	-0.10*	0.05
Hypertension	-5.60*	2.56	-1.58	1.24
Diabetes	1.58	2.42	0.68	1.17
Sleep apnea	4.88	2.89	-0.68	1.43
Baseline of DV	0.55**	0.12	0.72**	0.06
Baseline BMI	0.16	0.20	-0.22*	0.10
F	5.27**		38.07**	
R^2	0.42		0.84	
Model 1 Block 2				
12-Week BMI	-2.64*	1.10	--	--
F change	5.75*		--	
R^2 change	0.07		--	
Model 2 Block 2				
12-Month BMI	--	--	-0.69*	0.35
F change	--		4.00*	
R^2	--		0.01	

Note. Table shows hierarchical regression analyses that examined the predictive validity of 12-week and 12-month BMI changes after CR on cognitive function; there was no association between 12-week BMI and 12-week attention/executive function or between 12-month BMI and 12-month memory;

* $p \leq 0.05$;

** $p < 0.001$;

b = unstandardized beta coefficient; SE b = standard error; EF = executive function; DV = dependent variable; BMI = body mass index.