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Original Article

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Body Composition and Body Fat Distribution are Related to Cardiac

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Autonomic Control in Non-Alcoholic Fatty Liver Disease Patients

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Running Title: Body composition and Cardiac Autonomic Control

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23 **ABSTRACT**

24 **Background/Objectives:** Heart rate recovery (HRR), a cardiac autonomic control
25 marker, has been shown to be related to body composition (BC), yet this was not
26 tested in Non-Alcoholic Fatty Liver Disease (NAFLD) patients. The aim of this study was
27 to determine if, and to what extent, markers of BC and body fat (BF) distribution are
28 related with cardiac autonomic control in NAFLD patients.

29 **Subjects/Methods:** BC was assessed with Dual Energy X-ray Absorptiometry in 28
30 NAFLD patients (19 males, 51 ± 13 yrs, and 9 females, 47 ± 13 yrs). BF depots ratios
31 were calculated to assess BF distribution. Subjects' HRR was recorded 1 (HRR1) and 2
32 minutes (HRR2) immediately after a maximum graded exercise test.

33 **Results:** BC and BF distribution were related to HRR, particularly weight, trunk BF as
34 well as trunk BF-to-appendicular BF ratio showed a negative relation with HRR1 ($r=-$
35 0.613 ; $r=-0.597$ and $r=-0.547$; respectively, $p<0.01$) and HRR2 ($r=-0.484$; $r=-0.446$;
36 $p<0.05$ and $r=-0.590$; $p<0.01$, respectively). Age seems to be somewhat related to both
37 HRR1 and HRR2 except when controlled for BF distribution. The preferred model in
38 multiple regression should include trunk BF-to-appendicular BF ratio and BF to predict
39 HRR1 ($r^2=0.549$; $p<0.05$), and trunk BF-to-appendicular BF ratio alone to predict HRR2
40 ($r^2=0.430$; $p<0.001$).

41 **Conclusions:** BC and BF distribution were related to HRR in NAFLD patients. Trunk BF-
42 to-appendicular BF ratio was the best independent predictor of HRR and therefore
43 may be best related to cardiovascular increased risk, and possibly act as a mediator in
44 age related cardiac autonomic control variation.

45 **Keywords:** Regional Body Fat; Dual Energy X-ray Absorptiometry; Hepatic Steatosis;
46 Heart Rate Recovery; Parasympathetic Reactivation.

47 INTRODUCTION

48 **Paragraph number 1** Non Alcoholic Fatty Liver Disease (NAFLD) is a condition present
49 in up to 30% of developed countries, with a considerably higher prevalence in the
50 obese populations, particularly in the presence of abdominal or morbid obesity (1-5).
51 NAFLD was shown to result from hepatic fat metabolism imbalance and encompasses
52 several stages, from the initial hepatocyte fat accumulation (hepatic steatosis), to
53 hepatic inflammation (non-alcoholic steatohepatitis) along with a constellation of
54 other disturbances, that ultimately can lead to advanced fibrosis, cirrhosis, liver failure
55 and death (6). NAFLD patients have also been reported to have increased
56 cardiovascular risk compared with the general population (7). Insulin resistance and
57 obesity are major risk factors for NAFLD, yet BF accumulation, particularly that of the
58 abdominal region, besides being strongly associated with NAFLD and found to precede
59 presence of insulin resistance (8), may mimic the same metabolic abnormalities
60 triggered by insulin resistance alone (9, 10) and is also associated with other metabolic
61 disorders that can also increase the risk of NAFLD, therefore, BF may be a key factor in
62 the etiology of NAFLD (6).

63 **Paragraph number 2** Heart rate recovery (HRR) after exercise is a recognized cardiac
64 autonomic control marker mostly reflective of parasympathetic reactivation (11, 12).
65 Slow HRR is independently related to higher risk of mortality and other cardiovascular
66 and metabolic outcomes (13-20). Autonomic nervous system (ANS) imbalance,
67 including blunted HRR, has also been linked to obesity (21), higher body fat (BF)
68 accumulation (22, 23). Kreier and colleagues (24) presented a neuroanatomical
69 evidence for a reciprocal influence of BF, particularly intra-abdominal BF, and ANS, and
70 suggested a pathway for ANS mediated imbalance in several other biological functions

71 including liver fat metabolism, meaning it may be somewhat involved in the etiology,
72 progression, consequences and treatment of both obesity and NAFLD, however this
73 has been largely overlooked, particularly in the population of NAFLD, and research is
74 warranted in this field. Insulin resistance and obesity (main risk factors for hepatic fat
75 accumulation) have been shown to precede the presence of slow HRR (20, 25). Thus,
76 the BF accumulation and distribution has been suggested to be associated with ANS
77 imbalance (22, 26, 27), but this has not yet been tested in NAFLD patients.

78 **Paragraph number 3** Very few studies have focused on BF distribution and HRR
79 associations and it is unknown if such a relationship exists in NAFLD patients. The
80 purpose of the present study was to determine if, and to what extent, specific markers
81 of BC and BF distribution, are related with reduced parasympathetic reactivation
82 following maximal exercise, as assessed by heart rate recovery (HRR), in NAFLD
83 patients.

84

85 **SUBJECTS AND METHODS**

86 **Paragraph number 4 Subjects:**

87 This study was conducted at Exercise and Health Laboratory, from the
88 Interdisciplinary Centre for the Study of Human Performance (Faculty of Human
89 Kinetics, Technical University of Lisbon, Portugal). To be selected for the present study
90 subjects had to be over 18 years of age without history of hepatotoxic substances
91 intake (eg. steroids) and tobacco consumption. Exclusion criteria included alcohol
92 consumption over 20 gr/day; the presence of other potential causes for fatty liver
93 disease (viral hepatitis, auto-immune disease and others); any physical and/or mental
94 disabilities or any condition that constituted an absolute restriction to exercise, or

95 other diagnosed diseases, with mandatory specific pharmacologic therapy. Not
96 included in the exclusion criteria is the presence of metabolic and cardiovascular
97 disease (insulin resistance, hypertension or dyslipidemia). We studied 25 NAFLD
98 patients (19 males, 51 ± 13 yrs, and 9 females, 47 ± 13 yrs) who were diagnosed
99 through liver biopsy or ultrasound. Subjects were recruited from the outpatient
100 medical departments in Santa Maria Hospital and Curry Cabral Hospital; 59
101 consecutive patients were selected based on selection criteria; 37 of the selected
102 subjects accepted to participate and 28 were found eligible to enter the study after
103 exclusion criteria was considered. Subjects were taking one or more of the following
104 medication: platelet inhibitors, angiotensin-converting enzyme inhibitors, nitrates,
105 statins, ezetimibe, nicotinic acid and biguanides with similar use among both genders.
106 All participants signed an informed consent before being included in the present study
107 and undergoing any study procedure. All methods used in the present study comply
108 with ethics and Portuguese laws and were approved by Faculty of Human Kinetics
109 institutional review board for human studies. The present investigation also complies
110 with the principles outlined in the Declaration of Helsinki.

111 ***Paragraph number 5 Body composition:***

112 Body composition was assessed using Dual Energy X-ray Absorptiometry (DXA)
113 (Explorer W, Hologic; Waltham, MA, USA; Fan bean mode) whole body scans and
114 anthropometric measurements. Repeated measurements in 18 young adults showed a
115 coefficient of variation (CV) of 1.7% for total BF mass and 1.5% for total %BF. All scans
116 were performed in the morning after an overnight 12-hour fast. Quality control with
117 spine phantom was made every morning, and with step phantom every week. By
118 default the DXA software (QDR for windows, version 12.4) estimates the head, trunk,

119 arms and legs, both left and right, regional fat content, according to a three-
120 compartment model (fat mass, lean tissue and bone mass). The trunk region of
121 interest (ROI) (CV = 0.005%) includes chest, abdomen and pelvis. Appendicular ROI (CV
122 = 0.004 %) includes both arms plus both legs. All scans were submitted to additional
123 analysis by ROI to assess fat content of the abdominal and central abdominal regions
124 (CV = 0.01 %). The upper and lower limits of the abdominal and central abdominal ROI
125 were determined as the upper edge of the second lumbar vertebra to the lower edge
126 of the fourth lumbar vertebra, respectively (28-30). The lateral limits of the abdominal
127 ROI were determined as to include all trunk length, but exclude any upper limb scan
128 area (29, 30), whereas the vertical sides of central abdominal ROI were the
129 continuation of the lateral sides of the ribs cage, as to exclude the lateral
130 subcutaneous fat of the trunk, including the anterior and posterior subcutaneous
131 abdominal fat, as well as the intra-abdominal fat (28). Absolute and relative BF content
132 results were registered to the nearest 0.01kg and 0.1%, respectively. All scans and
133 analyses were made by the same observer.

134 **Paragraph number 6** Anthropometric measurements consisted of weight, height and
135 body mass index (BMI). Body weight was measured to the nearest 0.1kg, and height
136 was measured to the nearest 0.1 cm, on a scale with an attached stadiometer (model
137 770, Seca; Hamburg, Deutschland), according to standard protocol (31). Both weight
138 and Height were used to calculate the subject's BMI, by dividing the weight, in kg, by
139 the squared height, in meters ($BMI = \text{weight [kg]} / \text{height [m]}^2$).

140 **Paragraph number 7 Body fat distribution:**

141 BF distribution variables were calculated using ratios between BF content
142 absolute values of different fat depots, obtained by DXA, as done elsewhere (30). The

143 trunk BF-to-appendicular BF ratio, also called trunk-to-extremity fat ratio (32) or
144 central-to-peripheral fat mass ratio (33), was calculated as the trunk BF content
145 divided by the sum of the BF content of the arms and legs, both left and right. The
146 abdominal BF-to-trunk BF ratio was calculated as the fat content of the selected
147 abdominal ROI divided by the trunk BF. The abdominal BF-to-total BF was calculated as
148 the selected abdominal ROI fat content divided by the whole BF. Ratios were
149 registered to the nearest 0,01.

150 ***Paragraph number 8 Exercise testing:***

151 All subjects underwent a treadmill (Q-65, Quinton, Cardiac Science Corp; Bothell,
152 WA, USA) graded exercise test (GXT) using Bruce standard protocol (34). All GXT were
153 monitored using a 12 lead electrocardiogram PC-based acquisition module (Welch-
154 Allyn PCE-210, Welch Allyn Inc.; Skaneateles Falls, NY, USA) and the data, including
155 heart rate (HR), were monitored and recorded using Welch Allyn CardioPerfect
156 software (Welch Allyn Inc.; Skaneateles Falls, NY, USA). Oxygen uptake was monitored
157 during GXT using a MedGraphics CPX Ultima Cardio metabolic cart (Medical Graphics
158 Corp; St Paul, MN, USA) and data was recorded using Breeze Suite software (version
159 6.4.1, Medical Graphics Corp; St Paul, MN). Subjects exercised until at least two of the
160 following test termination criteria were reached (35): (1) subjects volitional fatigue; (2)
161 respiratory exchange ratio reached 1.1 or higher; (3) subjects reached age predicted
162 maximal HR (HR_{max}); (4) oxygen uptake did not increase in spite of increasing work
163 load.

164 ***Paragraph number 9 Heart Rate Recovery:***

165 When GXT termination criteria were reached patients started exercise recovery
166 with a speed of 1.5mph and incline of 2.5% on the treadmill. Subjects remained

167 walking with the recovery treadmill mechanical load for 2 minutes. After 2 minutes of
168 recovery the treadmill was stopped and subjects continued their recovery seated in an
169 armless standard chair. HR was recorded beat-by-beat and was averaged at 15 seconds
170 intervals for identifying HRmax. HR at the end of the first and second recovery minutes
171 were recorded from beat-by-beat records (HR1 and HR2, respectively). HRR was
172 calculated as the difference between observed HRmax and HR1 ($HRR1 = HRmax - HR1$)
173 and HR2 ($HRR2 = HRmax - HR2$). Cut off value for identifying slow HRR was considered
174 12bpm for HRR1 (13-15, 19). The 22bpm cut off value for identifying slow HRR2 was
175 developed using a supine recovery protocol (18, 36), however it has been used with
176 diverse exercise recovery protocols, including seated (37) and walking (20) recovery
177 protocols and therefore was adopted in the present study for descriptive purposes
178 only.

179 ***Paragraph number 10 Statistical methods:***

180 Descriptive statistics are presented as mean \pm SD and range for all analyzed
181 variables. The Gaussian distribution of the data was assessed with the Shapiro-Wilk
182 goodness-of-fit test. Partial and part, also called semipartial (38), correlations were
183 performed to assess the relations between dependent and independent variables
184 controlling for age and sex. When age was an independent variable the correlation was
185 controlled for sex and fat distribution. In order to accomplish a statistical power of 80%
186 ($\beta = 0.20$) at a statistical significance level of 5% ($\alpha = 0.05$), as has been used as a
187 convention (38), only coefficients of correlation equal or superior to 0.5, corresponding
188 to a large effect size, were considered significant and unexposed to type I and II errors
189 (38). Multiple linear regressions were conducted, using Enter method, between
190 dependent variables and correlated independent variables to analyze r square change

191 when using two predictors in the model. Stepwise regressions were performed to find
192 preferred models for the prediction of both dependent variables (HRR1 and HRR2).
193 The level of significance was set at $P < 0.05$ (two-tailed). Statistical calculations were
194 performed using the IBM SPSS Statistics version 19 (SPSS, inc, Chicago, IL).

195

196 **RESULTS**

197 **Paragraph number 11** Mean values for all studied variables are presented in Table 1.

198 No clinical test interruption criteria, such as electrocardiogram signs of ischemia, new
199 onset of arrhythmias, or excessive hypotensive/hypertensive response, were observed
200 in any GXT. All subjects met termination criteria for ending the GXT. From among the
201 25 studied NAFLD patients slow HRR1 was present in 6 (22.2%, 2 were female) and
202 slow HRR2 in 5 (18.5%, 2 were female) patients. Neither HRR1 nor HRR2 were different
203 between men and women ($p = 0.754$ and $p = 0.631$ obtained in an independent samples t
204 test comparison, respectively). Mean BMI of the studied sample was in the overweight
205 category, with no differences between sexes ($p = 0.075$ on independent samples t test).
206 BMI was also not related with age ($r = -0.218$; $p = 0.285$ on Pearson correlation).

207 **Paragraph number 12** Table 2 shows the results for partial and semipartial
208 correlations between each independent variable and each dependent variable (HRR1
209 and HRR2), controlled for sex and age (unless otherwise noted). Only the studied BF
210 compartments, not fat free mass, were related to HRR. On a whole body analysis only
211 weight was found negatively correlated with HRR1 ($p = 0.002$), in partial correlations
212 and semipartial correlations. The regional BC analysis showed that trunk BF ($p = 0.003$)
213 and central Abdominal BF ($p = 0.009$) were negatively correlated with HRR1 but not
214 with HRR2, both in partial and semipartial correlations, independently of sex and age.

215 The analysis of BF distribution indicated that the trunk BF divided by appendicular BF
216 was the only studied BF distribution marker related to HRR1 ($p=0.008$) and the only
217 studied independent variable to be related to HRR2 ($p=0.003$) in both partial and
218 semipartial correlations, when controlled for sex and age. Age, when controlled for sex
219 and BF distribution, was not related to neither HRR1 nor HRR2 ($p=0.596$ and $p=0.483$,
220 respectively).

221 **Paragraph number 13** All independent variables that showed significant relation with
222 HRR in partial and semipartial correlations were included in multiple linear regression
223 analysis shown in table 3. Regressions were performed using only trunk BF-to-
224 appendicular BF ratio and age, which has been suggested to influence HRR in healthy
225 adults (20), as predictors of either HRR1 or HRR2, and also between pairs of
226 independent variables to predict HRR1. Because trunk BF-to-appendicular BF ratio was
227 the only independent variable correlated with both dependent variables, it was chosen
228 as a fixed independent variable in multiple linear regressions. The higher R square
229 change in the prediction of HRR1 seems to be that obtained by adding weight to trunk
230 BF-to-appendicular BF ratio in the prediction model. In the prediction of HRR2 Trunk
231 BF-to-appendicular BF ratio alone was found to predict over 40% of the variation of
232 HRR2, in this sample of NAFLD patients.

233

234 **DISCUSSION**

235 **Paragraph number 14** To our knowledge this is the first study to focus on the
236 association between HRR, and BC and/or BF distribution, in NAFLD patients. Most
237 studies on HRR focus primarily on cardiovascular outcomes and have not included BC
238 variables (12-16). Some previous population-based reports showed slower HRR in

239 patients with higher BMI (25, 39). Nilsson and colleagues found similar results in elders
240 (27). In a recent report, BMI showed the highest odds ratio for slow HRR2 (OR=6.58)
241 over a 20 yr period, after controlling for baseline HRR (20). In our sample BMI was not
242 associated with either HRR1 or HRR2, after controlling for age and sex. Similar results
243 had also been found in a sample of type 2 diabetes mellitus patients (19). These
244 discrepancies may be explained by differences in studied samples as well as in research
245 protocols, including different HRR record timing criterion as well as considerable
246 exercise protocol differences either in the effort as in the recovery phase. Nevertheless
247 the development of slow HRR seems more likely in those who have more BF
248 accumulation (20, 25, 37).

249 **Paragraph number 15** A recent report showed that the sum of skinfolds accounted for
250 the greatest variance of both HRR1 and HRR2, as compared with BMI, waist
251 circumference (WC) and maximal oxygen consumption (23). They used mainly skinfolds
252 from the trunk region, including the abdominal skinfold, which can reinforce the
253 importance of central BC for appropriate ANS function. In accordance to this, the
254 present results showed trunk BF and CABd BF to be significant correlated with HRR1,
255 independent of age and sex. Few studies could be found using different BC markers,
256 besides BMI, when focusing on HRR, nevertheless some investigations have used WC
257 to assess central obesity or central as well as whole BF accumulation and found
258 concordant results to ours (20). Mean WC has been shown to be higher in patients
259 with slow HRR (20, 25). The association between slow HRR and WC has been shown to
260 be stronger than with BMI (adjusted for age, race and sex) (25) as well as with all
261 metabolic syndrome components (27). In the present study the results on central BF
262 variables, particularly abdominal fat and central abdominal fat, also show a negative

263 correlation with HRR1, but not with HRR2. Kim and colleagues (22) found somewhat
264 concordant results concerning the relation between visceral fat, particularly that
265 around the myocardium, and both HRR1 and HRR2. The only study we found focusing
266 on HRR and regional body composition analysis using DXA showed no differences in
267 HRR between overweight young adults and lean control subjects, in a sample of
268 overnight sleep apnea patients, even though overweight subjects were significantly
269 heavier, and had higher BMI, %BF and central abdominal BF (40).

270 **Paragraph number 17** In the present study Trunk BF: Appendicular BF ratio was the
271 only BF distribution marker that was related to HRR, moreover this BF distribution
272 marker was the only studied independent variable to show correlation magnitudes
273 with both HRR1 and HRR2 that correspond to a large effect size, even after removing
274 the effect of sex and age. Multiple regression also revealed that other BC variables
275 added little predictive capacity to Trunk BF-to-Appendicular BF ratio. These results
276 emphasize that BF distribution may be more important for ANS function than the
277 absolute or relative amount of BF. Because HRR has been considered a powerful
278 predictor of cardiovascular, as well as overall, mortality (13, 14, 17, 19, 41-44), the
279 present results suggest that a central BF distribution, particularly Trunk BF-to-
280 Appendicular BF ratio, can possibly relate more strongly to cardiovascular increased
281 risk. The importance of a central distribution of BF was noticed before, using HRV to
282 assess ANS function (26). In that study, abdominal-to-peripheral fat distribution, assess
283 by dividing abdominal by thigh DXA estimated fat contents, was found to explain a
284 significant variation of HRV (26). It is known that the ANS may influence adipocyte fat
285 metabolism by an endocrine pathway and a neuronal pathway (45, 46), and adipocytes
286 from different regions of the body respond differently to the intensity and duration of

287 the endocrine stimulation (47) and may also be controlled by different
288 branches/neurons of the ANS (24). Therefore, the fact that BF distribution was the
289 most consistent correlate with the studied autonomic markers, in the present study,
290 gives strength to the theory that ANS may be somewhat involved, either as a cause or
291 as a consequence, in BC and overall metabolic abnormalities associated with the
292 central BF accumulation phenotype, though this is still speculative at this point. The
293 potential implications of the ANS in the etiology, progression, consequences and
294 treatment of both adverse body fat accumulation patterns and NAFLD should warrant
295 further research.

296 **Paragraph number 18** Carnethon et al. (20) showed an association of HRR with aging.
297 In our cross-sectional study the relation of HRR1 and HRR2 with patient's age, was
298 absent if controlled for BF distribution. Christou and colleagues (26) had long proposed
299 that the changes in fat accumulation pattern that occurs with aging, resulting in BF
300 distribution changes, may contribute to the ANS variation commonly attributed to
301 aging. This is a matter that needs to be confirmed either in the general population as in
302 specific sub-populations such as the NAFLD patients and other metabolic impaired sub-
303 populations.

304 **Paragraph number 19** The prevalence of slow HRR in the present study is in
305 accordance with most of the published data, including that from the Cleveland Clinic
306 Foundation (13-15) that focused on patients referred for symptom-limited exercise
307 testing, as well as in patients with metabolic impairments (17, 19) or in even more
308 heterogeneous populations (25), in accordance to the understanding that metabolic
309 impairments are somewhat linked to abnormal ANS. Accordingly, when confronted
310 with healthy cohort data, as shown recently by Carnethon and colleagues (20) the

311 prevalence of slow HRR in the present sample was fairly high. The prevalence of high
312 levels of BMI, including obese and morbidly obese patients, in the present sample was
313 expected since obesity, along with insulin resistance, have been identified as the
314 strongest risk factors for NAFLD, and therefore highly prevalent in this sub-population
315 (1-4).

316 **Paragraph number 20** There are several strengths and limitations to this study. In the
317 present report autonomic nervous system assessment was restricted to HRR. Previous
318 studies have validated the use of HRR as a marker of parasympathetic reactivation,
319 however HRR is not a direct measure of autonomic nervous system dysfunction but
320 rather is an estimate of parasympathetic response to a specific physiologic challenge
321 (i.e., exercise) (11, 12). Further studies with measures of different components of
322 autonomic nervous system function (e.g., sympathetic input), as well as
323 sympathetic/parasympathetic balance and resting cardiac autonomic control, are
324 warranted to confirm our observations. Also our BC assessment method (DXA) albeit
325 being a gold standard instrument to assess BC in a three compartment model, is
326 unable to determine visceral adiposity independently from subcutaneous fat.
327 Nevertheless, recent studies indicate strong correlation between abdominal fat
328 estimated from selected ROI and visceral fat assessed by magnetic resonance imaging
329 (29) and computed tomography (48, 49). Because a cross-sectional approach was used,
330 a causal relation between cardiac autonomic control variation and BC or BF
331 distribution could not be established, based on the present results. Finally, the size of
332 the sample was rather constrained due to difficulties in the recruitment of such a
333 specific sub-population. 90 individuals were coveted to be included in the present
334 sample in the initial research project. This would allow coefficients of correlation as

335 low as 0.3, traditionally corresponding a moderate effect size, to be considered
336 significant and unexposed to type I and II errors (38). Unfortunately, despite all efforts
337 on behalf of everyone involved in this research project, only 28 NAFLD could be
338 recruited. This embodied acknowledged consequences in the statistical power of the
339 present results. Consequently, only associations equal or higher to $r=0.50$ could be
340 considered to attain minimal statistical power of 80% and statistical significance of 5%,
341 and could be considered fairly unexposed to type 1 and type 2 errors (38). However
342 the aim of the present study was not compromised, neither it's importance. This study
343 sought to find the best markers, which are found at the higher end of correlational
344 range, so the inability to find significant associations lower than $r=0.5$, though
345 interesting are not the aim of the present study. Moreover, the present results
346 represent a relevant preliminary analysis to establish the importance of BC and BF
347 distribution in the cardiac autonomic control of NAFLD patients.

348 **Paragraph number 21** In the present study BF content and distribution were
349 important contributors to HRR in NAFLD patients. Excess BF accumulated in the trunk
350 or abdominal regions is associated with poor HRR. BF distribution appears to be more
351 important than overall BF accumulation in explaining the variation of HRR and
352 therefore can possibly be a better predictor of cardiovascular risk in NAFLD patients.
353 Therefore, present results also highlight the importance of assessing BF distribution in
354 NAFLD patients, rather than just markers of generalized BF.

355

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359

360 **CONFLICT OF INTEREST**

361 The authors have nothing to disclosure.

362

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546 **TABLES:**

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548 Table 1. Descriptive data of the studied sample.

| Variables | NAFLD Patients (n=25) | |
|---------------------------------------|------------------------------------|-----------------------------|
| | Mean \pm sd * | Min. – Max. |
| Age, yr (median, yr) | 48.6 \pm 12.8 (49) | 25 – 68 |
| Sex, n female (% female) | 8 (30.8) | |
| VO ₂ max, ml/kg/min | 24.9 \pm 6.4 | 13.8 – 38.0 |
| Type 2 Diabetes Mellitus, n (%) | 8 (28.6) | |
| Insulin resistance, n (%) | 12 (42.9) | |
| HRR1, bpm | 19.4 \pm 10.1 | -4.0 – 37.0 |
| HRR2, bpm | 35.9 \pm 16.7 | -8.0 – 67.0 |
| Whole Body Analysis | | |
| Weight, kg | 88.0 \pm 12.8 | 66.2 – 115.8 |
| Stature, cm | 167.3 \pm 9.4 | 149.5 – 183.7 |
| BMI, kg/m ² (% obese) | 29.1 \pm 4.1 (34.6) | 22.6 – 42.2 |
| BF, kg (%) | 27.5 \pm 9.4 (31.52 \pm 8.29) | 13.7 – 51.2 (18.84 – 46.28) |
| FFM, kg (%) | 58.8 \pm 9.2 (68.48 \pm 8.29) | 39.6 – 77.7 (53.72 – 81.16) |
| Regional Body Analysis | | |
| Trunk BF, kg (%) | 15.4 \pm 5.2 (33.37 \pm 7.71) | 7.4 – 25.0 (20.87 – 48.01) |
| Trunk FFM, kg (%) | 29.9 \pm 4.0 (66.63 \pm 7.31) | 21.1 – 38.6 (51.99 – 79.13) |
| Appendicular BF, kg (%) | 11.0 \pm 4.8 (30.63 \pm 10.54) | 5.2 – 25.7 (13.63 – 50.40) |
| Appendicular FFM, kg (%) | 28.5 \pm 5.1 (80.40 \pm 6.56) | 19.2 – 36.7 (68.64 – 90.66) |
| Abdominal BF, kg (%) | 3.5 \pm 1.2 (37.99 \pm 6.67) | 1.7 – 6.3 (26.09 – 49.40) |
| Central Abdominal BF, kg (%) | 2.9 \pm 0.8 (35.94 \pm 5.78) | 1.6 – 5.0 (24.28 – 44.64) |
| Body Fat Distribution (Ratios) | | |
| Trunk BF-to-Appendicular BF ratio | 1.478 \pm 0.378 | 0.958 – 2.547 |
| Abdominal BF-to-Total BF ratio | 0.130 \pm 0.026 | 0.045 – 0.185 |
| Abdominal BF-to-Trunk BF ratio | 0.233 \pm 0.040 | 0.095 – 0.299 |

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* results are presented as mean \pm standard deviation, unless otherwise noted; VO₂max – maximal oxygen consumption; BF – body fat; BMI – body mass index; FFM – fat free mass; HRR1 – heart rate recovery at 1 min.; HRR2 – heart rate recovery at 2 min.; Máx. – highest observed value; Min. – lowest observed value.

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Table 2. Partial and semipartial correlations between dependent and independent variables.

| Variables | HRR 1 | | HRR 2 | |
|---------------------------------------|---------------------|---------------------|---------------------|---------------------|
| | r^{\dagger} | r^{\ddagger} | r^{\dagger} | r^{\ddagger} |
| Age | -0.120 [§] | -0.093 [¶] | -0.154 [§] | -0.115 [¶] |
| Whole Body Analysis | | | | |
| Weight, kg | -0.613 ** | -0.565 ** | -0.484 * | -0.440 * |
| Stature, cm | -0.176 | -0.162 | -0.161 | -0.147 |
| BMI, kg/m ² | -0.325 | -0.299 | -0.164 | -0.149 |
| BF, kg | -0.493 * | -0.453 | -0.313 | -0.285 |
| BF, % | -0.241 | -0.222 | -0.068 | -0.062 |
| FFM, kg | -0.190 | -0.172 | -0.144 | -0.129 |
| FFM, % | 0.235 | 0.213 | 0.192 | 0.172 |
| Regional Body Analysis | | | | |
| Trunk BF, kg | -0.597 ** | -0.550 ** | -0.446 * | -0.406 * |
| Trunk BF, % | -0.356 | -0.327 | -0.232 | -0.211 |
| Trunk FFM, kg | -0.211 | -0.192 | -0.151 | -0.135 |
| Trunk FFM, % | 0.288 | 0.262 | 0.259 | 0.232 |
| Appendicular BF, kg | -0.273 | -0.251 | -0.096 | -0.088 |
| Appendicular BF, % | -0.020 | -0.018 | 0.186 | 0.170 |
| Appendicular FFM, kg | -0.179 | -0.163 | -0.140 | -0.125 |
| Appendicular FFM, % | 0.171 | 0.156 | 0.144 | 0.129 |
| Abdominal BF, kg | -0.491 * | -0.451 * | -0.265 | -0.241 |
| Abdominal BF, % | -0.296 | -0.272 | -0.093 | -0.085 |
| Central Abdominal BF, kg | -0.553 ** | -0.508 ** | -0.335 | -0.304 |
| Central Abdominal BF, % | -0.376 | -0.345 | -0.170 | -0.154 |
| Body Fat Distribution (Ratios) | | | | |
| Trunk BF-to-Appendicular BF ratio | -0.547 ** | -0.503 ** | -0.590 ** | -0.537 ** |
| Abdominal BF-to-Total BF ratio | -0.150 | -0.138 | -0.042 | -0.038 |
| Abdominal BF-to-Trunk BF ratio | 0.086 | -0.079 | 0.260 | 0.236 |

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BF – body fat; BMI – body mass index; FFM – fat free mass; HRR1 – heart rate recovery at 1 min.; HRR2 – heart rate recovery at 2 min.; † – partial correlations controlling for age and sex (except when age is a variable); ‡ – semipartial correlations removing the effect of age and sex (except when age is a variable); § – partial correlation controlling for trunk BF/ Limb BF ratio and sex; ¶ – semipartial correlation removing the effect of trunk BF/ Limb BF ratio and sex. * - significant for p<0.05; ** - significant for p<0.01; *** - significant for p<0.001.

565 Table 3. Linear regressions with R square change analysis (Enter method) between dependent and
 566 related independent variables.

| Variables | Model † | R | R square | R square change | P |
|-------------------------------------|--------------------------|-------|----------|-----------------|-----------|
| HRR 1 ‡ | | | | | |
| Trunk BF-to-Appendicular BF ratio | | 0.617 | 0.380 | -- | 0.001 ** |
| | Weight, kg | 0.739 | 0.546 | 0.166 | 0.012 * |
| | BF, kg † | 0.741 | 0.549 | 0.169 | 0.011 * |
| | Trunk BF, kg | 0.724 | 0.524 | 0.144 | 0.020 * |
| | Abdominal BF, kg | 0.657 | 0.432 | 0.052 | 0.167 |
| | Central Abdominal BF, kg | 0.664 | 0.441 | 0.061 | 0.138 |
| | Age, yr | 0.625 | 0.391 | 0.011 | 0.346 |
| HRR 2 ‡ | | | | | |
| Trunk BF-to-Appendicular BF ratio † | | 0.655 | 0.430 | -- | 0.000 *** |
| | Weight, kg | 0.709 | 0.502 | 0.072 | 0.087 |
| | Trunk BF, kg | 0.698 | 0.487 | 0.057 | 0.131 |
| | Age, yr | 0.666 | 0.444 | 0.014 | 0.467 |

567 BF – body fat; HRR1 – heart rate recovery at 1 min.; HRR2 – heart rate recovery at 2 min.; † – Regressions were
 568 conducted using pairs of independent variables, which include always Trunk BF/Appendicular BF ratio plus one
 569 of the listed variables; ‡ – Dependent variable in the following regressions. * – significant for p<0.05; ** –
 570 significant for p<0.01; *** – significant for p<0.001.

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