

## Changes in weight after traumatic brain injury in adult patients: A longitudinal study.

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► **To cite this version:**

Pascal Crenn, Sabrina Hamchaoui, Alette Bourget-Massari, Mouna Hanachi, Jean-Claude Melchior, et al.. Changes in weight after traumatic brain injury in adult patients: A longitudinal study.. Clinical Nutrition, Elsevier, 2014, epub ahead of print. <10.1016/j.clnu.2013.06.003>. <inserm-00924236>

**HAL Id: inserm-00924236**

**<http://www.hal.inserm.fr/inserm-00924236>**

Submitted on 6 Jan 2014

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1 **Changes in weight after traumatic brain injury in adult patients: a longitudinal study**

2

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9

10 **Key words:** traumatic brain injury, weight change, behavioral dysexecutive syndrome, eating  
11 disorders

12

13 **Abbreviations:** TBI: traumatic brain injury. BMI: body mass index. WHO: World Health  
14 Organization.

15

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21

22 Word count of the text: 3736

23 3 tables and 1 figure

24

25 **Abstract**

26 **Background & Aims:** Although changes in weight have been reported after traumatic brain  
27 injury (TBI), their frequency and underlying factors are little known. Our aim was to  
28 determine the prevalence of weight changes and the associated factors during the recovery  
29 phase after TBI.

30 **Methods:** Longitudinal follow-up of adults with TBI. Multivariate analysis was carried out on  
31 weight change, demographic data, dysexecutive syndrome, eating behavior, physical activity,  
32 therapeutic classes and metabolic complications.

33 **Results:** 107 patients (81 males/26 females), age  $36\pm 13$  yrs, baseline BMI  $23.3\pm 3.9$ , followed  
34 for 38 (8–66) months, were included. In intensive care, patients lost a mean  $11\pm 6$  kg. End of  
35 follow-up, mean BMI was not different to pre-TBI BMI, but patients could be categorized in  
36 3 groups: stable (30%), loss (28%,  $-8\pm 7$  kg) and gain (42%,  $+9\pm 6$  kg). Sex, age, severity of  
37 TBI, intensive care weight loss, physical activity, therapeutic classes and the occurrence of  
38 metabolic syndrome did not differ between the groups. Factors related to weight gain were  
39 hyperphagia, OR 4.5 (IC95%, 1.6–12.1) and presence of a dysexecutive syndrome, OR 2.5  
40 (IC95%, 1.03–6.3). Factors related to weight loss were hypophagia, OR 4.1 (IC95%, 1.5–  
41 10.9) and higher pre-TBI BMI, OR 4.9 (IC95%, 1.7–14.0).

42 **Conclusions:** Over a median period of 38 months, 42% of TBI patients gained and 28% lost  
43 weight. Factors associated with these changes were the presence of a behavioral dysexecutive  
44 syndrome for weight gain, oral food intake and initial BMI, which were inversely associated  
45 with weight at end of follow-up. These findings highlight the importance of evaluating the  
46 time course of weight changes and providing specific nutritional care.

47

## 48 **Introduction**

49 Obese passengers are more likely to suffer a more severe head injury after frontal motor-  
50 vehicle crashes<sup>1</sup>. After traumatic brain injury (TBI), changes in weight and (or) food behavior  
51 have been reported in short studies<sup>2,3</sup> and various clinical case-reports<sup>4,5</sup>. Some reports have  
52 described hyperphagia and reduction of satiety<sup>6</sup>. Anorexia following TBI has also been  
53 reported<sup>7</sup>. A recent longitudinal study in 39 children with TBI showed that 15% were  
54 overweight 1 year after the TBI<sup>8</sup>. However, the frequency of weight changes and the  
55 underlying factors are currently unknown in adult patients. In a study of 20 TBI patients, the  
56 presence of other persons during meals, or the social factor, was a significant predictor of  
57 meal size for healthy control subjects, but not for brain-injured patients<sup>2</sup> indicating probable  
58 central involvement. In addition, a low prevalence (5.4%) of hypopituitarism, including GH  
59 deficiency and hypogonadism, which can contribute to obesity, was reported in a study of 112  
60 adult TBI patients<sup>9</sup>. The same trend was observed in 39 children, with a prevalence of 2.5%<sup>8</sup>.  
61 A higher prevalence -15%- was found in a cross-sectional study of 104 adult patients 13  
62 months after TBI<sup>10</sup>. Modification of food behavior – and thus of weight – can be expected to  
63 occur after TBI, due to the presence of a dysexecutive syndrome and because the  
64 hypothalamus is the main brain center involved in food intake in both animal experimental  
65 models and humans<sup>11</sup>. Cognition and behavior can also be impaired in moderately severe  
66 TBI, and is related to a “post-concussion syndrome”<sup>12</sup>.

67       Changes in nutritional intake after TBI appear to occur in two phases: a constant,  
68 prolonged and significant hypermetabolism during the stay in neurosurgical units and  
69 intensive care<sup>13,14</sup>, with a risk of severe undernutrition despite nutritional support, followed  
70 by a recovery phase which may follow different patterns. To determine the prevalence of  
71 changes in weight after TBI in the recovery phase, and the associated factors, we performed a

72 longitudinal study in a cohort of adult TBI patients followed in a specialized hospital for

73 neurocognitive rehabilitation.

74

**75 Patients and methods***76 Patients*

77 This was a single centre longitudinal study of a cohort of adult TBI patients. All the patients  
78 were initially cared for in different intensive care units and were then admitted to our physical  
79 medicine and rehabilitation (PMR) center between 2004 and 2009. The study protocol was  
80 approved by the local Ethics committee. Informed consent was obtained from the patients or  
81 their relations in compliance with the French regulations for observational clinical research.  
82 Inclusion criteria were: age 18–70 years at the time of the TBI, isolated TBI assessed with the  
83 cerebral Marshall CT-scan classification<sup>15</sup> and the Glasgow coma scale (GCS) [mild (score  
84 13–15), moderate (9–12), or severe (<9)] with no associated spinal cord injury or  
85 polytraumatic lesions of the viscera which required surgery. In addition, a follow-up of at  
86 least 6 months after the intensive care period and from the beginning of rehabilitation was  
87 mandatory.

88 We excluded patients below the age of 18 or above the age of 70, pregnant women, patients  
89 with paraplegia, tetraplegia and those in a persistent vegetative state, patients with no  
90 indications for rehabilitation and patients with previous surgery to the digestive system, in  
91 order to eliminate possible interference with the regulation of food intake.

92 Analyses of hypothalamic and pituitary hormones were carried out in patients for whom there  
93 was a clinical or biological suspicion of deficiency, such as polyuria or hypernatremia.

94 Enteral nutrition adapted to their needs in terms of energy and protein (1500 to 1800 kcal/d,  
95 56 to 67 g of proteins) was given to each patient in intensive care units by a nasogastric tube  
96 or gastrostomy and was continued during the transfer to PMR. Reduction and weaning from  
97 enteral nutrition was carried out under the supervision of the dietician during the stay in PMR  
98 (4 months in average) when the patient regained the ability to swallow without difficulty. A  
99 mixed feeding program was put into place, with a nocturnal enteral intake until oral intake

100 became sufficient, with an energy intake goal of 1600 to 1800 kcal/d for patients who were  
101 overweight before the TBI and 1800 to 2000 kcal/d for those who were not.

102

### 103 *Data collection*

104 Weight was noted at different time points: before the TBI (W1 i.e. usual weight recorded in  
105 previous medical files or noted during interviews with the patients or their relatives), at  
106 admission to, (W2) and discharge from (W3) the PMR center, and at the end (W4) of the  
107 follow-up period. BMI (body mass index) was calculated at each of these time points. The  
108 following potential explanatory factors were noted: age, sex, initial GCS score, duration of  
109 coma and stay in intensive care, level of physical activity according to the WHO (World  
110 health organization) criteria<sup>16</sup> and therapeutic classes of drugs taken by the patients.

111 Quantitative oral food intake was noted according to three categories which were relative to  
112 pre-TBI levels: lower, i.e. hypophagia (<1500 kcal/d), similar, and higher, i.e. hyperphagia  
113 (>2500 kcal/d). This was based on a dietary inquiry, verbal or visual analogue scales and  
114 eating behavior (number of meals per day, eating between meals, taste preferences (sweet or  
115 savory), binge eating and nocturnal eating) during the stay in PMR and following return  
116 home. No patients took topiramate<sup>17</sup> as an antiepileptic drug or were treated for a binge  
117 eating disorder. Oral food intake and eating behavior were determined during dietary inquiries  
118 with the patients and their families, at least twice in a 2-month period during a medical  
119 consultation or a phone call by a dietician and a physician. In addition, alcohol and tobacco  
120 abuse, presence or absence of addictions since TBI and before TBI, were recorded. The  
121 presence of a behavioral dysexecutive syndrome was scored dichotomously (yes/no) during  
122 the stay in PMR by an experienced neuropsychologist, based on the patient's performance on  
123 a standardized test<sup>18</sup>. Metabolic complications which were present before the TBI and any

124 occurring during the follow-up after TBI were recorded: arterial hypertension, diabetes  
125 mellitus or glucose intolerance and dyslipidemia.

126

### 127 *Statistics*

128 Data from all the patients included were used in the initial analysis. For this analysis, three  
129 classes of BMI were used: below 20 (underweight), 20 to 25 (normal), and above 25  
130 (overweight). Data were evaluated at four time points: W1: weight prior to TBI, W2:  
131 admission to PMR, W3: discharge from PMR and W4: end of the follow-up period. In a  
132 posthoc analysis, patients were categorized in three groups according to the amount of weight  
133 change at the end of the follow-up compared with pre-TBI weight: group 1 (weight loss),  
134 group 2 (weight stabilization:  $\pm 3\%$ ) or group 3 (weight gain). Student *t* tests and ANOVAs  
135 were used for quantitative data comparisons, and chi-squared tests for qualitative data. To  
136 study independent associated factors in relation to weight change, a logistic regression was  
137 used. SPSS software version 11.5 was used for the statistical analysis. Statistical significance  
138 was set at  $p < 0.05$ .

139



140 **Results**

141 During the period from 2004 to 2009, among the 280 patients admitted to our PMR center and  
142 classified as post-TBI, 107 patients met the inclusion criteria. Median duration of follow-up  
143 was 38 (8–66) months from the end of the intensive care period.

144

145 *Baseline*

146 Patient characteristics relating to the severity of TBI are listed in Tables 1 and 2.

147 Neurosurgical interventions were carried out in 28 patients (26%), mostly for compressive  
148 intracranial hematoma. A diffuse cerebral injury on the initial cerebral X-ray or MRI according  
149 to Marshall classification was found in 79 patients (74%) and was associated with a prefrontal  
150 lesion in 74 (69%) patients.

151

152 *End of follow up*

153 At the end of follow-up, 104 patients (97%) were discharged home and 3 (3%) were admitted  
154 to a specialized institution for patients with severe cognitive disorders. Fifty-three TBI  
155 patients (49%) had resumed work or school activities at the end of follow-up whereas 51  
156 (48%) were not able to return to a normal socio-professional life. Sixty eight patients (64%)  
157 were found to have a behavioral dysexecutive syndrome following assessment in the PMR  
158 department, including 12 (11%) with severe disinhibition. Only three patients had significant  
159 hypopituitarism, two had diabetes insipidus and took vasopressin (DDVAP) and one had a  
160 partial adenohypophysis deficiency that required hormonal supplementation.

161

162 *Weight changes*

163 W1 to W2:

164 All the patients received enteral nutrition (including 18 by gastrostomy) during intensive care  
165 and (or) neurosurgery, but still lost a mean  $11 \pm 6$  kg (IC 95%, 7.9–12) in 38 (7–364) days:  
166  $W1 = 71 \pm 13.5$  kg vs.  $W2 = 60 \pm 12.7$  kg ( $P < 0.001$ ).

167 W2 to W4:

168 All the patients were weaned from enteral nutrition, and from the gastrostomy, during their  
169 stay in PMR, once they recovered the ability to eat by mouth without any swallowing  
170 impairments. During the stay in PMR (W2 to W3), mean weight gain was  $8.8 \pm 7.8$  kg (IC 95  
171 % = 6.5–11.1,  $P < 0.001$ ). W3 was  $68.8 \pm 11.5$  kg and W2 was  $60 \pm 12.7$  kg, corresponding to  
172 a mean gain of 80% of weight lost during intensive care. This change occurred over  $3.9 \pm 3.4$   
173 months, the mean duration of stay in our PMR unit. Between the end of PMR (W3) and the  
174 end of follow-up (W4) there was a mean weight increase of  $3 \pm 6.6$  kg (IC 95% = 1.2–4.8)  
175 over a  $32 \pm 16.6$  month period.

176 W1 to W4:

177 Mean BMI at W4 ( $23.8 \pm 4.1$ ) was not different to W1 (pre-TBI) ( $23.3 \pm 3.9$ ) ( $P = 0.08$ ).  
178 However, we found that the patients could be categorized in three different groups at the end  
179 of the follow up: group 1, weight loss ( $n = 30$ , 28%:  $-8 \pm 7$  kg), group 2, weight stabilization  
180 ( $n = 32$ , 30%), and group 3, weight gain ( $n = 45$ , 42%:  $+9 \pm 6$  kg).

181

### 182 *Factors associated with changes in weight*

183 Changes in weight and BMI showed that patients with a lower initial BMI tended to become  
184 more overweight at the end of follow-up than patients with a higher initial BMI (Figure 1).

185 There was a statistically significant relationship between weight change at W4 and pre-TBI  
186 BMI class for each of the three groups ( $P = 0.015$ ): 54% of pre-TBI patients with a BMI  $> 25$   
187 lost weight during the follow-up compared to 14% with a BMI  $< 20$ . In addition, 59% of pre-  
188 TBI patients with a BMI  $< 20$ , and 25% of patients with a BMI  $> 25$  gained weight during the

189 follow-up. Thus the weight loss during intensive care reversed for all patients on entry to  
190 PMR (W2) and the weight time courses of each group crossed over during the stay in PMR  
191 (W2-W3) (see Figure 1).

192         There was a significant relationship between the presence of hypophagia or  
193 hyperphagia and W4 weight group ( $P = 0.001$ ). Patients in group 3 ate more than patients in  
194 the other two groups, with a 40% prevalence of hyperphagia compared with 16% and 10% in  
195 groups 1 and 2 respectively. In contrast, hypophagia was observed in 50% of group 1  
196 compared with 31% and 11% in groups 2 and 3 respectively. There was, however, no  
197 statistically significant difference between the three groups for eating behavior (Table 3). The  
198 prevalence of a behavioral dysexecutive syndrome was, however, more frequent in the weight  
199 gain group (Table 3). There was no significant difference between the severity of TBI,  
200 assessed by the initial type of cerebral lesions, the GCS and the duration of coma or intensive  
201 care, for any group. There were no significant differences in the number of drug prescriptions  
202 at discharge from PMR and during the follow-up between the three groups. Neither did the  
203 level of physical activity, sex, age, weight loss during intensive care, level of physical  
204 activity, or modification of alcohol or tobacco addiction differ significantly between the three  
205 groups.

206         The multivariate analysis included categories of oral food intake, behavioral  
207 dysexecutive syndrome and classes of initial BMI in the model. The results showed that the  
208 factors which were independently related to weight gain were hyperphagic food intake  
209 ( $P=0.003$ , OR 4.5 (IC 95%, 1.6–12.1) and the presence of a behavioral dysexecutive  
210 syndrome ( $P=0.04$ , OR 2.5 (IC 95%, 1.03-6.3). Two factors ( $P < 0.01$ ) were related to weight  
211 loss, hypophagic food intake (OR 4.1 (IC 95%, 1.5–10.9) and higher pre-TBI BMI (OR 4.9  
212 (IC 95%, 1.7–14.0).

213

214 *Metabolic complications*

215 Before the TBI, 15 patients (15%) had a pre-existing metabolic disease: 3 had type 2 diabetes,  
216 5 had arterial hypertension and 7 had dyslipidemias. The occurrence of new metabolic  
217 complications was 16% with no significant differences between the three weight change  
218 groups. During the post-TBI follow-up, 4 patients (4%) developed arterial hypertension, 3  
219 type 2 diabetes (3 %) and 9 (8 %) a dyslipidemia. In contrast, type 2 diabetes associated with  
220 hyperlipidemia resolved in one patient with a normalization of previous obesity.

221

**222 Discussion**

223           The results of this study showed that weight change during the rehabilitation period  
224 after TBI was correlated with level of oral food intake. Higher intakes (hyperphagia), and the  
225 presence of a behavioral dysexecutive syndrome, were associated with a higher weight gain  
226 than a low level of food intake (hypophagia), which was associated with lower weight gain or  
227 weight loss. The second, more paradoxical result, was that the patients with the lowest pre-  
228 TBI BMI had the highest BMI at the end of follow-up, while patients with the highest pre-  
229 TBI BMI had the lowest BMI at the end of follow-up. Patients could be categorized in three  
230 groups of weight change at the end of follow-up –loss, stabilization and gain. Only 3  
231 independent factors were associated with the time course of weight change: nutritional state  
232 before TBI, based on the BMI, level of food intake, which significantly changed in more than  
233 one half (57 out of 107) of the patients after TBI, and dysexecutive syndrome for the weight  
234 gain group. In contrast, type of medication, duration of intensive care, age, sex and physical  
235 activity were not related to the type of weight change. All patients lost weight during  
236 intensive care and gained weight during the stay in PMR. These findings suggest that the TBI  
237 is responsible for changes in weight through the modification of food intake such as  
238 hyperphagia and hypophagia, probably as a result of disruption of the appetite control  
239 network following the brain lesions. However, it is difficult to establish the direct impact of  
240 TBI on feeding behavior and weight. No significant association was found between the type  
241 of cerebral injury according to the initial CT scan, and changes in nutritional status and eating  
242 behavior at the subacute/chronic stage. However, this is not a surprising finding, as standard  
243 CT and/or MRI assessment at the acute stage lack sensitivity to detect diffuse traumatic  
244 axonal injury<sup>19</sup>. It was therefore very unlikely that subtle functional or structural lesions  
245 involving the hypothalamus, which could explain changes in eating habits, would be detected  
246 with these techniques. A more detailed exploration of morphological lesions could be useful

247 to gain a better understanding of behavioral changes, including interest in food after a brain  
248 injury. More sophisticated imaging techniques, such as functional magnetic resonance  
249 imaging or Diffusion Tensor Imaging may in the future help to understand the relationship  
250 between brain injuries, morphological lesions and eating behavior following TBI<sup>20</sup>.

251 It is well accepted that the brain, notably the hypothalamus and its complex network,  
252 plays a crucial role in the regulation of food intake<sup>11</sup>. It can therefore be expected that TBI  
253 will disrupt this regulation by altering the structures involved. Our study was the largest ever  
254 conducted in this field in a cohort of TBI patients of varying severity, with initial management  
255 in intensive care and secondary care in a specialized university center for TBI rehabilitation.  
256 To limit bias, we included only patients who were not in a neurovegetative state and who had  
257 not undergone previous digestive surgery that could disturb normal digestive physiology. We  
258 also controlled for potential associated factors, such as medication or physical activity in  
259 order to evaluate the specific effect of TBI on weight change. Given the low prevalence of  
260 hypopituitarism: 3% in our study, 5% in a multicenter study<sup>9</sup> and 2.5% in a cohort of children<sup>8</sup>,  
261 it is unlikely that this pathology plays a major role in weight change. It must, however, be  
262 noted that we did not carry out systematic hormonal examinations, as is considered to be  
263 optimal practice<sup>21</sup>. In addition, the results showed that there was no significant relationship  
264 between weight gain and the various drugs prescribed for these patients. Data regarding food  
265 intake and eating behavior were recorded at least twice in a 2-month period, through patient  
266 inquiries and confirmation by the family and relatives in the case of cognitive sequelae. No  
267 differences were found between the two inquiries, similarly to previous studies of alcohol  
268 consumption<sup>22</sup>. Most of the patients had recovered sufficient cognitive capacity to respond to  
269 the inquiry: for example 49% had resumed normal work or school activities by the end of the  
270 follow-up.

271 Weight change pattern after TBI was noteworthy: our results confirmed a constant  
272 significant weight loss in the intensive care unit of a mean 11 kg, despite the use of enteral  
273 nutrition. This is a well-known effect of severe post-traumatic aggression during the acute  
274 phase<sup>23</sup>. The goal of treatment is to maintain nitrogen balance but, more particularly, to  
275 control stress and reduce malnutrition through nutritional assistance. During their stay in  
276 PMR, patients regained an average of 8.8 kg, almost all of the weight lost, in an average time  
277 of  $3.9 \pm 3.4$  months, which is twice that spent in intensive care. This weight regain was  
278 comparable to that found by Brooke<sup>3</sup>, who evaluated the effect of nutritional status on  
279 functional outcome in 53 TBI patients. Their study describes a significant weight gain in  
280 patients during hospitalization in a rehabilitation unit, even exceeding their pre-morbid weight  
281 such that 60% of patients left PMR significantly overweight. Various factors probably  
282 contributed to the weight change during PMR. First of all, “intrinsic” factors related to the  
283 TBI *per se*, notably a dysexecutive syndrome which was present in 68 patients during the  
284 PMR hospitalization and was characterized by major cognitive disorders, including reduced  
285 inhibition which can lead to increased food intake<sup>24</sup>. This phenomenon is clinically similar to  
286 the genetic Prader-Willi syndrome<sup>25</sup>. Secondly, an “extrinsic” factor related to feeding  
287 management may have been involved. Because of the undernutrition during intensive care  
288 and the related potential complications such as bedsores, all the patients received  
289 hyperenergetic and hyperproteic diets, however no metabolic evaluations (for example with  
290 indirect calorimetry) were carried out to adapt the nutrition to each patient. Families also  
291 appear to have played a role in weight gain by overfeeding despite instructions from the  
292 dietician<sup>2</sup>. It appears that seeing their loved ones regain the capacity to eat and gain weight  
293 after a long period of artificial feeding and, for some, “wasting”, could be seen by relatives as  
294 part of the recovery process. These factors probably maintained and worsened disorders  
295 which were directly related to the TBI. This illustrates the complexity of nutritional

296 management after brain injury. A diet adapted to the metabolic level of each patient could  
297 help to improve the nutritional changes. To do this, the systematic assessment of metabolic  
298 and morphological clinico-biological status is necessary at admission to PMR. Resting energy  
299 expenditure and body composition could be evaluated by bioelectric impedance or DEXA  
300 according to available resources, for example. Unfortunately, this was not done in the present  
301 study. Indeed, the changes we observed in weight could be related to fat deposition in the  
302 phase of regaining weight.

303         This study has important clinical implications. TBI is an important cause of eating  
304 disorders, particularly in the subacute phase, i.e. during rehabilitation. The correlation of  
305 weight change with hyperphagia or hypophagia and dysexecutive syndrome, suggests that  
306 TBI strongly disturbs the central areas which control food intake and weight set-points. It is  
307 therefore necessary to forestall these disturbances through a better assessment of nutritional  
308 needs, tailored to each patient, depending on available resources and technical feasibility. It is  
309 important to keep a watchful eye throughout the medium- and long-term follow-up, since we  
310 found that 42% of patients significantly gained weight and 28% significantly lost weight over  
311 a median of 38 months. The metabolic consequences of this weight gain in the medium term  
312 were demonstrated in 16% of patients who developed arterial hypertension, dyslipidemia or  
313 type 2 diabetes, although their average age was only 36 years. This could be a significant  
314 problem. Monitoring in the longer term might reveal many more metabolic complications,  
315 and therefore the extended follow-up of these patients should include weight and metabolic  
316 management. The weight loss found in 28% of patients could also have significant somatic  
317 consequences, since chronic undernutrition can impair functional recovery<sup>26</sup>.

318



319 **Conclusion**

320 The results of this study showed that over 42% of adult TBI patients gained weight and 28%  
321 lost weight over a period of 38 months. The amount of weight change was related to the level  
322 of food intake and was strongly determined by behavioral changes. Initial weight was  
323 inversely related to weight at the end of follow up and the time course of weight change was  
324 independent from the amount of weight loss during intensive care. The results of this study  
325 therefore suggest that TBI strongly disturbs quantitative eating behavior and body weight set-  
326 points. A study of the long-term metabolic consequences appears justified since in this 38-  
327 month study, 16% of patients developed a metabolic syndrome. Future studies should  
328 evaluate the related changes in body composition, mainly fat free mass and fat mass.

329

330 **Conflict of interest**

331 The authors declared no potential conflicts of interest with respect to the authorship and/or  
332 publication of this article.

333

334 **Statement of authorship**

335 PC, SH, JCM and PA contributed to the study design, analysis and interpretation. SH, ABM  
336 and MH collected the data. The paper was written by PC, SH and PA and all the authors read  
337 and approved the final manuscript.

338

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399 **Table 1. Baseline characteristics of patients ( $n = 107$ ) before traumatic brain injury**

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Sex ratio: M/F	81/26
Age (mean $\pm$ SD)	36 $\pm$ 13
Weight (mean $\pm$ SD)	71 $\pm$ 13.5
BMI (mean $\pm$ SD)	23.3 $\pm$ 3.9
BMI classes:	
BMI < 20	22 (21%)
BMI between 20 and 25	61 (57%)
BMI > 25	24 (22%)
Metabolic disorders ( $n$ )	15 (15%)

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403 **Table 2. Criteria of severity of traumatic brain injury (*n* = 107)**

404

	<b>Mean ± SD or number (%)</b>	<b>Min</b>	<b>Max</b>
Initial Glasgow coma scale score (GCS)*	6.8 ± 3.1	2	15
Mild TBI (GCS 13–15)	8 (9)	13	15
Moderate TBI (GCS 9–12)	10 (10%)	9	12
Severe TBI (GCS < 9)	76 (81%)	3	8
Coma duration (days)	15 ± 10	0	68
ICU duration (days)	38 ± 40	2	364
Post-traumatic amnesia (days)	72 ± 89	2	510
Cerebral lesions**	Diffuse injury	n=79	
	Evacuated mass lesion	n=28	

405 \* For 94 patients in whom the GCS was documented

406 \*\* according to Marshall CT-scan classification

407 TBI: traumatic brain injury

408

409 **Table 3. Characteristics of patients ( $n = 107$ ) according to weight change groups at the**  
 410 **end of follow-up [38(8-66) months after traumatic brain injury]**

	Total	Weight loss	Weight stabilization	Weight gain	$p$ (between the 3 groups)
Patients ( $n$ )	107	30	32	45	
Sex ratio: M/F	81/26	20/10	26/6	35/10	NS
Age (yr)	36	38	37	33	NS
T1 BMI (pre-TBI)	$23.3 \pm 3.9$	$25.0 \pm 4.4$	$22.5 \pm 2.5$	$22.6 \pm 4.1$	$P = 0.014$
Intensive care duration (d)	$38 \pm 40$	$31 \pm 18$	$30 \pm 17$	$46 \pm 56$	NS
Coma duration (d)	$15 \pm 10$	$15 \pm 10$	$16 \pm 11$	$14 \pm 11$	NS
$\Delta$ BMI in intensive care	$-3.2 \pm 1.5$	$-3.3 \pm 1.6$	$-2.9 \pm 1.4$	$-2.8 \pm 1.4$	NS
T4 BMI (end of follow-up)	$23.8 \pm 4.1$	$22.4 \pm 4.0$	$22.7 \pm 2.6$	$25.4 \pm 4.5$	$P = 0.01$
Behavioral dysexecutive syndrome ( $n$ )					
	68	16	18	34	$P = 0.04$
Hyperphagia ( $n$ )*	26	5	3	18	$P = 0.004$
Hypophagia ( $n$ )**	31	15	10	6	$P = 0.003$
Number of meals $> 3/d$ ( $n$ )	15	3	3	9	$P = 0.09$
Eating between meals ( $n$ )	35	10	8	17	NS
Taste preferences sweet ( $n$ )	30	11	6	13	NS
Taste preferences savoury ( $n$ )	36	12	9	15	NS
Physical activity ( $n$ ) ***	36	8	13	15	NS
Drugs ( $n$ )					
neuroleptics	19	9	3	7	$P = 0.09$
antidepressants	38	13	9	16	NS
analgesics	45	15	11	19	NS
anticonvulsants	46	13	13	20	NS
Addictions: alcohol/tobacco ( $n$ )	4/17	2/5	0/4	2/8	NS
Onset of metabolic disorders (including dyslipidemia) ( $n$ )					
	16(9)	6(2)	2(2)	8(5)	NS

411 \* defined by an increase of usual food intake compared with pre-TBI (T1); \*\* defined by a  
 412 decrease in usual food ingestion compared with pre-TBI (T1)

413 \*\*\* According to WHO (World Health Organization)

414 TBI: traumatic brain injury

415

416 **Figure 1: Time course of body mass index in 107 adult traumatic brain injured patients**

417

418 P+: weight gain group. P-: weight loss group. P=: weight stabilization group.

419 T1: before traumatic brain injury. T2: end of intensive care. T3: discharge from rehabilitation.

420 T4: end of follow-up.

421 BMI = body mass index

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