

V. Florent · B. Gaudrat

Emotional Eating Is not What You Think It Is and Emotional Eating Scales Do not Measure What You Think They Measure

Bongers P, Jansen A (2016) *Front Psychol* [1]

In eating research, it is common practice to group people into different eater types, such as emotional, external and restrained eaters. This categorization is generally based on scores on self-report questionnaires. However, recent studies have started to raise questions about the validity of such questionnaires. In the realm of emotional eating, a considerable number of studies, both in the lab and in naturalistic settings, fail to demonstrate increased food intake in emotional situations in self-described emotional eaters. The current paper provides a review of experimental and naturalistic studies investigating the relationships between self-reported emotional eater status, mood, and food consumption. It is concluded that emotional eating scales lack predictive and discriminative validity; they cannot be assumed to measure accurately what they intend to measure, namely increased food intake in response to negative emotions. The review is followed by a discussion of alternative interpretations of emotional eating scores that have been suggested in the past few years, i.e., concerned eating, uncontrolled eating, a tendency to attribute overeating to negative affect, and cue-reactive eating.

V. Florent (✉)

Service de nutrition, Centre spécialisé obésité (CSO),
centre hospitalier d'Arras, 3, boulevard Besnier, CS 90006,
F-62022 Arras cedex, France
Unité Inserm U1172, centre de recherche Jean-Pierre-Aubert,
59000 Lille, France
e-mail : vincent.florent@ch-arras.fr

B. Gaudrat

Service de nutrition, Centre spécialisé obésité (CSO),
centre hospitalier d'Arras, 3, boulevard Besnier, CS 90006,
F-62022 Arras cedex, France
Laboratoire PSITEC EA4072, université Lille-III,
domaine du Pont-de-Bois, BP 60149,
F-59653 Villeneuve-d'Ascq cedex, France
e-mail : bulle.gaudrat@ch-arras.fr

Commentaires : *Cet article questionne la validité des mesures et du concept même d'alimentation émotionnelle fréquemment utilisé pour caractériser le comportement alimentaire des patients en situation d'obésité [2]. Une revue poussée de la littérature sur le sujet permet d'appréhender les biais méthodologiques et théoriques du concept d'alimentation émotionnelle. Les auteurs insistent sur le développement et la validation nécessaire de paradigmes expérimentaux implicites permettant d'éviter les écueils des mesures actuelles.*

Intuitive Eating Is Inversely Associated with Body Weight Status in the General Population-Based NutriNet-Santé Study

Camilleri GM, Mejean C, Bellisle F, et al (2016) *Obesity* [3]

Objective: To examine the relationship between Intuitive Eating (IE), which includes eating in response to hunger and satiety cues rather than emotional cues and without having forbidden foods, and weight status in a large sample of adults.

Methods: A total of 11,774 men and 40,389 women aged ≥ 18 years participating in the NutriNet-Santé cohort were included in this cross-sectional analysis. Self-reported weight and height were collected as well as IE levels using the validated French version of the Intuitive Eating Scale-2. The association between IE and weight status was assessed using multinomial logistic regression models.

Results: A higher IE score was strongly associated with lower odds of overweight or obesity in both men and women. The strongest associations were observed in women for both overweight [quartile 4 vs. 1 of IE: odds ratio, 95% confidence interval: (0.19, 0.17–0.20)] and obesity (0.09, 0.08–0.10). Associations in men were as follows: for overweight (0.43, 0.38–0.48) and obesity (0.14, 0.11–0.18).

Conclusions: IE is inversely associated with overweight and obesity which supports its importance. Although no causality can be inferred from the reported associations, these data suggest that IE might be relevant for obesity prevention and treatment.

Commentaires : *Cette étude issue de l'analyse de la base de données NutriNet-Santé confirme sur un large échantillon français les résultats déjà observés dans de nombreuses recherches [4] et qui ont donné lieu au développement de prises en charge préventives et curatives centrées sur l'alimentation intuitive plutôt que sur la restriction alimentaire [5,6], fût-elle flexible [7].*

Is it Time to Consider the “Food Use Disorder?”

Laurence JN (2017) *Appetite* [8]

In the contemporary milieu, the term “addiction” brings to mind issues of physical dependence, uncontrolled behavior, psychoactive substances, and disease. Thus, the use of the term “food addiction” which has become common in research on binge eating and obesity, suggests a disease state characterized by craving, compulsive eating and, possibly, the presence of food constituents with drug-like properties which weaken the will power to abstain from consumption. In this commentary, the case is made that, following the trends in substance use disorder terminology, adoption of “food use disorder” as a term for compulsive eating associated with subjective loss of control may foster continued research in this area without the connotations suggested by “food addiction”.

Commentaires : *L'auteur questionne le concept « d'addiction alimentaire » mettant en avant les inconvénients d'une telle terminologie (tant sur le plan nosographique, sociétal et des conséquences en termes de prise en charge) et les différences qui persistent entre les différentes formes de troubles du comportement alimentaire chez le patient obèse et le concept d'addiction dans sa définition historique. L'auteur propose d'introduire le concept de « trouble de l'usage alimentaire » permettant d'éviter les écueils associés au terme « addiction ».*

Are All Metabolically Healthy Individuals with Obesity at the Same Risk of Diabetes Onset?

Navarro-Gonzalez D, Sanchez-Inigo L, Fernandez-Montero A, et al (2016) *Obesity* [9]

Objective: To examine the risk of diabetes and the development of an unhealthy status according to metabolic health. To assess the effect of changes in metabolic health among participants with metabolically healthy obesity (MHO) on the risk of diabetes.

Methods: A total of 4,340 subjects were included. Unhealthy metabolic status was defined as having three or more risk factors of the Adult Treatment Panel-III criteria.

A Cox proportional-hazard analysis was conducted to estimate the hazard ratio (HR) of developing diabetes across the change in the metabolic status of subjects with MHO.

Results: After 40,622 person-years of follow-up, the risk of becoming unhealthy was 1.53 times higher for participants with MHO, compared with lean or overweight healthy subjects. A greater risk of diabetes was found in MHO, but it was attributable to those who progressed to an unhealthier status over time: HR of 4.78 (95% CI: 3.38–6.78). The combination of being metabolically unhealthy and obesity heightened the risk of diabetes: HR of 10.09 (95% CI: 4.82–21.55).

Conclusions: The increased risk of diabetes in MHO is attributed to the progression to an unhealthier state. “Healthy obesity” is not a permanent situation but a transitory state.

Commentaires : *Le concept d'obésité métaboliquement saine est maintenant bien connu. Plusieurs études ont pu démontrer que ces patients ne présentent pas de facteurs de risque cardiométabolique [10,11]. Les auteurs s'intéressent ici au risque de survenue de diabète de type 2 en fonction du statut métabolique de 4 340 sujets obèses d'une cohorte préexistante. Ils avancent que le risque de présenter un syndrome métabolique est 1,53 fois plus important chez les sujets métaboliquement sains obèses que métaboliquement sains non obèses ou en surpoids. De plus, les sujets obèses métaboliquement sains ont un risque accru de présenter un diabète de type 2 au cours des neuf années de suivi. La controverse reste entière !*

Gut Permeability Is Related to Body Weight, Fatty Liver Disease, and Insulin Resistance in Obese Individuals Undergoing Weight Reduction

Damms-Machado A, Louis S, Schnitzer A, et al (2017) *Am J Clin Nutr* [12]

Background: Obesity and associated metabolic disorders are related to impairments of the intestinal barrier.

Objective: We examined lactulose:mannitol (Lac:Man) permeability in obese individuals with and without liver steatosis undergoing a weight-reduction program to test whether an effective weight-loss program improves gut barrier function and whether obese patients with or without liver steatosis differ in this function.

Design: Twenty-seven adult, nondiabetic individuals [mean \pm SD body mass index (BMI; in kg/m²): 43.7 \pm 5.2; 78% with moderate or severe liver steatosis] were included in the follow-up intervention study ($N = 13$ by month 12). All patients reduced their weight to a mean \pm SD BMI of 36.4 \pm 5.1 within 12 mo. We assessed barrier functions by the oral Lac:Man and the fecal zonulin tests. Insulin resistance was assessed by the homeostatic model assessment

index (HOMA), and liver steatosis by sonography and the fatty liver index (FLI).

Results: The Lac:Man ratio and circulating interleukin (IL) 6 concentration decreased during intervention from 0.080 (95% CI: 0.073, 0.093) to 0.027 (95% CI: 0.024, 0.034; $P < 0.001$) and from 4.2 ± 1.4 to 2.8 ± 1.6 pg/ml ($P < 0.01$), respectively. At study start, the Lac:Man ratio was higher in patients with moderate or severe steatosis than in those without any steatosis ($P < 0.001$). The Lac:Man ratio tended to correlate with HOMA ($\rho = 0.55$, $P = 0.052$), which correlated with FLI ($\rho = 0.75$, $P < 0.01$). A multiple-regression analysis led to a final model explaining FLI best through BMI, waist circumference, and the Lac:Man ratio.

Conclusions: Intestinal permeability is increased in obese patients with steatosis compared with obese patients without. The increased permeability fell to within the previously reported normal range after weight reduction. The data suggest that a leaky gut barrier is linked with liver steatosis and could be a new target for future steatosis therapies. This trial was registered at clinicaltrials.gov as NCT01344525.

Commentaires : Le microbiote intestinal est aujourd'hui au cœur de l'actualité de recherche en nutrition. Ici, les auteurs s'intéressent non pas au microbiote lui-même, mais à l'impact de la perméabilité intestinale sur l'insulinorésistance et la stéatose hépatique chez des patients obèses, avant et au cours d'une perte de poids par suivi médical. Dans cette étude, il est mis en évidence sur 27 patients obèses sévères une perméabilité intestinale significativement accrue surtout si l'obésité est associée à une stéatose hépatique ou à un HOMA élevé, et de façon graduelle avec la sévérité de ceux-ci. De plus, après perte de poids, cette hyperperméabilité intestinale s'amende totalement. De quoi remettre en question les facteurs étiologiques des hépatopathies dysmétaboliques.

The Association of Change in Physical Activity and Body Weight in the Regulation of Total Energy Expenditure

Drenowatz C, Hill JO, Peters JC, et al (2017) Eur J Clin Nutr [13]

Background/Objectives: The limited success in addressing the current obesity epidemic reflects the insufficient understanding of the regulation of energy balance. The present study examines the longitudinal association of body weight with physical activity (PA), total daily energy expenditure (TDEE) and total daily energy intake (TDEI).

Subjects/Methods: A total of 195 adults (52% male) between 21 and 35 years of age with no intention for weight loss were followed over a 2-year period. Body weight, fat

mass and fat-free mass were measured every 3 months. Participants were stratified into three groups based on change in body weight using a 5% cutpoint. TDEE and time spent in different PA intensities were determined via a multisensor device at each measurement time. TDEI was calculated based on change in body composition and TDEE.

Results: At 2-year follow-up, 57% of the participants maintained weight, 14% lost weight and 29% gained weight. Average weight change was -6.9 ± 3.4 and 7.1 ± 3.6 kg in the weight-loss and weight-gain groups, respectively. Average TDEE and TDEI did not change significantly in any weight change group ($P > 0.16$). Moderate-to-vigorous PA, however, increased significantly in the weight-loss group (35 ± 49 min/day; $P < 0.01$) and decreased in the weight-gain group (-35 ± 46 min/day; $P < 0.01$).

Conclusions: Results of this observational study indicate an inverse association between body weight and PA to maintain a stable TDEE and allow for a stable TDEI over time. Sufficient PA levels, therefore, are an important contributor to weight loss maintenance. Eur J Clin Nutr advance online publication, 14 December 2016.

Commentaires : Dans cette étude réalisée sur 195 adultes sans velléité de perte de poids, les auteurs ont contrôlé tous les trois mois la dépense énergétique de repos par calorimétrie indirecte et la composition corporelle par DEXA, en parallèle d'une mesure de l'activité physique par accéléromètre. Ces données ont été comparées aux variations physiologiques du poids sur deux années consécutives. Ainsi, il est mis en évidence que malgré des variations, conséquences de poids au cours du suivi (± 14 kg), celles-ci ne sont pas accompagnées de variation significative de la dépense énergétique de repos malgré une nette modification de composition corporelle en DEXA. Les auteurs avancent que même si l'excédent d'activité physique est connu pour majorer la dépense énergétique à court terme, celui-ci s'accompagne d'une perte de poids à l'origine d'une réduction de dépense énergétique afin d'équilibrer l'homéostat énergétique sur le long terme. Ces résultats suggèrent qu'il faut prendre du recul sur l'interprétation des mesures calorimétriques.

Références

1. Bongers P, Jansen A (2016) Emotional eating is not what you think it is and emotional eating scales do not measure what you think they measure. *Front Psychol* 7:1932
2. Cardi V, Leppanen J, Treasure J (2015) The effects of negative and positive mood induction on eating behaviour: a meta-analysis of laboratory studies in the healthy population and eating and weight disorders. *Neurosci Biobehav Rev* 57:299–309
3. Camilleri GM, Mejean C, Bellisle F, et al (2016) Intuitive eating is inversely associated with body weight status in the general population-based NutriNet-Santé study. *Obesity (Silver Spring)* 24:1154–61

4. Van Dyke N, Drinkwater EJ (2014) Relationships between intuitive eating and health indicators: literature review. *Public Health Nutr* 17:1757–66
5. Humphrey L, Clifford D, Neyman Morris M (2015) Health at every size college course reduces dieting behaviors and improves intuitive eating, body esteem, and anti-fat attitudes. *J Nutr Educ Behav* 47:354–60e1
6. Bacon L, Stern JS, Van Loan MD, et al (2005) Size acceptance and intuitive eating improve health for obese, female chronic dieters. *J Am Diet Assoc* 105:929–36
7. Tylka TL, Calogero RM, Danielsdottir S (2015) Is intuitive eating the same as flexible dietary control? Their links to each other and well-being could provide an answer. *Appetite* 95:166–75
8. Nolan LJ (2017) Is it time to consider the “food use disorder?”. *Appetite* pii: S0195-6663(16)30862-5. doi: 10.1016/j.appet.2017.01.029. [Epub ahead of print]
9. Navarro-Gonzalez D, Sanchez-Inigo L, Fernandez-Montero A, et al (2016) Are all metabolically healthy individuals with obesity at the same risk of diabetes onset? *Obesity (Silver Spring)* 24:2615–23
10. Guo F, Garvey WT (2016) Cardiometabolic disease risk in metabolically healthy and unhealthy obesity: stability of metabolic health status in adults. *Obesity (Silver Spring)* 24:516–25
11. Meigs JB, Wilson PW, Fox CS, et al (2006) Body mass index, metabolic syndrome, and risk of type 2 diabetes or cardiovascular disease. *J Clin Endocrinol Metab* 91:2906–12
12. Damms-Machado A, Louis S, Schnitzer A, et al (2017) Gut permeability is related to body weight, fatty liver disease, and insulin resistance in obese individuals undergoing weight reduction. *Am J Clin Nutr* 105:127–35
13. Drenowatz C, Hill JO, Peters JC, et al (2017) The association of change in physical activity and body weight in the regulation of total energy expenditure. *Eur J Clin Nutr* 71:377–82