Could intermittent fasting diets increase diabetes risk?

EMBARGOED UNTIL 00.01 CEST SUNDAY 20 MAY 2018

Fasting every other day to lose weight impairs the action of sugar-regulating hormone, insulin, which may increase diabetes risk, according to data presented in Barcelona at the European Society of Endocrinology annual meeting, ECE 2018. These findings suggest that fasting-based diets may be associated with long-term health risks and careful consideration should be made before starting such weight loss programmes.

Type-2 diabetes is a growing global epidemic that is often attributed to poor diet and a sedentary lifestyle, so is closely linked to obesity. Blood sugar is partially regulated by the hormone insulin, which is produced by the pancreas, if insulin levels are too low, or the body becomes resistant to its effects, type-2 diabetes results and high blood sugar levels can cause serious health issues, including heart, kidney and eye damage. In addition to medical strategies used to treat type-2 diabetes, patients are also advised to make lifestyle and dietary changes to lose weight. Recently, intermittent fasting diets have gained general popularity for weight loss, however, evidence on their success has been contradictory and there is a lack of knowledge and some debate on their potentially harmful long-term health effects. Previous research has also shown that short-term fasting can produce molecules called free radicals, which are highly reactive chemicals that can cause damage to the body at a cellular and may be associated with impaired organ function, cancer risk and accelerated aging.

In order to investigate whether an intermittent fasting diet could also generate damaging free radicals, Ana Bonassa and colleagues, from the University of Sao Paulo in Brazil, examined the effects of fasting every other day on the body weight, free radical levels and insulin function of normal, adult rats, over a 3-month period. Although the rats' body weight and food intake decreased as expected over the study period, the amount of fat tissue in their abdomen actually increased. Furthermore, the cells of the pancreas that release insulin showed damage, with the presence of increased levels of free radicals and markers of insulin resistance were also detected.

Ana Bonassa comments, "This is the first study to show that, despite weight loss, intermittent fasting diets may actually damage the pancreas and affect insulin function in normal healthy individuals, which could lead to diabetes and serious health issues."

The researchers now plan to investigate how this diet impairs pancreas and insulin function. There are many conflicting reports on the benefits and disadvantages, and many different types of intermittent fasting diets. Although these data were obtained in normal weight rats with positive effects on weight gain and food intake, the results suggest that in the long-term harm may be caused and that more investigation is needed to assess how people may be affected, particularly those with existing metabolic issues.

Ana cautions, "We should consider that overweight or obese people who opt for intermittent fasting diets may already have insulin resistance, so although this diet may lead to early, rapid weight loss, in the long-term there could be potentially serious damaging effects to their health, such as the development of type-2 diabetes."

Abstract

605

Intermittent fasting for three months decreases pancreatic islet mass and increases insulin resistance in Wistar rats.

Ana Cláudia Munhoz Bonassa, Angelo Rafael Carpinelli
University of São Paulo, São Paulo, Brazil.

Introduction: It is known that fasting causes several physiological changes in the endocrine pancreas, such as insulin secretion, pancreatic islet metabolism and beta cells redox state. However, there is still no consensus about the effects of intermittent fasting (IF), a fad diet widespread by the media and adopted by individuals seeking rapid weight loss. In the present study, we sought to study the effects of the IF diet for three months in an animal model.

Methods: Thirty-day-old female Wistar rats were submitted to IF for three months. During this time body weight and food intake were recorded. After the treatment the animals were killed, and pancreatic islets, perigonadal white adipose tissue, extensor digitorum longus muscle tissue and liver were collected for different analyses.

Results: IF decreased body weight and food intake. The stomach was greatly increased in size. There was an increase in adipose tissue and a decrease in muscle tissue. IF caused elevation of plasmatic insulin levels, both baseline and after glucose administration. *In vitro*, IF pancreatic islets had increased insulin secretion, glucose metabolism and net reactive oxygen species production, while decreased their mass. In addition, impairment in AKT phosphorylation was observed in peripheral tissues indicating insulin resistance.

Discussion: Previous studies showed an increase in orexigenic neurotransmitters production in IF, inducing hunger and hyperphagia in the *ad libitum* feeding days. Our experiments demonstrate that, despite the weight loss, IF treatment induces undesirable effects on tissue homeostasis. Therefore, the hyperinsulinemia registered *in vivo* and *in vitro*, associated with the impairment of glucose tolerance and the decrease in AKT phosphorylation, make clear the occurrence of peripheral insulin resistance. The increased metabolism of pancreatic islets dispersed cells, after IF treatment, indorses the higher insulin secretion. Furthermore, the decrease in the pancreatic islet mass indicates that three months of IF treatment cause severe impairment in glucose homeostasis. In conclusion, intermittent fasting diet may not be healthy to be adopted by individuals seeking rapid weight loss.

Notes for Editors

1. The study “Intermittent fasting for three months decreases pancreatic islet mass and increases insulin resistance in Wistar rats” is a poster presentation at the European Congress of Endocrinology at the Centre Convencions Internacional Barcelona, Spain.

2. For other press enquiries please contact the ECE 2018 press office:

Aida de Heras
Communications Executive
European Society of Endocrinology
Tel: (+44) (0)1454 642 206
Mob: (+44) (0)7876824027
Email: aida.heras@bioscientifica.com

3. The European Congress of Endocrinology is held at Centre Convencions Internacional Barcelona, Spain on the 19-22 May 2018. See the full scientific programme.

4. The European Society of Endocrinology was created to promote research, education and clinical practice in endocrinology by the organisation of conferences, training courses and publications, by raising public awareness, liaison with national and international legislators, and by any other appropriate means.