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Research report

Influence of depression on cardiometabolic responses to a lifestyle intervention in at-risk individuals

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ABSTRACT

Background: Cardiometabolic diseases and depression are public health problems that are often related. The benefits of behavioral interventions on lifestyle are well documented. However, the influence of depression in these interventions is unclear.
Objective: To evaluate whether depression affects the impact of a lifestyle intervention on cardiometabolic response in an at-risk sample.
Methods: 129 individuals identified by the public health system to be at risk for cardiometabolic disease were allocated to 18-month interventions on diet and physical activity. Socio-demographic and clinical data were obtained. Depressive symptoms were assessed by the Beck Depression Inventory. Changes by at least 10% in each of 6 cardiometabolic risk factors were used to define responses to intervention. Logistic regression models were employed for each gender.
Results: Approximately 42% of individuals had depressive symptoms. They had higher adiposity, cholesterol, and blood pressure levels and lower quality of life and physical activity levels than non-depressed individuals. In adjusted models, only women with depression at baseline had lower chance of improving plasma glucose (OR: 0.32) and lower chance of improving mean blood pressure (OR: 0.29) after the follow-up, compared with non-depressed women.
Limitations: The small sample size may have diminished the power of the results and the instrument used to measure depression does not provide clinical diagnosis according to DSM criteria.
Conclusion: Depression at baseline of lifestyle interventions predicted a lower chance of improving long-term cardiometabolic risk, particularly in women, suggesting that screening and management of depression as part of lifestyle interventions can potentially improve cardiometabolic risk profile.

1. Introduction

Depression may affect quality of life (QoL) including social relationships, occupational productivity, and general well-being. Prospective studies have shown that even subclinical depression can impair health and interfere in daily living activities (Lecrubier, 2000; Spitzer et al., 1994). The worldwide prevalence rates for depression ranges from 0.8% to 9.6%, affecting a greater proportion of women than men (Demyttenaere et al., 2004; Simon, 2000). By 2030, depression is expected to be the leading cause of disease burden worldwide (Chisholm, 2006). Disease severity is inversely proportional to QoL (Strine et al., 2009) and directly linked to disability. Even mild depression reduces QoL and productivity this highlighting the importance of depression management (Lecrubier, 2000; Spitzer et al., 1994).

A substantial number of depressed patients are not appropriately diagnosed and treated despite frequently accessing primary health care services (Kessler et al., 2005; McQuaid et al., 1999). Their risk of coronary heart disease is high (Whang et al., 2009) and mortality rates are 1.8-fold greater than in the general population (Chang et al., 2010).

As a result of economic and technological progress, a number of lifestyle modifications among general population have taken place. These changes have contributed to an increase in cardiometabolic disturbances. It is generally accepted that changes in dietary habits and physical activity can reduce the risk associated with cardiometabolic diseases (Alberti et al., 2007; Tuomilehto et al., 2001). Difficulties of adherence to a healthy lifestyle may be partially explained by the presence of psychological disorders, such as depression. Associations between depression and cardiometabolic diseases such as obesity (Simon et al., 2008), type 2 diabetes

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mellitus (T2DM) (Renn et al., 2011; Gois et al., 2012a, 2011) and others have been described. One in four individuals with T2DM has suffered from depression during their lifetime (Pouwer et al., 2006). Depressed individuals are 2.4 times more likely to develop metabolic syndrome than non-depressed individuals (Kahl et al., 2012). Coexistence of diseases increases morbidity, health care costs, and mortality (Bogner et al., 2007; Katon et al., 2009).

Inflammation could be the underlying mechanism involved with both depression and cardiometabolic diseases (Valkanova et al., 2013). Depression-induced activation of the hypothalamic–pituitary–adrenal axis increases cortisol secretion and enhances cortisol concentration. This contributes to the development of a pro-inflammatory state increasing abdominal obesity and insulin resistance, which in turn increases the risk of cardiometabolic diseases.

A lifestyle intervention for the prevention of T2DM was shown to improve emotional and physical aspects of QoL (Cezaretto et al., 2012). In addition, depressive symptoms decreased during follow-up, and individuals with reduced depression scores had the most metabolic benefit. However, whether the presence of depression at baseline of intervention programs influences cardiometabolic profile is unclear. This study evaluated whether depression affects the impact of a lifestyle intervention on cardiometabolic profile in a sample of the Brazilian population.

2. Material and methods

A total of 438 individuals aged 21 to 79 years, treated under the public health system of the São Paulo city, Brazil, between 2008 and 2010, were screened for T2DM using a locally developed questionnaire aiming to develop a prevention-based program. Those individuals who were found to be at risk were invited to a clinical examination and laboratory tests including a 75-g oral glucose tolerance test. Those with prediabetes (fasting glycemia between 100 and 125 mg/dL or 2-h glucose between 140 and 200 mg/dL) were invited to participate in the 18-month lifestyle intervention for diabetes prevention (www.fsp.usp.br/prevsm). Individuals with metabolic syndrome were considered for this study, since the presence of the condition has been associated with a 4.6-fold greater risk of T2DM (Ford et al., 2008; Gami et al., 2007). Individuals with a medical history of neurological or severe psychiatric disturbances, thyroid, liver, renal or infectious diseases were excluded. The Institutional Ethics Committee approved the study and written consent was obtained from all participants. This trial was registered (RBR #65N292) in the Brazilian registry center of the World Health Organization International Clinical Trials Registry Platform (www.ensaiosclinicos.gov.br).

For this study, 230 individuals were eligible and 183 agreed to participate in one of the two types of 18-month interventions on lifestyle. Ninety seven subjects were allocated to the interdisciplinary intervention and 86 to the traditional intervention (de Barros et al., 2013). Among those who refused to participate, there was a predominance of men; however, non-participants did not differ from participants in terms of baseline socio-demographic, anthropometric, metabolic variables, or average depression scores. The reasons for refusals were related to distance and timing as the intervention occurred during business hours. Of the 183 individuals enrolled, 129 completed the intervention (Fig. 1). Individuals who dropped out during the follow-up were younger than those who remained for the whole period (50.6 SD 12.9 *vs.* 56.3 SD 11.8 years, $p=0.006$).

The methodology of the two intervention plans was previously described elsewhere (Cezaretto et al., 2012; Siqueira-Catania et al., 2013). Briefly, the traditional intervention consisted of quarterly medical visits with an endocrinologist, in which participants received usual written guidelines for changing diet and physical activity, advocated by the Brazilian public health system. In addition to medical visits, the interdisciplinary intervention with the psychoeducational approach included an individual appointment with a nutritionist and 16 group sessions conducted by a multiprofessional team (endocrinologist, psychologist, nutritionist, and physical educator). The present analysis is based on a sample pooled from both the intervention groups. Results comparing clinical, behavioral, and psychological data between interventions have been described elsewhere (Cezaretto et al., 2012; Siqueira-Catania et al., 2013).

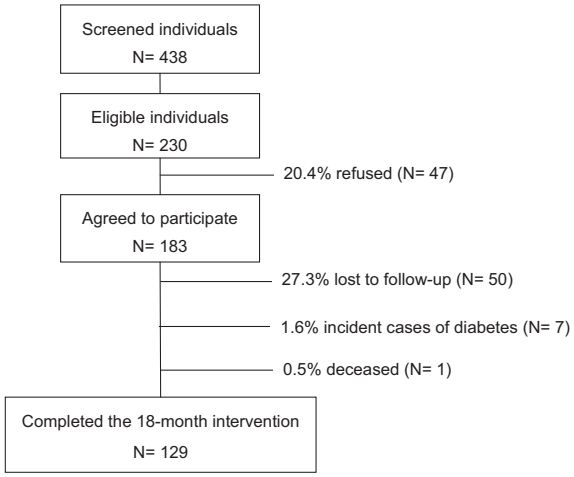

Fig. 1. Flowchart of individuals at each stage of the study.

Standardized questionnaires were used for studying socio-economic and general health status. Physical activity level was evaluated by utilizing long version of the International Physical Activity Questionnaire (Craig et al., 2013). Dietary habits were assessed using three 24-h recalls (two weekdays and one weekend day) and data was analyzed by using the Nutrition Data System for Research software (Nutrition Coordinating Center (NCC), 2005). Depression symptoms were assessed using the Beck Depression Inventory (BDI), of which depression scores ranged from between 0 and 63 (Beck et al., 1988) BDI scores greater than or equal to 12 were interpreted as indicating the presence of depression. QoL was assessed by the Medical Outcome Study 36-Item Short-Form Health Survey (Ware et al., 1998). This questionnaire includes eight QoL domains (physical function, physical role, bodily pain, general health, vitality, social functioning, emotional role, and mental health). The scores obtained were combined to calculate the SF-36 physical and mental component summaries. These health scales are scored from 0 to 100, indicating worse to better QoL, respectively (Ware et al., 1998).

Biochemical tests (*i.e.* plasma glucose, lipid profile, and adiponectin, C-reactive protein—CRP, and TNF-α) in conjunction with clinical examinations were performed at the study baseline and after 18 months of follow-up. Height was measured using a fixed stadiometer and weight was taken with individuals wearing light clothes and no footwear on a Filizola digital scale. Waist circumference was measured at the midpoint between the bottom of the rib cage and above the top of the iliac crest during minimal respiration. Blood pressure (BP) was measured at rest in a sitting position, three times with a five minute interval, using an automatic blood pressure device (Omron HEM-712C, Omron Health Care, USA). The average of the last two measurements was used in the analysis. Mean arterial blood pressure was calculated by the formula: "Systolic BP + (2 × Diastolic BP)/3".

2.1. Statistical analysis

Mean and standard deviation (SD) were reported for continuous variables including; depression scores, QoL, dietary intake,