Effects of educational and counselling intervention on daily physical activity and prevention of cardiovascular risk factors, in primary health care

Ana Isabel Valongo Ramôa de Castro
Porto, 2016

KEYWORDS: physical activity, cardiovascular risk, regulation for exercise, primary care
The European Regional Development Fund through the Operational Competitiveness Programme (FCOMP-01-0124-FEDER- 020180), and the Portuguese Foundation for Science and Technology (FCT) granted the study encompassing this Doctoral thesis and the research unit CIAFEL within the projects reference PTDC/DES/122763/2010 and UID/DTP/00617/2013, respectively.
DEDICATORY

I wish to dedicate this manuscript to my parents Celina Ramôa e Emanuel Castro who always supported and inspired me in all my projects.
Acknowledgements

This doctoral journey has been one of the hardest challenges I had to face in my entire life.
I’m grateful to my academic supervisors, José Oliveira and Fernando Ribeiro. Thanks for your help, availability, encouragement and the time you invested in helping me.
I’m grateful to all the colleagues and professionals of USF Espaço Saude. Thank you for your patience whenever this project collided with your work and time, for all the availability and for the strange persons in the health center.
To Helena and Lucimére that made part of this project. To Ana Alves e Silvia Camões for all the help and patience as well.
To Cristina Sousa, you have been an encouragement to finish my PhD.
To Anelise Gaya, that made me start this journey.
To Gustavo, thanks for your help with statistics every time I needed.
To everyone that participated directly or indirectly in this work.
To all my family that supported me in my hardest moments, particularly Michel, with patience through what has been an emotional roller coaster over the past few years. Thank you for being there for me every step of the way during this five years, between my health unit work, PhD work, two pregnancies and the specialty degree.
To my daughters Ana Maria e Mariana. You are my world!
# TABLE OF CONTENTS

FUNDING .................................................................................................................. IV  
DEDICATORY.................................................................................................................. V  
Acknowledgements....................................................................................................... VII  
TABLE OF CONTENTS.................................................................................................. IX  
LIST OF FIGURES ......................................................................................................... X  
LIST OF TABLES ........................................................................................................... XI  
ABSTRACT .................................................................................................................... XIII  
RESUMO ...................................................................................................................... XV  
List of abbreviations...................................................................................................... XVII  

Chapter I: Introduction .................................................................................................. 1  
Aims and Structure of the Thesis .................................................................................... 7  

Chapter II: Literature Review ...................................................................................... 9  
Chapter II: Literature Review ....................................................................................... 11  
ABSTRACT .................................................................................................................. 11  
INTRODUCTION ......................................................................................................... 13  
METHODS .................................................................................................................. 14  
RESULTS ..................................................................................................................... 16  
DISCUSSION ............................................................................................................... 22  
CONCLUSION .............................................................................................................. 24  

Chapter III: Intervention Study ................................................................................... 25  
STUDY AIMS .............................................................................................................. 27  
METHODS .................................................................................................................. 27  
INTERVENTION: EDUCATIONAL AND COUNSELLING PROGRAM OF HEALTHY  
LIFESTYLES .................................................................................................................. 28  
MEASUREMENTS .......................................................................................................... 29  
Daily physical activity ................................................................................................... 29  
Regulation for exercise ................................................................................................. 30  
Resting Blood Pressure Measurements ....................................................................... 31  
Blood collection and biochemical analysis ................................................................... 31  
Dietary intake ............................................................................................................... 32  
RESULTS ..................................................................................................................... 34  
*Lipid profile, metabolic parameters and inflammatory biomarkers* ............................. 40  
DISCUSSION ............................................................................................................... 42  

Chapter IV: Conclusions ............................................................................................. 53  
References .................................................................................................................... 57  
REFERENCES .............................................................................................................. 59  

Appendix ...................................................................................................................... I  
Appendix I: Patient file ................................................................................................ III  
Appendix II: Informal Consent ....................................................................................... V  
Appendix III: Booklet ..................................................................................................... VII  
Appendix IV: BREQ-2 Questionnaire ........................................................................... XIII  
Appendix V: 4-Day Food Recall Questionnaire ............................................................ XV  
Appendix VI: List of the conference communications and journal papers related with this  
work .............................................................................................................................. XXI
LIST OF FIGURES

FIGURE 1 - STUDY IDENTIFICATION AND SELECTION FLOW DIAGRAM................................................................. 15
FIGURE 2 - FLOW DIAGRAM OF PARTICIPANT THROUGH THE STUDY.......................................................... 34
LIST OF TABLES

Table 1 - Studies of the effects of educational intervention in physical activity ........................................... 18
Table 2 - Frequency of cardiovascular risk factors at baseline in the entire sample, and within the two groups ................................................................................................................................. 36
Table 3 - Changes in anthropometrics [Data are mean (SD) or median (25th - 75th percentile)] .......... 37
Table 4 - Data for daily physical activity levels [Data are mean (SD) or median (25th - 75th percentile)] ............................................................................................................................................... 38
Table 5 - Changes in motivation [Data are mean (SD) or median (25th - 75th percentile)] ................. 39
Table 6 - Changes in resting blood pressure [Data are mean (SD) or median (25th - 75th percentile)] .... 39
Table 7 - Changes in lipids, metabolic parameters, and inflammatory biomarkers in the study groups [Data are mean (SD) or median (25th - 75th percentile)] ........................................... 41
ABSTRACT

Purposes: review systematically the impact of educational interventions on primary prevention settings to promote physical activity and evaluate the effects of an educational and counselling intervention on changes in physical activity, regulation for exercise, and cardiometabolic risk factors, and biomarkers of inflammation.

Review: Methods: A computer-aided search on PubMed and Scopus was performed to identify relevant studies published from January 2000 to November 2014. Two authors independently selected studies for inclusion and extracted data, including intervention characteristics and outcome measures, namely physical activity and cardiovascular risk or risk factors. Results: Of the 151 identified studies, 14 met the inclusion criteria. The 14 studies enrolled 7713 participants; the sample size varied between 136 and 878 adults. Thirteen studies assessed physical activity by questionnaire and only one study used accelerometry. Eight of the 14 studies showed improvements in the physical activity levels after the intervention. The majority of the studies reported significant positive effects of the educational interventions on cardiovascular risk factors, mainly on lipid profile, blood pressure and cardiovascular risk score. Conclusion: The educational interventions, in primary care, seem to improve daily physical activity, cardiovascular risk factors and risk score.

Intervention: Methods: one hundred thirty-five patients participated in this study with the inclusion criteria: i) to be registered in the primary health care unit clinical file database; ii) to be aged between 18 and 65 years old; and iii) to have the presence of at least one cardiovascular risk factor. Patients were assigned to an intervention group (age: 55.7 ± 7.6 years) or to a control group (age: 56.9 ± 6.6 years). The intervention group participated in an educational program of healthy lifestyle lasted 5-month and included educational group sessions, information material, text messages to reinforce the information material, and demonstration sessions about types of leisure physical activities whereas the control group received usual medical care. At baseline and after 5 months the following parameters were assessed: anthropometrics (weight, height and waist circumference), daily physical activity, regulation for exercise, resting blood pressure, lipids and metabolic parameters (lipid profile, Glucose, Insulin, HbA1c) and inflammatory biomarkers (high-sensitivity C-reactive protein (hsCRP), Interleukin-6, Tumour Necrosis Factor-alpha, leptin and adiponectin) and dietary intake. Body mass index, Homeostasis model assessment (HOMA) and quantitative insulin-sensitivity check index (QUICKI) were calculated. Results: The main findings of the present study indicate that the intervention did not promote significant changes in physical activity, lipids and metabolic parameters and inflammatory biomarkers, with the exception of waist circumference (P=0.006) as well as amotivation (P=0.002) and intrinsic regulation (p<0.001) in the intervention group when compared to control group. Conclusions: Intervention group did not have effects in physical activity, cardiometabolic risk factors, and biomarkers of inflammation. Intervention group had important repercussions on regulation for exercise.

KEYWORDS: physical activity, cardiovascular risk, regulation for exercise, primary care
RESUMO

Objetivos: Revisão sistemática acerca do impacto da intervenção educacional para promover a prática de atividade física e avaliar o efeito de uma intervenção educacional e aconselhamento em estilos de vida saudáveis, com aumento da atividade física, motivação para o exercício, fatores de risco cardiometabólicos e biomarcadores inflamatórios.

Revisão: Métodos: foi realizada uma pesquisa bibliográfica na PubMed e Scopus de maneira a identificar estudos relevantes publicados de Janeiro de 2000 a Novembro de 2014. Dois autores selecionaram independentemente estudos que cumpriam os critérios de inclusão, incluindo características da intervenção e medidas de avaliação, nomeadamente da atividade física, risco cardiovascular e fatores de risco cardiovasculares. Resultados: dos 151 estudos identificados, 14 cumpriram os critérios de inclusão. Os 14 estudos incluíram 7713 participantes; as amostras variavam entre 136 a 878 adultos. Treze dos estudos mediaram a atividade física por questionário e apenas 1 usou acelerometria. Oito dos 14 estudos mostraram aumento dos níveis de atividade física após intervenção. A maioria dos estudos reportaram efeitos positivos e significativos das intervenções educacionais nos fatores de risco cardiovasculares, nomeadamente no perfil lipídico, tensão arterial e score de risco cardiovascular. Conclusão: As intervenções educacionais nos cuidados de saúde primários parecem aumentar a atividade física diária, diminuir fatores de risco cardiovascular e score de risco cardiovascular.

Intervenção: Métodos: participaram no estudo 135 pacientes com os seguintes critérios de inclusão: inscritos na unidade de saúde familiar, idade entre 18 a 65 anos e pelo menos um fator de risco cardiovascular. Os participantes foram divididos em dois grupos, grupo de intervenção (idade: 55.7±7.6 anos) e controlo (idade: 56.9±6.6 anos). O grupo de exercício participou num programa de educação e aconselhamento de 5 meses, composto por 3 sessões de grupo, material informativo, mensagens telefónicas para reforço da informação, assim como sessões de demonstração de atividade física enquanto o grupo controlo recebeu o seguimento médico habitual. Foram medidos os seguintes parâmetros no momento de início do programa e aos 5 meses: Antropométricos (peso, altura), atividade física diária, motivação para o exercício, tensão arterial em repouso, estudo analítico (perfil lipídico, HbA1c, Proteína C reativa de alta sensibilidade (hsCRP), interleucina-6 (IL-6), fator de necrose tumoral-alfa (hsTNF-α), leptina e adiponectina e ingestão alimentar diária. Resultados: as principais conclusões deste estudo mostram que a intervenção não teve influência na atividade física, função lipídica, parâmetros metabólicos e biomarcadores inflamatórios, com a exceção do perímetro abdominal (P=0.006) assim como na amotivação (P=0.002) e regulação intrínseca (P<0.001) em comparação com o grupo controlo. Conclusões: o grupo de intervenção não teve efeitos na atividade física, fatores de risco cardiometabólicos e biomarcadores inflamatórios em comparação com o grupo controlo. O grupo de intervenção teve efeitos importantes na regulação para o exercício.

PALAVRAS-CHAVE: atividade física, risco cardiovascular, motivação para o exercício, prevenção primária
List of abbreviations

ACE-inhibitors: angiotensin converting enzyme inhibitors;
ARBs: angiotensin II receptor blockers
BMI: body mass index
BREQ 2: The Behavioural Regulation in Exercise Questionnaire
CAD: coronary artery disease
CG: control group
CHAMPS: community Healthy Activities Model Program for seniors
CRP: c-reactive protein
CVD: cardiovascular diseases
CVR: cardiovascular Risk
DBP: diastolic blood pressure
DECODE: Diabetes epidemiology: collaborative analysis of diagnostic criteria in Europe
DGS: General Health Direction
F: Female
HDL: high-density lipoprotein
HEARTSCORE: european cardiovascular estimation risk system
hs: high-sensitivity
IG: interventional group
IL-6: interleukin-6
IPAQ: International Physical Activity Questionnaire
M: male
MBP: mean blood pressure
MVPA: moderate to vigorous physical activity
NHANES III: Third National Health and Nutrition Examination Survey
OECD: organisation for economic co-operation and development
PA: physical activity
PAI: plasminogen activator inhibitor
PEDro scale: Physiotherapy evidence database
QUICKI: quantitative insulin-sensitivity check index
SBP: systolic blood pressure
SD: standard deviation
SDT: self-determination theory
SQUASH: short questionnaire to assess health-enhancing physical activity
T2DM: type 2 diabetes mellitus
TG: triglycerides
TNF-α: tumour necrosis factor alpha
VLDL: very low density lipoprotein
WC: waist circumference
Chapter I: Introduction
Introduction

Cardiovascular diseases (CVD) still remain the main cause of premature mortality, accounting for 33% of all deaths in OECD countries (OECD). It is recognized by public health agencies that a substantial proportion of these deaths (46%) occurred in people younger than 70 years, which is the most productive period of life. In Portugal, a survey report of Portuguese General Health Direction (DGS), in 2013, indicate that 30% of total deaths and 16% of premature deaths (before 70 years) are due to cardiovascular diseases (Bordalo A, 2015).

The Framingham Heart Study (Kannel et al., 1976) created the concept and term cardiovascular risk factor based on evidence from observational (Ragland & Brand, 1988; "Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: final report of the pooling project. The pooling project research group", 1978) and intervention (Hjermann et al., 1981; "Multiple risk factor intervention trial. Risk factor changes and mortality results. Multiple Risk Factor Intervention Trial Research Group", 1982) studies. From those studies it was established that those non-modifiable factors (age, sex, familiar history of CVD) and modifiable risk factors (related to lifestyle and environment) predispose to early sub-clinical initiation and progression of atherosclerosis and later on to the clinical manifestation of signs and symptoms of coronary artery disease (CAD). Indeed, prolonged exposure to risk factors like smoking, sedentariness, high blood pressure, hypercholesterolemia, overweight and obesity, and diabetes mellitus increase the risk of CVD, including CAD, which makes the early identification and diagnosis important from an epidemiological perspective, but also from a clinical point of view in the primary health care setting. Moreover, the INTERHEART study showed that nine modifiable risk factors (smoking, low levels of physical activity, diet, obesity, lipids, hypertension, diabetes, alcohol consumption, and psychosocial factors) account for over 90% of the risk to first acute myocardial infarction (Yusuf et al., 2004). According to a survey report published by DGS in 2015, the prevalence of risk factors was 25% for smoking, 46% of for physical inactivity, 14% for obesity and 54% for overweight, 47% for hypercholesterolemia, 42% for hypertension, and 13% for diabetes (Bordalo A, 2015).
It is well documented that cardiovascular risk factors tend to cluster. A good example is the metabolic syndrome that consists in the coexistence in the same individual of multiple risk factors (Reaven). This clustering seems to directly promote the initiation and progression of atherosclerosis and clinical CVD (Mottillo et al.). The following are considered metabolic risk factors: atherogenic dyslipidaemia (increased serum triglycerides and apolipoprotein B, decreased concentration of HDL cholesterol), hypertension, increased fasting glucose, pro-thrombotic and pro-inflammatory state (Graham et al., 2007). These factors are strongly associated with diabetes mellitus and/or to the risk of its development. Currently it is not clear that metabolic syndrome has a single origin and may be triggered by multiple underlying risk factors such as obesity and insulin resistance (Graham et al., 2007). The study NHANES III (Third National Health and Nutrition Examination Survey) ("Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report", 2002) estimated that the age-adjusted prevalence of metabolic syndrome (according the criteria of NCEP ATP III) is 23.7%, although the existence of a significant variation according ethnicity, being more prevalent in Hispanics (31.9 %) than in Caucasians (23.8%) and African-Americans (21.6%) (Hu et al., 2004). The overall prevalence was similar in both genders, but the sub-analysis by ethnic group revealed predominance in men among Caucasians (men: 24.8%; women: 22.8%) predominance in women within African-Americans (women: 25.7%; men: 16.4%) and Hispanics (women: 35.6%, men: 28.3%) (Hu et al., 2004).

In Europe, based on the DECODE study group, the overall prevalence of the metabolic syndrome in European non-diabetic adults is 15%, being the age-standardized prevalence slightly higher in men (15.7%) than in women (14.2%) (Hu et al., 2004). In Portugal, three studies were conducted (Correia F et al., 2004; Correia et al., 2006; Santos et al., 2004), reporting the prevalence of metabolic syndrome, which ranges from 32.9% to 70.3%. The Portuguese studies, in general, enrolled small samples (Correia F et al., 2004; Correia et al., 2006), which may explain the contrasting findings in our country compared to with the European reality. Thus, those studies could give an imprecise picture of the Portuguese reality.
Atherosclerosis, the pathology underlying many cardiovascular diseases, is multifactorial in nature (genetic, environmental, dietetic, metabolic, hemodynamic and inflammatory). Furthermore, this risk factors interact in an exponential form (DGS, 2013). Inflammation contributes and is present in all stages of atherosclerosis, from lesion initiation to progression and plaque formation and destabilization. In addition, inflammation regulates thrombotic potential in the plaque itself and the pro-thrombotic and anti-fibrinolytic capacity of blood in the fluid phase (Libby & Theroux, 2005). The threatening presence of inflammation in atherosclerosis has prompted the evaluation of certain key inflammatory factors in cardiovascular risk prediction (Packard & Libby, 2008). The experimental and clinical evidence shows that prolonged exposure to cardiovascular and metabolic risk factors induces vascular changes, which can be characterized by endothelial dysfunction (increased expression of adhesion molecules, diminished synthesis and bioavailability of endothelial dependent vasodilators such as, for instance, nitric oxide), low grade chronic inflammation with elevated circulating levels of pro-inflammatory of cytokines such as interleukin-6 (IL-6) and tumour necrosis factor alpha (TNF-α) and acute phase proteins (e.g. C-reactive protein), a pro-thrombotic state - with increased concentration of pro-coagulant (e.g. fibrinogen and factor VII) and a anti-fibrinolytic phenotype (e.g., plasminogen activator inhibitor PAI-1) (Danesh et al., 2004; de Lemos & Lloyd-Jones, 2008). Based on this evidence, several biomarkers of inflammation has been studied as predictors of cardiovascular events, with the results suggesting that the predictive diagnostic value may increase by the evaluation of these emergent risk factors (Ribeiro et al., 2009).

The increase of pro-inflammatory cytokines in circulation, including IL-6, TNF-α can also be derived from adipocytes further stimulating the hepatic synthesis of CRP (Ribeiro et al., 2009). In obesity, plasmatic levels of IL-6 and TNF-α are elevated and predict CVD morbidity (Libby & Theroux, 2005). C-reactive protein is a systemic inflammatory biomarker (Packard & Libby, 2008). High-sensitivity CRP has shown consistency across large prospective studies as a risk factor integrating multiple metabolic and low-grade inflammatory factors underlying the development of unstable atherosclerotic plaques, with a magnitude of effect matching that of classical
major risk factors. This marker was used in individuals showing a moderate level of risk from clinical assessment of major CVD risk factors (Perk et al., 2012).

Leptin is an adipokine released by white adipose tissue controlling the appetite and energy spending. Leptin deficiency or deficient leptin signalling results in excessive obesity and type 2 diabetes (Antonopoulos et al., 2015). Hyperleptinemia exerts proatherogenic effects by producing endothelial dysfunction (Antonopoulos et al., 2015). Conversely, Adiponectin, a product of adipose tissue, has insulin sensitizing, antiatherogenic, and anti-inflammatory properties (Packard & Libby, 2008). Adiponectin exerts multiple biologic effects essential to cardiovascular biology, including increasing insulin sensitivity, reducing visceral adipose mass, reducing plasma triglycerides, and increasing high-density lipoprotein (HDL) cholesterol (Packard & Libby, 2008).

The prevention of CVD presents a long-term challenge to all health care professionals and for public health policy as well. Family medicine is in a strategic position to promote the goals of patient empowerment and self-management (WONCA, 2011). Longitudinal care, a multidisciplinary approach, a strong relationship based on a unique consultation process and on trust, a person-centred approach, are the starting points for a continuous educational process aimed to empower the patient (WONCA, 2011). The care plan for all patients should include measures to assess and minimize cardiovascular risk (Fauci A, 2008). The calculation of the global cardiovascular (CV) risk as estimated synergism derived from the simultaneous presence of several individual risk factors, does not only identify users with a high risk but also how to model the intensity of therapeutic intervention in the effective control of risk factors, motivate users in an intervention strategy with the full implementation of the measures to modify lifestyle and to adhere to pharmacological therapy (DGS, 2013). On the other hand, the purpose of prevention of CVD in clinical practice should consist in reducing the overall CV risk with family physicians dealing with patients, not with isolated risk factors (DGS, 2013). The European Cardiovascular estimation risk system (HEARTSCORE) is a validated prediction system of fatal CV events in 10 years on the level of Primary Prevention (Perk et al., 2012).
‘Lifestyle’ is commonly based on established behavioural patterns. These patterns are enclosed during youth and adolescence by an interface of environmental and genetic factors, and are preserved or even encouraged by the individual’s social environment as an adult (Albert et al., 2006; Stringhini et al., 2010). However, marked differences in health behaviour between individuals but also between social groups can be observed (Orrow et al., 2012). In addition, these factors hamper the ability to adopt a healthy lifestyle, as does complex or confusing advice from medical caregivers. Increased awareness of these factors facilitates empathy and counselling (simple and explicit advice), thus facilitating behavioural change (Perk et al., 2012). Therefore, physicians must counsel patients regarding health risk behavioural patterns, such as tobacco use, unhealthy diet and physical inactivity (Fauci A, 2008). A sedentary lifestyle is a traditional risk factor for CVD (Lusis, 2000). Epidemiological studies have shown a positive association between sedentary behaviours (sitting, TV viewing, bed rest) and/or low levels of physical activity (PA) below the recommended doses (ACSM, 2010) and the presence of multiple cardiovascular risk factors. Epidemiological studies showed that the incidence of CVD in active individuals is lower by about 50% compared to sedentary subjects (Eriksson et al., 2009). It is assumed that PA plays a major role in primary and prevention of CVD having a positive impact on modifiable traditional risk factors and on the regression of CVD clinical symptoms (Goldhammer et al., 2005; Hamer et al., 2014; Nahrendorf & Swirski, 2015). Physical activity, exercise, diet, stress, and sleep are receiving attention as environmental modifiers of chronic inflammatory diseases, including atherosclerosis (Nahrendorf & Swirski, 2015). Regular physical activity and exercise are protective against all-cause mortality through suppressing pro-inflammatory cytokine production, enhancing anti-inflammatory mediators and antioxidant development, and promoting fibrinolytic activity (Chen et al., 2014). Primary care setting seems to be well placed to promote behaviour changes in PA among sedentary adults. In developed countries, 70-80% of adults visit their general practitioner at least once a year, and patients are concerned in discussing health promotion issues with primary care health professionals (Orrow et al., 2012). Interventions to promote smoking cessation in primary care have shown effectiveness (Stead et al., 2008) and have been recognized as important preventive
activities for respiratory and cardiovascular diseases (DGS, 2013). However, evidence to support the effectiveness of physical activity promotion in primary care is less robust (Orrow et al., 2012). A Cochrane systematic review of interventions to promote physical activity in community dwelling adults showed a moderate effect on self-reported physical activity (pooled SMD random effects model 0.28 95% CI 0.15 to 0.41) and cardiorespiratory fitness (pooled standardized mean difference random effects model 0.52 95% CI 0.14 to 0.90) at a minimum of six months of follow-up (Hilsdon M, 2005). Physical activity can be promoted in primary care setting in different ways, including delivery of face-to-face advice, group sessions, provision of written materials, and referral to an exercise program (Hilsdon M, 2005). Despite the differences in the way how this interventions are made, the promotion of PA at the population level is a public health priority (Heath et al., 2012).

In order to promote PA improvements and maintenance at individual and/or populational level, a proper understanding of the correlates and determinants of PA is needed (Friederichs et al., 2015). One determinant thought contributing substantially to PA adhesion and compliance is the motivation to become physically active (Bauman et al., 2012; Friederichs et al., 2015). The Self-Determination Theory (SDT) offers a theoretical framework for the understanding why the individuals adhere and sustain compliant with physical activity or exercise (Teixeira et al., 2012). There are validated instruments to assess the regulation for exercise (Murcia et al., 2007), with five categories that include different construct dimension of motivation (Mullan E, 1997). Indeed, the success of any intervention aiming to change behaviours, including PA, should take into account the initial subjects beliefs and try to modify the self-regulation status to adopt active life-style (Mullan E, 1997).

**Aims and Structure of the Thesis**

Despite the advent in last decades of multiple treatments that reduce mortality and morbidity, cardiovascular disease (CVD) due to atherosclerosis still remains the leading cause of death in the developed countries, including Portugal. Given the dramatic impact of CVD in the population, health professionals and politicians must remain vigilant about screening and preventing risk factors. Subjects with major risk
factors or high-risk scores should be aggressively treated with preventive therapies, including interventions to decrease sedentary time and improve physical activity levels. In this sense, this thesis aims to determine:

1) To review systematically the impact of educational interventions on primary prevention settings to promote physical activity
2) To evaluate the effects of an educational and counselling intervention on changes in physical activity, regulation for exercise, and cardiometabolic risk factors, and biomarkers of inflammation.

This thesis is organized into 6 chapters. The first chapter, Introduction, aims to guide the readers in the rational for the study and the presentation of the thesis aims. The second chapter, encompass a systematic review aiming to assess the effectiveness of primary care educational interventions designed to promote healthy lifestyles on physical activity levels and cardiovascular risk. The intervention study (aims, methods, results and discussion) is presented in chapter III. The main conclusions of the thesis are outlined in chapter IV. The references used in the entire thesis are listed in chapter V. Finally, in the Appendix (chapter VI) we exhibit several documents about instruments used for the study included in this thesis.
Chapter II: Literature Review
Chapter II: Literature Review

Impact of educational interventions on primary prevention of cardiovascular disease: a systematic review with a focus on physical activity

ABSTRACT

Background: Evidence from epidemiological and experimental studies illustrates the beneficial impact of healthy lifestyle behaviours on cardiovascular risk.

Objective: To assess the effectiveness of primary care educational interventions designed to promote healthy lifestyles on physical activity levels and cardiovascular risk.

Methods: A computer-aided search on PubMed and Scopus was performed to identify relevant studies published from January 2000 to November 2014. Two authors independently selected studies for inclusion and extracted data, including intervention characteristics and outcome measures, namely physical activity and cardiovascular risk or risk factors.

Results: Of the 151 identified studies, 14 met the inclusion criteria. The 14 studies enrolled 7713 participants; the sample size varied between 136 and 878 adults. Thirteen studies assessed physical activity by questionnaire and only one study used accelerometry. Eight of the 14 studies showed improvements in the physical activity levels after the intervention. The majority of the studies reported significant positive effects of the educational interventions on cardiovascular risk factors, mainly on lipid profile, blood pressure and cardiovascular risk score.

Conclusion: The educational interventions, in primary care, seem to improve daily physical activity, cardiovascular risk factors and risk score.

Keywords: Systematic reviews and meta-analyses; Health education; Prevention; General practice/family medicine, general
KEY MESSAGES

- Educational interventions increase daily physical activity levels.
- Primary care interventions focusing healthy lifestyles improve cardiovascular risk score and risk factors.
INTRODUCTION

The mortality attributed to the cardiovascular diseases (CVDs) has fallen considerably in the last decades, nonetheless it remains the major cause of premature death in Europe and worldwide (Perk et al., 2012). The most recommended management strategy to reduce the cardiovascular risk and cope with the modifiable CVDs risk factors, including sedentary behaviour, overweight/obesity and hypertension (Perk et al., 2012), is the change of unhealthy lifestyle behaviours (Foster et al., 2013; Orrow et al., 2012; Richards, Hillsdon, et al., 2013; Richards, Thorogood, et al., 2013). Primary health care interventions, by preventing and modifying CVDs risk factors (Eriksson et al., 2006), are a frontline strategy to fulfil this purpose. However, many barriers hamper the implementation of the recommended ‘high-risk’ approach, such as health professionals’ difficulties to assimilate multiple risk factors into an accurate assessment of cardiovascular risk (van Steenkiste et al., 2007). Indeed, the adherence to the guidelines and lifestyle counselling is less than optimal (van Steenkiste et al., 2007) and often abandoned by primary caregivers (Parra-Medina et al., 2011).

Cardiovascular risk reduction programs tend to be multidisciplinary with self-care components tailored to individual risk factors (Wister et al., 2007). The beneficial impact of healthy lifestyle behaviours on cardiovascular risk was demonstrated in a 3-year randomized trial in the primary care setting (Eriksson et al., 2009).

Previous reviews (Buchholz et al., 2013; Foster et al., 2013; Orrow et al., 2012; Richards, Hillsdon, et al., 2013; Richards, Thorogood, et al., 2013) on this topic, focusing on a specific type of intervention (e.g. text messaging, face-to-face interventions), were conducted. By not focusing on one type of intervention, the present review is broader and intends to highlight intervention features that make it most likely to increase daily physical activity (PA). In the present review we intend to provide a critical review of the literature linking healthy lifestyles, cardiovascular risk and/or risk factors and PA in primary care, and to discuss the impact of those interventions on PA. Having in mind the importance of PA, the aim of this review is to analyse the effectiveness of educational
interventions for change of lifestyle, with particular emphasis on PA and cardiovascular risk, in primary care.

METHODS

Databases and Search Strategy

A search of PubMed and Scopus was performed to identify studies in English language that evaluated the effects of educational interventions focused on change of lifestyle in primary care, on humans, published from January 2000 to November 2014. The searching terms were: (counselling OR education OR intervention OR health promotion) AND (primary care) AND (cardiovascular risk) AND (physical activity OR healthy lifestyles) AND (randomized controlled trials). Reference lists of studies identified by electronic searches were then searched to identify further articles relating to the topic of the review and to ensure that appropriate articles were obtained. In addition, in an effort to avoid retrieval bias, we manually searched the reference lists of landmark studies and background articles on this topic to look for any relevant citations that electronic searches might have missed.

Selection criteria for studies

All studies retrieved from our search needed to meet the following inclusion criteria: randomized controlled trials; adult human subjects in primary prevention; submitted to an educational intervention in healthy lifestyles; with PA as endpoint, regardless being the primary or secondary endpoint. Studies were excluded on the basis of the following: review papers; letters or editorial articles; studies not involving an educational intervention; studies involving interventions with supervised exercise sessions; studies with children and adolescents and those only involving individuals aged over 65 years old.

Study selection

Two authors (AR and FR) determined whether studies fulfilled the criteria for inclusion in this review through screening titles, abstracts and keywords of the studies identified in the electronic search. When both authors failed to reach an agreement, the full text of the respective study was obtained and analysed to
establish suitability. All studies classified as relevant by either of the authors were retrieved. Then, a standardized form was used to determine the eligibility for inclusion in the review based on the information within the full paper. A third author resolved disagreements (NLO). Figure 1 shows the process of the study identification and selection.

**Figure 1 - Study identification and selection flow diagram**

![Flow diagram](#)

**Data Extraction and Methodological Quality Assessment**

Two authors (AR and FR) independently extracted data relevant to the review using a customized form. Data were extracted for study design, population, type and characteristics of the intervention, and outcomes, namely PA and cardiovascular risk or risk factors. Two authors using the PEDro scale independently assessed methodological quality of each study. Disagreements were resolved by a third author (NLO).
RESULTS

A total of 151 studies were identified in the electronic databases search. After reviewing the title and abstract, 13 papers were excluded by being duplicates (i.e. coming from the two different databases). Of the remaining 138 papers, 125 were further discarded due to the following reasons: comprised supervised exercise interventions (n = 16), the studies did not assess a measure of PA and type of intervention (n = 33), enrolled participants with established disease (for instance ankylosing spondylitis) (n = 19), review papers (n = 8), comments (n = 1), participants were children or old adults (n = 10), papers describing the study protocol for a randomized controlled trial (n=38) (Figure 1). Therefore, only 14 studies were included in this review (Table 1).

The studies had a mean methodological quality score of 7.2 out of 10, ranging from 5 to 9 on the PEDro scale (Table 1). Lack of blinding was the most evident methodological flaw in the studies. Failure to conceal allocation was another general methodological limitation.

The 14 studies enrolled a total of 7713 participants; the sample size ranged from 136 (Armit et al., 2009) to 878 adults (Elley et al., 2003). Among these studies, seven enrolled adults eligible for CVDs risk assessment or presenting at least one cardiovascular risk factor (Cochrane et al., 2012; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009), one (Lakerveld et al., 2013) enrolled adults at risk of diabetes or CVDs, two studies (Armit et al., 2009; Elley et al., 2003) encompassed sedentary participants, two (Davies et al., 2008; Griffin et al., 2014) included participants with diabetes, or adults with hypertension or diabetes (Parra-Medina et al., 2011), and another study included family members of patients with coronary artery disease (Reid et al., 2014).

Regarding the methodology used to assess PA, the great majority of studies used a questionnaire. Only one study used an objective measure (accelerometry) together with the Norfolk Physical Activity Questionnaire (Griffin et al., 2014). The International Physical Activity Questionnaire (IPAQ) (Davies et al., 2008; Hardcastle et al., 2008; Hardcastle et al., 2013) and the Community Healthy Activities Model Program for Seniors (CHAMPS)
questionnaire (Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Parra-Medina et al., 2011) were used in three studies each. The other 7 studies used different questionnaires (Table 1).

The health education interventions were delivered in several ways, including face-to-face sessions (Cochrane et al., 2012; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012), by telephone (Parra-Medina et al., 2011; Wister et al., 2007), group sessions (Armit et al., 2009; Davies et al., 2008) and a combination of face-to-face, telephone, and group sessions (Elley et al., 2003; Griffin et al., 2014; Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Lakerveld et al., 2013; Reid et al., 2014). The method most used was face-to-face sessions plus telephone. There were differences between the frequency of sessions and the length of the interventions. Interventions varied in length from 6 hours (Davies et al., 2008) to 12 months (Cochrane et al., 2012; Griffin et al., 2014; Lakerveld et al., 2013; Parra-Medina et al., 2011; Reid et al., 2014).
Table 1 - Studies of the effects of educational intervention in physical activity

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participants</th>
<th>Intervention</th>
<th>Follow up</th>
<th>PA assessment</th>
<th>PA results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Griffin et al., 2014</td>
<td>478 participants with T2DM. IG: 239 (89 women), 59.5±7.5 yrs, CG: 239 (91 women), 59.8±7.5 yrs</td>
<td>Intensive treatment plus a theory-based behaviour change intervention: 1h introductory meeting + six 30 min meetings + 4 brief phone calls</td>
<td>12 months</td>
<td>4 days accelerometry and Norfolk PA Questionnaire</td>
<td>PA improved in both groups. No significant differences between groups at 1 year in PA both objectively and subjectively measured</td>
</tr>
<tr>
<td>Reid et al., 2014</td>
<td>426 family members of patients with CAD. IG: 211 (128 women), 52.0±11.9 yrs, CG: 215 (133 women), 51.1±11.3 yrs</td>
<td>17 counselling sessions (1 face-to-face + 16 telephone); weekly for the first 12 weeks and then at weeks 16, 20, 26, 39 and 52</td>
<td>12 months</td>
<td>Modified Godin Leisure-Time Exercise Questionnaire</td>
<td>The IG showed higher PA levels than the CG</td>
</tr>
<tr>
<td>Lakerveld et al., 2013</td>
<td>622 adults at risk for T2DM and CVDs. IG: 314 (178 women), 43.6±5.1 yrs, CG: 308 (185 women), 43.4±5.5 yrs</td>
<td>Cognitive behavioural program: six 30-min counselling sessions + 3-monthly sessions by phone for 1 year + health brochures</td>
<td>12 months</td>
<td>SQUASH Questionnaire</td>
<td>PA improved in both groups. The lifestyle intervention was not more effective than health brochures.</td>
</tr>
<tr>
<td>Hardcastle et al., 2013</td>
<td>334 participants with CVDs risk factors. IG: 203 (N of women n/a), 50.1±10.7 yrs, CG: 131, 50.4±10.0 yrs</td>
<td>Standard exercise and nutrition information plus up to five face-to-face motivational interviewing sessions</td>
<td>6 months</td>
<td>IPAQ</td>
<td>The IG significantly increased walking</td>
</tr>
<tr>
<td>Cochrane et al., 2012</td>
<td>601 participants with Framingham score ≥20%. IG:236 (32 women), 63.3±6.4 yrs, CG: 365 (36 women), 63.9±6.5 yrs</td>
<td>NHS Health Check service + support for lifestyle change based on the motivational interview/counselling model</td>
<td>Support up to 12 months</td>
<td>General Practice PA Questionnaire</td>
<td>PA scores improved in both the CG (NHS Health Check only group) and the IG (NHS Health Check plus additional lifestyle support)</td>
</tr>
<tr>
<td>Harris et al., 2012</td>
<td>699 participants either aged 50–64 years old or aged 40–55 yrs with hypertension or dyslipidaemia. IG: 384 (232 women), mean age n/a, CG: 315 (169 women)</td>
<td>Brief lifestyle advice and motivational counselling: 1 individual session + four 1.5-hour sessions over the first 3 months and a further two follow-up sessions at 6 and 9 months.</td>
<td>3 months</td>
<td>The Brief PA Assessment Tool</td>
<td>PA increased to a greater extent in the IG</td>
</tr>
<tr>
<td>Parra-Medina et al., 2011</td>
<td>266 women with hypertension or diabetes. IG: 136, mean age n/a, CG: 130</td>
<td>Theory-based lifestyle intervention targeting PA and dietary fat intake: tailored telephone counselling and tailored newsletters</td>
<td>12 months</td>
<td>CHAMPS Questionnaire</td>
<td>The IG showed higher total and leisure-time moderate-to-vigorous PA at 6 months but not at 12 months</td>
</tr>
<tr>
<td>Koelwijn-van Loon et al., 2010</td>
<td>589 patients eligible for cardiovascular risk management. IG: 304 (174 women), 56±10 yrs, CG: 285 (151 women), 58±10 yrs</td>
<td>2 face-to-face consultations (15-20 min) + 10 min telephone consultation (or a face-to-face consultation)</td>
<td>12 weeks</td>
<td>CHAMPS Questionnaire</td>
<td>PA improved in both groups</td>
</tr>
<tr>
<td>Koelwijn-van Loon et al., 2009</td>
<td>589 patients eligible for cardiovascular risk management. IG: 304 (174 women), 56±10 yrs, CG: 285 (151 women), 58±10 yrs</td>
<td>2 face-to-face consultations (15-20 min) + 10 min telephone consultation (or a face-to-face consultation)</td>
<td>12 weeks</td>
<td>CHAMPS Questionnaire</td>
<td>No changes in PA</td>
</tr>
<tr>
<td>Armit et al., 2009</td>
<td>136 participants not meeting PA recommendations (82 women). IG (ES): 45 (31 women), mean age n/a, IG (ES+P): 45 (27 women), CG: 46 (24 women)</td>
<td>ES: GP usual care +30 min PA counselling based on the transtheoretical model. ES+P: As for ES group, with goal setting (steps/day) and self-monitoring focusing on a pedometer</td>
<td>12 weeks (24 wks follow up)</td>
<td>Active Australia PA Questionnaire</td>
<td>PA improved in all groups. At week 24, the ES+P group were more likely to report meeting PA guidelines than the CG</td>
</tr>
<tr>
<td>Reference</td>
<td>N</td>
<td>IG/CG Details</td>
<td>Intervention/Outcome</td>
<td>Time Period</td>
<td>Measure</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>---------</td>
<td>--------------------------------------------------------------------------------</td>
<td>----------------------</td>
<td>-------------</td>
<td>---------</td>
</tr>
<tr>
<td>(Davies et al., 2008)</td>
<td>824</td>
<td>Patients with T2DM. IG: 437 (47 women), 59 yrs (SD n/a) CG: 387 (43 women), 60 yrs</td>
<td>6 hour structured group educational programme focused on lifestyle factors, such as food choices and PA</td>
<td>12 months</td>
<td>IPAQ</td>
</tr>
<tr>
<td>(Wister et al., 2007)</td>
<td>315</td>
<td>Participants with a Framingham score ≥10%. IG: 157 (86 women), 55.8±5.5 yrs CG: 158 (98 women), 55.1±5.2 yrs</td>
<td>Report card showing person’s risk profile + telehealth-guided self-care management system at every 6 months</td>
<td>12 months</td>
<td>5-Point Ordinal Scale</td>
</tr>
<tr>
<td>(Hardcastle et al., 2008)</td>
<td>334</td>
<td>Participants with CVDs risk factors. IG: 203 (N of women n/a), 50.1±0.7 yrs CG: 131, 50.4±0.9 yrs</td>
<td>Patient-centred counselling intervention that incorporated standard exercise and nutrition information + up to 5 face-to-face motivational interviewing sessions</td>
<td>6 months</td>
<td>IPAQ</td>
</tr>
<tr>
<td>(Elley et al., 2003)</td>
<td>878</td>
<td>Sedentary participants. IG: 451 (301 women), 57.2±10.8 yrs CG: 427 (281 women), 58.6±11.5 yrs</td>
<td>A prompt card, stating the stage of change, + oral and written advice by GP in the consultation + at least 3 telephone calls (lasting 10-20 min) over the next 3 months</td>
<td>3 months</td>
<td>3-Month PA Recall Questionnaire</td>
</tr>
</tbody>
</table>

Legend: CAD, coronary artery disease; CG, control group; CHAMPS, community healthy activities model program for seniors; CVDs, cardiovascular diseases; ES, Exercise Scientist group; GP, general practitioner; IG, intervention group; IPAQ, international physical activity questionnaire; n/a, not available; PA, physical activity; P, pedometer; Min, minutes; SD, standard deviation; SQUASH, short questionnaire to assess health-enhancing physical activity; T2DM, type 2 diabetes mellitus; yrs, years;
Lifestyle Outcomes

Behavioural outcomes included PA, diet, alcohol consumption and smoking status. Significant improvements in the PA levels of the interventional group (IG) compared with the control group (CG) were reported in eight studies (Armit et al., 2009; Davies et al., 2008; Elley et al., 2003; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Parra-Medina et al., 2011; Reid et al., 2014) (Table 1). Armit et al. showed an increase in PA at weeks 12 and 24 with no significant group differences; nonetheless, at week 24, the group receiving behaviour change advice plus a pedometer was more likely to report meeting PA guidelines than the CG group (Armit et al., 2009). Additionally, three studies (Cochrane et al., 2012; Griffin et al., 2014; Lakerveld et al., 2013) found a significant increase in the PA levels of both groups. In these studies, the CG received also an intervention, i.e. they received more than the usual care. In the Cochrane et al. study the CG received the NHS health check plus the usual general practitioner care (Cochrane et al., 2012); Lakerveld et al. provided the CG with brochures with information and guidelines with regard to healthy PA levels, a healthy diet and, if relevant, smoking cessation (Lakerveld et al., 2013); in the study of Griffin et al. the CG received an intensive treatment which included among other features dietary counselling, more frequent contacts with the general practitioner and theory-based education (Griffin et al., 2014). Only three studies (Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Wister et al., 2007) reported a lack of significant effects of the intervention on PA levels.

Dietary intake was reported in ten studies (Cochrane et al., 2012; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Lakerveld et al., 2013; Parra-Medina et al., 2011). Harris et al. reported a daily enhancement in the number of portions of fruit and vegetable consumed in the IG at 6 months, but showed no significant differences at 12 months (Harris et al., 2012). Koelewijn-Van Loon et al. showed a significant lower intake of fat in the IG and a significant higher percentage of participants meeting the recommendations for vegetable intake than in the control group (Koelewijn-van Loon et al., 2009). Hardcastle et al. described a lower fat intake at 6 and 18 months (Hardcastle et al., 2013).
Smoking and alcohol consumption were measured in eight studies (Cochrane et al., 2012; Davies et al., 2008; Griffin et al., 2014; Harris et al., 2012; Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Lakerveld et al., 2013; Reid et al., 2014). Only Davies et al. reported a decrease in smoking status in the intervention group at 8 and 12 months follow-up compared with the control group (Davies et al., 2008).

**Cardiovascular Risk Outcomes**

All studies used physiological outcomes to assess the effects of the educational interventions. They measured HbA1c (Davies et al., 2008), fasting blood glucose (Harris et al., 2012; Reid et al., 2014), blood pressure (Armit et al., 2009; Cochrane et al., 2012; Davies et al., 2008; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Reid et al., 2014), lipid profile (Cochrane et al., 2012; Davies et al., 2008; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Reid et al., 2014), body mass index (Cochrane et al., 2012; Harris et al., 2012; Reid et al., 2014; Wister et al., 2007), waist circumference (Cochrane et al., 2012; Harris et al., 2012; Reid et al., 2014; Wister et al., 2007), body weight (Cochrane et al., 2012; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012), Heart Score (Davies et al., 2008; Koelewijn-van Loon et al., 2010), Framingham score (Cochrane et al., 2012; Elley et al., 2003; Wister et al., 2007), and type 2 diabetes mellitus risk score (Lakerveld et al., 2013).

Davies et al. showed reductions in body weight compared with control groups with a decrease of 3.1% at 4 months and 3.2% at 12 months (Davies et al., 2008). The effects of educational interventions on waist circumference were significant in 2 studies (Cochrane et al., 2012; Reid et al., 2014) and not significant in other 2 studies (Harris et al., 2012; Wister et al., 2007). In some studies, risk factors, such as high cholesterol levels (Davies et al., 2008; Hardcastle et al., 2008; Harris et al., 2012; Koelewijn-van Loon et al., 2009; Reid et al., 2014) and blood pressure (Armit et al., 2009; Cochrane et al., 2012; Harris et al., 2012; Reid et al., 2014), did not improve during the study period. However, other studies showed a significant decreased in blood pressure (Hardcastle et al., 2008; Hardcastle et al., 2013; Wister et al., 2007), total cholesterol (Hardcastle et al., 2008;
Hardcastle et al., 2013; Wister et al., 2007) and triglycerides (Davies et al., 2008) after the intervention.

Regarding the Heart Score, Davies et al. reported a greater improvement in the IG in comparison to the CG (Davies et al., 2008). Wister et al. also reported a decrease in the Framingham Risk Score of 3.1% (Wister et al., 2007) and Cochrane et al. reported a decrease of 2.98% (Cochrane et al., 2012).

**DISCUSSION**

*Main findings*

Despite the differences between studies in the methods and instruments used to assess PA, the available evidence suggests that educational interventions are successful in the modification of PA levels in primary prevention. Overall, the educational interventions seem to have positive repercussions on cardiovascular risk factors, mainly on lipid profile, blood pressure and cardiovascular risk score.

*Strengths and limitations*

The majority of the studies included in this review showed high methodological quality and, with two exceptions (Armit et al., 2009; Parra-Medina et al., 2011), have conducted a priori power analysis to determine the sample size. We found heterogeneity both in the interventions and PA assessment tools. PA was assessed using different questionnaires, which may have limited the observation of the real impact of the interventions. Self-reported PA measures have low sensitivity, high variance, are less accurate, and frequently overestimate the PA levels (Dyrstad et al., 2014). On the other hand, some of the questionnaires do not stratify PA by intensity levels (light, moderate, vigorous or very vigorous), precluding the analysis of the PA by intensities. Objective data is needed to measure sedentary time and/or the time spent in sedentary behaviours (sitting, TV viewing, time in bed rest) because it has been questioned whether the accomplishment of the recommended amount of daily physical activity time at moderate-to-vigorous intensities is sufficient to overcome long periods of sedentary behaviours, and yet protect the individuals against cardiovascular risk.
factors (Garber et al., 2011).

The majority of the studies in this review did not consider the influence of environmental factors such as seasonal changes in daily physical activity. This cannot be disregarded when studying the long term effects of educational interventions in the modification of PA levels, since it was already shown there are significant differences in the amount and patterns of PA between winter and other seasons (Buchowski et al., 2009; Tucker & Gilliland, 2007).

One limitation of this review is potential publication bias, as it included only articles published in English, the selection of the studies was based on title and abstract and unpublished work was not included.

**Interpretation**

The studies that showed significant changes in PA have adopted face-to-face, at least one session, plus remote interventions and used motivational techniques for behavioural change such as motivational interviewing sessions. Interventions incorporating cognitive behavioral strategies, including goal-setting, self-monitoring, face-to-face contacts, feedback and reinforcement are more likely to induce changes (Artinian et al., 2010). Despite assuming that the PA behaviour of the individuals might be influenced by these intervention characteristics, we cannot assume that this is the most effective delivery method because these common methodological traits were not substantially different from some of the studies not reporting changes in PA after the intervention.

In addition to the improvements in PA, several cardiovascular risk factors and risk score were also positively change by the interventions, which make it clinically important. Indeed, even small but sustained lifestyle changes can substantially reduce cardiovascular risk, morbidity and mortality (Artinian et al., 2010).

**Implications for further research**

In the majority of studies the sample was mixed gender cohorts. Therefore, future studies should include sub-analysis by gender to ascertain whether between gender differences exist. Future studies should also include measures of the time spent at different PA intensities, namely at moderate-to-vigorous PA, and
sedentary behaviours, because sedentary time has been consistently related to deleterious health outcomes (Eckel et al., 2014). Environmental factors should be monitored, and PA contexts (occupational, leisure-time physical activity) must be differentiated. Since motivation or self-determination for exercise is an intra-personal correlate or determinant for the behavioural changes (Ingledew & Markland, 2008), future studies should also report motivation levels or states of self-determination at baseline and how these features change throughout the interventions.

**CONCLUSION**

The current research provides evidence that educational interventions are successful in the modification of PA levels in primary prevention. The educational interventions seem to have also positive impact on cardiovascular risk factors, mainly on lipid profile, blood pressure and cardiovascular risk score.
Chapter III: Intervention Study
Intervention Study

STUDY AIDS

To evaluate the effects of an educational and counselling intervention on changes in physical activity, regulation for exercise, and cardiometabolic risk factors, and biomarkers of inflammation

METHODS

Study design

This study was conducted at a primary health care unit in Oporto from 2011 to 2014. Eligibility criteria to be included in the study were: i) to be registered in the primary health care unit clinical file database; ii) to be aged between 18 and 65 years old; and iii) to have the presence of at least one of the following cardiovascular risk factors: hypertension (SBP/DBP at least 140/90 mmHg or hypertension treatment), dyslipidaemia (at least total cholesterol 190 mg/dl or triglycerides 150 mg/dl or HDL less than 40 mg/dl in men and 46 mg/dl if women or hypercholesterolemia treatment), diabetes (fasting glucose > 126 mg/dl or diabetes treatment), obesity (BMI>30 Kg/m2), abdominal obesity (WC > 102 in men and >88 in women), smoking or family history of premature cardiovascular disease (CVD) (Beeres, 2004). Volunteers were excluded if they met the following conditions: physical disabilities precluding autonomous physical activity, diagnosis of cardiovascular and/or respiratory and renal diseases, hepatic disorders, chronic infectious diseases, history of alcohol abuse, cancer, and thyroid, auto-immune or psychiatric/cognitive disorders.

To meet the age inclusion criteria, an age filter was applied in the 8000 clinical file database to exclude those aged below 18 and over 65 years old, remaining 4600 individuals. From those, 23.5% (n=1083) were randomly selected using a computer generated sequence of random numbers. Subjects were invited to participate through phone calls, and those who answered and agreed to participate in the study, underwent a first appointment with a physician to verify
eligibility and undertook clinical evaluation (Appendix I) as well as to collect information on pre-existing clinical conditions and use of medications. In addition, in the first appointment, participants underwent measurements of anthropometrics, resting blood pressure, dietary habits, determination of smoking status, regulation for exercise by questionnaire, and accelerometers were delivered for the purpose of assessment of daily PA. Within 7 days, participants return to health care unit, after a fasting overnight, for blood collection and return accelerometers.

Participants were allocated into two groups by convenience according their availability: an intervention group (IG) receiving an educational program of healthy lifestyles lasting 5-month, plus usual medical follow-up; and a control group (CG), which only received usual medical care (i.e., regular appointments with a Family doctor and optimized medication).

All the participants were re-evaluated after 5-month for all the above mentioned outcomes. The North Health Administration Ethics Committee granted ethical approval (reference 25/2010), and all procedures were conducted according to the Declaration of Helsinki. All participants who agreed to participate provided written informed consent (Appendix II).

**INTERVENTION: EDUCATIONAL AND COUNSELLING PROGRAM OF HEALTHY LIFESTYLES**

The educational program of healthy lifestyle lasted 5-month and included educational group sessions, information material (booklet; see Appendix III), text messages to reinforce the information material, and demonstration sessions about types of leisure physical activities.

After the initial consultation and assessment, and during the first 3 weeks the IG have participated in three group sessions (one per week), each one lasting approximately 60 minutes. The sessions were delivered by a physician and a physical fitness instructor. The content of the sessions consisted of: i) information about the physiopathology of atherosclerosis underlying several cardiovascular diseases (mainly ischemic heart disease) as well as the modifiable risk factors, including those that are moderated by unhealthy behaviours; ii) management and
control of risk factors and their impact on health status and quality of life; iii) importance of adoption of healthy lifestyles and behaviours that prevent clinical manifestations of cardiovascular disease with particular emphasis on physical activity and exercise; iv) importance of adherence and compliance with medication and physical activity; v) information about guidelines for healthy related PA, including frequency, intensity, duration, and types of PA, and preferable day periods to perform PA and/or daily living tasks with the desirable levels of exertion to achieve the recommended levels of PA; vi) the procedures to monitor exertion during PA, and risks and emergency procedures.

During the last session of education and counselling, subjects received a manual with all the information given in the sessions. After the group sessions, mobile text messages reinforcements about the need to be physically active and to perform PA was given at the 4th, 5th, 6th, 7th, 8th, 10th, 12th, 14th, 16th and 19th weeks.

**MEASUREMENTS**

**Anthropometrics**

Body weight was measured to the nearest 0.1 kg with an electronic weight scale (Tanita, Inner Scan BC-522, Tokyo, Japan). Participants were weighed barefoot wearing light clothing. Height was measured to the nearest 1 mm with a standard stadiometer. Body mass index (BMI) was determined as weight divided by height squared (kg/m²). The participants with BMI ≥ 25 Kg/m² were classified as overweight/obese.

Waist circumference (cm), as an indicator of central obesity, was measured with a flexible tape around the participant’s bare midriff, at the midpoint between the lowest rib and the iliac crest, after an exhalation (Klein et al., 2007). The participant was standing with arms hanging freely (Klein et al., 2007).

**Daily physical activity**

Daily PA was assessed using accelerometers (Actigraph GT1M, Actigraph LLC, Pensacola, FL). Participants were instructed to wore the accelerometer over the
right hip, for 7 consecutive days, during the waking hours, except while bathing and water-based activities.

ActiLife software (Actigraph, Florida, USA, version 6.9) was used to reduce raw activity data into daily PA. The average min per day spent at different physical activity intensities, which were calculated by relating counts/min to physical activity intensity according to cut points (sedentary < 100 counts/min, Light 100-2019 counts/min, Moderate 2020-5998 counts/min, Vigorous > 5998 counts/min, Moderate-to-vigorous ≥ 2020) defined by Troiano et al. (Troiano et al., 2008)

**Regulation for exercise**

Regulation for exercise was assessed by questionnaire (The Behavioural Regulation in Exercise Questionnaire – BREQ 2; see Appendix IV) validated for the Portuguese population (Palmeira, 2007). The BREQ 2 has 5 subscales, amotivation (items 5, 9, 12, 19), external regulation (1, 6 11, 16), introjected regulation (2, 7, 13), identified regulation (3, 8, 14, 17), and intrinsic regulation (4, 10, 15, 18). Amotivation is a lack on intention to involve in a behaviour (Markland, 2009). External regulation when there is purpose to obtain rewards or to avoid punishments administered by significant others (Markland, 2009; Mullan E, 1997). The introjected regulation of behaviour follows internalization of external control, which is then applied to the self through the administration of sanctions and other self-controlling behaviours (Markland, 2009; Mullan E, 1997). Identified regulation is a more internalised and autonomous form of regulation, involving a conscious acceptance that the behaviour is important in order to achieve personally valued outcomes (Markland, 2009). Intrinsically motivated behaviours are involved in for the inherent interest and satisfaction of participating (Markland, 2009).

Each item is scored from zero to four and for each subscale the final score was calculated by the sum of the points recorded in its items.
Resting Blood Pressure Measurements

Participants were instructed to refrain strenuous physical activities prior to the assessment, and to avoid smoking, heavy meals or caffeine at least 4 hours before the evaluation. A trained physician performed blood pressure measurements. After resting 5 minutes in seated position, brachial blood pressure was performed (Colin, model BP 8800; Critikron, Inc., Tampa, FL; USA) in the left arm and systolic and diastolic blood pressure (SBP and DBP, respectively) were computed as the average of 3 measurements, and subsequently used to calculate mean arterial pressure.

Blood collection and biochemical analysis

Twelve-hour fasting blood samples were collected by venipuncture of the antecubital vein into serum separator and EDTA coated tubes, which were centrifuged for 15 minutes at 2000-x g. Serum and plasma samples were separated, aliquot and stored at -80 °C for further analysis.

Glycated haemoglobin (HbA1c) was determined by an ion-exchange HPLC system with a D-10™ Bio-Rad® analyser (Bio-Rad). Serum glucose, total cholesterol, high density lipoprotein (HDL) cholesterol and triglycerides were measured in an automated clinical chemistry Olympus AU5400 (Beckman-Coulter). Low density lipoprotein (LDL) cholesterol was calculated using the Friedewald equation. Plasma insulin was determined in a Cobas E411 automated analyzer (Roche).

The homeostasis model assessment (HOMA) was calculated as described by Matthews et al. (Matthews et al., 1985).

The quantitative insulin-sensitivity check index (QUICKI) was calculated from fasting glucose and insulin values (Katz et al., 2000) \(\text{QUICKI} = 1/[\log(I_0) + \log(G_0)]\), where \(I_0\) is fasting insulin (microunits per milliliter) and \(G_0\) is fasting glucose (milligrams per decaliter). Because QUICKI is the reciprocal of the log-transformed product of fasting glucose and insulin, it is a dimensionless index without units (Chen et al., 2005).
Serum high-sensitivity C-reactive protein (hsCRP) was determined using a particle-enhanced immunonephelometric assay in a Dimension Vista 1500 nephelometer (Siemens).
To determine plasma levels of Interleukin-6 (hsIL-6), Tumor Necrosis Factor alpha (hsTNF-α) and leptin were used a high sensitive Milliplex map kit (Millipore) assayed in a Luminex 200™ analyzer (Luminex Corporation). Adiponectin plasma levels were determined using a commercial enzyme-linked immunosorbent assay (Mercodia AB).
All assays were performed according to the manufacturer’s instructions.

**Dietary intake**
Patients recorded their intakes of food and beverage for 4 days (one weekend and 3 week days) (Appendix V). Experienced nutritionists performed comprehensive nutrient analysis using an adapted Portuguese version of the software Food Processor Plus® (ESHA Research Inc., Salem, Oregon, USA). For the purposes of this study only total energy intake (Kcal/day) was used.

**Statistical analysis**
Before analysis, logarithm or square root transformations of data were performed to normalize skewed distributions. Nevertheless, the data are presented in the original scale for clarity, as mean and standard-deviation or median and interquartile interval (25th-75th). At baseline, between groups comparisons were performed through Student's independent t-test and Chi-square test, as appropriate. Comparisons of changes in outcomes between treatments over time (treatment*time) were performed through the General Linear Model (Repeated measures ANOVA), adjusting for age and sex, with Bonferroni correction. When a significant treatment*time interaction was observed, a univariate general linear model (treatment as fixed factor) was performed to ascertain the differences at the final assessment between treatments. Statistical significance was set at p < 0.05 for all tests. SPSS 21.0 (SPSS Inc., Chicago, IL, USA) was used for all analysis.
RESULTS

The flow of participants through the study is shown in the Figure 2. Of the 1082 patients selected to the study, 264 refused to participate and 321 were unable to communicate (did not respond to the phone calls). We excluded 343 for not meeting inclusion criteria due to the following reasons: previous cardiovascular disease (n=31), osteoarticular disorders precluding regular physical activities (n=35), 14 had hepatic disorders, renal disease (n=2), respiratory disorders (n=23), psychiatric disorders (n=28), cancer (n=28), endocrine disorders (n=17), HIV in 5, alcoholism in 5 patients and 155 in other disturbs. From the remaining 154 that agreed to participate, 70 were assigned to the control group (CG) and 84 to the intervention group (IG). During the study, we had 12 dropouts in the CG and 7 in the IG.

Figure 2 - Flow diagram of participant through the study

The sample encompassed male (47.4%) and female (52.6%) without statistical differences in the proportions in the two groups. The mean age of the entire
sample was 56.3 ± 7.1 years old, and no mean differences were found between groups (Control Group = 55.7 ± 7.6; Intervention Group = 56.9 ± 6.6).

Baseline characteristics of all participants, and of those included in each group, regarding the frequency of cardiovascular risk factors and medications are presented in Table 2.

No significant differences between groups were found for the prevalence of cardiovascular risk factors and use of medications (Table 2). Regarding medications, the level or type did not change during the intervention period (data not reported).
Results

Table 2 - Frequency of cardiovascular risk factors at baseline in the entire sample, and within the two groups

<table>
<thead>
<tr>
<th>Cardiovascular Risk factors</th>
<th>All (n=135)</th>
<th>Control Group (n=58)</th>
<th>Intervention Group (n=77)</th>
<th>χ²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>114 (84.4)</td>
<td>52 (89.7)</td>
<td>62 (80.5)</td>
<td>0.160</td>
<td></td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>115 (85.2)</td>
<td>49 (84.5)</td>
<td>66 (85.7)</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>37 (27.4)</td>
<td>16 (11.9)</td>
<td>21 (15.6)</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>Smokers</td>
<td>29 (21.5)</td>
<td>14 (18.1)</td>
<td>15 (25.8)</td>
<td>0.298</td>
<td></td>
</tr>
<tr>
<td>Overweight/Obesity</td>
<td>120 (88.8)</td>
<td>54 (40.0)</td>
<td>66 (48.9)</td>
<td>0.350</td>
<td></td>
</tr>
<tr>
<td>Central obesity</td>
<td>90 (66.7)</td>
<td>40 (69)</td>
<td>50 (64.9)</td>
<td>0.713</td>
<td></td>
</tr>
<tr>
<td>Familiar CVD</td>
<td>41 (30.4)</td>
<td>17 (29.3)</td>
<td>24 (31.2)</td>
<td>0.852</td>
<td></td>
</tr>
</tbody>
</table>

Cardiovascular Risk factors

<table>
<thead>
<tr>
<th>Medications</th>
<th>B-blockers</th>
<th>ACE-inhibitors</th>
<th>Fibrates</th>
<th>ARBs</th>
<th>Diuretic</th>
<th>Calcium Antagonists</th>
<th>Statins</th>
<th>Aspirin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>23 (17.0)</td>
<td>28 (20.7)</td>
<td>4 (3.0)</td>
<td>55 (40.7)</td>
<td>52 (38.5)</td>
<td>18 (13.3)</td>
<td>63 (46.7)</td>
<td>12 (8.8)</td>
</tr>
<tr>
<td></td>
<td>6 (10.3)</td>
<td>13 (22.4)</td>
<td>3 (5.1)</td>
<td>26 (44.8)</td>
<td>27 (46.6)</td>
<td>8 (13.8)</td>
<td>26 (44.8)</td>
<td>4 (6.9)</td>
</tr>
<tr>
<td></td>
<td>17 (22.1)</td>
<td>15 (19.5)</td>
<td>1 (1.2)</td>
<td>29 (37.7)</td>
<td>25 (32.5)</td>
<td>10 (13.0)</td>
<td>37 (48.0)</td>
<td>8 (10.4)</td>
</tr>
</tbody>
</table>

CVD: cardiovascular disease; ACE-inhibitors: angiotensin converting enzyme inhibitors; ARBs: angiotensin II receptor blockers

In addition, at baseline, there was significant differences between groups in PA, namely for the time spent at sedentary PA (432.8 ± 101.1 Control vs 468.1 ± 80.5 Intervention; p=0.026), and light physical activity (338.5 ± 95.9 Control vs 299.5 ± 89.2 Intervention; p=0.016). Regarding the regulation for exercise there was differences between groups in introjected (3.44 ± 2.88 control vs 5.44 ± 3.36 intervention; p<0.001) and identified regulations (10.2 ± 3.3 control vs 11.7 ± 3.6 intervention; P=0.012). For the inflammatory biomarkers the two groups showed significant differences at baseline for IL-6 (3.92 ± 3.25 control vs 2.57 ± 2.29 intervention; p=0.009) and TNF-α (3.97 ± 1.58 control vs 3.36 ± 1.39; p=0.024).
Results

To all the other variables the groups were identical at baseline (to see the means or median values please see Table 3-7).

**Anthropometrics and fat mass percentage**

After intervention, interaction time*group was significant for weight, BMI and waist circumference (Table 3), however at the final of the study intervention final differences between groups were significant only for waist circumference with reductions favouring the intervention group (-4.8 cm; p=0.05) (Table 3).

**Table 3 - Changes in anthropometrics [Data are mean (SD) or median (25th - 75th percentile)]**

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Intervention Group</th>
<th>time* group</th>
<th>Difference at final assessment Mean (95% CI)</th>
<th>Final Dif</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Height (cm)</strong></td>
<td>160.4 (9.0)</td>
<td>-</td>
<td>159.9 (10.1)</td>
<td>-</td>
<td>0.013</td>
<td>0.112</td>
</tr>
<tr>
<td><strong>Weight (Kg)</strong></td>
<td>75.2 (66.4 - 82.4)</td>
<td>75.8 (68.2 - 83.8)</td>
<td>74.2 (64.5 - 83.0)</td>
<td>73.7 (63.7 - 81.4)</td>
<td>0.017</td>
<td>0.111</td>
</tr>
<tr>
<td><strong>BMI (Kg/m2)</strong></td>
<td>29.7 (3.7)</td>
<td>29.8 (3.6)</td>
<td>29.0 (3.8)</td>
<td>28.8 (3.9)</td>
<td>-1.0 (-3.2 to 0.2)</td>
<td>0.111</td>
</tr>
<tr>
<td><strong>WC (cm)</strong></td>
<td>99.2 (9.5)</td>
<td>100.9 (9.5)</td>
<td>97.2 (10.1)</td>
<td>96.1 (10.1)</td>
<td>-4.8 (-3.2 to -1.4)</td>
<td>0.006</td>
</tr>
</tbody>
</table>

BMI: body mass index; WC: waist circumference

* Variables transformed for analysis (not normally distributed)

**Daily physical activity levels**

The analysis of daily PA according the time spent in intensity categories revealed there was not significant time*group interactions, and between group mean differences at the final of the intervention period (Table 4).
Table 4 - Data for daily physical activity levels [Data are mean (SD) or *median (25th-75th percentile)]

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Intervention Group</th>
<th>time* group P</th>
<th>Difference at final assessment Mean (95% CI)</th>
<th>final difference P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sedentary</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASELINE</td>
<td>432.8 (101.1)</td>
<td>433.3 (99.8)</td>
<td>468.1 (80.5)</td>
<td>458.6 (82.2)</td>
<td>0.426</td>
</tr>
<tr>
<td>FINAL</td>
<td>466.9 (96.4)</td>
<td>476.2 (94.7)</td>
<td>522.8 (105.8)</td>
<td>507.0 (89.1)</td>
<td>0.259</td>
</tr>
<tr>
<td><strong>Light</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASELINE</td>
<td>326.8 (276.8 - 387.5)</td>
<td>316.8 (270.7 - 375.3)</td>
<td>282.1 (230.6 - 372.4)</td>
<td>292.0 (246.7 - 342.5)</td>
<td>0.440</td>
</tr>
<tr>
<td>FINAL</td>
<td>360.2 (247.4 - 429.1)</td>
<td>366.9 (250.7 - 376.8)</td>
<td>324.1 (270.6 - 372.4)</td>
<td>351.0 (247.5 - 346.7)</td>
<td>0.046</td>
</tr>
<tr>
<td><strong>Moderate</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASELINE</td>
<td>33.6 (17.6 - 49.9)</td>
<td>36.7 (21.1 - 62.8)</td>
<td>25.6 (18.0 - 45.6)</td>
<td>30.0 (20.7 - 60.5)</td>
<td>0.243</td>
</tr>
<tr>
<td>FINAL</td>
<td>28.0 (17.6 - 35.4)</td>
<td>31.1 (20.7 - 50.5)</td>
<td>25.6 (18.0 - 45.6)</td>
<td>30.0 (20.7 - 60.5)</td>
<td>0.243</td>
</tr>
<tr>
<td><strong>Vigorous</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASELINE</td>
<td>0.00 (0.00 - 0.00)</td>
<td>0.00 (0.00 - 0.14)</td>
<td>0.00 (0.00 - 0.00)</td>
<td>0.00 (0.00 - 0.04)</td>
<td>0.238</td>
</tr>
<tr>
<td>FINAL</td>
<td>0.00 (0.00 - 0.00)</td>
<td>0.00 (0.00 - 0.14)</td>
<td>0.00 (0.00 - 0.00)</td>
<td>0.00 (0.00 - 0.04)</td>
<td>0.238</td>
</tr>
<tr>
<td><strong>MVPA</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASELINE</td>
<td>33.7 (17.6 - 50.2)</td>
<td>37.1 (21.1 - 62.8)</td>
<td>25.6 (18.0 - 46.7)</td>
<td>30.0 (21.0 - 60.5)</td>
<td>0.254</td>
</tr>
<tr>
<td>FINAL</td>
<td>38.2 (24.7 - 54.5)</td>
<td>41.6 (27.1 - 60.4)</td>
<td>36.6 (25.0 - 46.7)</td>
<td>41.0 (26.0 - 55.5)</td>
<td>0.254</td>
</tr>
</tbody>
</table>

MVPA: moderate to vigorous physical activity
* Variables transformed for analysis (not normally distributed)

**Regulation for exercise**

From baseline to the final assessment amotivation and intrinsic regulation improved significantly in the intervention group and with significant differences between groups at the final measurement (Table 5). Identified regulation showed a trend to improve in the Intervention group compared to Control, being the mean differences at the final assessment statistically significant. On average, Introjected regulation at the final assessment was superior in the Intervention group (mean final difference of 2.23 points; p<0.001), despite the absence of significant time*group interaction. To other dimensions of regulation for exercise, the two groups were identical in both periods of assessment.
Results

Table 5 - Changes in motivation [Data are mean (SD) or median (25th-75th percentile)]

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Intervention Group</th>
<th>time* group p</th>
<th>Difference at final assessment Mean (95% CI) p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amotivation</td>
<td>0.00 (0.00 - 3.00)</td>
<td>0.00 (0.00 - 3.00)</td>
<td>0.00 (0.00 - 0.00)</td>
<td>0.001</td>
</tr>
<tr>
<td>External Regulation</td>
<td>0.00 (0.00 - 2.60)</td>
<td>2.00 (0.00 - 4.00)</td>
<td>0.00 (0.00 - 0.00)</td>
<td>0.587</td>
</tr>
<tr>
<td>Introjected Regulation</td>
<td>3.44 (2.88)</td>
<td>4.14 (2.80)</td>
<td>5.44 (3.36)</td>
<td>6.36 (2.87)</td>
</tr>
<tr>
<td>Identified Regulation</td>
<td>10.2 (3.3)</td>
<td>9.9 (3.7)</td>
<td>11.7 (3.6)</td>
<td>12.5 (2.7)</td>
</tr>
<tr>
<td>Intrinsic Regulation</td>
<td>10.6 (3.9)</td>
<td>10.6 (3.9)</td>
<td>11.0 (4.2)</td>
<td>13.0 (3.2)</td>
</tr>
</tbody>
</table>

a Variables transformed for analysis (not normally distributed)

Resting blood pressure

In the present study we did not observe any significant time*group interaction for resting systolic, diastolic, and mean blood pressures (P>0.05) (Table 6). However, the mean systolic pressure has diminished in the intervention group (Baseline: 133.2 mmHg; Final: 126.6 mmHg) and comparisons at final assessment revealed a significant mean difference between groups, favouring the intervention group (-5.3 mmHg; p=0.035).

Table 6 - Changes in resting blood pressure [Data are mean (SD) or median (25th-75th percentile)]

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Intervention Group</th>
<th>time* group p</th>
<th>Difference at final assessment Mean (95% CI) p</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>136.5 (17.7)</td>
<td>132.0 (14.7)</td>
<td>133.2 (15.6)</td>
<td>126.6 (14.2)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>78.6 (10.0)</td>
<td>76.0 (8.9)</td>
<td>78.1 (10.2)</td>
<td>75.5 (9.3)</td>
</tr>
<tr>
<td>MBP (mmHg)</td>
<td>97.9 (11.9)</td>
<td>94.6 (10.1)</td>
<td>96.4 (11.5)</td>
<td>92.5 (10.3)</td>
</tr>
</tbody>
</table>

SBP: systolic blood pressure; DBP: diastolic blood pressure; MBP: mean blood pressure
Results

**Lipid profile, metabolic parameters and inflammatory biomarkers**

From baseline to the final assessment there were no significant changes or final differences between groups (Table 7) for lipids, glucose, glycated haemoglobin, insulin, HOMA-IR or QUICKI.

The category classification according to the clinical predictive value of C-reactive protein for future cardiovascular events of low (<1 mg/L), moderate (1-3 mg/L) and high (> 3mg/L) risk indicated that at baseline 24.4% (n=33) of the patients exhibited C-reactive protein levels inferior to 1 mg/L, 45.2% levels between 1-3 mg/L (n=61) and only 30.4% (n=41) levels superior to 3 mg/L (Ridker, 2003). The circulating levels of CRP, did not change from baseline to the end of the study in both groups (P>0.05). Despite the lack of significant time*group interactions for the biomarkers IL-6, TNF-α, at the final assessment the intervention group showed significant lower values compared to Control group for these two biomarkers (Table 7). Regarding adiponectin and leptin, no significant time*group interactions were observed as well mean differences between groups at final assessment.
Table 7 - Changes in lipids, metabolic parameters, and inflammatory biomarkers in the study groups [Data are mean (SD) or median (25th75th percentile)]

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Intervention Group</th>
<th>time*</th>
<th>Difference at final assessment</th>
<th>Final Diff</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BASELINE</td>
<td>FINAL</td>
<td>BASELINE</td>
<td>FINAL</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>P</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cholesterol (mg/dl)*</td>
<td>203.5 (175.5 - 223.2)</td>
<td>194.5 (174.5 - 216.0)</td>
<td>206.0 (181.5 - 232.5)</td>
<td>203.0 (181.0 - 230.5)</td>
<td>0.710</td>
</tr>
<tr>
<td>HDL (mg/dl)*</td>
<td>55.5 (46.5 - 71.3)</td>
<td>50.5 (40.8 - 63.3)</td>
<td>53.0 (45.0 - 63.5)</td>
<td>50.0 (43.0 - 59.5)</td>
<td>0.072</td>
</tr>
<tr>
<td>TG (mg/dl)*</td>
<td>115.8 (76.3 - 160.8)</td>
<td>112.5 (78.5 - 157.3)</td>
<td>124.0 (84.5 - 172.0)</td>
<td>111.0 (83.0 - 153.5)</td>
<td>0.539</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>120.4 (37.7)</td>
<td>121.4 (35.4)</td>
<td>125.5 (39.1)</td>
<td>126.6 (38.1)</td>
<td>0.984</td>
</tr>
<tr>
<td>VLDL (mg/dl)*</td>
<td>23.2 (15.3 - 32.2)</td>
<td>22.5 (15.7 - 31.5)</td>
<td>24.8 (16.9 - 34.4)</td>
<td>22.2 (16.6 - 30.7)</td>
<td>0.539</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>100.5 (89.0 - 111.0)</td>
<td>93.5 (85.0 - 110.0)</td>
<td>98.0 (87.5 - 117.0)</td>
<td>95.0 (86.0 - 112.0)</td>
<td>0.855</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.70 (5.50 - 6.05)</td>
<td>5.70 (5.40 - 6.03)</td>
<td>5.70 (5.30 - 6.05)</td>
<td>5.60 (5.20 - 6.00)</td>
<td>0.459</td>
</tr>
<tr>
<td>Insulin (mU/ml)*</td>
<td>9.9 (7.1 - 12.9)</td>
<td>9.6 (7.5 - 14.5)</td>
<td>9.9 (6.6 - 13.4)</td>
<td>10.3 (7.0 - 15.5)</td>
<td>0.480</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>0.14 (0.10 - 0.19)</td>
<td>0.13 (0.09 - 0.18)</td>
<td>0.13 (0.09 - 0.20)</td>
<td>0.14 (0.09 - 0.22)</td>
<td>0.565</td>
</tr>
<tr>
<td>QUICKI</td>
<td>0.34 (0.03)</td>
<td>0.34 (0.03)</td>
<td>0.33 (0.03)</td>
<td>0.33 (0.03)</td>
<td>0.545</td>
</tr>
<tr>
<td>PCR (mg/dL)</td>
<td>0.20 (0.11 - 0.29)</td>
<td>0.24 (0.16 - 0.40)</td>
<td>0.17 (0.07 - 0.37)</td>
<td>0.23 (0.15 - 0.48)</td>
<td>0.758</td>
</tr>
<tr>
<td>IL6 (pg/mL)</td>
<td>2.73 (1.16 - 6.02)</td>
<td>2.88 (1.70 - 5.43)</td>
<td>1.90 (0.96 - 2.99)</td>
<td>1.90 (1.06 - 2.88)</td>
<td>0.980</td>
</tr>
<tr>
<td>TNFα (pg/mL)</td>
<td>3.77 (3.05 - 4.83)</td>
<td>4.05 (3.45 - 5.41)</td>
<td>3.32 (2.28 - 4.53)</td>
<td>3.53 (2.31 - 4.69)</td>
<td>0.144</td>
</tr>
<tr>
<td>Adiponectin (mg/mL)*</td>
<td>8.61 (6.55 - 12.35)</td>
<td>8.97 (6.20 - 12.73)</td>
<td>9.26 (7.12 - 12.25)</td>
<td>8.87 (6.85 - 12.05)</td>
<td>0.096</td>
</tr>
<tr>
<td>Leptin (ng/mL)</td>
<td>11.3 (6.9 - 21.0)</td>
<td>13.6 (7.1 - 22.7)</td>
<td>12.0 (6.0 - 19.9)</td>
<td>9.4 (5.6 - 20.8)</td>
<td>0.621</td>
</tr>
</tbody>
</table>

HDL: high density lipoprotein; TG: triglycerides LDL: low density lipoprotein; VLDL: very low density lipoprotein; HOMA: homeostasis model assessment; QUICKI: quantitative insulin-sensitivity check index; CRP: C-reactive protein; hs: high-sensitivity; IL: interleukin; TNF: tumour necrosis factor.

* Variables transformed for analysis (not normally distributed)

Dietary intake

No differences between baseline and final assessment were observed between groups in dietary intake (group*time: P=0.656) and in final assessment (p=0.185).
DISCUSSION

Methodology discussion

To our best knowledge, this is the first Portuguese study in the context of primary prevention and familiar medicine, aiming to evaluate the impact of an educational program on daily physical activity behaviour, and simultaneously assessing regulation to exercise, traditional cardiovascular risk factors, and inflammatory biomarkers. This was a pragmatic intervention study, and its relevance and originality is based on the use of the existing human resources in Portuguese primary care units.

A major strength of this study is the context where the intervention was conducted. It is often described in the literature (Mancia et al., 2013) that preventive medicine at primary health care level is crucial to reduce the burden of non-communicable diseases, and particularly cardiovascular diseases related to the progression of atherosclerosis. Indeed, early sub-clinical initiation and progression of atherosclerosis develops silently without any manifestation of signs and symptoms (Mancia et al., 2013), but very often the individuals are under exposure of life-style risk factors such as reduced levels of physical activity and/or inappropriate foods and nutritional habits and smoking, and have also biological risk factors like overweight or obesity, hypertension, dyslipidemia, metabolic syndrome, type II diabetes, and low-grade chronic inflammation (Mancia et al., 2013; Stone et al., 2014). Therefore, early detection and management of risk factors have been recommended (Beeres, 2004; "Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: final report of the pooling project. The pooling project research group", 1978) under the lead of caregivers at primary care ("Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: final report of the pooling project. The pooling project research group", 1978). Previous studies showed that primary care interventions aiming to change behaviours and manage risk factors are successful in reducing risk factors and cardiovascular risk (Armit et al., 2009; Davies et al., 2008; Elley et al., 2003;
Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Parra-Medina et al., 2011; Reid et al., 2014).

Based on the aforementioned, we would like to emphasize that in the present study we have recruited and selected individuals that at least presented one risk factor, which is within the scope for early treatment of the possible causes of development of atherosclerosis and future manifestation of cardiovascular diseases. This can be considered a major positive feature of this study, since the target sample is within those individuals that need more attention because their cardiovascular risk level is negatively altered (Mancia et al., 2013; Stone et al., 2014), and eventually can benefit more from an educational and counselling intervention.

Although not being a randomized controlled trial, this study included a control group so that we can appreciate the independent effect of the intervention. Although the allocation in the two arms of the study was made by convenience of the participants, we did not observed any difference between the groups’ characteristics at baseline including motivation, which minimized the possible bias in allocation.

Regarding the intervention, this study complied with the methodology used in other studies. Indeed, in this study the sessions were face-to-face, although provided in group sessions. Furthermore, the duration of intervention was, in general, more extended than similar interventions previously reviewed (for ref’s see Chapter II).

Regarding the primary outcome of this intervention study, PA was our choice since it was previously showed that insufficient levels of PA is one of the most prevalent CVRF, and the first among those classified as being life-style modifiable CVRF (Goff et al., 2014). Moreover, the INTERHEART study showed that regular PA reduces the risk of acute myocardial infarction by 14% and the population attributable risk for the lack of PA was 12.2% (Yusuf et al., 2004). Reduced leisure time physical activity and a sedentary occupation are associated with an increased risk of ischemic heart disease death (Salonen et al., 1988). Therefore, interventions targeting the promotion of PA are within the scope of primary health care.
Another strong point in the present study was the use of accelerometry to evaluate daily physical activity. Within its scope (education and counselling) and clinical context (primary prevention), and taken into consideration the primary outcome (physical activity), this study is the first using objective measures of daily PA (accelerometry). In previous studies, physical activity was mostly assessed by questionnaire, although they usually over- or underestimate true physical activity energy expenditure and rates of inactivity (Prince et al., 2008). Objective measures offer more precise estimates of energy expenditure and remove many of the issues of recall and response bias (Dyrstad et al., 2014; Prince et al., 2008). The correlation between self-reported and objective measures of physical activity is known to be low-to-moderate (Dyrstad et al., 2014; Helmerhorst et al., 2012). Since it has been reported that beliefs and regulatory motives matters for any behaviour change, being either correlates or determinants or potential confounders, we strongly believe that the assessment of exercise motivation is a strength of our study design. In this study we reported an objective measure of different constructs of the regulation for exercise, and the effect of the educational program in the beliefs and regulatory motives toward exercise practice. Previous studies aiming to change lifestyle, namely the daily physical activity levels, have reported the use of techniques to modify the regulation for exercise, although they did not describe any objective measure for the different constructs of self-determination theory (Armit et al., 2009; Lakerveld et al., 2013).

Another strength of this study is related with the evaluation of some biological indicators associated to the most prevalent cardiovascular disease (ischemic heart disease) underlying atherosclerosis physiopathology. The selection of these indicators was performed according to the evidence arising from numerous studies showing that they are determinants or correlates of CVD (Orrow et al., 2012; Packard & Libby, 2008). For instance, the INTERHEART study reported that nine easily measured risk factors are associated with more than 90% of the risk of an acute myocardial infarction (Yusuf et al., 2004). Taking into account the results of the INTERHEART study, we have assessed physical activity, smoking, diet, obesity, lipids, hypertension, diabetes, which is a broad number of traditional risk factors.
Various studies reported that chronic low-grade inflammation is a main feature of atherosclerosis, and that it is associated with clinical manifestations of CVD. In the last years, investigations showed that inflammatory biomarkers can be modulated by exercise and/or PA and the traditional risk factors (Hamer et al., 2012; Rana et al., 2011; Ribeiro et al., 2010). A meta-analysis showed that exercise was associated with a decrease of CRP and IL-6 levels and that programs with a larger number of sessions tend to induce greater reduction in IL-6 levels (Hayashino et al., 2014).

Regarding the relationships between inflammatory biomarkers and traditional risk factors, Ryo et al showed that adiponectin levels were negatively correlated with waist circumference, visceral fat, serum triglycerides, fasting plasma glucose, fasting plasma insulin, and systolic and diastolic blood pressure in males and females, and positively correlated with HDL (Ryo et al., 2004). As the mean number of metabolic syndrome components increased, plasma adiponectin levels decreased (Ryo et al., 2004). Multiple studies have shown that elevated levels of IL-6 are associated with metabolic syndrome and increasing levels are associated with more severe metabolic syndrome (assessed by hypertriglyceridemia, hypertension, and fasting glucose levels) (Srikanthan et al., 2016). In a study of middle-aged adults with metabolic syndrome, elevated levels of TNF-α and other pro-inflammatory cytokines were associated with insulin resistance and hypertriglyceridemia. The TNF-α, IL-6, and leptin levels in these patients were higher than those levels in the control group, indicating that these cytokines directly correlated with metabolic syndrome (Balasoiu et al., 2014). CRP is a strong independent predictor of CVD (Srikanthan et al., 2016).

Similar findings were reported for dietary behaviors. Eating fruit and vegetables together with exercise and avoiding smoking could lead to about 80% lower relative risk for myocardial infarction (Yusuf et al., 2004).

Results discussion
The main findings of the present study indicate that the intervention did not promote changes in the primary outcome, the daily physical activity levels. Similar
findings were observed for secondary outcomes, such as lipids and metabolic parameters, and inflammatory biomarkers. However, there were significant changes in self-regulation for exercise, namely amotivation and intrinsic regulation for exercise, and waist circumference.

Regarding the primary outcome of this trial, physical activity, the results contrast with other RCT which we have systematically reviewed (Armit et al., 2009; Davies et al., 2008; Elley et al., 2003; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Reid et al., 2014).

In what concerns to the characteristics of the interventions (content, duration and way of delivering) in the aforementioned studies, compared to ours, were similar in the content (targeted behaviours, program messages), but somewhat different in the duration and way of delivering. For instance, regarding the duration, the interventions where PA has improved, ranged from 6 hours in one day or two halve days (Davies et al., 2008) up to 1 year (Parra-Medina et al., 2011; Reid et al., 2014). Considering the broad range of durations compared to that of our study we can’t attribute the lack of changes in PA in our study to this study design variable.

One important difference in our study to the other studies reviewed in which PA has improved, was the way of delivery the intervention. In our study sessions were designed for groups (approximately 10 participants in each group), while in the reviewed studies with positive results on PA, the sessions were delivered individually. It can be speculated that sessions delivered individually could result in a more effective retention on the message related with the need and the mode to change behaviours. However, this possible explanation could be somewhat denied by the fact that 6 out of 14 studies systematically reviewed have used the same approach (individual sessions) and no changes on PA were observed (Cochrane et al., 2012; Griffin et al., 2014; Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Lakerveld et al., 2013; Wister et al., 2007).

Another important topic that we must address is the use techniques for behaviour change. Indeed, although we have evaluated the self-regulation to PA, it is important to note that those who have the responsibility for delivering the sessions do not have the expertise on this motivational techniques, and haven’t
received any kind of previous training. This is an important issue, given that qualifications, experience and personal attributes of those who are in the leadership of the interventions could have influence on the success of changing the outcomes (Morgan et al., 2016), in particular PA.

Despite the above, it must be also emphasized that our results are similar to other RCT in which no changes in PA were found or PA changed both in intervention and control groups. Interestingly, Griffin et al (Griffin et al., 2014) described subjective and objective measures of PA, and reported no changes in PA after the intervention.

Of importance to the interpretation of our results is the fact of our participants in both groups were at baseline, on average, complying with the recommended amount of daily MVPA (intervention group: 33.6 min; Controls: 41.1 min), although this applies to apparently healthy people free of disease and not classified at high risk for cardiovascular events (ACSM, 2010; Myers, 2003). This could be one possible explanation for the failure to significantly increase PA in our study.

We are not saying that those who comply can’t improve, but additional changes are probably harder to achieve, eventually requiring more time for changing and/or the overcoming of external barriers not related with personal beliefs or attitudes regarding PA. It should be expected within the population a wide range of variation in the amount of daily PA, and therefore, changes related with interventions cannot disregard the initial levels of PA. This issue is a matter of interesting that should be addressed in future studies covering sensitive analysis to examine differences in response to interventions in those who are more physically active comparing to those that are insufficiently active.

Regarding regulation to PA, the differences observed in amotivation suggest that the educational program was successful to decrease the lack on intention to change PA behaviour. In addition, we also observed that the program was able to change the interest and satisfaction of participating in PA, as the item of intrinsic regulation improves significantly in the intervention group compared to the control group. According to Identified regulation, there was no significant group*time interaction (p=0.090) but differences between groups at final
measurements were observed. These are important results meaning that, at least, the participants in the intervention group start to consider that PA is an important health related issue that deserves their attention and eventually should be changed (ACSM, 2010; Beeres, 2004; Bordalo A, 2015). Despite these results, the daily levels of PA did not change significantly. According to the Transtheoretical Model of behaviour change (Prochaska et al., 1992) behavioural changes follow different stages and are time-dependent. Although not being identified by us, it is possible that the participants in our study are in the contemplation stage, meaning that the individuals were aware that a problem exists in relation to PA behaviour and they seriously think of overcoming it, but they still had not yet taken action or made any preparations; or they were in the the preparation stage, where individuals have decided to take action in the months after the intervention. Thus, the intervention duration could be insufficient to cover all the stages of behaviour change.

In this study we decided to recruit participants that have, at least, one cardiovascular risk factor. Overall, overweight/obesity (88.8%), dyslipidaemia (85.2%) and hypertension (84.4%) registered high frequencies. The same can be said to Type II Diabetes (27.4%). Interestingly, the frequency of medications used to treat this conditions ranged from 3% (fibrates) to 40.7% (angiotensin II receptor blockers), which leads us to refer that not all were under pharmacological therapies. These data also revealed that participants in this study are exposed to a number of independent CVD risk factors that are identified as triggers of atherosclerosis (DGS, 2013; Mancia et al., 2013).

We have reviewed systematically studies that used anthropometric and biological outcomes to assess the effects of the educational interventions. They measured body weight (Cochrane et al., 2012; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012), body mass index (Cochrane et al., 2012; Harris et al., 2012; Reid et al., 2014; Wister et al., 2007), waist circumference (Cochrane et al., 2012; Harris et al., 2012; Reid et al., 2014; Wister et al., 2007), blood pressure (Armit et al., 2009; Cochrane et al., 2012; Davies et al., 2008; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Reid et al., 2014), lipid profile
(Cochrane et al., 2012; Davies et al., 2008; Hardcastle et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Reid et al., 2014), fasting blood glucose (Harris et al., 2012; Reid et al., 2014), and HbA1c (Davies et al., 2008).

Davies et al. showed reductions in body weight compared with control groups with a decrease of 3.1% at 4 months and 3.2% at 12 months (Davies et al., 2008).

The effects of educational interventions on waist circumference were significant in 2 studies (Cochrane et al., 2012; Reid et al., 2014) and not significant in other 2 studies (Harris et al., 2012; Wister et al., 2007). In some studies, risk factors, such as high cholesterol levels (Davies et al., 2008; Hardcastle et al., 2013; Harris et al., 2012; Koelewijn-van Loon et al., 2009; Reid et al., 2014) and blood pressure (Armit et al., 2009; Cochrane et al., 2012; Harris et al., 2012; Reid et al., 2014), did not improve during the study period. However, other studies showed a significant decreased in blood pressure (Hardcastle et al., 2008; Hardcastle et al., 2013; Wister et al., 2007), total cholesterol (Hardcastle et al., 2008; Hardcastle et al., 2013; Wister et al., 2007) and triglycerides (Davies et al., 2008) after the intervention.

In this study we didn’t observe changes in body weight and BMI, lipid profile, fasting blood glucose, and HbA1c, which agreed with several studies above-referred.

In this study we found a significant group*time interaction on WC favouring the intervention group, and a significant difference at final assessment in SBP (-5.5 mmHg in the intervention group compared to control), despite the lack of a significant group*time interaction. A possible explanation for changes in WC or SBP might be the increment in light physical activity in the intervention group as documented in earlier trials (Howard et al., 2015). However, given that the changes in PA levels were not different between the two study arms, other reasons are candidate to explain the results. In regard to WC, the observed differences can be related with the inherent error of measurement, or eventually, to changes in diet, which can be extended to SBP, although we can’t truly examine this possible effect since we only have controlled the total energy intake (Kcal/day) and not the foods and nutrient composition. Changes on diet was a content approached in the appointments with physicians and in group sessions.
for the intervention group, but not a main target in the reinforcements deliveries. Therefore, future studies should go beyond the analysis of total energy intake and look to foods and nutrients.

Atherosclerosis is defined as proceeding from endothelial dysfunction (increased expression of adhesion molecules, diminished synthesis and bioavailability of endothelial dependent vasodilators such as, for instance, nitric oxide, and low grade chronic inflammation, which can be assessed through circulating levels of proinflammatory biomarkers such as acute phase proteins (e.g. C-reactive protein) and cytokines (for instance, IL-6 and TNF-) (Danesh et al., 2004; de Lemos & Lloyd-Jones, 2008). Taking into consideration the frequencies of several CVD risk factors, we should expect, at baseline, the values are elevated. However, looking at the biomarkers and comparing with median or average serum concentrations measured in healthy populations (Himmerich et al., 2006; Kim et al., 2011; Kleiner et al., 2013) the values we found are within the expectable to adult healthy people even considering the large age range. As for cytokines, also the values of measured adipokines could be considered as normal (Gijon-Conde et al., 2015). Nevertheless, the values of inflammatory biomarkers are in agreement with previous interventional studies in primary care settings (Koelewijn-van Loon et al., 2010; Koelewijn-van Loon et al., 2009; Lakerveld et al., 2013; Wister et al., 2007). Collectively, the results of this trial, drive us to raise the hypothesis that participants were not yet in an advanced stage of atherosclerosis progression, and consequently, the inflammatory biomarkers do not reflect the observed frequencies of CVD risk factors. Moreover, the fact that no differences were detected in daily physical activity levels in combination with the relatively small observation period, lead us to conclude it would not be expectable to see differences in secondary endpoints (the effect of PA is time and dose dependent). On the one hand, the high prevalence of CVRF, these factors are controlled according to the mean values. On the other hand, the baseline levels of PA already meet the recommendation for PA (ACSM, 2010).
Discussion

Study Limitations
Some limitations of the present study should be addressed. The sample size is relatively small compared to international studies. The allocation to the control and intervention groups was not randomized. This limitation was minimized by the fact that the two groups were similar at baseline regarding potential confounders such as clinical conditions, use of medications, age, sex, and for the majority of the study variables.
Another potential limitation is the fact that evaluators were not blind to the group assignment. However, this study intends to be a pragmatic intervention, and therefore it would be almost impossible that the evaluators were not aware of allocation.

Perspectives for Future Research
Implementing lifestyle interventions in everyday practice is challenging and requires further investigation. More research is needed to evaluate the benefits of group programs compared with brief counselling in Primary care. Primary care has high population reach and an important role in assessing risk and informing patients, and preventing chronic diseases.
Chapter IV: Conclusions
CONCLUSIONS

Considering the purposes defined for the present study and based on our findings, it seems reasonable to emphasize the following conclusions:

The systematic review of data previous published showed that educational interventions increase daily physical activity levels and that primary care interventions focusing healthy lifestyles improve cardiovascular risk score and risk factors.

The results of the present interventional study showed that intervention group did not improve physical activity, cardiometabolic risk factors, and biomarkers of inflammation compared to control group.

Intervention group had important repercussions on regulation for exercise concluding that educational program was successful to decrease the lack on intention to change PA behaviour. In addition, we also observed that the program was able to change the interest and satisfaction of participating in PA
REFERENCES


References


References


WONCA. (2011). *The European Definition of General Practice/Family medicine*. WONCA Europe. Dissertação de apresentada a

Appendix I. Patient file

Nome: 

Idade: 
Data de Nascimento: 

Peso: 
Altura: 
PC: 

Número acelerômetro: 
TA: 
FC: 

dia de colocação: 
código Acelerômetro: 

Fumador: 

Hipertensão: 

Diabetes: 

Dislipidemia: 

Educação: 

Doença CV familiar prematura: 

Medicação: 

Exercício: 
CONSENTIMENTO INFORMADO, LIVRE E ESCLARECIDO PARA REALIZAÇÃO DE ESTUDO: FACTORES DE RISCO METABÓLICOS E CARDIOVASCULARES, ACTIVIDADE FÍSICA E APTIDÃO CARDIORESPIRATÓRIA. ESTUDO DAS REPERCUSSÕES DE PROGRAMA DE EDUCAÇÃO E INTERVENÇÃO DE PREVENÇÃO PRIMÁRIA.

Confirmo que expliquei ao utente, doente ou seu representante, de forma adequada e inteligível, os objectivos e procedimentos necessários ao estudo acima referido, estando também à disposição para responder a todas as dúvidas e mencionados, detalhes relevantes sobre o estudo e/ou riscos eventuais e significativos. Também expliquei ao doente que todos os encargos são da responsabilidade da entidade do centro de investigação. O presente estudo decorrerá no âmbito do programa Doutoral em Actividade Física e Saúde, do Centro de Investigação em Actividade Física, Saúde e Lazer (CIADFEL) da Faculdade de Desporto da Universidade do Porto e suportará as teses de Doutoramento da Dra Ana Ramôa Castro e Drª Helena Leal.

É garantido que haverá ocultação de dados de identificação da pessoa, assim como serão minimizados e optimizados as deslocações ao centro de saúde. É igualmente garantido que a presente autorização pode ser retirada, em qualquer altura, sem que isso cause qualquer prejuízo ou afete os cuidados a prestar a pessoa.

Nome dos profissionais de saúde e responsáveis pela proposta:

Ana Ramôa Castro
Helena Leal

Data: .../.../...... Assinatura ..........................................................

(Este documento é feito em duplicado)
Por favor, leia com atenção todo o conteúdo deste documento. Não hesite em solicitar mais informações se não estiver completamente esclarecido/esclarecida. Verifique se todas as informações estão corretas. Se tudo estiver conforme, então assine este documento.

Declaro que concordo com o que me foi proposto e explicado pelo profissional de saúde que assina este documento, tendo podido fazer todas as perguntas sobre o assunto. Autorizo a realização do estudo analítico, procedimentos e de dados indicados nas condições em que me foram explicadas.

... ... ... ... ... ... ... ... (local), ... ... ... ... ... (data)
Assinatura

Se não for o próprio a assinar:
Nome: ...
BI/CD No: ...
Grau de parentesco ou tipo de representação: ...
Assinatura
Appendix

Appendix III: Booklet

Risco Cardiovascular e Actividade Física

Porquê preocupar-se com o colesterol?
- O infarto agudo do miocárdio e os acidentes vasculares cerebrais são algumas das principais causas de mortalidade em Portugal.
- Valores elevados de colesterol no sangue contribuem para obstruir as artérias, dificultando a circulação do sangue e a adequada irrigação do coração ou do cérebro e, em consequência, aumentando o risco de ter uma doença cardiovascular ou cerebrovascular.

Quando deve efectuar análises ao sangue para avaliar o seu colesterol?
- Isto dependerá da sua idade e do perfil de risco cardiovascular.
- O seu médico decidirá de acordo com as suas características com que periodicidade deve efectuar a avaliação do colesterol.

Existem vários tipos de colesterol?
- Eléutrico que sim. De início, avalia-se o colesterol total. Se o colesterol total estiver elevado, então será necessário determinar o chamado "boon" colesterol (colesterol HDL) e o chamado "mau" colesterol (colesterol LDL).
- Qual o valor de colesterol que o devo ter?
- Idealmente, o colesterol total deve ser inferior a 200 mg/dl e será bom que o colesterol LDL seja inferior a 100 mg/dl.
- Contudo, os valores normais variam de pessoa para pessoa conforme o risco cardiovascular de cada um.

Que factores podem contribuir para aumentar o risco de ter doença cardiovascular?
- Ser fumador
- Ter um estilo de vida sedentário
- Ter diabetes
- Ter hipertensão arterial (acima de 140/90 mmHg)
- Ter obesidade ou excesso de peso
- A idade: homens acima dos 45 anos e mulheres acima dos 55 anos têm um risco aumentado
- Ter antecedentes de doença cardíaca em familiares com idade abaixo dos 60 anos
O que é a hipertensão arterial? Qual o significado dos números?

Os valores da pressão arterial (tensão arterial) são representados por duas medidas, dois números separados por uma barra (por exemplo 120/80).

Em Portugal, existe o hábito de introduzir uma casa decimal e dizer 120,0/80,0 em vez de 120/80, o que seria mais correto e que se pratica no nível mundial, que é dizer 120/80.

O primeiro valor (120) corresponde ao pressão arterial sistólica e ocorre no momento em que o coração se contrae e bombeia o sangue para o organismo. É o maior pressão que o sangue atinge e dá a designação popular de "pressão".

O segundo valor (80) corresponde ao pressão arterial diastólica e ocorre quando o coração relaxa para se enchêr de sangue. Nesse momento, a pressão arterial atinge o seu valor mais baixo e dá a designação popular de "pressão mínima".

São de hipertensão arterial quando uma pessoa apresenta em pelo menos duas ocasiões diferentes um dos valores (máximo ou mínimo) ou ambos acima de 140/90. A pressão arterial normal corresponde a um valor inferior ou igual a 120/80. Entre 120/80 e 140/90 diz-se que a pessoa se encontra num estádio de pré-

hipertensão, pois apresenta um risco maior de vir a ter hipertensão arterial.

Porque é preocupante?

Imagine que numa casa a água chega na cozinha com uma pressão superior àquela para que a cozinha fique construída. Em poucos tempos surgirão avermutes, fugas, desgaste da bomba, mau funcionamento das torneiras, etc. É exactamente isso que se passa no corpo humano. Uma vez diagnosticada, a hipertensão arterial é um problema crónico que no futuro poderá provocar problemas como enfarte, insuficiência cardíaca, acidentes vasculares cerebrais e insuficiência renal.

Quais os sintomas?

Uma das preocupações da hipertensão arterial relaciona-se com o facto de, na maioria das vezes, um problema silencioso, ou seja, uma pessoa pode ser hiper tensiva durante anos sem sentir qualquer sintoma. Da importância da medida da pressão arterial.

Nalguns casos, principalmente quando a pressão arterial atinge valores muito elevados (ex. 200/110) podem ocorrer sintomas como cefaleias (dores de cabeça), tonturas, cansaço fácil ou perda de visão transitória.

O que pode fazer?

O primeiro passo do tratamento de hipertensão arterial passa pela adoção de estilos saudáveis de vida:

- Não fumar.
- Reduzir a ingestão de sal, bebidas alcoólicas e cafeína.
- Abrace o seu peso ideal.
- Pratique exercício físico de forma regular.
- Deixe rica em frutas, vegetais e pobre em gorduras.

Também é muito importante:

- Cumpra corretamente o tratamento, seguindo as indicações do seu médico.
- Não fale aos familiares de vaiagem.
- Não tire mais medicamentos por sua conta, pois podem agravar os valores da pressão arterial.
- Se é diabético, ainda é ainda mais importante que contegem um bom e canal de coordenação da sua diabetes e da sua hipertensão arterial.

Quando deve consultar o seu médico de família?

- Quando a sua pressão arterial não está bem controlada (superior a 140/90) apesar de cumprir correctamente o tratamento.
- Deve recorrer ao seu médico de forma mais rápida se lhe for dado a pressão arterial acima de 150/100.
- E será ainda mais urgente se esses valores forem acompanhados de dor de cabeça muito intensa, tonturas ou vomitivo, visão turva, dor no peito ou sensação de falta de ar.
Actividade Física

Porque devo fazer exercício?

O exercício físico pode ajudar:
* a prevenir infartos e problemas cardiacos
* a reduzir o stress
* a aumentar a sensação de bem-estar
* a melhorar a qualidade do sono
* a perder peso - mas para isso DEVE ajustar os seus hábitos alimentares
* a diminuir os riscos de coelheiros
* a normalizar o pressão arterial
* a diminuir zonas musculares e adiposas

O que devo e não deve sentir durante o exercício?

Dever:
* Respirar mais rapidamente – contudo DEVE poder falar normalmente enquanto faz exercício
* Sentir-se mais quente
* Transpirar
* Sentir o coração a bater mais depressa

Não dever:
* Sentir dor no peito - se sentir de devar e desconfortável
  * Esperar algum tempo até que a dor desapareça antes de continuar
  * Procurar o seu Médico de Família

Que precauções devo ter quando vou fazer exercício?

* Use roupas leves e o calçado adequado.
* Escolha momentos do dia nos quais a temperatura não seja muito elevada nem muito baixa.
* Procure fazer exercício antes de grandes refeições.
* Não fazer exercício em jejum.

Como sei que o exercício que faço é adequado?

* No dia seguinte ao exercício não se sinta demasiadamente cansado.
* Respira mais rapidamente, mas consegue falar normalmente enquanto faz exercício.
* Sente-se mais quente e transpira durante o exercício.
* Com o passar dos dias sente a sua capacidade para fazer exercício aumentar.

Aspectos Importantes a lembrar sobre o exercício

* Lembre-se que o exercício não provoca uma leve falta de ar (deve sentir a necessidade de respirar mais rapidamente do que em repouso) durante e após a sua execução não está a tomar-lhe mais do que física.
* Provavelmente vai ter dias bons e dias ruins. Nos dias ruins não tente fazer mais do que o planeado. Nos dias maus não procure uma resposta exata, intuitivamente, se não tiver feito muito mais do que o habitual nos últimos dias, isso pode explicar o porquê de se sentir mais cansado.
* Almoxar sempre antes e após o exercício.
* Siga o plano de exercício – tentar fazer muito mais do que o recomendado pode abalar a sua recuperação.
* Não ignore a presença de dor no peito.
Appendix

À medida que seu objetivo é alcançar pelo menos 30 minutos de exercício de intensidade moderada pelo menos 5 dias por semana (preferencialmente todos os dias),

**Objetivo**
- O seu objetivo é realizar pelo menos 30 minutos de exercício de intensidade moderada pelo menos 5 dias por semana (preferencialmente todos os dias).

**Onde passar fazer exercício?**
- Nos seus jardins, parques ou em casa.
- Procure fazer exercício em locais frequentados por outras pessoas. Evite locais isolados.
- Procure locais com terreno plano e plano regular.

**O que tipo de exercício posso fazer?**
- Caminhar
- Correr
- Andar de bicicleta

**Como fazer mais de 10 minutos?**
- A sua capacidade física ainda não está a desejável, mas está a melhorar e ainda pode melhorar mais!
- Continue a fazer as suas caminhadas a uma velocidade mais lenta do que uma vez por dia.
- Proporcione um período de 10 minutos para dois períodos (por exemplo, 10 min + 5 min = conseguir fazer 10 + 10 minutos).
- Faça um período de exercício matinal e outro ao final do dia ou antes de ir ao trabalho e ao final do dia.
- Quando conseguir a parte do exercício que suporta em dois períodos de exercício com facilidade (por exemplo, realizar o exercício com raça velocidade fácil dois dias seguidos), procure aumentar a duração de um dos períodos de dia. De tal modo que fique entre raça velocidade fácil e raça velocidade difícil.
- O seu objetivo agora será caminhar durante 20 minutos por dia.

**Como fazer mais de 20 minutos?**
- Esta no bom caminho, está próximo da meta dos 30 minutos e um pouco mais de vontade vai lá chegar!
- Continue a fazer as suas caminhadas e procure atingir 30 minutos de duração.
- Por exemplo, aumente em 5 minutos a duração da caminhada.
- Quando perceber que suporta o exercício com facilidade (por exemplo, realizar o exercício com raça velocidade fácil dois dias seguidos) aumente mais 5 minutos a duração, ou seja, caminhe 30 minutos de forma contínua.

**Plano de exercício**

- A sua capacidade física está longe da desejável, mas você pode melhorar o mais possível do seu.
- Precisa de aumentar a sua capacidade física.
- Comece por fazer caminhada.
- Não perca distâncias quanto não desenhe mais do que o tempo de caminhada que consegui realizara.
- Lembre-se que deve caminhar e a um velocidade que o faça respirar mais rapidamente e sentir-se mais quente.
- Quando começar a perceber que suporta o exercício com maior facilidade (por exemplo, caminhar o exercício com raça velocidade fácil dois dias seguidos), procure aumentar a sua duração. De tal modo que fique entre raça velocidade fácil e raça velocidade difícil.
- O seu objetivo agora será caminhar mais de 10 minutos.
Appendix

Como posso ainda melhorar?

- Quanto mais bem e gaste mais tempo no exercício, mais benefícios você obterá.
- Se aumentar a duração do treino de 30 minutos para 60 minutos por dia, pode-se aumentar a duração do exercício.
- Se você não tem disponibilidade de tempo para fazer mais do que 30 minutos de exercício, divida o exercício para mais do que um período por dia (por exemplo, 30 + 15 = 30 + 30).

Como se manter ativo ao longo da vida?

- Incorpora o exercício na sua rotina diária.
- Use as escadas em vez do elevador ou dos escadas rolantes.
- Desloque-se sempre que possível e peça por exemplo, para ir ao banheiro e para o café, para o trabalho.
- Permanece em redor.
- "Recrear" amigos para as suas caminhadas – não convirna no estô, convirna a caminhar.

Como ganhar do exercício e não desistir?

Algumas pessoas sentem dificuldade em manter regularmente a prática de exercício físico, porque precisam passar o tempo livre ou a ver televisão ou simplesmente a repousar sentada no sofá.

Se é assim, você pode até ver os benefícios em fazer o exercício em suas rotinas diárias que gostam e que têm real prazer com ela.

Existem muitos exercícios que podem ser feitos com o apoio de um amigo.

Sugestões para escolher o tipo de exercício:

- O exercício deve ser divertido: Se não for, vai acabar por desistir.
- Comece com o que gostar, seja caminhar, andar de bicicleta, etc. (tente o que você gosta e faça o que você gostar). (tente o que você gosta e faça o que você gostar).
- Se você faz regularmente exercício físico, pode começar a tentar diferentes tipos de exercício antes de experimentar um novo.
- Se você faz regularmente exercício físico, pode começar a tentar diferentes tipos de exercício antes de experimentar um novo.
- Para ter benefícios e para que se sinta bem é goste realmente de fazer exercício, este deve ser adequado.
- Se não tiver motivação suficiente para fazer exercício, talvez ele não seja o melhor para o seu nível de condicção física.

- Se possível, faça exercício com alguém misturado. Ou seja, procure fazer diferentes tipos de exercício durante e entre os períodos de exercícios.
- Tente incorporar o exercício em sua rotina diária. Assim torna-se um hábito, tal como ir ao supermercado!
- Lembre-se que o exercício não tem que necessariamente ser organizado ou para ser efetivo. Caminhar em passo rápido 30 a 60 minutos por dia é tão bom para si como praticar um esporte ou ir ao ginásio!
**Exemplos de Exercícios de Alargamento**

Faça cada exercício de alargamento antes e depois de exercícios. Deve sentir um ligeiro desconforto (não dor) no músculo que está a alargar. Faça 3 alargamentos de 15 a 20 segundos de cada músculo. Deve fazer os alargamentos de 1º, os procurar de apoio para se equilibrar apoie-se em algo estável.

1. Alargamento dos músculos anteriores da coxa (quadricipites)

2. Alargamento dos músculos de perna (tibíáceo sural)

3. Alargamento dos músculos posteriores da coxa (sócio-glúteo)

4. Alargamentos dos músculos da coluna

5. Outros Alargamentos
Appendix IV. BREQ-2 Questionnaire

Estamos interessados nas razões das pessoas, para se envolverem ou não na prática de exercício físico. Usando a escala abaixo, por favor indique qual o nível mais verdadeiro para si. Relembramos que não há respostas certas ou erradas nem perguntas traçoeiras. Queremos apenas saber como é que se sente em relação ao exercício.

<table>
<thead>
<tr>
<th>PORQUE FAZ EXERCÍCIO?</th>
<th>Não é verdade para mim</th>
<th>Algumas vezes é verdade para mim</th>
<th>Muitas vezes é verdade para mim</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Faço exercício porque outras pessoas dizem que devo fazer…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Sinto-me culpado/a quando não faço exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Dou valor aos benefícios/vantagens do exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Faço exercício porque é divertido…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Não vejo porque é que tenho de fazer exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Participo no exercício porque os meus amigos/família dizem que devo fazer…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Sinto-me envergonhado/a quando falto a uma sessão de exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. É importante para mim fazer exercício regularmente…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Não percebo porque é que tenho de fazer exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Gosto das minhas sessões de exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Faço exercício porque os outros vão ficar insatisfeitos comigo se não fizer…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Não percebo o objetivo de fazer exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Sinto-me fracassado/a quando não faço exercício durante algum tempo…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Penso que é importante fazer um esforço por fazer exercício regularmente…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Aacho o exercício uma atividade agradável…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Sinto-me pressionado/a pela minha família e amigos para fazer exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Sinto-me irrequieto/a se não fizer exercício regularmente…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18. Fico bem disposto e satisfeito por praticar exercício…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19. Penso que o exercício é uma perda de tempo…</td>
<td>0 □ 1 □ 2 □ 3 □ 4 □</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix V. 4-Day Food Recall Questionnaire

INSTRUÇÕES PARA O PREENCHIMENTO DO DIÁRIO ALIMENTAR DE 4 DIAS

Este diário alimentar tem como objectivo avaliar a sua ingestão alimentar e consiste no registo de tudo aquilo que comeu e bebeu ao longo do dia. Procure preencher o registo de uma forma sincera, indicando aquilo que realmente come e não o que pensa que seria correcto comer.

Por favor, anote tudo o que comer ou beber durante 4 dias desta semana (3 dias da semana e 1 dia de fim-de-semana).

Faça descrições pormenorizadas dos alimentos e bebidas consumidos como, por exemplo, o tipo de pão (trigo, mistura, integral, etc.) ou o tipo de leite (gordo, meio gordo ou magro). Mencione também o tipo de confeção culinária como, por exemplo, carne de vaca grelhada, ovo estrelado, lombo assado, costeleta de porco frita em margarina. No caso de pratos compostos tente mencionar a quantidade aproximada de cada alimento. Mencione sempre que possível a marca do alimento. Quando ingerir chá ou outros alimentos em que adicione açúcar ou outro edulcorante, não se esqueça de os referir, bem como as suas quantidades. Inclua sempre tudo aquilo que comeu e/ou bebeu fora de casa (p.ex., num café ou em casa de um amigo). Não se esqueça, também, de registar tudo o que comeu e/ou bebeu no intervalo das refeições como, por exemplo, bolachas, sumos, açúcar, bolos, etc.

Como fazer o registo: inicie com a página correspondente ao dia de registo; assegure-se, por favor, que preencheu todas as partes correspondentes à – HORA, LOCAL, ALIMENTOS E BEBIDAS CONSUMIDOS, QUANTIDADE.

Quanto às quantidades e aos tamanhos das porções

Use medidas caseiras como, por exemplo, 1 colher de chá, 4 colheres de sopa, 1 tigela, 1 chávena almoçadeira. Seguidamente é apresentado um quadro sumário de como pode ser efectuada a descrição da quantidade de alimentos/bebida.

<table>
<thead>
<tr>
<th>Alimento</th>
<th>Medidas (exemplos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sopa</td>
<td>1 concha de sopa ou 1 tigela ou 1 prato chelo</td>
</tr>
<tr>
<td>Massa, arroz, leguminosas (feijão, grão, lentilhas, etc.)</td>
<td>1 colher de sopa ou 1 colher de servir ou ½ prato</td>
</tr>
<tr>
<td>Batata</td>
<td>1 batata média (tamanho de um ovo), grande ou pequena</td>
</tr>
<tr>
<td>Carne, peixe</td>
<td>1 porca média, grande, pequena ou 1 sardinha, fariná, douxada, etc.</td>
</tr>
<tr>
<td>Legumes cozidos ou crus</td>
<td>1, ½, ¼ prato ou 1 chávena almoçadeira</td>
</tr>
<tr>
<td>Fruta</td>
<td>1 peça média, grande, pequena ou 1 rociela ou 1 fatia</td>
</tr>
<tr>
<td>Flambre, queijo, mortadeira, chouriço</td>
<td>1 fatia grossa, fina</td>
</tr>
</tbody>
</table>
## Appendix

### Pequeno-Almoço

<table>
<thead>
<tr>
<th>Horário</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td>8h</td>
<td>Casa</td>
<td>Leite meligordão com cereais (Macarrão) sem açúcar</td>
<td>160ml de leite + 3 colheres dessa em cereais</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pão integral com fiasbre</td>
<td>1 pão médio (40g) + 1 fãta fina de fiasbre</td>
</tr>
</tbody>
</table>

### Meio-dia

<table>
<thead>
<tr>
<th>Horário</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:30</td>
<td>Casa</td>
<td>Sopa de legumes (cuscuz, batata, couve, cebola)</td>
<td>1 prato (2 conícos)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bife de vaca grelhado</td>
<td>1 prato médio 100g</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Arroz de ervilhas</td>
<td>2 colheres dessa de arroz + 1 colher de sopa de ervilhas</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alface sem adição de aneto</td>
<td>½ prato</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maçã + água</td>
<td>1 maçã média + 1 conico</td>
</tr>
</tbody>
</table>

### Almoço

<table>
<thead>
<tr>
<th>Horário</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td>15h</td>
<td>Casa</td>
<td>Pão de trigo com manteiga</td>
<td>1 pão médio + 1 colher de chá de manteiga</td>
</tr>
</tbody>
</table>

### Jantar e Ceia

<table>
<thead>
<tr>
<th>Horário</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td>19h</td>
<td>Casa</td>
<td>Sopa de legumes (cuscuz, batata, couve, cebola)</td>
<td>1 prato (2 conícos)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Massa com cebola de porco grelhada</td>
<td>1 conetela média (100g) + 4 colheres dessa de massa</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Morango com açúcar</td>
<td>5 morangos + 1 colher de chá de açúcar</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Soro (nectar de pira)</td>
<td>200ml</td>
</tr>
<tr>
<td>22h</td>
<td>Casa</td>
<td>Leite meio-gordão simples</td>
<td>1 copo (79 ml)</td>
</tr>
</tbody>
</table>
## Dia da Semana: ______________

## Data: __/__/_____

<table>
<thead>
<tr>
<th>Hora</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Dia da Semana: ________________  Data: __/__/______

<table>
<thead>
<tr>
<th>Hora</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Dia da Semana: ________________  Data: __/__/______

<table>
<thead>
<tr>
<th>Hora</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
O próximo dia de registo será um dos dias do fim-de-semana (sábado ou domingo). Por favor, registe tudo o que comeu e bebeu desde a hora que se levantou até à hora de dormir.

Dia de **FIM-DE-SEMANA:** ________________  Data: __ / __ / _______

<table>
<thead>
<tr>
<th>Hora</th>
<th>Local</th>
<th>Alimento</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Indique por favor, se tomou (durante o ano) algum suplemento alimentar (multivitaminico, etc.):  □ sim □ nunca

Se sim, indique o número __________ e a frequência: □ todos os dias □ 1-3x/por ano □ 4-9x/por ano □ 10x/ano
Appendix VI. List of the conference communications and journal papers related with this work

Comunications


LUCIMERE BOHN, HELENA LEAL, ANA RAMOA, NORTON OLIVEIRA, GUSTAVO SILVA, JOSE OLIVEIRA (2014). Physical inactivity and arterial stiffness did not change after a lifestyle educational and counselling intervention. Comunicação em poster apresentada no World Congress of Cardiology, Melbourne, Austrália, 4-7 Maio

ANA RAMOA, LUCIMERE BOHN, HELENA LEAL, JOSE OLIVEIRA (2014). Physical activity motivation: impact of educational program in primary care. Comunicação em poster apresentada no World Congress of Cardiology, Melbourne, Austrália, 4-7 Maio


Appendix

Publications


LUCIMERE BOHN, HELENA LEAL, ANA RAMOA, NORTON OLIVEIRA, GUSTAVO SILVA, JOSÉ OLIVEIRA (2014). Physical inactivity and arterial stiffness did not change after a lifestyle educational and counselling intervention. Global Heart, 9(1S): e131-e132
