Biomarkers, 2008; 13(7–8): 671–679

informa
healthcare

Effect of intensive lifestyle intervention on C-reactive protein in subjects with impaired glucose tolerance and obesity. Results from a randomized controlled trial with 5-year follow-up

JONAS ANDERSSON[1], KURT BOMAN[1], JAN-HÅKAN JANSSON[1], TORBJÖRN K. NILSSON[2], & BERNT LINDAHL[3]

[1]*Department of Medicine and Geriatrics, Skellefteå County Hospital, Skellefteå, Sweden,*
[2]*Department of Clinical Chemistry, Örebro University Hospital, Örebro, Sweden and*
[3]*Department of Public Health and Clinical Medicine, Umeå university, Umeå, Sweden*

Abstract
C-reactive protein (CRP) is a marker of metabolic and cardiovascular disease. To study the effects of lifestyle on CRP in a high-risk population we conducted a randomized controlled trial on 200 obese subjects (BMI >27 kg m^{-2}) with impaired glucose tolerance recruited from primary care settings. They were randomized to either a 1-month stay at a wellness centre focusing on diet, exercise and stress management (intervention group) or 30–60 min of oral and written information on lifestyle intervention (control group). A significant reduction of CRP was observed after 1 month and 1 year in the intervention group. They reduced their CRP levels more than the control group 1 year after intervention ($p=0.004$). In conclusion lifestyle intervention can decrease CRP in obese individuals with impaired glucose tolerance for up to 1 year. Further research is needed to evaluate whether the CRP level reduction translates into a decreased risk for cardiovascular morbidity.

Keywords: *Health behaviour, exercise, weight loss, C-reactive protein, insulin resistance, obesity*

(Accepted 1 December 2008)

Introduction

Obesity and impaired glucose tolerance are associated with a wide range of cardiovascular diseases (CVDs) (DeFronzo & Ferrannini 1991, Reaven 1998). Obese individuals have an elevated high-sensitivity C-reactive protein (CRP) level, possibly mediated via interleukin (IL)-6 secreted from adipose tissue (Yudkin et al. 1999). This is suggested to reflect a systemic low-grade inflammation (Visser et al. 1999). Even CRP elevations at this low level are independently associated with an increased risk of an array of CVDs in individuals with no previous history of CVD as well as after established CVD (Bassuk et al. 2004).

CRP has also been associated with several features of the metabolic syndrome (Fröhlich et al. 2000), and is an independent predictor for the development of type 2

Correspondence: Jonas Andersson, Department of Medicine, Skellefteå County Hospital, Skellefteå, S-931 86, Skellefteå, Sweden. Tel.: +46 910 771 294. E-mail: jonas.so.andersson@vll.se

ISSN 1354-750X print/ISSN 1366-5804 online
DOI: 10.1080/13547500802661266

diabetes (Pradhan et al. 2001). Altogether, CRP might not only be a predictor of atherosclerotic disease, the inflammatory disease of the vessels (Ross 1999), but also a measure of the components of the metabolic syndrome and obesity leading to atherosclerosis. Today we have an increasing body of evidence of the beneficial effects of lifestyle change (Galani & Schneider 2007), but there is still little knowledge of how to obtain and maintain this healthier lifestyle.

We hypothesize that increased physical activity and weight reduction should result in a decrease in CRP. Therefore our primary aim was to study whether or not CRP could be reduced through lifestyle intervention in obese individuals with impaired glucose tolerance. A secondary aim was to evaluate if changes of CRP could be maintained over time.

Methods

Between the years 1985 and 1994 the Västerbotten Intervention Programme (VIP) (Weinehall 1997), a community intervention programme on CVD and diabetes, examined approximately 28 000 subjects aged 30, 40, 50 and 60 years. All participants with a normal fasting glucose value were offered an oral glucose tolerance test (OGTT) according to World Health Organization (WHO) standards (WHO 1985). From the VIP database an invitation was sent out by mail to the 650 individuals who fulfilled the inclusion criteria defined as an abnormal OGTT and a body mass index (BMI) >27 kg m^{-2}; 345 individuals expressed interest in participation and accepted the randomized design of the study.

We excluded 41 subjects who had already participated in a lifestyle modification programme and three subjects who were deemed too ill to participate. The Glucose Tolerance Study within VIP (GT-VIP) was formed by the remaining 301 subjects of whom 100 were randomized to the intensive intervention group (IIG) and 100 to the usual care group (UCG) (Lindahl 1999). The remaining subjects ($n=101$) were assigned as substitutes. Twenty substitutes were enrolled in the IIG and eight in the UCG. Another six participants withdrew their participation from the UCG before the study start, but for logistic reasons they were not replaced by substitutes. The participants in the IIG were admitted to the wellness centres within 4–9 weeks from randomization. The reason for this delay was purely practical as the centres could not admit all subjects at once. Baseline was set to the time of admittance to the wellness centre. In the present substudy, one subject in the IIG was excluded as the CRP level at baseline was missing, thus leaving 99 participants in the IIG and 94 participants in the UCG.

The standard programme (usual care group, UCG)

The participants randomized to the UCG underwent a health survey with physical examination, an OGTT and blood sampling at their health-care centre or by their general practitioner. The survey ended with a counselling session for 30–60 min conducted by a specially trained nurse. The participants were given both oral and written advice (see Appendix) focusing on lifestyle intervention towards impaired glucose tolerance and obesity. The protocol and a new short counselling session were repeated at follow-up after 12 months.

The intensive intervention programme (intensive intervention group, IIG)

The participants randomized to the IIG were divided into two groups and 50 individuals were simultaneously admitted for a 1-month stay with full room and board at Sorsele ($n=20$) or at Vindeln ($n=30$) wellness centres owned by the County Council. A physical examination with blood sampling and an OGTT were conducted after an overnight fast on the first or second morning from admittance.

The programme included approximately 140 h of scheduled activities including exercise of low to moderate intensity daily for 2.5 h, e.g. brisk walks, gymnastics, cycling and swimming. The diet served contained approximately 20% of energy from fat and a relatively high fibre content. Recommended portion sizes were calculated to approximately 7.6 MJ (1800 Kcal) for men and 6.3 MJ (1500 Kcal) for women leading to a slow but persistent weight decline. Alcoholic beverages were not allowed and smoking cessation was strongly encouraged. Useful and health-promoting coping strategies together with stress management and relapse-prevention techniques were emphasized (Marlatt & Gordon 1985). The participants were encouraged to make plans on how to incorporate healthy lifestyle changes in everyday life. The examination protocol, together with additional learning sessions, were repeated during a 4-day stay at follow-up 12 months later. A phone call, asking for the status of the lifestyle change, was placed to the participants in the IIG at 6 months and 2 years from the study start. Furthermore, in both study groups similar follow-up visits, including a health examination and blood sampling, were carried out at 3 and 5 years.

A power calculation showed that 99 participants in each group with a standard deviation of 5 would detect a decrease in CRP from 3 mg l^{-1} to 1 mg l^{-1} at a power of 80%. As CRP levels >3 mg l^{-1} are associated with higher cardiovascular risk and <1 mg l^{-1} with lower cardiovascular risk such a decrease would potentially have a clinical importance (Bassuk et al. 2004, Pearson et al. 2003).

Measurements

Body weight was measured in light indoor clothing and recorded to the nearest kilogram. Height was measured to the nearest centimetre without shoes. Smokers were defined as those reporting daily smoking; ex-smokers and occasional smokers were defined as non-smokers. An OGTT was performed according to the WHO standard using a 75-g anhydric glucose load with measurement of plasma glucose after 2 h (WHO 1985). Venous blood was sampled in K3-EDTA tubes in the morning after an overnight fast for the analysis of fasting plasma insulin, plasma total cholesterol and triglyceride concentrations. Insulin was measured using a highly sensitive two-site sandwich enzyme-linked immunosorbent assay (Andersen et al. 1993). Total cholesterol and triglycerides were analysed using kits from Roche/Boehringer (Mannheim, Germany). Low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol were measured by direct, homogeneous assays based on detergent treatment of the serum or plasma (N-geneous™ HDL-c and N-geneous™ LDL reagents, respectively, from Genzyme Corporation, Cambridge, MA, USA). CRP was determined in plasma with Tina-quant CRP (Latex) high-sensitivity assay using a Hitachi 911, a chemistry analyser with an ISE System and phonometric measuring device. All samples were thawed and analysed at the same time.

Statistical analysis

Statistical Package for the Social Sciences version 11.5.1 (SPSS Inc., Chicago, IL, USA) was used for the statistical analysis. Several variables including CRP had a skewed distribution and analysis was in those cases performed using non-parametric tests. The Mann–Whitney *U* test was used for calculations between the two treatments groups and the Wilcoxon signed ranks for calculations within the groups. In linear regression analysis the dependent variable CRP was log-transformed. Relationships between changes in CRP (from baseline to a maximum decrease in CRP) and changes in body weight, glucose and insulin were analysed with linear regression in the IIG. Differences between groups of categorical variables were calculated using χ^2 tests with $p < 0.05$ considered statistically significant. Analysis was made according to intention to treat.

Approvals

The study was approved by the Research Ethics Committee at Umeå University. The participants gave informed consent according to the Helsinki Declaration.

Results

Baseline data on the 193 patients enrolled in the study are described in Table I. There were no differences between treatment groups in age, blood pressure, total

Table I. Baseline characteristics.

	Intense intervention ($n=99$) Mean±SD (Q1/median/Q3)	Usual care ($n=94$) Mean±SD (Q1/median/Q3)	*p*-Value
Age (years)	52.5±9.1 (50/60/60)	53.8±8.3 (50/60/60)	0.33
Sex (M/F)	33/66	37/56	0.37
Smokers (yes/no) (missing)	7/75 (17)	9/76 (9)	0.79
C-reactive protein (mg l^{-1})	4.9±6.6 (1.5/2.8/5.9)	3.3±3.7 (1.2/2.1/3.6)	0.021
Body weight (kg)	86.7±10.4 (79.1/85.5/93.9)	84.1±11.0 (76.6/81.5/91.4)	0.031
Body mass index (kg m^{-2})	31.2±3.3 (29.0/30.5/33.1)	30.2±3.4 (28.0/29.3/32.0)	0.018
Systolic blood pressure (mmHg)	140.4±19.1 (125/142/155)	141.3±19.2 (130/140/150)	0.84
Diastolic blood pressure (mmHg)	84.4±9.8 (77/85/92)	85.9±9.8 (80/85/92)	0.47
Total cholesterol (mmol l^{-1})	5.7±1.25 (4.8/5.6/6.6)	5.7±0.93 (5.0/5.6/6.2)	0.97
Triglycerides (mmol l^{-1})	1.89±0.86 (1.24/1.67/2.38)	2.09±0.87 (1.39/1.98/2.53)	0.048
Fasting plasma insulin (pmol l^{-1})	55.6±32.7 (33/47/71)	73.6±54.0 (45/61/68)	0.00
Use of statin (yes/no)	12/84	17/76	0.32
Use of antihypertensive drug (yes/no)	26/73	23/71	0.87
Previous myocardial infarct (yes/no)	1/98	2/92	0.61

cholesterol or proportion of men and women. Neither were there any differences in numbers of smokers or use of antihypertensive or statin treatment. At baseline the IIG had a higher BMI, higher CRP level and lower triglycerides and fasting plasma insulin level than the UCG. The IIG had a greater number of CRP values above 10 mg l^{-1} although the difference was not significant (10 vs 5 mg l^{-1}, $p = 0.22$).

Table II shows the *p*-values for CRP at the different times of follow-up. As smokers have higher CRP levels and patients treated with statins have lower CRP levels, results were also calculated excluding these groups. Data were also analysed excluding CRP levels >10 mg l^{-1} as those subjects could be biased by a clinical infection.

At the 1-year follow-up there was no difference in CRP between the two treatment groups. However, when comparing the change in the CRP level from baseline to the 1-year follow-up between the groups there was a significant reduction of the CRP level in the IIG group compared with the UCG group (Table II). At the 3-year follow-up 87 subjects remained in the IIG and after 5 years 79 subjects remained. For the UCG the numbers were 85 and 84, respectively.

Regardless of whether all subjects were analysed or if cases were excluded, as seen in Table II there was always a strong significant reduction in CRP in the IIG group

Table II. *p*-Values at different times between and within groups. *p*-Values after exclusion of smokers, statin users and C-reactive protein (CRP) levels >10, respectively, are also shown.

	Within groups		Between groups
CRP (mg l^{-1})	Intense intervention Q1/Median/Q3 (*p*-value)	Usual care Q1/Median/Q3 (*p*-value)	*p*-Value
Baseline	1.48/2.79/5.90	1.22/2.14/3.59	*0.021*
After 1 month	0.69/1.64/3.30 (*0.000*)	–	–
After 1 year	1.05/1.99/5.00 (*0.001*)	1.01/1.96/4.24 (0.55)	0.75
After 3 years	1.23/2.47/4.92 (0.09)	1.23/2.20/4.78 (0.57)	0.46
After 5 years	1.33/2.22/5.63 (0.10)	1.15/2.35/4.72 (0.55)	0.41
0–1 year	–	–	0.04
0–3 year	–	–	0.10
Smokers excluded			
Baseline	1.14/2.01/3.28	1.41/2.74/5.87	*0.016*
After 1 month	0.67/1.45/3.06 (*0.000*)	–	–
After 1 year	1.02/1.93/4.37 (*0.005*)	0.99/1.87/3.93 (0.26)	0.40
After 3 years	1.14/2.37/4.66 (0.074)	1.16/1.91/4.05 (0.71)	0.29
Statin users excluded			
Baseline	1.48/2.64/5.89	1.11/2.05/3.51	*0.03*
After 1 month	0.68/1.60/3.44 (*0.000*)	–	–
After 1 year	1.06/2.05/5.04 (*0.005*)	0.94/1.87/4.22 (0.92)	0.49
After 3 years	1.27/2.65/5.74 (0.11)	1.13/2.20/4.78 (0.29)	0.40
CRP >10 excluded			
Baseline	1.36/2.47/5.22	1.19/2.01/3.28	0.05
0–1 year	–	–	0.03
After 1 month	0.66/1.29/2.57 (*0.000*)	–	–
After 1 year	1.00/1.79/4.31 (*0.004*)	0.99/1.88/3.83 (0.87)	0.93
After 3 years	1.12/2.37/4.51 (0.66)	1.16/1.94/4.05 (0.42)	0.50

after 1 month at a wellness centre. This decrease persisted up to 1 year. In the UCG no difference in CRP was observed at any time.

In a univariate linear regression model on all participants smokers (beta = 0.203, $p = 0.005$), higher BMI (beta = 0.245, $p = 0.005$) and female sex (beta = 0.260, $p = 0.000$) were correlated to a higher CRP level at baseline.

In a multiple linear regression analysis with CRP as a response variable and age, sex, BMI, use of statin, ever been smoking, cholesterol, systolic blood pressure and fasting plasma insulin level as explanatory variables, smokers, female sex and higher BMI remained independently associated with CRP at baseline ($p = 0.006$, 0.004 and 0.009, respectively).

To study how the change in CRP related to the changes in metabolic variables we analysed the change in these variables from baseline to the 1-month of follow-up (when the maximum decrease in CRP occurred) in the IIG. In this time CRP decreased 1.43 mg l^{-1}, fasting glucose 0.47 mmol l^{-1}, body weight 4.1 kg and fasting plasma insulin 11.2 pmol l^{-1}. Only the change in fasting glucose ($p = 0.031$) correlated to the change in CRP.

Discussion

This randomized trial demonstrates that intensive lifestyle intervention, effected through a 1-month stay at the wellness centres, can significantly decrease the CRP level in obese individuals with impaired glucose tolerance up to 1 year after admittance. In the UCG who received thorough lifestyle advice on only one occasion, no significant difference in CRP was observed at any time. The difference in treatment effect after 1 year was statistically significant in favour of the IIG.

Previous intervention studies have shown that weight reduction and increased physical activity reduced not only BMI but also CRP (Esposito et al. 2003, Milani et al. 2004). It has also been shown in weight-loss studies that elevated CRP concentrations are confined to insulin-resistant obese individuals further implicating the relevance of studying a population with the characteristics of those reported here (McLaughlin et al. 2002).

There are at least two mechanisms that may account for the reduction in CRP level during lifestyle intervention. First, adipose tissue secretes several cytokines and at least one of them, IL-6, stimulates CRP production in the liver and it has been shown that caloric-restriction inducing weight loss decreases CRP (Nicklas et al. 2004, Tchernof et al. 2002). Secondly, the CRP level can be lowered by exercise training possibly through more direct effects on the inflammatory process itself, as this effect cannot be explained by weight reduction induced by training (Kasapis & Thompson 2005, Lakka et al. 2005).

Our regression analysis showed that smoking, female sex and higher BMI are independently related to CRP. Relationships between sex and CRP have been observed previously in obese populations with higher CRP levels in females (Khera et al. 2005). Unfortunately, our data did not have enough power to analyse males and females separately, but further research on this issue would be of great interest. The change in CRP, from baseline to 1 month, in the IIG group was not related to changes in body weight or plasma insulin and weakly to change in plasma glucose.

It is always important to give advice on lifestyle changes when one meets a patient in a metabolic or cardiovascular risk group. Our intensive intervention population received an intensive intervention programme which had strong positive effects on the CRP level. This effect was lost within 3 years illustrating the difficulty of maintaining a lifestyle change.

This study used CRP as a marker of the risk of CVD in a high-risk population, but it did not show that the lower CRP level decreases the risk of CVD. In future studies, it would be of great interest both to find out how to best reduce an increased CRP level and how to encourage a persisting change in lifestyle.

There are to date only a few studies published on the effects of lifestyle change on CRP, and even fewer with 5 years follow-up. Hardly any of them used a randomized design aimed at obese individuals with impaired glucose tolerance, a group with an especially high risk for developing CVD. The long follow-up period is a strength of this study and shows the difficulties associated with maintaining a change in lifestyle.

Limitations in the present study

The IIG had a significantly higher BMI at baseline which was associated with a higher level of CRP. The higher BMI and CRP level at baseline in the IIG makes the result difficult to interpret as it might be easier to get a decrease from a higher baseline. Differences at baseline must be a random phenomenon due to the limited sample size of the study.

The fact that no CRP sampling were made at 1 month in the UCG means that analysis between groups is impossible at that time point. Since the effects of lifestyle intervention are hopefully maintained for several years and in our case persisted at least at the 1-year follow-up, this lack of comparability after 1 month feels of minor importance.

In summary, the present randomized intervention study on subjects with impaired glucose tolerance and obesity showed a significant decrease in CRP up to 1 year after an intensive lifestyle intervention programme. This calls for further prospective intervention trials aimed at reducing CRP in high-risk populations through lifestyle intervention to study whether or not the reduced CRP level translates into a decreased risk for cardiovascular morbidity and mortality. As it is evident that the intervention group did not maintain the reduction in CRP we also need more knowledge on how to maintain a lifestyle change that may prove beneficial for health.

Acknowledgements

We thank Professor Knut Borch-Johnsen at the Steno Diabetes Center for technical assistance (insulin determinations). The study was supported by the joint Committee of the Northern Sweden Health Care Region, the Swedish Public Health Institute, Västerbotten County Council and the Foundation of Medical Research in Skellefteå.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

Andersen L, Dinesen B, Jorgensen P, Poulsen F, Roder M. 1993. Enzyme immunoassay for intact human insulin in serum or plasma. Clinical Chemistry 39:578–582.

Bassuk S, Rifai N, Ridker P. 2004. High-sensitivity C-reactive protein: clinical importance. Current Problems in Cardiology 29:439–493.

DeFronzo R, Ferrannini M. 1991. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care 14:173–194.

Esposito K, Pontillo A, Palo CD, Giugliano G, Masella M, Marfella R, Giugliano D. 2003. Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial. JAMA 289:1799–1804.

Fröhlich M, Imhof A, Berg G, Hutchinson WL, Pepys MB, Boeing H, Muche R, Brenner H, Koenig H. 2000. Association between C-reactive protein and features of the metabolic syndrome. Diabetes Care 23:1835–1839.

Galani C, Schneider H. 2007. Prevention and treatment of obesity with lifestyle interventions: review and meta-analysis. International Journal of Public Health 52:348–359.

Kasapis C, Thompson P. 2005. The effects of physical activity on serum C-reactive protein and inflammatory markers: a systematic review. Journal of the American College Cardiology 45:1563–1569.

Khera A, McGuire D, Murphy S, Stanek HG, Das SR, Vongpatanasin W, Wians FH, Grundy SM, de Lemos JA. 2005. Race and gender differences in C-reactive protein levels. Journal of the American College Cardiology 46:464–469.

Lakka T, Lakka H, Rankinen T, Leon AS, Rao DC, Skinner JS, Wilmore JH, Bouchard C. 2005. Effect of exercise training on plasma levels of C-reactive protein in healthy adults: the HERITAGE Family Study. European Heart Journal 26:2018–2025.

Lindahl B, Nilsson TK, Jansson JH, Asplund K, Hallmans G. 1999. Improved fibrinolysis by intense lifestyle intervention. A randomized trial in subjects with impaired glucose tolerance. Journal of Internal Medicine 246:105–112.

Marlatt G, Gordon J. 1985. Relapse Prevention. New York: The Guilford Press. p. 1985.

McLaughlin T, Abbasi F, Lamendola C, Liang L, Reaven G, Schaaf P, Reaven P. 2002. Differentiation between obesity and insulin resistance in the association with C-reactive protein. Circulation 106:2908–2912.

Milani R, Lavie C, Mehra M. 2004. Reduction in C-reactive protein through cardiac rehabilitation and exercise training. Journal of the American College of Cardiology 43:1056–1061.

Nicklas BJ, Ambrosius W, Messier SP, Miller GD, Penninx BWJH, Loeser RF, Palla S, Bleecker E, Pahor M. 2004. Diet-induced weight loss, exercise, and chronic inflammation in older, obese adults: a randomized controlled clinical trial. American Journal of Clinical Nutrition 79:554–551.

Pearson T, Mensah G, Alexander R, Anderson JL, Cannon RO, Criqui M, Fadl YY, Fortman SP, Hong Y, Myers GL, et al. 2003. Markers of inflammation and cardiovascular disease: application to clinical and public health practice. A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation 107:499–511.

Pradhan A, Manson J, Rifai N, Buring J, Ridker P. 2001. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. JAMA 286:327–334.

Reaven G. 1988. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 37:1596–1607.

Ross R. 1999. Atherosclerosis – an inflammatory disease. New England Journal of Medicine 340:115–126.

Tchernof A, Nolan A, Sites C, Ades P, Poehlman E. 2002. Weight loss reduced C-reactive protein levels in obese postmenopausal woman. Circulation 105:564–569.

Visser M, Bouter L, McQuillan G, Wener M, Harris T. 1999. Elevated C-reactive protein levels in overweight and obese adults. JAMA 282:2131–2135.

Weinehall L. 1997. Partnership for health. On the role of primary health care in a community intervention programme. Umeå University Medical Dissertations 1997, New series No 531.

WHO. 1985. Expert committee on diabetes mellitus. Technical Report Series 727. Geneva: World Health Organization.

Yudkin J, Stehouwer C, Emeis J, Coppack S. 1999. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction. Arteriosclerosis, Thrombosis. and Vascular Biology 19:972–978.

Appendix A

This is a translated version of the written information given to the usual care group.

IMPAIRED GLUCOSE TOLERANCE

What is it and what can you do yourself?

1. What is it?

You have done an oral glucose tolerance test. The test is done by measuring the blood glucose level two hours after digesting 75 g sugar dissolved in water. The test showed that your glucose concentration was higher than normal but not high enough to be diagnosed as diabetic. This situation is called impaired glucose tolerance.Having impaired glucose tolerance means that you in the future have a higher risk of developing diabetes (diabetes of the adult) and cardiovascular disease (angina pectoris).

2. What can you do yourself?

1. Try to lose weight, preferably to normal weight if possible. See facts.
2. Regular physical activity, preferably several times a week. Adjust the exercise to your capacity and remember that it is low energy exercise (brisk walks, swimming, cycling) that is best suited to burn fat.
3. If you are smoker: Try to quit smoking!
4. Decrease you alcohol consumption – especially if you have a high intake.

FACTS

How to calculate your normal upper weight limit:

Your length in metres times your length in metres times 25. If you're 1.75 m tall your upper normal weight limit is $1.75 \times 1.75 \times 25 = 76.6$ kg. If you are few kg over this limit then it does not have so much importance. Look up for more substantial overweight.

Important to consider regarding overweight:

1. Regular meals: eat breakfast, lunch, dinner and a couple of between-meals. Eat sparsely in the evening.
2. Use the plate model. (Figure showing ½ plate with fruits and vegetables, ¼ potatoes, rice or pasta and ¼ meat or fish).
3. Control your eating between meals.