A workplace lifestyle intervention programme: Effect on anthropometric risk factors for cardiovascular disease and type 2 diabetes

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Background: A lifestyle intervention programme (LIP) was developed to deliver to employees from Carmarthenshire Health service and Tata Steel Llanelli aged 40 years or over, whom a 10 year cardiovascular disease (CVD) risk estimate was found to be ≥20% at a CVD health check undertaken as a component of Prosiect Sir Gâr (the Carmarthenshire Project).

Methods: Employees received eight 75 min sessions delivered at their workplace, by a dietitian (7 sessions) and an exercise specialist (1 session) with an emphasis on education and motivation for behaviour change. Weight, BMI, waist circumference, and a health and lifestyle questionnaire (HLQ) score were collected at programme commencement and completion. Participant satisfaction was also captured. A maximum of 10 participants were enrolled on a single programme. All employees who undertook the initial risk screen are reassessed in 6 months, 12 months or 5 years depending on their initial risk profile.

Results: To date, six 8-week programmes have been evaluated and 20 participants have completed. Fifteen participants demonstrated weight reduction post-LIP with mean BMI 35.24 kg/m² (range 29.89–40.20 kg/m²) post-LIP, and mean BMI 34.30 kg/m² (range 29.89–40.20 kg/m²) post-LIP, respectively. Among the fifteen participants mean percentage weight loss was 2.65% (range 0.31–5.31%) post-LIP. Among the fifteen weight reducers 2 participants had a raised waist circumference (WC) post-LIP however mean reduction in WC was 4.86 cm (range 1.2–13 cm). Sixteen participants completed the HLQ of which, 15 demonstrated improved scores.

Summary: Positive anthropometric and health and lifestyle questionnaire results were observed with the pilot phase of this workplace-based intervention. The results suggest a lifestyle intervention programme represents a positive start towards behavioural change. As participants are re-evaluated an examination of whether these positive changes are maintained may point towards an effective workplace-based strategy in CVD risk reduction.


Effect of historical changes in HDL-cholesterol measurements on cardiovascular risk assessment

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HDL-cholesterol (HDL-C) measurements are incorporated in algorithms for cardiovascular disease (CVD) risk assessment. We have previously reported substantial changes in patient mean HDL-C over the last decade. We have modelled the effects of such HDL-C changes on calculated CVD risks with the Framingham and QRISK risk equations.

CVD risks were calculated using the Framingham and QRISK equations, for patients aged 30–74 years. Total cholesterol and HDL cholesterol values, patient ages and genders were taken from samples analysed between January and December 2009 at Birmingham Heartlands Hospital. These were incorporated into the Framingham and QRISK equations to calculate CVD risks. The population frequency of other risk factors was imputed and HDL-C values for each patient were varied over the mean range observed from 1999 to 2009. At all values of HDL-C, QRISK gave significantly higher median risk scores than Framingham. The percentage of the population with treatable CVD risks (>20% over 10 years) was also higher with QRISK. As the mean HDL-C increased from 1.10 to 1.50 mmol/L (observed over the last decade), median QRISK scores fell from 8.8% to 7.3% (Townsend score 1), 12.2% to 10.3% (Townsend score 10) and 8.2% to 5.8% (Framingham). The percentage with CVD risks ≥20% fell from 15.2% to 8.5% (QRISK Townsend Score 1), 22.1% to 12.8% (QRISK Townsend Score 10) and 9.6% to 2.6% (Framingham). In younger subjects Framingham risks were higher than QRISK, but the converse was found in older patients, with the change from a positive to negative bias occurring in patients in their 50s.

Observed historical changes in HDL-C values have had a major impact on calculated CVD risks. QRISK and Framingham CVD risk equations are not interchangeable, and contrary to other published data QRISKs are not always lower than Framingham risks.


Genotyping of patients with familial hypercholesterolaemia from lipid clinics in Wales

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The aim of this project was to determine the mutation detection rate and spectrum of mutations for patients clinically diagnosed with familial hypercholesterolaemia (FH) from Lipid Clinics across Wales. These are patients identified as possible probands as part of the Wales FH Cascade Testing Service. 175 individuals underwent genotyping for mutations in the low-density lipoprotein receptor (LDLR), ApoB and PCSK9 genes. Information was collected on LDL-cholesterol levels, the presence of tendon xanthomata and a personal or family history of premature coronary heart disease. Mutations were identified in 95/175 (54%) individuals. This includes 21 sequence variants of uncertain significance (unclassified variants, UVs). A total of 57 different mutations were detected. Eight mutations had not been described in previous published literature. 65 (88%) pathogenic mutations occurred in the LDLR gene, 7 (9%) in the ApoB gene and 2(3%) in the PCSK9 gene. Of the pathogenic mutations, 40 (54%) were missense, 8 (11%) frame shift, 7 (9%) nonsense, 9 (12%) splice site and 10 (14%) large rearrangements. Mutations in exons 3 and 4 accounted for 36% of pathogenic mutations with the commonest single mutation (c.301G>A) in exon 3 being found in 6 patients. Individuals with pathogenic mutations had significantly higher LDL-cholesterol (8.2 mmol/L) compared to those with a clinical diagnosis of possible FH but no detectable mutation (6.3 mmol/L). LDL-cholesterol levels were lower in individuals with ApoB 3500 compared to LDLR and PCSK9 mutations. In this sample no statistical differences in phenotype were identified between individuals with LDLR missense mutations and larger genetic alterations (frame shift, nonsense, splice site and large rearrangements). Genotyping of lipid clinic patients has demonstrated a wide range of mutations including a range of new mutations and unclassified variants. Genetic testing can refine the clinical diagnosis of FH and be the starting point for family cascade testing.