

**Aim** To evaluate the impact of this program in risk factors control and events.

**Methods** 514 consecutive MI patients hospitalised in six public hospitals. Data collection was obtained by a review of medical records and a 12-month interview (anthropometric and biochemical measurements, lifestyle information and pharmacological treatment). Predictors of good control were evaluated with multilevel analysis.

**Results** follow-up was available in 398 patients (77.4%), 75% were male, aged 62.1 years (SD  $\pm$ 11.7). At the time of interview 8.6% were smokers; 24% reported regular physical activity; 78.6% were overweight or obese. The proportion of patients with raised systolic blood pressure was 46.3% and raised diastolic blood pressure was found in 35.4%. 28.9% had LDL cholesterol  $\geq$ 100 mg/dl and 21.1% glucose  $\geq$ 100 mg/dl. In diabetic patients (24.1%), 52% had glycosylated haemoglobin  $\geq$ 7.0%. The use of drug therapies at month 12 was: aspirin 95.5%,  $\beta$ -blockers 70.6%, ACE inhibitors 64.0% and statins 89.2%. One year mortality was 6.8%. Predictors for good control of risk factors were statin use (OR 2.64; CI 1.16 to 5.98) and control by cardiologist (OR 1.13; CI 1.01 to 1.27); diabetic patients have a poor control (OR 0.30; CI 0.15 to 0.61).

**Conclusion** Patients with MI have unhealthy lifestyles and a high proportion not achieved the goal for cholesterol and blood pressure management. A multidisciplinary approach is needed to improve secondary prevention in MI patients.

**P2-219 RACIAL/ETHNIC DISPARITIES IN THE TIMING OF DEATH DURING EARLY CHILDHOOD AMONG CHILDREN WITH CONGENITAL HEART DEFECTS**

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<sup>1</sup>W Nembhard,\* <sup>1</sup>J Salemi, <sup>1</sup>J Thumm, <sup>2</sup>M Ethen, <sup>3</sup>D Fixler, <sup>2</sup>M Canfield. <sup>1</sup>University of South Florida, College of Public Health, Tampa, Florida, USA; <sup>2</sup>Birth Defects Epidemiology and Surveillance Branch, Texas Department of State Health Services, Austin, Texas, USA; <sup>3</sup>Division of Cardiology, Department of Pediatrics, University of Texas Southwestern Medical Center, Dallas, Texas, USA

Infants with congenital heart defects (CHD) have increased risk of childhood mortality; previous research indicates racial/ethnic differences in timing of death during infancy. However, less is known about racial/ethnic disparities in timing of death during early childhood. Texas Birth Defect Registry data were used in a retrospective cohort study of 19 406 singleton, live-born infants, born with a CHD between 1 January 1996 and 31 December 2003 to non-Hispanic (NH) white, NH-black, or Hispanic women. Registry data were linked to death records to ascertain deaths through 31 December 2005. Kaplan–Meier survival estimates were computed and HRs and 95% CIs were calculated from multivariable Cox-proportional hazard regression models to determine the adjusted effect of maternal race/ethnicity on mortality for each specific CHD during the neonatal, post-neonatal and childhood periods. Racial/ethnic disparities in mortality were most pronounced during the post-neonatal period and persisted into early childhood. Among children who survived infancy, NH-Blacks with tetralogy of Fallot (HR=3.61; 95% CI 1.25 to 10.47), coarctation of the aorta (HR=3.13; 95% CI 1.15 to 8.54) and ventricular septal defect (HR=2.60; 95% CI 1.31 to 5.19) were more likely to die in early childhood compared to similarly affected NH-Whites. No statistically significant differences in timing of death after infancy were found for Hispanics vs NH-Whites. Racial/ethnic disparities in timing of death in childhood for specific CHD diagnoses are present but of unknown aetiology. Elucidation of factors associated with early childhood CHD mortality will aid in development of public health and clinical strategies to reduce racial/ethnic disparities in childhood mortality.

**P2-220 WITHDRAWN**

**P2-221 THE EFFECT OF ENVIRONMENTAL EXPOSURE TO PHTHALATES ON TESTICULAR CARCINOGENESIS**

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B Nguyen,\* J Gomes. University of Ottawa, Ottawa, Canada

Testicular germ cell tumour is the most common malignancy in young males and its incidence has been rising in recent years. Environmental and occupational exposures are believed to increase the risk of testicular cancer. The exposures along with genetic susceptibility may influence the risk of developing Testicular germ cell tumour even further. Phthalates are endocrine disruptors and are abundantly used as industrial plasticisers. Human exposure to phthalates occurs from the use of this substance in commerce. Di-2-(ethylhexyl) phthalate (DEHP) is the most common phthalate found in consumer products. Post exposure DEHP is rapidly hydrolysed into its active form, mono-(2-ethylhexyl) phthalate (MEHP), a testicular toxicant and a carcinogen. The objective of this research was to determine the toxicity of MEHP. Possible mechanisms that may be involved in the pathogenesis of testicular atrophy from exposure to MEHP include FAS signalling, ROS signalling, NF- $\kappa$ B, PPAR and cAMP pathway. Alternative mechanisms may also be associated with the regulation of germ cell apoptosis leading to testicular atrophy. Relevant genes of interest that may be affected are Testisin, GSTP1, and MGMT. The expression of these genes was examined by RT-PCR in testicular germ cells exposed to MEHP in a dose- and time-dependent manner at concentrations of 1  $\mu$ M, 10  $\mu$ M, and 100  $\mu$ M at 24, 48, 72 and 96 h time points. The findings of this study will allow for a better understanding of the role of phthalates in altering expressions in testicular germ cells and a better understanding of the process of testicular carcinogenesis.

**P2-222 RISK MARKERS FOR CORONARY HEART DISEASE AND TYPE 2 DIABETES IN CHILDHOOD: COMPARISON OF INDIAN CHILDREN LIVING IN INDIA AND THE UK**

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<sup>1</sup>C Nightingale,\* <sup>2</sup>G Krishnaveni, <sup>1</sup>A Rudnicka, <sup>1</sup>C Owen, <sup>2</sup>S Veena, <sup>3</sup>J Hill, <sup>1</sup>D Cook, <sup>3</sup>C Fall, <sup>1</sup>P Whincup. <sup>1</sup>St. George's, University of London, London, UK; <sup>2</sup>Epidemiology Research Unit, CSI Holdsworth Memorial Hospital, Mysore, India; <sup>3</sup>Medical Research Council Epidemiology Resource Centre, Southampton General Hospital, Southampton, UK

**Introduction** UK Indian adults have higher risks of coronary heart disease (CHD) and type 2 diabetes (T2D) than Indian and UK European adults. With growing evidence that CHD and T2D risks begin before adulthood, we compared risk factor patterns in Indian children living in India and the UK.

**Methods** We compared markers of adiposity and cardiometabolic risk in 9–10 year-old Indian children in the Mysore Parthenon birth cohort study, India (n=538) and in the cross-sectional Child Heart Health Study, England (n=483), which used comparable survey methods in 2007–2008 and 2004–2007 respectively. Small mean age and gender differences between studies were adjusted for in analyses.

**Results** UK Indian children were taller and had markedly higher levels of BMI (mean difference 3.2 kg/m<sup>2</sup>, % difference 22%, 95% CI 20 to 25%) combined skinfold thickness (% difference 36%, 95% CI 29 to 44%), LDL-cholesterol (mean difference 0.4, 95% CI 0.3 to 0.5 mmol/l), systolic BP (mean difference 11.3, 95% CI 9.9 to 12.8 mm Hg) and fasting insulin (% difference 141%, 95% CI 121 to 163%). These differences were similar in boys and girls; differences in LDL-cholesterol, blood pressure and insulin remained marked after adjustment for adiposity markers and pubertal status.

**Conclusions** Substantial differences in cardiometabolic risk between UK Indian and Indian children are apparent before puberty. They do

not depend on differences in adiposity and are likely to have an environmental basis. Strategies for chronic disease prevention need to include measures to combat the emergence of chronic disease risks in childhood or earlier.

**P2-223 THE COMBINED EFFECT OF CHEWING THOROUGHLY AND EATING UNTIL FULL ON CHILDHOOD OVERWEIGHT: RESULTS OF 1999–2009 SCHOOL-BASED SURVEY IN JAPAN**

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<sup>1</sup>H Ochiai,\* <sup>1</sup>T Shirasawa, <sup>1</sup>N Shimada, <sup>2</sup>R Nishimura, <sup>2</sup>A Morimoto, <sup>1</sup>T Ohtsu, <sup>1</sup>H Hoshino, <sup>1</sup>A Kokaze. <sup>1</sup>Department of Public Health, Showa University School of Medicine, Tokyo, Japan; <sup>2</sup>Division of Diabetes, Metabolism and Endocrinology, Department of Internal Medicine, Jikei University School of Medicine, Tokyo, Japan

**Introduction** The aim of the present study was to investigate the relationship of overweight to chewing thoroughly and eating until full and to examine the combined effect of chewing thoroughly and eating until full on overweight among schoolchildren in Japan.

**Methods** Subjects included all fourth-grade schoolchildren (9 or 10 years of age) in Ina-town, Saitama prefecture, Japan, during 1999–2009. Information about subject's sex, age, and lifestyle, including chewing thoroughly and eating until full, was collected using a self-administered questionnaire. Measurements of height and weight were made for each child. Overweight in children was defined according to the criteria of the International Obesity Task Force. To calculate the OR and 95% CI for overweight, a logistic regression model was used.

**Results** Data from 4027 children were analysed. Chewing thoroughly revealed significantly decreased OR for overweight when compared to not chewing thoroughly (OR: 0.39, 95% CI 0.33 to 0.48). Eating until full showed significantly increased OR for overweight compared with not eating until full (1.24, 1.02 to 1.51). Among children who reported chewing thoroughly, OR of eating until full was not statistically significant (0.97, 0.76 to 1.24). On the other hand, eating until full illustrated significantly increased OR among the not chewing thoroughly group (1.67, 1.21 to 2.30).

**Conclusion** Chewing thoroughly and eating until full were associated with overweight. Furthermore, a combined effect of not chewing thoroughly and eating until full on overweight was noted. This study suggests that chewing thoroughly is useful for the prevention of childhood overweight.

**P2-224 PATHWAYS TO DIAGNOSIS DO NOT EXPLAIN ALL OF THE INCREASE IN THYROID CANCER: RESULTS OF A POPULATION BASED CROSS-SECTIONAL STUDY**

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<sup>1</sup>D O'Connell,\* <sup>1</sup>C Kahn, <sup>1,2</sup>L Simonella, <sup>2</sup>M Sywak, <sup>3</sup>S Boyages, <sup>4</sup>O Ung. <sup>1</sup>Cancer Council NSW, Sydney, New South Wales, Australia; <sup>2</sup>University of Sydney, Sydney, New South Wales, Australia; <sup>3</sup>Clinical Education and Training Institute NSW, Westmead, New South Wales, Australia; <sup>4</sup>Royal Brisbane and Women's Hospital, Brisbane, Queensland, Australia

**Background** The incidence of thyroid cancer has increased over the last few decades in many countries, particularly in developed countries and markedly in the Australian state of New South Wales (NSW). To investigate whether these changes may be due to increased detection we studied the clinical pathways leading to the diagnosis of thyroid cancer.

**Methods** Newly diagnosed cases of thyroid cancer were identified and recruited through the population-based NSW Central Cancer

Registry. Participants completed a questionnaire and diary of doctor visits and investigations that led to their diagnosis. Tumour characteristics were obtained from pathology reports.

**Results** 452 people (76% female) with thyroid cancer completed the study. The median age at diagnosis was 48 years for women and 53 for men. Only 40% of diagnoses occurred after the patient reported a lump or symptom and 60% of diagnoses were serendipitous. The pathways to diagnosis varied significantly with tumour size ( $p=0.001$ ) and by age in men ( $p=0.008$ ) and place of residence in women ( $p=0.05$ ). Not all of the increase in incidence is explained by increased detection. Allowing for cases diagnosed serendipitously, the estimated age-standardised incidence rates for men (3.83 per 100 000) and women (10.65 per 100 000) were well below those observed (4.65 and 15.3 respectively).

**Conclusion** As the diagnosis of only 40% of thyroid cancers was patient initiated, the reported incidence of thyroid cancer is likely to be influenced by diagnostic technology and medical surveillance practices. This, however, probably only partly explains the observed rise in incidence of thyroid cancer in NSW.

**P2-225 PROSTATE-SPECIFIC ANTIGEN TESTING AWARENESS AND PARTICIPATION IN NEW SOUTH WALES, AUSTRALIA: DEMOGRAPHIC, LIFESTYLE AND HEALTH-RELATED FACTORS**

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<sup>1</sup>D O'Connell,\* <sup>1</sup>L Carmichael, <sup>1</sup>D Smith, <sup>2</sup>M Gattellari, <sup>3</sup>S Chambers, <sup>4</sup>C Pinnock, <sup>5</sup>T Slevin, <sup>6</sup>J Ward. <sup>1</sup>Cancer Council NSW, Sydney, New South Wales, Australia; <sup>2</sup>University of NSW, Sydney, New South Wales, Australia; <sup>3</sup>Griffith University, Brisbane, Queensland, Australia; <sup>4</sup>Repatriation General Hospital, Daws Park, South Australia, Australia; <sup>5</sup>Cancer Council WA, Perth, Western Australia, Australia; <sup>6</sup>University of Ottawa, Ottawa, Ontario, Canada

**Background** Although the prostate-specific antigen (PSA) test is widely used to screen for prostate cancer, there is very little information on the characteristics of men who are aware of the PSA test, and their patterns of PSA testing.

**Methods** A cross-sectional study used computer assisted telephone interviews to collect data in New South Wales, Australia. Multinomial logistic regression identified the factors independently associated with the awareness of, and participation in PSA testing.

**Results** Of the 6100 men, 39% were unaware of the PSA test, 12% were aware of the PSA test but never tested, 14% had a non-recent PSA test, and 35% had a recent PSA test. Unaware men were more likely to be born outside Australia (OR=1.19; 95% CI 0.88 to 1.60), have a blue-collar occupation (OR=1.38; 95% CI 1.00 to 1.91), be a current smoker (OR=1.99; 95% CI 1.30 to 3.05), or have benign prostatic hyperplasia (BPH) (OR=1.70; 95% CI 1.07 to 2.71), and less likely to have completed a higher school certificate (OR=0.44; 95% CI 0.24 to 0.79), or live in inner regional areas (OR=0.59; 95% CI 0.44 to 0.80). Men who did not have a recent test, were more likely to visit the doctor (OR=1.38; 95% CI 1.05 to 1.82), or have BPH (OR=2.70; 95% CI 1.74 to 4.20), and were less unsure of their risk of developing prostate cancer (OR=0.61; 95% CI 0.37 to 1.00). Men who had a recent test were more likely to visit the doctor (OR=2.57; 95% CI 1.99 to 3.33), have BPH (OR=3.87; 95% CI 2.58 to 5.81), or have a higher perceived risk of developing prostate cancer (OR=1.99; 95% CI 1.22 to 3.26), and less likely to be other than married (OR=0.65; 95% CI 0.47 to 0.91).

**Conclusions** As men's PSA testing experience varied by demographic, lifestyle and health-related factors, it is important for policymakers and physicians to consider these when communicating about PSA testing.