Thursday, 10 September

Parallel session B

Life course obesity

025  BODY MASS INDEX THROUGH LIFE AND ADULT MORTALITY: RESULTS FROM THE BRITISH 1946 BIRTH COHORT

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Background: Adult body mass index (BMI) has been consistently related to mortality, but little is known about the impact of earlier life BMI on adult mortality.

Objective: Investigate the impact of childhood, adolescent and early adult BMI on premature adult mortality and assess whether any associations are explained by adult BMI.

Design: Cohort study with prospective information on BMI at ages 2, 4, 6, 7, 11, 15, 20, 26 and 36 years, and mortality follow-up from age 26 to 60 years. BMI was standardised at each age, separately for men and women.


Participants: 2525 males and 2136 females.

Outcome measure: All-cause mortality (332 deaths).

Results: Splines were used to model the non-linear associations between BMI and mortality. In both genders, adult BMI from 20 years onwards showed a consistent U-shaped relationship with adult mortality (overall p-value <0.05 for BMI at ages 20, 26 and 36 years). In females, a similar relationship was observed for adolescent BMI at 15 years (p=0.02); the hazard ratio (HR) comparing females with low BMI (2 standard deviations (SDs) below mean) vs mean BMI was 2.96 (95% CI 1.26 to 6.97). The corresponding HR for females with BMI 2 SDs above the mean was 1.97 (0.95 to 4.10). In males, increased mortality rates were only seen for low adolescent BMI. BMI in childhood was generally not associated with adult mortality. The exception was BMI at age 4 years in females, where a U-shaped relationship was observed (p = 0.02); HR for low BMI (2 SDs below mean) at 4 years vs mean BMI was 2.13 (0.97 to 4.70). The HR for females with BMI 2 SDs above the mean was 1.67 (0.85 to 3.28), and for females with BMI 3 SDs above mean it was 3.08 (1.21 to 7.85). This association was not explained by subsequent BMI change, adult BMI, smoking, childhood social class or adult educational level.

Conclusions: High and low childhood and adolescent BMI are related to adult premature mortality, especially in women. Interventions to reduce under- and overweight in childhood are required to prevent increasing premature adult mortality in more recent cohorts with greater numbers of overweight children.

026  TRAJECTORIES OF BODY MASS INDEX AND OVERWEIGHT IN EARLY ADULTHOOD AND BLOOD PRESSURE AT 53 YEARS

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Background: Body mass index (BMI) is positively associated with blood pressure (BP) throughout adulthood. However, it is not known whether being overweight (BMI>25 kg.m2) earlier in adult life is associated with higher BP independent of current BMI, or whether there are sensitive periods for weight gain in adulthood.

Objectives: To explore the pathways by which being overweight in adulthood is associated with BP at 53 years.

Design: Prospective birth cohort study with information on BMI at 20, 26, 36, 43 and 53 years and BP at 53 years.


Participants: 1451 males and 1479 females.

Outcome: BP at age 53 years. Censored regression models, accounting for individuals on antihypertensive medication, were used. To investigate a cumulative influence, we estimated the effect of time from being first overweight. To investigate sensitive period(s), we used standardised conditional BMI velocities for each interval. Lastly, an overweight trajectory was defined for each individual based on their status at 26, 36 and 53 years to try to formally assess the evidence for an accumulation or sensitive period pathway.

Results: Overweight at all ages was associated with a higher mean BP at 53 years. After adjusting for current BMI, only overweight at age 43 in men contained any additional information on BP at 53 years. Men who became overweight at 26 had a SBP 8.7 mm Hg higher (95% CI 4.4 to 13.0) than those first overweight at 53. Similar patterns but smaller associations were seen in women (p(interaction)<0.001). All periods of adult weight gain (26–36, 36–43, 43–53 years) were associated with a higher BP. The association varied little between periods, in males it ranged from a 2.7–3.6 mm Hg increase in SBP per SD increase in BMI velocity. BMI tracked strongly through adulthood, the BMI at 53 years in men first overweight at 26 was 30.9 kg.m2 (95% CI 30.5 to 31.4) compared to 26.4 kg.m2 (95% CI 26.3 to 26.6) in those first overweight at 53. Few individuals moved to a normal weight once overweight, this prevented a reliable estimation of the excess risk associated with prolonged overweight and meant it was difficult to disentangle a sensitive period or accumulation pathway using the different trajectories of overweight.

Conclusion: Early adult overweight and all periods of adult weight gain irrespective of earlier BMI were associated with a higher later life BP. Associations at younger ages appear to be largely mediated through BMI tracking. This highlights the importance for later health of preventing overweight in early adulthood.

Results: In terms of incidence, in 1980, the overall ASR varied widely across countries, ranging from 18 per 100 000 in Belarus to 309 per 100 000 in Switzerland. Between 1980 and 2002, prostate cancer incidence increased in all 20 countries. The OPC was the smallest in Denmark (+40%) and highest in Italy (+35%).

Generally, countries with lower incidence in early years had the highest OPCs. In North-America and Australia incidence peaked around 1994, whereas in most European countries rates rose throughout the study period. The increase was most pronounced among men aged 50–74, and in a few countries, the OPC for men aged ≥75 years was less than zero. Mortality rates decreased in North-America and some western European countries (eg France, England, Italy, Switzerland), remained stable in others (eg Scotland, Sweden, Denmark) and increased in Eastern Europe. In countries where rates fell, the decline was more pronounced among younger, than older, men. In some countries (eg France), mortality began to fall before incidence peaked.

Conclusions: International variations in prostate cancer incidence and mortality were observed. The different trend in incidence by age suggests an impact of earlier diagnosis/PSA testing. While the increasing trend in incidence is mainly due to improvements in treatment or earlier detection, they could also be affected by changes in death certificate coding or competing causes of death.