

## CUTTING EDGE METHODOLOGY

## P1-6 MEASURES OF SOCIO-DEMOGRAPHIC FACTORS FOR CHILD HEALTH RESEARCH

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**Background** *H pylori* infection is one of the major causes of health problems with considerable morbidity and mortality. *H pylori* seroprevalence is common in populations with poor standards of sanitation and hygiene.

**Methods** A two-stage cluster sampling technique was employed to draw the required sample. A crowding index with three categories (low, moderate, high) was constructed by dividing the number of individuals per household by the number of the rooms. Assessment of socioeconomic status (SES) was calculated by Hollingstead Index (HI).

**Results** Serum of 1976 children was tested. *H pylori* seropositivity in 1–5 years were 53.5%. It increased with moderate crowding index (CRI) of 2–4 to 45.9% and to 51.2% with CRI >4. In middle SES, seropositivity was 331 (50.5%) while in lower SES 500 (47.1%). Multivariate analysis showed *H pylori* seroprevalence was high in 6–10 and 11–15 years (OR: 1.5, 95% CI 1.2 to 1.9 and OR: 1.9, 95% CI 1.56 to 2.47, respectively), in lower-middle SES (OR: 1.6, 95% CI 1.2 to 2.1 and OR: 1.5, 95% CI 1.10 to 2.0, respectively) and uneducated fathers (OR: 1.58, 95% CI 1.27 to 1.95).

**Conclusion** *H pylori* seropositivity increased with age, in low-middle SES and is related to father's educational status.

## P1-7 INFLUENZA RECYCLING AND EPIDEMIOLOGIC EVOLUTION: AN ALTERNATIVE TO OMRAN'S EPIDEMIOLOGIC TRANSITION THEORY FOR POPULATION CHANGE

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The sustained increase in longevity initiated during the second half of the 20th Century poses huge challenges upon the public and private healthcare and pensions' systems. Current theories do not explain the observed phenomenon, what precludes predictions of its future trends. The authors goal was to go back to Omram's original project—to develop an epidemiologic theory of the demographic change—but looking for an explanation alternative to the epidemiologic transition proposed by him. The study expands upon a previous one, that showed, for the first time, a cohort association between the mortality burden of 1918 Influenza Pandemic and the 20th Century rise in CHD mortality. Based on an age-period-cohort analysis of USA and UK mortality (and natality) data (1933–2005) and on the epidemiology of influenza as we know it, it is shown that, overall, temporal changes in mortality and natality accompany the recycling of influenza A viruses, that is, the re-exposure of human populations, from time to time, to influenza A subtypes that circulated in the past. Mortality (and natality) and main causes of death change as birth cohorts (whole population and maternal) primed at early life with one (period-specific) influenza A sub-type, course through subsequent influenza A environments over time.<sup>1</sup> The implications of this new theory to demography and to the epidemiology of several diseases are revolutionary.

## REFERENCE

1. <http://www.actuaries.org.uk/research-and-resources/documents/influenza-recycling-and-secular-trends-mortality-and-natality>.

## P1-8 PRINCIPAL COMPONENTS ANALYSIS OF DIET IS NOT GOOD AT IDENTIFYING FOODS THAT ARE CAUSALLY LINKED TO DISEASE: A SIMULATION STUDY

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**Introduction** Dietary patterns derived empirically from food frequency questionnaire (FFQ) data using principal components analysis (PCA) are widely employed for the investigation of diet-disease relationships. The aim of the study was to investigate whether PCA performed better at identifying associations between diet and disease than an analysis of each individual food in the FFQ separately after adjusting for multiple testing, a process we refer to as exhaustive single food analysis (ESFA).

**Methods** Using simulated data employing a known model for the associations between food intakes and disease, and a realistic joint distribution of food intakes, we investigated the performance of PCA and ESFA in correctly identifying associations between diet and disease. Performance was assessed in terms of the power with which we could identify at least one association between a food intake and disease, and the power and false discovery rate (FDR) for identifying specific food intakes that were causally linked to disease in the model.

**Results** ESFA had greater power than PCA to detect an association of at least one food with disease, and greater power and lower FDR for identifying specific foods causally linked to disease. With both methods FDRs increased with sample size, even using an FDR-controlling adjustment. However, when we adjusted the ESFA for foods that were significant in univariate analyses, FDRs were controlled at the specified level.

**Conclusions** An exhaustive analysis of single foods out-performed PCA in identifying associations between diet and disease using FFQ data.

## P1-9 THE HEALTH IMPACT FUND-MEETING THE CHALLENGE OF HEALTH IMPACT ASSESSMENT

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**Introduction** The Health Impact Fund (HIF) is a publicly funded international agency proposed to enable pharmaceutical innovators to register a product for health impact rewards in exchange for selling it worldwide at cost. Supplementing the current patent regime, the HIF would improve incentives to research diseases concentrated among the poor. A workable HIF presupposes a consistent, predictable, and contractible method of health impact assessment.

**Methods** We reviewed the literature using search terms, "health impact assessment tools" and an exploratory workshop for all stakeholders was held at the National Institute for Health and Clinical Excellence in April 2010.

**Results** Although there are many challenges with the nature of current epidemiological data and their application to global health, there is scope for improvement and the HIF may help to trigger and sustain such enhancements. Moreover, the HIF would use much more information than the present system. The following steps in health impact assessment need to occur for each registered product: defining subgroups, establishing baseline treatments, defining incremental health impact by subgroup, measuring the numbers of patients treated in each subgroup, and a process of appeal.

**Conclusion** Health impact assessment is a new science, and complexities involved in assessing new drugs in the global context are formidable. However, initial models suggest that the HIF could significantly change the focus of drug innovation. Only pilot studies will properly test the HIF's underlying principles and uncover the practical challenges which will determine its implementation and effectiveness. The creation of the HIF could bring great advances in epidemiological data collection and its application.

**P1-10 DEVELOPMENT AND PSYCHOMETRIC PROPERTIES OF A SCALE TO MEASURE HEALTH EFFECTS OF DISCRIMINATION**

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**Introduction** The development of discrimination scales is an emerging field of enquiry in the area of social determinants of health. However, published scales cannot distinguish health consequences of discrimination as a result of the exposure to differential treatments of any kind from the strict attribution of these events to discrimination. We report the development of a scale that may clarify the relative importance of the effects of these cognitive mechanisms for health outcomes.

**Methods** Successive versions of the instrument were developed based on a systematic review of racial discrimination scales, focus groups and an evaluation by a panel of experts. Instrument refinement was achieved via cognitive interviews and pilot-testing, so that the final scale version was administered to 424 university students in Rio de Janeiro, Brazil. Structural dimensionality, two types of reliability and construct validity were assessed.

**Results** Exploratory factor analysis corroborated the hypothesis of unidimensionality, and the experts indicated that scale items were face and content valid. Internal consistency, estimated by Cronbach's  $\alpha$ , was 0.8, and test-retest reliability was higher than 0.5 for 14 out of the 18 items, according to the weighted  $\kappa$  statistics. The scale's score was statistically higher among socially disadvantaged individuals and correlated with adverse health behaviours and outcomes. Nevertheless, the low test-retest reliability and the observed invariance of specific items indicate that the scale should be assessed in other population domains.

**Conclusion** The proposed scale will enable the investigation of aspects of the relationship between discrimination and health not previously documented in the literature.

**P1-11 WITHDRAWN**

**P1-12 DETECTING DIFFERENCES BETWEEN TREATMENTS IN CRITICAL CARE TRIALS USING MIXTURES OF PARAMETRIC SURVIVAL DISTRIBUTIONS**

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**Background** Traditional methods in survival analysis inadequately summarise the timing of outcomes in critical care trials because they accommodate only one clinical endpoint. We sought to develop an

analytical approach to detect differences when there are two clinical endpoints where one may preclude the other.

**Methods** We used a mixture of parametric survival distributions that belong to the three-parameter generalised  $\gamma$  family (generalised  $\gamma$ ,  $\gamma$ , Weibull, log-normal, and exponential) to model the timing and frequency of two clinical endpoints jointly. Study outcomes were hospital mortality at 60 days and time to unassisted breathing (UAB) at 28 days. We used data from a trial of methylprednisolone vs placebo in 180 critically ill patients with persistent ARDS to show our approach.

**Results** The best model to fit these data was a mixture of log-normal distributions. Patients who received methylprednisolone achieved UAB earlier than did those who received placebo ( $p=0.05$ ); however, this effect decreased over time: by day 5, 55% more patients (95% CI 16% to 79%) achieved UAB in the methylprednisolone group while by day 20, 25% more patients (95% CI 6% to 42%) achieved UAB. The overall probability of achieving UAB was similar between both study groups ( $p=0.82$ ), as were the times to death ( $p=0.15$ ).

**Conclusions** Times of UAB between the study groups were not proportional over time and are unlikely to be proportional in any trial where duration of mechanical ventilation is affected. Furthermore, our approach can easily accommodate mixtures of several well-known parametric distributions under a single comprehensive family, which simplifies hypothesis testing.

**P1-13 MEAT INTAKE AND URINARY TRACT TUMOURS RISK ASSESSMENT THROUGH PROMOTING LATENT VARIABLES MODELS**

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**Introduction** Generalize Linear Latent and Mixed Models are scarcely used in cancer epidemiology, having been basically used multilevel and generalised linear and mixed models. Using flexible models allow including random effects, common factors coupled to a multilevel structure for unobserved heterogeneity. Cancer is the main cause of death worldwide. In Córdoba, (Argentina) bladder cancer is the fourth most incident cancer among men and ninth in the overall population. Previous studies have suggested that fruits, lean meats, some cereals and cereal products, and vegetable oils would prevent against these tumours, while some fatty meats and use of sweeteners, may increase the risk. In the present work new methodological strategies are used in order to explore the dietary influence on the disease outcome.

**Objective** To define some possible promoting dimensions related to meat intake and combine with a disease model including some bio-socio-cultural characteristics.

**Methods** A case-control study, conducted in Córdoba, including 221/472 cases/controls is used. Subjects were interviewed using a validated FFQ containing biological, educational and lifestyle characteristics, and 127 food items. A two steps model was proposed: common factor modelling as confirmatory factor analysis to explore the dimensionality of constructs from the diet information; and a disease model, which arises from the composition of exposure and measurement models.

**Results and Conclusion** Two constructs were identified, a promoting and a protective one. The direct and indirect covariates effects were also estimated as risk. This work improves the understanding about the diet-cancer relationship.