DOWNLOAD PDF THE LIFE COURSE PLOT IN LIFE COURSE ANALYSIS TIM COLE

Chapter 1 : Epidemiological Methods in Life Course Research - Oxford Scholarship

This chapter considers the life course in the context of an outcome in later life as related to early growth status, where each subject is assumed to have been measured as a child at two or more pre-specified ages.

Advanced Search Abstract The fetal origins of adult disease hypothesis postulates that the inverse association between birth weight and later adverse outcome reflects fetal programming that increases the risk of later disease. However, low birth weight is associated with catch-up after birth, and weight gain is itself a risk factor for later disease. It is difficult to disentangle the effects on outcome of the size and growth components of weight change through time. This paper presents the life course plot, a device to display both size and growth effects simultaneously. It is based on the multiple-regression analysis of the outcome on the various weights, expressed as z-scores, and the plot displays the coefficients plotted against the corresponding ages of measurement. Examples from Brazil Pelotas and the Phillippines Cebu relate blood pressure in adolescence to weight through childhood. They show small inverse weight effects in infancy, but early weight is less important than weight and weight gain during adolescence. In addition, birth length in the Cebu study affects the strength of the relationship between weight and blood pressure in adolescence. This suggests a fetal programming effect, with children who were relatively long at birth having a more sensitive relationship between blood pressure and weight at age Whether this is a good or a bad thing is not immediately clear. There is considerable evidence that size early in life, particularly weight at birth, is related inversely to later chronic disease. Infants of low birth weight are more likely to suffer from such disease as hypertension, diabetes or coronary heart disease as adults. The original interpretation of this inverse association was within the framework of the fetal origins of adult disease hypothesis, i. Many of the markers used as proxies for adult chronic disease for example, elevated blood pressure or insulin or cholesterol are strongly positively related to body weight at the time of measurement. This is in contrast to the inverse association between the various markers and weight at birth. In many of the studies reporting this inverse association, the association was strengthened by including a concurrent adjustment for current weight 1. The outcome for example, blood pressure can be adjusted for the combined effects of weight at birth and weight in adulthood using multiple regression analysis, where the two regression coefficients of weight are of opposite sign. The coefficients can be made comparable by converting the weights at the two ages to sd scores z-scores , with a mean of 0, an sd of 1 and a normal distribution. Once this is done the two coefficients can be compared directly. And it then becomes apparent that the regression equation can be rearranged. Assume the equation is as follows, where W1 and W2 are the early and later weight z-scores and a1 and a2 are the corresponding regression coefficients: This equation can be rearranged as follows: The second term is still later weight, but with a smaller coefficient than before. This is the way that growth charts are interpreted clinically, so that z-score change is a natural way of expressing divergence from normal growth, i. The rearranged equation shows that the combination of an inverse birth weight association and a direct adult weight association can be interpreted in two quite different ways. The conventional view is that birth weight is a proxy for previous fetal growth looking backward in time , whereas the alternative view is that birth weight is a baseline for weight change in postnatal life, which highlights increasing overweight as a risk factor for chronic disease in later life looking forward in time. Both interpretations are valid as far as they go, but the first is couched in terms of size i. Unfortunately, the two interpretations have diametrically opposed implications for public health. The size approach implies the need to improve maternal nutrition in pregnancy and hence fetal growth, whereas the growth approach urges interventions in postnatal life to control weight gain and overweight. Clearly, this duality of interpretation raises important practical questions. Low-birth-weight infants tend to grow faster i. The key question is whether rapid later weight gain is worse, in terms of outcome, among those with a low birth weight. Related to this is the question of whether the timing of the rapid growth period has a critical effect on outcome. The aim of this paper is to develop a statistical framework for visualizing the impact of size

and growth in early life on later outcome. Two examples are used, based on data from published studies of hypertension in adolescence from the developing world. Further details of the studies can be found in the original publications 3 , 4. For the present analysis, weights at each age were converted to z-scores using the CDC reference 5. The statistical life-course problem is how to present the relationship between weight, as it changes through childhood, and outcome. Growth is special in that it represents the rate of change of sizeâ€"weight velocity is the rate of change of weight. Thus, plotting successive weight measurements for an individual on a weight centile chart allows the data to be interpreted both cross-sectionally, as the position of each measurement on the chart, and longitudinally, in terms of the slope of the line joining successive weight measurements. In the context of life course, it is not the size or growth of the child that matters, but rather the strength of the association between size and growth on the one hand, and outcome on the other. Multiple regression analysis is conventionally used to establish the separate and independent associations between outcome and weight at each distinct age. The analogy of position and slope representing size and growth can be used here, but applied now to the regression coefficients from the regression analysis. Each coefficient is an age-specific measure of association between outcome and size, whereas the change in coefficient from one age to another indicates the association between outcome and growth over the corresponding time interval. This is in the sense of Lucas et al. To make the regression coefficients at each age comparable, weight is expressed as a z-score for age and sex. In this way both the values of the coefficients analogous to size and their change from one age to another analogous to growth are displayed together. Such a chart is here termed a life-course plot. The first illustration of the life-course plot is the example used by Lucas et al. Figure 1 shows the regression coefficients of weight z-score at each age, with split pro-insulin at 10 y as the outcome, from a study of children born preterm. The y-axis shows the effect on insulin of a 1-sd change in weight at each age. The steep slope of the line joining the two points also indicates the importance of weight gain i. Figure 1 also shows that the adverse outcome is associated with a particular pattern of growthâ€"a child whose z-score increases from below 0 at 18 mo to well above 0 at 10 y has the greatest risk of a high insulin level. The life-course plot can be thought of as the shape of the growth curve associated with the outcome. A subsidiary interest is the way that early size interacts with later growth, which tests whether the importance of growth depends on early size. This can be added to the multiple regression equation as an interaction term between early and later size 2 , but it is complicated to include in the life-course plot. Instead, it is easier to split the data into two parts on the basis of early size weight or length , using the median as the cut point, and produce life-course plots for each group separately. This allows differences in the size and growth coefficients to be displayed graphically. They can also be tested formally for significance using appropriate nested regression models. Figure 2 shows separate life-course plots for systolic and diastolic BP at 15 y against weight at four ages from birth to 15 y. It shows a negative effect of birth weight on systolic BP and a larger positive effect of weight at 15 y on systolic and less so on diastolic BP. Taken together, the data for the early and later ages indicate that weight centile crossing from birth to 15 y has a dramatic and consistently linear effect on systolic BP at 15 y, with no evidence of a critical period when weight gain is particularly important. The slope of the line indicates that BP increases on average by 0. For diastolic BP the effect of weight in early life is less clear, but weight gain from 4 to 15 y is still an important influence. The plot shows linear regression coefficients for weight at each age, with an essentially linear trend through childhood for systolic BP but steeper later for diastolic BP. The second example is from the Cebu study, which originally concluded that among males, those most at risk of high BP were relatively thin at birth and heavy in adolescence, whereas there were no obvious early risk factors among females 4. Thinness at birth i. Thus the difference between low weight and thinness is length, and as a proxy for fetal growth, birth length is just as valid as birth weight. Interactions of either with later weight would constitute evidence in favor of fetal programming. Figure 3 shows the findings for males, with separate life-course plots for subjects above and below the 33rd CDC centile for birth length, which is the median for the cohort. For the groups together the coefficients in early life are all small, whereas those at 8 and 16 y are highly significant and of opposite sign, indicating that weight gain from 8 to 16 y i. The plot

shows logistic regression coefficients for weight at each age; associations above or below the median differ according to birth length. An increase in weight centile from 1 to 2 y increases the risk in long infants, but reduces it in short infants. Weight at age 16 has more than twice the impact on risk among those who were long in infancy. This shows that weight gain from 8 to 11 y has a similar effect on later high BP irrespective of birth length, but long infants are sensitive to weight gain from 11 to 16 y i. At the same time, the plots highlight the importance of weight gain i. The importance of later weight, and of weight gain from early to later ages, suggests that obesity is the relevant factor in terms of growth. Indeed, Adair showed previously 4 that the pattern of growth predicting high BP was the same as that predicting high BMI at the same age. Weight and weight change in early life appear to be less important in the two examples here, up to 2 y in Cebu and 4 y in Pelotas. Birth weight affects systolic but not diastolic BP in Pelotas, and there is no sign of a trend in coefficient from birth to 2 y in either study. There is conflicting evidence from the literature about early weight gain, for a variety of outcomes. Barker and colleagues have claimed that first-year weight gain is inversely associated with later heart disease in the Helsinki cohort 6 , though their interpretation of the evidence is not universally accepted 7. In the opposite direction, Ong 8 found that weight gain in the first 2 y is positively associated with obesity at age 5 in a recent British cohort ALSPAC , whereas Stettler 9 reported a positive link between weight gain in the first 4 mo and obesity at age 7 in a large sample of U. Most recently, Singhal 10 has highlighted the importance of rapid weight gain in the first two weeks, among those born preterm, as a risk factor for insulin resistance at ages 13 to There may be a difference between the developing and developed worlds here. There are two particular issues regarding interpretation of the life-course plot. The first question is exactly what the plot represents. The second is how it helps to distinguish between the fetal-origins and postnatal-origins explanations for the early weight association. The life-course plot indicates the age-specific effect on the outcome of body weight 1 sd above the median throughout the age range, as compared with median weight. It also shows the effect on the outcome of weight change from one age to another. However, its most important function is to emphasize the dual nature of size and growth, so that both appear in the same graph. Regarding the second question, whether the life-course plot supports the fetal or postnatal explanation for the association between early growth and later outcome, all the examples show that later weight is by far the most important factor in predicting outcome. Thus, evidence in support of fetal programming must affect this later regression coefficient, as indeed it does in Figure 3. The effect of later growth on high BP is twice as great in those who were long at birth compared with those who were short; the excess of 0. Therefore, greater body length at birth leads to greater sensitivity to body weight of BP in adolescence. However, the fact that the sensitivity of the association between later weight and BP depends on birth length fits with the idea of programming. Of course, it is possible that the risk factor is reduced sensitivity in those born short, rather than the converse. As an analogy, flow-mediated dilatation, which measures the elasticity of the vessel wall as a proxy for vascular health, is reduced in individuals with vascular disease Thus, in this context reduced elasticity i.

Chapter 2 : - NLM Catalog Result

The life course plot in life course analysis Tim Cole 7. Methods for handling missing data Paul Clarke and Rebecca Hardy 8. An overview of models and methods for life course analysis Andrew Pickles and Bianca De Stavola 9.

Advanced Search Abstract Background Growth curve analysis is a statistical issue in life course epidemiology. Height in puberty involves a growth spurt, the timing and intensity of which varies between individuals. Such data can be summarized with individual Preeceâ€"Baines PB curves, and their five parameters then related to earlier exposures or later outcomes. But it involves fitting many curves. Curves for individuals are matched to the mean curve by shifting their curve upâ€"down representing differences in mean size and leftâ€"right for differences in growth tempo , and the age scale is also shrunk or stretched to indicate how fast time passes in the individual i. These three parameters per individual are estimated as random effects while fitting the curve. The outcome is a mean curve plus triplets of parameters per individual size, tempo and velocity that summarize the individual growth patterns. Conclusions The SITAR growth curve model is a useful epidemiological instrument for the analysis of height in puberty. By its nature the growth curve consists of a series of highly correlated measurements, and it is important to reduce the dimensionality of the data to simplify comparisons between individual children. The traditional approach is to identify a suitable parametric model for the given measurement and age range, for example the Jenssâ€"Bayley curve 3 for weight in early life or the Preeceâ€"Baines PB curve 4 for height in puberty. This article focuses on height in puberty, where the PB curve with its five estimated parameters fits individual growth curves extremely well, with a residual standard deviation SD of 6â€"7 mm. The ideal approach would be to fit a form of curve to all subjects simultaneously, and to estimate the subject-specific parameter values as subject random effects. Such an approach could in principle be applied to the PB curve. However, Beath 6 introduced a considerable simplification to the analysis of growth curves by describing a shape invariant model of infant weight. This consists of a single growth curve, which can be applied to all subjects by applying just three subject-specific translations and rotations of the curve to fit the individual subject growth curves. The aim here is to fit the model described by Beath 6 and show how effectively it summarizes height growth around the time of puberty. The second aim is to show how the estimated subject-specific parameters can be related to earlier exposures and later outcomes. Methods Data sets Two data sets are used as examples, both of height in puberty. These were linked to follow-up data on adults, some 50 years later, when inter alia height and insulin-like growth factor 1 IGF-1 levels were measured. The cohort has been described in detail previously, 7â€"9 and an inverse association between age at peak height velocity APHV and later IGF-1 has been reported. The second data set came from a randomized clinical trial of oxandrolone to increase final height in girls with Turner syndrome TS. This is a chromosomal disorder where the second X chromosome is missing or malformed, leading to short stature and primary ovarian failure. A total of girls with TS already on a standard dose of growth hormone were randomized to receive either oxandrolone or placebo from the age of 9 years or the age at recruitment if later until final height was reached. One girl dropped out immediately and 13 more later on, whereas 92 girls remained in the study and 82 had reached final height before the time of analysis. A total of heights were included in the analysis median 12 per child, IQR 9â€"17, range 1â€" The trial, which also included a second randomization to early or late oestrogen for induction of puberty, is reported in detail elsewhere EJ Gault et al. The SITAR model The method used to summarize the individual growth curves followed that of Beath, 6 and was also an extension of the model developed independently by the first author and colleagues. Their interpretations are as follows: Geometrically it corresponds to a shrinking or stretching of the age scale. For this reason the parameter is termed velocity. Note that its effect is to make growth curves steeper or shallower, so it effectively rotates each curve this terminology makes clear its similarity to a random slope effect, though strictly it is a scale change rather than a rotation. Figure 1 illustrates the meanings of the three parameters geometrically, i. Size is an upâ€"down shift on the height axis

red dashed lines and tempo is a right-left shift on the age axis blue long dashed lines , while velocity is a shrinkingâ€”stretching of the age axis green dot-dashed lines. The principle is that by suitable choice of these subject-specific parameters, smoothed estimates of each individual growth curve can be obtained by transformation of the mean curve. This leads to a particularly simple way of assessing goodness of fit. Height and age were fitted both untransformed and log transformed, to identify the optimal underlying scales. Growth related to exposure and outcome In the CHS cohort, the relationships between the SITAR parameters and the outcome measures of height and IGF-1 at follow-up 50 years later were investigated using correlation and regression. This was done in three ways. Comparing the mean values of each growth parameter in the two arms using t-tests. Fitting the SITAR model to the data for each trial arm separately, to demonstrate differences in the two mean curves. The first two approaches test the effect of the oxandrolone intervention on the parameters, while the third shows its effect on the mean growth curve. Table 1 shows the deviances and residual standard deviations RSDs for the alternative models in the CHS and TS datasets, confirming that for CHS log age provided a far better fit than age deviance units smaller , and height was better than log height. Despite this, the effect on the RSD was small. For the TS data, height and age were slightly better than height and log age by 19 units of deviance.

DOWNLOAD PDF THE LIFE COURSE PLOT IN LIFE COURSE ANALYSIS TIM COLE

Chapter 3 : Table of contents for Epidemiological methods in life course research

Growth curve analysis is a statistical issue in life course epidemiology. Height in puberty involves a growth spurt, the timing and intensity of which varies between individuals.

Anne Holmes Tim Cole, Founder and CEO of The Compass Alliance, says that his goal is writing this book is to help his readers by providing lessons that will help generate a sustainable and successful career. He points out early in the book that young adults setting out on a career path can expect to spend â€" at minimum â€" , hours building a career. And yet, too many of us drift into careers â€" or merely take on a series of unrelated jobs â€" without giving our future success much thought. Which leads too many of us to condemned to spending our entire working lives dissatisfied, frustrated, and â€" at best â€" enduring the time that not only makes up fully one-third of our lives, but also impacts our total life satisfaction. The Compass Solution was written for those who want to win their career, not just endure it. Cole says he sees this book as a definitive guide to successful career navigation. A practical resource that balances personal experience with proven theory. A guide for both the newcomer struggling to get started, and the veteran lost and essentially wandering. He opens the book with his own story. Telling the reader that his career evolved â€" as so many of us Boomers could also attest if discussing our own careers â€" from trial and error. He reports that he would work hard, gain responsibility, and think his career was soaring â€" only to have the rules changed on him â€" resulting in his being dumped back to earth, somewhat like Icarus, the mythical Greek figure who dared to fly too close to the sun on wings built of feathers and wax. Once this happened a few times, Cole figured out that his career course was terribly flawed. He would never be able to plot a path to success based on the unpredictability of his supervisor, his company or his industry. Instead, he realized, he needed a constant â€" a True North â€" using himself as the constant. He had to invest in himself. With his lodestar, or True North, being Personal Accountability. And the remaining compass points being People, Process and Perspective. The rest of Part One: Which is perfect, because we all learn better when stories are involved. Obviously, this is a perfect book to gift a favorite protege, offering a lifetime of experience in one book, sharing the secrets most never learn. But you may not immediately realize what the book can do for you â€" and others of us who have already spent decades in the workplace. And further, if challenges you to consider becoming a mentor, or Whisperer, for others. Finally, once you invest the time to read this book, you may find that it helps you figure out what you will do with the rest of your life, once you retire. What inspires you and excites you? What do you find yourself doing outside of work? Think hard about this. Because as Boomers, most of us are going to have decades of time ahead of us, once we reach the current official retirement age of The Compass Solution just may help point you into your new direction â€" the starting block for your second â€" or even third â€" career. At time of publishing this review, the Kindle version of The Compass Solution is available at no charge. What a great gift from the author. We have no idea how long this special pricing will continue.

Chapter 4 : ECU Libraries Catalog

Life course approach to adult health series Life course approach to adult health series. UNAUTHORIZED: The life course plot in life course analysis / Tim Cole.

Chapter 5 : Epidemiological methods in life course research (eBook,) [racedaydvl.com]

View Notes - Chap 6, life course plot from PUBLIC HEA life-cours at National Taiwan University. HUMAN DEVELOPMENI. UFE COURSE. INTERVENTION AND HEALTH Karoly LA.

Chapter 6 : SITAR--a useful instrument for growth curve analysis.

DOWNLOAD PDF THE LIFE COURSE PLOT IN LIFE COURSE ANALYSIS TIM COLE

Contents: Introduction: development and progression of life course ideas in epidemiology / Michael Wadsworth, Barbara Maughan, and Andrew Pickles -- Measurement and design for life course studies of individual differences and development / Jane Costello and Adrian Angold -- Measurement and design for life course studies of the social.

Chapter 7 : Life course plot in life course analysis - Oxford Scholarship

Life course epidemiology is concerned with how factors from the early development period, alongside genetic and environmental influences, can shape the future health of a person.

Chapter 8 : Epidemiological methods in life course research (Book,) [racedaydvl.com]

Tim J Cole This chapter considers the life course in the context of an outcome in later life as related to early growth status, where each subject is assumed to have been measured as a child at.

Chapter 9 : Epidemiological methods in life course research - ECU Libraries Catalog

Life course epidemiology is concerned with the origins of risk, resilience, and the processes of ageing, and how this information can be of value in a public health context - particularly for preventive health care.