the proportion of cases with hypertensive cardiomyopathy is of only 15% [6].

After exploring the clinical characteristics of the patients studied by Bestetti et al. [1], it was found that data related to a higher risk for malignant arrhythmias were more present in the chagasic group than in the hypertensive group, such as the occurrence of ventricular premature complexes (46% versus 18%, $p < 0.005$), and the necessity for the use of amiodarone (39% versus 19%, $p < 0.005$) and of an implanted cardioverter-defibrillator (16% versus 0.7%, $p < 0.001$), despite a similar left ventricular ejection fraction (35% ± 13% versus 36% ± 10%, ns).

In conclusion, I do believe that the main reason for the poorer outcome in patients with CHF due to Chagas heart disease, in comparison to those with CHF secondary to hypertensive cardiomyopathy and similar left ventricular ejection fractions, is the occurrence of malignant ventricular arrhythmias leading to SCD in chagasic patients.

References

[1] Bestetti PR, Otaviano AP, Fantini JP, et al. Prognosis of patients with chronic systolic heart failure: Chagas disease versus systemic arterial hypertension. Int J Cardiol 2013;168:2990–1.
[2] Freitas HF, Chizzola PR, Paes AT, Lima AC, Mansur AJ. Risk stratification in a Brazilian hospital-based cohort of 1220 outpatients with heart failure: role of Chagas' heart disease. Int J Cardiol 2005;102:239–47.
[3] Rassi Jr A, Rassi A, Little WC, et al. Development and validation of a risk score for predicting death in Chagas heart disease. N Engl J Med 2006;355:799–808.
[4] Rassi Jr A, Rassi A, Rassi SG. Predictors of mortality in chronic Chagas disease: a systematic review of observational studies. Circulation 2007;115:1101–8.
[5] da Silva RM, Távora MZ, Gondim FA, et al. Predictive value of clinical and electrophysiological variables in patients with chronic chagasic cardiomyopathy and nonsustained ventricular tachycardia. Arq Bras Cardiol 2000;75:33–47.
[6] Hookana E, Junttila MJ, Puurunen VP, et al. Causes of nonischemic sudden cardiac death in the current era. Heart Rhythm 2011;8:1570–5.

http://dx.doi.org/10.1016/j.ijcard.2014.04.012

Tracking of cardiovascular risk factors from childhood to young adulthood — the Pune Children's Study ☆

Suyog M. Joshi [a,1], Prachi A. Katre [b,1], Kalyanaraman Kumaran [a,c,*,1], Charudatta Joglekar [a,1], Clive Osmond [c,1], Dattatray S. Bhat [a,1], Himangi Lubree [a,1], Anand Pandit [d,1], Chittaranjan S. Yajnik [a,1], Caroline H.D. Fall [c,1]

[a] Kamalnayan Bajaj Diabetology Research Centre, King Edward Memorial Hospital Research Centre, Pune, India
[b] Persistent Systems Ltd., Pune, India
[c] Medical Research Council Lifecourse Epidemiology Unit, University of Southampton, UK
[d] Department of Paediatrics, King Edward Memorial Hospital Research Centre, Pune, India

ARTICLE INFO

Article history:
Received 4 April 2014
Accepted 6 April 2014
Available online 13 April 2014

Keywords:
Tracking
Cardiovascular risk factors
Childhood
Young adults
India

To target preventive measures appropriately in early life, it is important to know what childhood cardiovascular (CVD) risk profiles mean in terms of predicting adult risk. Studies from high-income countries have reported tracking of individual risk factors from childhood to adulthood [1–7]; there are no reports of child–adult tracking in low- and middle-income countries (LMICs). The Pune Children's Study is a cohort of 477 individuals born in the KEM Hospital, Pune, India in 1987–1989. We measured a range of CVD risk factors at 8 [8] (1996–7) and 21 (2009–11) years of age, using similar methods, providing the first opportunity to assess child–adult tracking in a LMIC. Ethical permission was obtained from the KEM Hospital Ethics Committee and informed consent was obtained from all the participants.

Weight was measured to the nearest 5 g, and height and waist circumference to the nearest 0.1 cm. Biceps, triceps, subscapular and suprailiac skinfolds were measured to the nearest 0.2 mm using calipers. Blood pressure was measured supine using a digital monitor; the average of two readings made 5 min apart was used for analysis. Fasting venous blood was drawn for plasma lipid, glucose, insulin and leptin measurements. An oral glucose tolerance test was performed giving 1.75 g/kg (8 years) and 75 g (21 years) anhydrous glucose in water, followed by a 120-minute blood sample (WHO protocol). Plasma glucose, cholesterol, HDL-cholesterol, and triglyceride concentrations were measured using standard enzymatic methods. Plasma insulin and leptin were measured using an immunoenzymometric assay and RIA respectively at 8 years, and a Delfia technique and ELISA at 21 years. Insulin resistance (HOMA-IR) was calculated at both ages using the online Oxford model (http://www.dtu.ox.ac.uk). Overweight was defined as BMI ≥ 25 kg/m^2 and <30 kg/m^2, obesity as BMI ≥ 30 kg/m (WHO criteria) and central obesity as waist circumference ≥90 cm in men and ≥80 cm in women (IDF criteria). Hypertension was defined as systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥85 mm Hg (IDF criteria). Impaired fasting glucose (IFG) was defined as fasting glucose ≥100 mg/dl and <126 mg/dl, impaired glucose tolerance (IGT) as 120-minute glucose ≥140 mg/dl and <200 mg/dl, and diabetes mellitus (DM) as fasting glucose ≥126 mg/dl or 120-minute plasma glucose ≥200 mg/d (ADA criteria). Hypercholesterolaemia was defined as total cholesterol ≥200 mg/dl (NCEP criteria), hypertriglyceridaemia as triglycerides ≥150 mg/dl, and low HDL-cholesterol as HDL-cholesterol <40 mg/dl for men and <50 mg/dl for women (IDF criteria).

☆ Funding source: The Wellcome Trust (083460/Z/07/Z), UK.
* Corresponding author at: MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton General Hospital, Tremona Road, Southampton SO16 6YD, UK. Tel.: +44 23 80777624; fax: +44 23 80704021.
E-mail address: kk@mrc.soton.ac.uk (K. Kumaran).
[1] This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

Table 1
Tracking of CVD parameters from childhood to young adulthood.

Age and sex specific variables	8 to 21 years (n = 354)					
	r (p-value)	% in highest fourth of the distribution at 8 who remained there at 21 y	Relative risk (95%CI)	% in lowest fourth of the distribution at 8 who remained there at 21 y	Relative risk (95% CI)	Cohen's kappa (p-value)
BMI	0.53 (<0.001)	50.6	2.87 (2.05, 4.01)	47.7	2.86 (2.02, 4.05)	0.33 (<0.001)
Height	0.44 (<0.001)	43.4	2.43 (1.70, 3.46)	44.9	2.36 (1.68, 3.31)	0.23 (<0.001)
Waist circumference	0.44 (<0.001)	48.3	2.67 (1.91, 3.74)	40.9	2.12 (1.49, 3.02)	0.28 (<0.001)
Sum of skinfolds	0.44 (<0.001)	49.4	2.80 (2.00, 3.93)	47.7	2.73 (1.94, 3.85)	0.31 (<0.001)
Systolic BP	0.21 (<0.001)	31.3	1.37 (0.93, 2.02)	30.6	1.37 (0.93, 2.03)	0.07 (0.03)
Diastolic BP	0.28 (<0.001)	35.3	1.64 (1.13, 2.38)	25.3	1.00 (0.66, 1.53)	0.14 (<0.001)
Cholesterol	0.53 (<0.001)	57.5	4.19 (2.94,5.97)	49.4	2.88 (2.05, 4.06)	0.36 (<0.001)
Triglycerides	0.33 (<0.001)	37.4	1.79 (1.25, 2.56)	40.2	2.07 (1.45, 2.96)	0.22 (<0.001)
HDL-cholesterol	0.26 (<0.001)	36.0	1.64 (1.14,2.35)	38.9	1.94 (1.36, 2.77)	0.18 (<0.001)
Fasting glucose	0.17 (0.006)	32.2	1.47 (1.00,2.15)	31.0	1.37 (0.93, 2.01)	0.07 (0.03)
120-minute glucose	0.07 (0.15)	27.4	1.20 (0.79, 1.82)	30.5	1.30 (0.87, 1.93)	0.06 (0.06)
Fasting insulin	0.14 (0.009)	28.4	1.18 (0.79, 1.77)	32.1	1.46 (1.00, 2.15)	0.08 (0.02)
HOMA-IR	0.14 (0.006)	35.6	1.65 (1.14, 2.38)	33.7	1.58 (1.08, 2.32)	0.11 (0.002)
Leptin	0.24 (<0.001)	40.5	1.99 (1.37, 2.87)	39.0	1.86 (1.27, 2.69)	0.23 (<0.001)

BMI: body mass index; BP: blood pressure; IR: insulin resistance.

Statistical methods: We assessed tracking between the two ages using Pearson correlation coefficients. We categorized each risk factor by quartiles and calculated the relative risk of persisting in the highest or lowest categories between 8 and 21 years. We assessed the agreement between category status at the two ages using Cohen's kappa statistic. We calculated sensitivity, specificity, and positive and negative predictive values for being in the highest risk category at 8 years and disease outcomes at 21 years (overweight/obesity, central obesity, hypertension, dyslipidaemia, hyperglycemia). Statistical analyses were performed using SPSS 16.0.

Of 477 children studied at 8 years, 357 (75%) participated at 21 years (191 males). Non-participants had higher 8-year BMI ($p = 0.05$) and larger 8-year subscapular ($p = 0.03$), triceps ($p = 0.02$), and biceps ($p = 0.05$) skinfolds but similar values for all other measurements compared to participants. Of the participants, 18.5% were overweight, 2.5% were obese, 4.8% were hypertensive, 5.6% had hypercholesterolaemia, 7.6% had hypertriglyceridaemia, and 69.3% had low HDL-cholesterol at 21 years. Three participants were known to have diabetes; a further 18.5% were found to be hyperglycaemic (11.2% IFG, 5.9% IGT and 1.4% DM).

All the 8-year measurements were significantly positively correlated with their corresponding 21-year measurements, except 120-minute glucose concentration (Table 1); findings were similar in both sexes. Correlation coefficients for the age- and sex-adjusted values ranged from $r = 0.53$ (total cholesterol and BMI) to $r = 0.14$ (fasting insulin and HOMA-IR) ($p < 0.05$ for all). They were also similar using BMI-adjusted variables.

The risk of an individual being in the highest-risk category at 21 years was increased if they were in the same category at 8 years; these associations were strongest for cholesterol and BMI and weakest for fasting insulin, glucose and systolic blood pressure (Table 1). There was a similar tendency to persist in the lowest-risk category between 8 and 21 years. The strength of tracking showed similar results; the closest agreement between categories at 8 and 21 years was observed for cholesterol (Cohen's kappa = 0.36), while the weakest was observed for 120-minute glucose (Cohen's kappa = 0.06).

Positive predictive values of being in the high-risk group at 8 years for developing disease outcomes ranged from 73% (low HDL-cholesterol) to 8% (hypertension) (Table 2). Negative predictive values ranged from 98% (hypercholesterolaemia) to 32% (low HDL-cholesterol). Sensitivity values were less than 50% except for hypercholesterolaemia (75%). Specificity values were 70–80%.

These are the first data on child–adult tracking of a range of CVD risk factors from a LMIC. We found significant tracking for all risk factors, similar in both sexes. Measures of adiposity (BMI, skinfolds, waist circumference) and total cholesterol showed strong tracking. Intermediate tracking effects were seen for blood pressure, triglycerides, HDL-cholesterol, fasting glucose and insulin resistance while tracking was weakest for 120-minute glucose concentrations.

Our findings are consistent with studies from high-income countries [4–7]. Tracking may occur due to persistence of environmental factors through childhood and into adulthood. A meta-analysis showed that dietary behavior and physical activity tracks from childhood into adulthood [9]. As both are modifiable factors, it suggests intervening earlier in the lifecourse may be beneficial. Tracking may also be due to genetic factors. Another possible reason could be 'programming' of risk factors during early life, and may suggest stability of epigenetic changes established earlier. Our previous finding linking birthweight to insulin resistance at four years [10] supports this possibility adding weight to interventions before and during pregnancy to improve fetal growth.

Despite significant correlations of child and adult risk factors, predictive values do not support their use as screening tools. This suggests that measures to reduce risk factors in children should be

Table 2
Positive and negative predictive values, sensitivity and specificity of childhood risk categories for disease outcomes at young adulthood.

Upper fourth risk category at 8 years	Disease outcome at 21 years	Positive predictive value	Negative predictive value	Sensitivity	Specificity
BMI	Overweight/Obesity	42%	85%	48%	82%
Waist circumference	Central obesity	43%	82%	44%	81%
Systolic BP	Hypertension	8%	96%	43%	76%
Diastolic BP	Hypertension	8%	96%	43%	76%
Cholesterol	Hypercholesterolaemia	17%	98%	75%	78%
Triglycerides	Hypertriglyceridaemia	14%	94%	50%	76%
HDL-cholesterol	Low HDL	73%	32%	26%	78%
Fasting glucose	Hyperglycaemia	26%	83%	33%	77%

BMI: body mass index; BP: blood pressure.

public health interventions, such as encouraging regular physical activity and healthier diets, rather than individually targeted interventions.

References

[1] Deshmukh-Taskar P, Nicklas TA, Morales M, Yang SJ, Zakeri I, Berenson GS. Tracking of overweight status from childhood to young adulthood: the Bogalusa Heart Study. Eur J Clin Nutr 2006;60:48–57.

[2] Nicklas TA, von Duvillard SP, Berenson GS. Tracking of serum lipids and lipoproteins from childhood to dyslipidemia in adults: the Bogalusa Heart Study. Int J Sports Med 2002;23(Suppl 1):S39–43.

[3] Lambrechtsen J, Rasmussen F, Hansen HS, Jacobsen IA. Tracking and factors predicting rising in 'tracking quartile' in blood pressure from childhood to adulthood: Odense Schoolchild Study. J Hum Hypertens 1999;13:385–91.

[4] Webber LS, Srinivasan SR, Wattigney WA, Berenson GS. Tracking of serum lipids and lipoproteins from childhood to adulthood. The Bogalusa Heart Study. Am J Epidemiol 1991;133:884–99.

[5] Nguyen QM, Srinivasan SR, Xu JH, Chen W, Kieltyka L, Berenson GS. Utility of childhood glucose homeostasis variables in predicting adult diabetes and related cardiometabolic risk factors: the Bogalusa Heart Study. Diabetes Care 2010;33:670–5.

[6] Bayer O, Kruger H, Kries R von, Toschke AM. Factors associated with tracking of BMI: a meta-regression analysis on BMI tracking. Obesity (Silver Spring) 2011;19:1069–76.

[7] Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. Circulation 2008;117:3171–80.

[8] Bavdekar A, Yajnik CS, Fall CH, et al. Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? Diabetes 1999;48:2422–9.

[9] Craigie AM, Lake AA, Kelly SA, Adamson AJ, Mathers JC. Tracking of obesity-related behaviours from childhood to adulthood: a systematic review. Maturitas 2011;70:266–84.

[10] Yajnik CS, Fall CH, Vaidya U, et al. Fetal growth and glucose and insulin metabolism in four year-old Indian children. Diabet Med 1995;12:330–6.

http://dx.doi.org/10.1016/j.ijcard.2014.04.105

Rheumatic heart disease in modern urban america: A cohort study of immigrant and indigenous patients in Chicago

Rami Doukky [a,b,*], Salaheldin A. Abusin [b], Yohannes A. Bayissa [c], Russell F. Kelly [a,b], Asimul H. Ansari [b]

[a] Division of Cardiology, Rush University Medical Center, Chicago, IL, United States
[b] Division of Adult Cardiology, John H. Stroger, Jr. Hospital of Cook County, Chicago, IL, United States
[c] Department of Medicine, John H. Stroger, Jr. Hospital of Cook County, Chicago, IL, United States

ARTICLE INFO

Article history:
Received 18 April 2014
Accepted 19 April 2014
Available online 26 April 2014

Keywords:
Rheumatic heart disease
Valvular heart disease
Indigenous
Immigrant
Minority
Urban

It is widely reported that the natural history of rheumatic heart disease (RHD) is more aggressive in developing countries, manifesting symptoms and requiring interventions at a younger age than in industrialized nations [1–5]. As the demographics in urban America shift with growing immigrant minorities from Latin America and other developing countries, it is possible that the demographics and natural history of RHD in the U.S. are changing as well. We compared the natural history and disease burden between immigrant and indigenous patients with RHD receiving care at Cook County Health and Hospitals System, a large public health care network serving inner-city Chicago. The study was internally funded and approved by the Institutional Review Board of John H. Stroger, Jr. Hospital of Cook County.

A retrospective cohort study design was implemented. In a query of electronic health records, we identified 1257 adult patients with clinical or echocardiographic documentation of rheumatic valvular heart disease, heart valve prosthesis, or valvular intervention in the period between January 1, 2005 and December 31, 2011. A detailed chart review excluded 1153 subjects with valvular surgeries for indications other than RHD. Review of echocardiographic images of the remaining 104 patients excluded 13 patients for lacking diagnostic rheumatic valvular deformity [6]. One patient was lost to follow-up. In the 90 remaining subjects, the latest echocardiographic examination on records and the last one prior to valvular intervention (if different) were reviewed to determine the presence and severity of stenotic and regurgitant valvular lesions [7,8]. The pulmonary arterial systolic pressure was calculated [9].

Among the 90 subjects included in the study 33 (37%) were *immigrants*, raised and acquired the disease outside the U.S. and 57 (63%) were *indigenous* raised and acquired RHD in the U.S. Women comprised 79% of both groups. All immigrant patients migrated from developing countries [Latin America (16); East Asia (7); South Asia (3); Sub-Saharan Africa (3); Middle East (2); East Europe (2)]. African Americans comprised 98% of the indigenous group, which is significantly greater than their proportion in the general echocardiography laboratory population (81%) [odds-ratio = 13.1, 95% confidence interval (CI) = 1.8–95.5, $P < 0.001$].

The study cohort was followed for a mean of 10 ± 8 years (900 patient-years). During follow-up, immigrants had higher event rate of the primary endpoint of valvular intervention [22 (67%) vs. 25 (44%), hazard-ratio = 3.0 (CI = 1.6–5.5); $P < 0.001$] and had their first intervention at younger age, by an average of 11 years. Similarly, immigrants had a higher rate of the secondary endpoint of symptomatic onset of RHD [28 (85%) vs. 35 (61%), hazard-ratio = 2.9 (CI = 1.7–4.8); $P < 0.001$] and had their symptom onset at younger age, by an average of 13 years (Fig. 1, Table 1). By age 40 years, immigrants were more likely to be diagnosed with RHD (33% vs. 14%, $P = 0.03$), have symptom onset (33% vs. 9%, $P = 0.008$), and undergo valvular interventions (33% vs. 7%, $P = 0.02$). Immigrants were more likely to present with atrial fibrillation as a first manifestation of the disease (Table 1).

* Corresponding author at: Rush University Medical Center, Division of Cardiology, 1653 W. Congress Pkwy, Chicago, IL 60612, United States. Tel.: +1 312 563 7473; fax: +1 312 563 7475.
E-mail address: rami_doukky@rush.edu (R. Doukky).