Defining the research priorities to fight the burden of cardiovascular diseases in Latin America

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Journal of Hypertension 2008, **26**:1886–1889

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Received 6 April 2008 **Accepted** 14 May 2008

Introduction

Latin America is undergoing a burden of cardiovascular diseases (CVD) and diabetes mellitus type 2 that are responsible for 46% of the total reported deaths [1]. This is a general phenomenon in developing countries; thus by 2001, 79% of all chronic disease-related deaths in the world happened in developing countries [2]. An increase in life expectancy in Latin American countries is one of the factors that are influencing the burden of CVD. For instance, in Colombia, life expectancy has increased from 55 to 72.2 years in the last 50 years. Moreover, the adaptation to occidental life styles in developing countries has given rise to an increase in the prevalence of overweight, obesity, hypertension, metabolic syndrome, diabetes mellitus type 2 and CVD [3]. Changes in nutritional habits and physical activity are the main characteristics involved in the fast economic transition experienced by these countries in recent years [4]. Obesity and overweight in children and adolescents are of particular relevance in developing countries. Results from the most recent US National Health and Nutrition Examination Survey (NHANES) showed higher rates of overweight and obesity in children from minority groups when compared with non-Hispanic whites, thereby suggesting that the Hispanic pediatric population has predisposition to develop increased adiposity [5]. This result is supported by the 'thrifty phenotype' hypothesis [6], which suggested that when the fetal environment is poor, there is an adaptive response, which optimizes the growth of key body organs to the detriment of others and leads to an altered postnatal metabolism, which is designed to enhance postnatal survival under conditions of intermittent or poor nutrition. Suboptimal intrauterine nutrition may result in the loss of structural units, such as nephrons, cardiomyocytes or pancreatic B cells during the development of organ systems [7]. Those adaptations during fetal programming may prove to be detrimental if food becomes more abundant [8–10]. Transient nutritional stimuli occurring at critical ontogenic stages may have lasting influences on the expression of various genes by interacting with epigenetic mechanisms and altering chromatin conformation and transcription factor accessibility [11]. Converging data are now available to support the hypothesis that, in addition to 'thrifty genotype' inheritance, individuals with metabolic syndrome underwent incorrect 'epigenetic programming' during fetal/postnatal development because of inadequate maternal nutrition and metabolic disturbances [12]. These individuals may also display 'transgenerational effects' because of the inheritance of epigenetic changes first experienced either by their parents or grandparents, or both [12]. Thus, in developing countries, restriction of maternal and fetal nutrition or the restriction of placental growth and function can result in adaptations that may enhance the probability of fetal survival, but they may also contribute to the association between an adverse intrauterine environment and an increased risk of hypertension, metabolic syndrome, diabetes mellitus type 2 and CVD in adult life, as nowadays the transplantation to a western lifestyle is contributing to a high intake of high-calorie food, animal fat and processed sugars [13,14]. The impact of this nutritional transition on the risk of myocardial infarction in Latin America was recently demonstrated by the global study of risk factors for acute myocardial infarction (INTERHEART study) [15,16], which identifies risk factors for acute myocardial infarction and provides information about the population attributable risk (PAR) in Latin America. Although the nine risk factors for coronary heart disease account for 90% or more of the PAR for both men and women worldwide, an important difference was noted in Latin American countries, in which the PAR for abdominal obesity was more important (48.5%) than in the rest of the world (30.2%). Moreover, in a similar population-based control study [17] in Costa Rica, the PAR for myocardial infarction among 889 individuals who had no history of diabetes mellitus type 2, hypertension or prior medical therapy, abdominal obesity (PAR = 29.3%) was the leading risk factor, especially in women (PAR = 35%). Recently, in 325 schoolchildren (mean age 10.0 years) selected from the school population of Bucaramanga, Colombia [18], a positive correlation was shown among BMI, systolic blood pressure (SBP) and C-reactive protein (CRP), demonstrating an interrelationship between adiposity and the increase in subclinical inflammation.