Health in southeast Asia

Neglect of human rights that compromises health outcomes, the combination of high population density and domestic livestock that encourages zoonoses, claims over viral sovereignty, and an emphasis on health tourism that creates dual standards of care: too often health in southeast Asia is in the news for the wrong reasons. Today's Lancet reports good news from the region as well as disappointments, and provides opportunities to improve care locally by analysing how a variety of health systems in different settings within the region are responding to rapid socioeconomic change and shifting threats to health. The six theme papers in the Series on health in southeast Asia1–6 show a range of responses to public health challenges, some of which might inform policy in other countries at similar stages of development. This is the first Lancet Series to focus on a region. In doing so, health professionals from different disciplines in the ten member countries of the Association of Southeast Asian Nations (ASEAN)—Brunei, Myanmar, Cambodia, Indonesia, Laos, Malaysia, Philippines, Singapore, Thailand, and Vietnam—collaborated to identify common themes and individual approaches to problems that challenge health systems locally and around the world.

The strength of southeast Asia is its diversity: social, geographic, religious, and economic. But these same factors can also be weaknesses that limit intercountry cooperation on levels deeper than superficial self-interest. Health care offers a path to better mutual understanding by developing and sharing best practice and, as local capacity is developed (eg, the Mekong Basin Disease Surveillance Network), to forge more meaningful and substantial surveillance and clinical links between countries to improve health across the region. The Series addresses aspects of concern to all health systems: the burden of disease, prevention, and treatment;1–4 human resources for health;5 and financing.6

Other topics were too controversial, for example, human rights. To advocate for health based on human rights was an agreed goal of WHO’s 6th Global Conference on Health Promotion, held in Bangkok, Thailand, in 2005. Human rights are also enshrined in the 2008 ASEAN charter, to which all ten countries in the Series subscribe. But they are too often absent in health and the social determinants of health in southeast Asia. For example: limited sanitation to marginalised rural ethnic minorities in Vietnam and discrimination against people with HIV/AIDS in Cambodia. Addressing the social determinants of health, such as sanitation, education, nutrition, and equitable access to care are fundamental to improving the health of the population.

There are examples of hope. Six ASEAN countries—Cambodia, Indonesia, Laos, Philippines, Thailand, and Vietnam—are signatories to the International Covenant on Economic, Social and Cultural Rights, which forms a core for health-related rights. The Philippines and Vietnam recognise the right to health in their constitution. The Philippines also makes access to information a right in law (as does Thailand) and has legislated for access to essential medicines. Another marker of good practice is Malaysia’s charter for patients’ rights. In Myanmar, the release from prolonged house arrest of pro-democracy advocate Aung San Suu Kyi, as the Series went to press, may herald a change in attitude that could allow greater external collaboration to improve the country’s health performance.

To establish a rights-based approach to health and improve health outcomes in southeast Asia requires not only political will and investment, but also leadership by health professionals. In doing so, health professionals must stand apart from the politics and narrow interests that have too often fettered, rather than fostered, progress in the region. This leadership needs to come from within the members of ASEAN, so that regional identity and focus is clear (by contrast to the WHO regions that subdivide southeast Asia). Until public health trumps private wealth, progress in health across the region will be disjointed and inequitable. Only by placing human rights at the heart of development will the right of the region’s 580 million people to the highest attainable standard of health begin to be realised.

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Addressing the complexity of cardiovascular disease by design

Myocardial infarction and its main determinant, coronary artery disease, together the major cause of death and disability worldwide, are strongly heritable. From a genetic standpoint these diseases are classified as complex, because they do not segregate in affected families following the patterns of Mendelian genetics (i.e., autosomal dominant, recessive, or X-linked). Their inheritance is therefore believed to arise from several different genetic variants, which interact among themselves and with environmental exposures.

Genome-wide association studies have made major steps in deciphering the genetics of such complex traits as cardiovascular diseases. These hypothesis-free scans of the human genome, which simultaneously interrogate a large proportion of known common single-nucleotide polymorphisms (SNPs; common is defined according to the HapMap project—i.e., with a minor allele frequency above 5%), have identified several hundred SNPs associated with common diseases. In coronary disease, nine studies have found 14 chromosomal loci at which one or more common SNPs are associated with coronary artery disease or myocardial infarction or both (table). Interestingly, almost all studies consistently reported an association for non-coding SNPs at the 9p21.3 locus next to CDKN2A and CDKN2B, which can be considered the most widely and consistently replicated genetic risk factor for coronary artery disease and myocardial infarction.

Despite the achievements of genome-wide association studies, much of the genetic risk that underlies the development of coronary artery disease and myocardial infarction is unexplained by common risk-SNPs. Large meta-analyses of data from genome-wide association studies that include several thousand patients and controls are underway but, even with sample sizes in the order of 100 000, are unlikely to explain more than 15–20% of the heritability of coronary artery disease and myocardial infarction. The application of next-generation DNA sequencing with the improved catalogue of low-frequency genetic variation provided by the 1000 Genomes Project promises to reveal much of the unknown heritability of complex traits, and to unravel the interplay between common risk-SNPs identified by genome-wide association studies and all surrounding genetic variation in determining the predisposition to common diseases.

However, even if it was possible to re-sequence the entire genome in several cases and controls with coronary artery disease and myocardial infarction, the phenotypic complexity of these disorders would still represent an obstacle to a full understanding of the underlying genetics. Indeed, the transition from a normal artery to myocardial infarction involves several pathological processes: the initiation and progression of an atherosclerotic plaque and its rupture, arterial thrombosis, and infarction of cardiac tissue. Each process is probably influenced by particular genetic risk factors. By contrast with this complex scenario, genetic association studies have rarely addressed the issue of different genetic risk factors contributing to either coronary artery disease or myocardial infarction.

In The Lancet, Muredach Reilly and colleagues’ address the heterogeneity of these different cardiovascular phenotypes in genome-wide association studies. The investigators adopted an original study design and criteria for the definition of cases and controls, tailored to show genetic predisposition to angiographically defined coronary artery disease (study A) or myocardial infarction in the context of coronary artery disease (study B). In study A, they compared individuals affected by coronary artery disease with individuals without stenosis at coronary angiography, thereby circumventing the relatively high prevalence of coronary atherosclerosis.