SHORTCOMINGS & MISSTEPS?

MALAYSIA'S EFFORTS IN ADDRESSING CHILD STUNTING

Derek Kok

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Introduction

Despite the government's policy introductions, the rate of child stunting in Malaysia has not improved, but in fact worsened to its 1999 level – this is in contrast to the steadily improving global rate.

Drawing from a review of recent evidence, this paper argues that this poor performance can be attributed, in part, to a global trend which erroneously views stunting as an individual-level nutritional diagnosis instead of its intended purpose as a statistical proxy for a population's general welfare. This approach leads to child stunting being viewed solely as a nutritional issue instead of a multicausal problem that is brought about by social determinants that require structural solutions.

In making this argument, this paper first provides the context of child stunting in Malaysia and its consequences at both the individual and national levels. It then proceeds to trace the changing definitions of stunting and how it evolved from a population-level mathematical proxy to become a biological diagnosis for nutrition at the individual/child level. Naturally, this leads to policy responses that focuses on individual parental behaviour with little impact on stunting rates.

Contextualising Stunting in Malaysia[1]

Stunting simply means that a child is short for their age. Stunting reflects the deficiencies in a child's growth environment during the critical 1,000-day period covering pregnancy and the first two years of the child's life. It is during this period that a child's growth and brain development is at its most rapid and most sensitive to nutritional disruptions (Cusick & Georgieff 2018).

Stunting wields devastating and practically permanent health effects, such as increased risk of child mortality and morbidity, and diseases like high blood pressure in adulthood (de Onis & Branca 2016). A large body of research exists that also shows a strong link between stunting and cognitive development. These studies indicate that stunted children usually display delayed development of motor skills such as crawling and walking, and exhibit diminished exploratory behaviour (Brown & Pollitt 1996). In a 2013 study of 8062 children in Ethiopia, India, Peru, and Vietnam, stunted children were found to have lower mathematics achievement, reading comprehension, and receptive vocabulary than children who were never stunted (Crookston et al. 2013). Stunting can also lower the future income-earning ability of stunted children. It is estimated that adults who were stunted during their childhood earn 20% less in their working life compared to non-stunted individuals – each additional centimetre of adult height can be associated with an almost 5% increase in wage rates (Grantham-McGregor et al. 2007).

Childhood stunting as such has potentially serious social and economic costs for the labour market and overall economic development of the country. A 2018 World Bank study by

[1] At time of publication, most recent data on child stunting under the National Health & Morbidity Survey 2023 had yet to be released; as such this paper uses the then latest available data from the National Health & Morbidity Survey 2019.

Galasso and Wagstaff sought to calculate how much lower a country's per capita income is today due to the effects of childhood stunting on its adult workers. It was shown that countries lose, on average, 7% of per capita income because of stunting (Galasso & Wagstaff 2018). These effects of stunting will be amplified even more in poorer segments of society.

The consequences of stunting can go beyond one lifetime and transcend future generations. Women who were themselves stunted as children, are at greater risk of bearing stunted children (Black et al 2013; Addo et al 2013). As stunting is largely irreversible, stunted girls grow up to become stunted adult women, who then give birth to stunted children. This may perpetuate a vicious cycle of poverty and stunting for children from lower income groups that are already at higher risk of stunting compared to their more affluent counterparts (Menezes et al 2011).

These effects of stunting make its high prevalence in Malaysian children deeply concerning. However, despite the promulgation of the National Plan of Action for Nutrition of Malaysia 2016-2025 and the 2022-2030 National Strategic Plan to Address the Problem of Stunting in Children, policy outcomes on this front are far from encouraging. Although stunting rates have been decreasing over the past several decades globally, Malaysia's child stunting rate is on a worsening trend. In 2019, its stunting prevalence rose to 21.8% from 20.7% in 2016 (NHMS 2019). This is higher than its 1999 level and for the first time ever, worse than the global stunting rate.

Despite being an upper middle-income country, Malaysia's stunting rate is higher than some lower middle-income and low-income countries such as Ghana and Senegal. In fact, Malaysia's stunting prevalence is far worse than West Bank & Gaza (7.4%) and comparable to Iraq's stunting rate (22.6%) at the end of the American invasion in 2011 (Kok 2019). The prevalence of stunting in upper middle-income countries stands at 6.0%, a striking distance from Malaysia's 21.8%. In fact, Malaysia's current rate far exceeds the target of 11.0% to be achieved by the year 2025 under the National Plan of Action for Nutrition of Malaysia 2016-2025 (Ministry of Health Malaysia 2016). A pre-pandemic study found that about 22% of children living in low-cost flats in Kuala Lumpur are stunted, more than double the Malaysian capital's average (UNICEF 2018). This large disparity in follows a general pattern nationwide where stunting is more prevalent in lower-income segments of the population. In 2019, the bottom 40% of Malaysian households in terms of income recorded a stunting rate of 22.4%, in contrast to 16.8% of top 20% households (NHMS 2019). Stunting rates are expected to be worsen significantly due to the effects of the COVID-19 lockdowns and the slow post-pandemic recovery in Malaysia.

Two policy blindspots may explain this poor performance: (i) a conceptual misunderstanding of what stunting means on a statistical and biological level; and consequently, flowing from that conceptual misunderstanding, (ii) an overtly narrow policy focus on nutritional behavioural change at the individual level.

Shifting Definitions of Stunting

Stunting means that a child is short for their age and is technically defined as a child with a height-for-age Z-score (HAZ) less than minus two standard deviations (<−2 SD) below the median of a reference height-for-age standard (WHO 1995; 2006). HAZ is calculated by

subtracting an age- and sex-appropriate median value from a standard population and dividing by the SD of the reference standard population.

The reference standard used is the World Health Organisation (WHO) Child Growth Standards, which was developed from the Multicentre Growth Reference Study (WHO 2006). The Multicentre Growth Reference Study was mooted by the WHO after its review of anthropometric references in the early 1990s found that the main growth reference being used was inadequate for assessing the growth pattern of healthy breastfed infants. The development of new international standards (as opposed to a reference merely describing how children grew) was recommended, using a novel approach that would describe how children should grow when free of disease and when their care follows healthy practices such as breastfeeding and non-smoking (de Onis 2015). The key assumption underlying the WHO's efforts to develop the universal child growth standards is that growth is driven by "the biological reality that environmental differences rather than genetic endowments are the principal determinants of disparities in physical growth" (Garza & de Onis 2004).

From 1997 to 2003, the Multicentre Growth Reference Study collected data on the growth of 8500 children aged 0-5 years from six sites (Brazil, Ghana, India, Norway, Oman, and the USA) with various ethnic backgrounds and cultural settings. To reduce the influence and impact of environmental variation on growth, only children from privileged and healthy populations were included in the study, while their mothers had to follow a strict set of recommended practices and behaviours associated with healthy outcomes such as breastfeeding their children and not smoking during and after pregnancy.

This study discovered that average growth is strikingly similar around the world when conditions for growth are optimal. The study found only about 3% variability in foetal growth, which suggests that children from different genetic and cultural backgrounds are likely to grow on a similar trajectory until the age of five years when given this optimal environment. Any variability in the children's growth were observed to be caused by factors unrelated to genetics or ethnicity, and were more influenced by nutrition, environment, and healthcare differences. The Multicentre Growth Reference Study resulted in the new WHO Child Growth Standards, which included standards for height-for-age, weight-for-age, weight-for-height, BMI-for-age, head circumference-for-age, arm circumference-for-age, subscapular skinfold-for-age, triceps skinfold-for-age, and motor development milestones.

As de Onis et al put it, "the scrutiny that the WHO standards have undergone is without precedent in the history of developing and applying growth assessment tools, whether national or international" (de Onis et al 2012). Some of initial objections to the new WHO Child Growth Standards included the likes of Hui et al (2008) who wrote that that ''these growth standards could be invalid or even misleading for the one fifth of the global population in East Asia'' while Wright et al (2008) opined that the new standards "may not be simply transferable to the United Kingdom", both expressing that the study sample should have been a random selection from the collection of all sites with healthy populations, not just the six countries.

By 2011, however, the WHO Child Growth Standards have been widely implemented; results of a 178-country survey showed that the WHO standards are universally used in paediatric care, whereby 125 countries had adopted the standards (de Onis 2012 et al). In Southeast Asia for instance, only one country had not adopted them when surveyed. The findings and applicability of the WHO Child Growth Standards has recently been reinforced by the multi-

country study Intergrowth-21st. Using a similar methodology to the Multicentre Growth Reference Study, the study reported similar findings across its eight study sites whereby foetal growth and new-born length was similar across these diverse geographical settings when environmental constraints are few (Villar et al 2014). Strikingly, although the Intergrowth-21st study was conducted several years after the Multicentre Growth Reference Study, the mean birth length for term new-borns for both studies was highly similar with a mere difference of 0.1cm.

The past decade has seen an unprecedented rise in attention on child undernutrition, as witnessed by the proliferation of global nutrition initiatives, the creation of worldwide goals for nutrition indicators, and high-level publications on the subject matter. After a prolonged period of 'neglect' due to the difficulties in visually identifying stunted children and the lack of routine assessment of linear growth in primary healthcare (de Onis et al 2016), stunting is now framed as the key global nutrition challenge. Stunting is now firmly in the global agenda as a major development priority and the centre of high-profile initiatives such as Scaling Up Nutrition, the Zero Hunger Challenge, and the Nutrition for Growth Summit. Stunting is also one of the six global nutrition targets for 2025 that was adopted in 2012 by the World Health Assembly, and is also a key target under the United Nations' Sustainable Development Goal (SDG) 2 of achieving 'zero hunger'.

While this spotlight on stunting has contributed to garnering wide support for tackling child undernutrition throughout the world, a growing body of literature asserts that this focus has also borne 'misunderstanding about the meaning of stunting among researchers, donors, and agencies active in nutrition' (Leroy & Frongillo 2019). The links between child linear growth and environmental conditions have been well established as early as the mid-1800s, with child height being used as a proxy to measure the economic development of populations for more than 200 years (Fogel 2004). However, Perumal et al (2018) argue that the conceptual and practical interpretation of stunting, as defined by the proportion of children with HAZ <–2 SD, has "evolved dramatically" over the past several decades. Stunting is now widely used as a classifier of disease at the individual-child level as a direct reflection of undernutrition. The authors however assert that this is a "misuse" of the concept, as the prevalence of stunting was originally used as an indicator of a population's "general level of living and welfare".

This line of thinking is not new. Tracing back to the 1980s, scholars have asserted that "height is only important as a proxy for social deprivation" (Waterlow, 1988). A significant echo of voices had started to caution against using HAZ to imply that stunting was a form of malnutrition, as opposed to an indicator for environmental deprivations surrounding the child. Beaton (1989) opined that nutritionists have been "mistaken in labelling the problem malnutrition rather than what it is, growth failure consequent to environmental constraints". As Van Lerberghe puts it (Waterlow, 1988):

> "*I think it's dangerous to talk about the 'problem of stunting.' Is 'stunting' a problem, or is 'poverty' a problem for which stunting could be an indicator? If we go on using the term 'stunting problem,' we might be formulating 'stunting control programs,' which could create the illusion that we can solve the 'stunting problem.' But why should we solve a so-called 'stunting problem' as such and let the problem of poverty remain? Such 'stunting control programs' would divert attention from looking at the real solutions,*

> *which conceptually are much more difficult to develop and will certainly go far beyond what is considered to be the territory of the nutritionist*." (p 37)

Broadly, it was viewed that the use of stunting as a direct indicator of children's nutritional status had shifted the emphasis away from social and environmental determinants of growth to "a disproportionate emphasis on dietary determinants of linear growth" (Perumal et al 2018).

One of the criticisms levelled against this conception of stunting as a nutritional condition in its own right, is the use of HAZ <–2 SD. The definition of stunting itself is entirely dependent on a child's height being classified as HAZ <–2 SD, or two standard deviations below the median of the WHO Child Growth Standards. The use of HAZ <–2 SD as an indicator enables comparisons of height-for-age distributions across time as well as across populations (and between comparison and intervention groups). This is acceptable as long as it is used to measure patterns and make inferences about the population as a whole, and not about individual children within the population itself. Current uses of the technical definition of stunting (HAZ <–2 SD) however suffers from a misinterpretation of the meaning of the –2 SD cut-off point, leading to numerous pitfalls.

Firstly, using HAZ <–2 SD as an individual-child signifier instead of its intended purpose as a statistical indicator of a population's height distribution can lead to an underestimation of linear growth faltering, which is defined as a failure to reach one's linear growth potential. The number of children suffering from linear growth faltering is much higher than the number of children that are stunted. In poor contexts, all children in that population would likely be smaller than their potential if they had been in a growth-conducive environment (Frongillo et al 2019). In fact, even in in a healthy population, ~2.5% of all children have a HAZ <–2SD. This means that using stunting as an indicator can end up underestimating the number of children who suffer from linear growth faltering because the <–2 SD signifier implies that only children under that cut-off point have growth deficits. As de Onis (2000) puts it,

> *"In reality, there are not two distinct populations—one well-nourished and the other malnourished—but rather a continuous gradation of nutritional status. That is, the risk of undesirable health outcomes such as mortality does not change dramatically by simply crossing the cut-off line: significant deterioration within the 'normal' range may in fact carry greater risk."*

Yip & Scanlon (1994) even go as far as to argue that there is no biological basis for the –2 SD threshold:

> "*The –2 SD criterion is not unique to anthropometry; it is widely used for many other clinical laboratory tests. Such a cut-off based criterion is useful for individual-based applications in screening for disease, and for population-based applications in public health monitoring. It was never intended to be an all-or-none definition or diagnostic criteria for disease or nutritional disorders. All too often, we forget about the origin as well as the proper way to interpret a cut-off based a statistical criterion. The cut-off criterion of 2 SD below the mean … can be viewed as an arbitrary point to facilitate uniform applications. In reality, the risk of undesirable outcomes*

including mortality does not change drastically when crossing the magic cut-off point."

Indeed, the use of the –2 SD criterion seems to be rooted in its ubiquitous use in other clinical studies. Frongillo et al (2019) expands on Yip & Scanlon's argument by positing six reasons there is so much focus on the –2 SD threshold of stunting: first, linear growth is relatively stable and unaffected by daily variation compared to other measures such as dietary intake; second, linear growth can be measured easily and accurately on a large scale compared to other child development indicators such as cognitive and socioemotional status; third, the conceptual confusion of the construct of nutrition with the measure of growth; fourth, the mistaken assumption that the usefulness of measuring linear growth (e.g., monitoring population-level prevalence) is automatically useful for other purposes (e.g., estimating impact of interventions); fifth, the ignorance of the complexity of human growth for the simplicity of linear growth measures; and finally, the misframing of nutrition around child size due to its comparative visibility relative to other manifestions of undernutrition.

This view that the –2 SD criterion lacks biological basis is supported by studies that have found a lack of a threshold effect for adverse outcomes among children classified as "stunted" using the –2 SD threshold (Olofin et al 2013; Pelletier et al 1993; Grantham-McGregor 1982). For instance, a study by Sudfeld et al (2015) established that there was no threshold effect at –2 SD or any other cut-point for cognitive, communication, and motor development among children, with a positive correlation throughout the HAZ range. The implication of this lack of threshold effects at the –2 SD cut off point means that the criterion cannot be used to "diagnose" stunting and differentiate "normal" from "growth-restricted" children.

Despite these critical interventions, the notion that stunting is a health condition in its own right has become a mainstay since the 1990s. Stunting and its –2 SD threshold is now the accepted proxy for "chronic malnutrition" and is used as a target indicator at every level from global nutrition initiatives to household food aid programmes – Malaysia is not alone in this. The problem of labelling stunting as a proxy for chronic child undernutrition is that it implies that the child's impaired linear growth is primarily the result of insufficient quality and quantity of food. To quote Perumal et al (2018): "… it is unsurprising that the burden of responsibility for a child's slow growth often ends up placed on the child's mother and family, rather than being viewed as an indicator of a community-wide problem for which the major causal factors are upstream of the household". This narrative has taken hold at the highest levels of governance. In a 2014 WHO report, caregivers and parents were referred to as "the main protagonists of healthy child growth and development (providing appropriate feeding, care and stimulation)"; while stunting is "an outcome of maternal undernutrition and inadequate infant and young child feeding". In India, a high-level governmental committee recommended individual-focused determinants such as encouraging breastfeeding and complementary feeding practices in its national policy strategy to address the country's high levels of child stunting (Haroon et al 2013; Lassi et al 2013; Bhutta et al 2013). The unintended consequence of this framing is that caregivers are wrongly blamed for not ensuring their children's adequate food intake – if these parents simply made better decisions, their children would grow normally, goes the logic.

Such thinking is dominant in both research and practice (Baum & Harris 2006; Raphael 2009), although numerous studies show that an individual child's dietary intake plays a minimal role in their growth (de Onis et al 2013). For instance, the Lancet's proposed adoption of ten

nutrition-specific individual-focused interventions are estimated to result only in a modest effect on stunting rates (Bhutta et al 2013). In fact, decades of research on real-life nutrition interventions show not just minimal but mostly null improvements on height, although the same interventions have highly positive results in other nutritional indicators. For instance, vitamin A supplementation and exclusive breastfeeding significantly lower morbidity and mortality, but do not impact stunting (Black et al 2013). This however means that if stunting is used as an indicator to evaluate effectiveness of these interventions, it could lead to well-designed working programmes that aim to improve social and environmental conditions being wrongly assessed as failing to meet its intended purpose if stunting is the only indicator used in evaluations (Frongillo et al 2019; Leroy & Frongillo 2019).

Disproportionate Focus on Individual Factors

Globally, policies and programmes that are designed to tackle child stunting tend to focus on 'a standard set of risk factors that represent the immediate, underlying, and basic causes of stunting' (Vaivada et al 2020). These causes of stunting are typically depicted by multilevel conceptual frameworks, with the most prevalent model for the last 30 years being the UNICEF Conceptual Framework for Undernutrition (Figure 1).

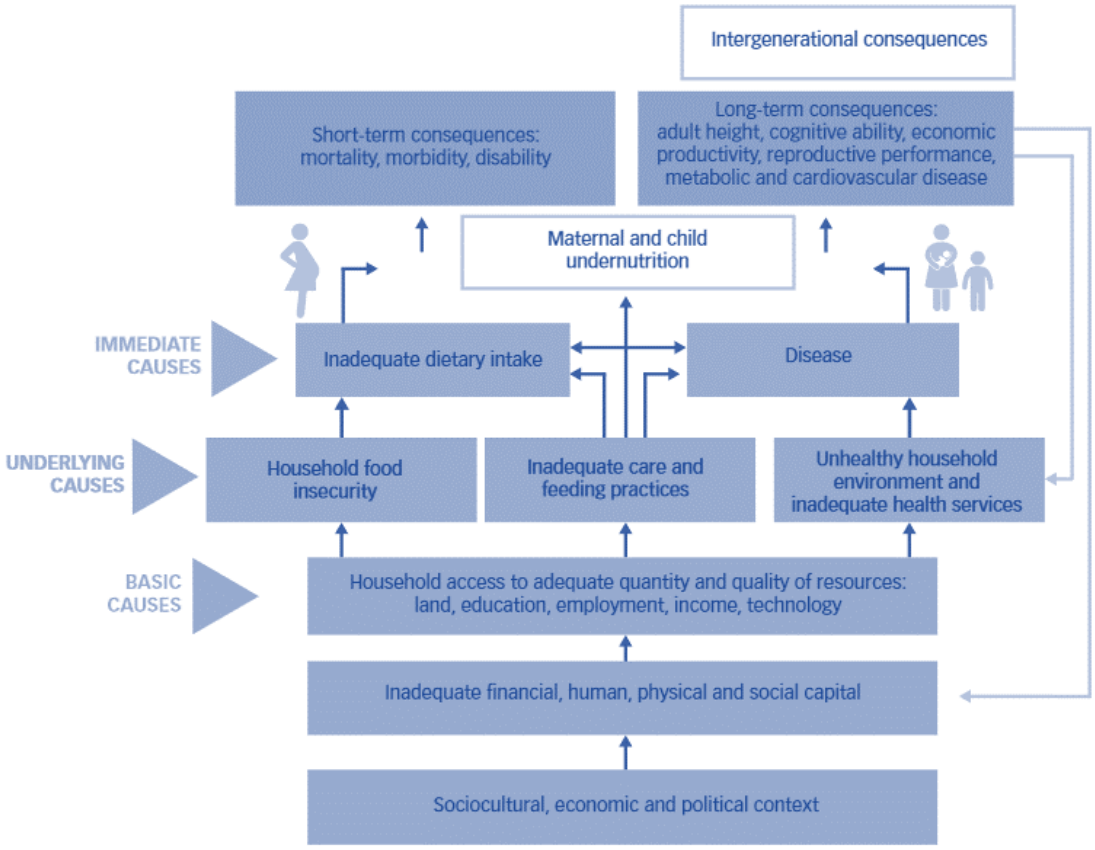

Figure 1: UNICEF Conceptual Framework for Undernutrition

The UNICEF framework illustrates that maternal and child nutrition are inextricably linked and caused by a complex interaction of various influences. Crucially, this framework shows how individual challenges are shaped by factors at the household, community, and national level. For example, the immediate causes of inadequate dietary intake and disease are influenced by underlying food security, caregiving practices, and environmental conditions, which are in turn shaped by systemic-level factors which determine how resources are distributed and accessed (Reinhardt & Fanzo 2014).

While it is a strength of the UNICEF framework that it mentions the 'sociocultural, economic, and political context'; 'inadequate financial, human, physical and social capital"; and "household access to resources" as basic causes of stunting (Stewart et al 2013), this theoretical acknowledgment does not state how exactly these structural determinants can moderate, exacerbate, or facilitate each of the risk factors at the basic, underlying, and immediate levels. These effects are merely assumed in the UNICEF framework, without the necessary empirical evidence (Vaivada et al 2020).

In particular, the social determinants of health are not named, but are subsumed in the UNICEF framework under the broad catch-all phrase of 'basic causes'. This is despite the fact that social determinants of health account for between 30-55% of health outcomes (Marmot 2018).

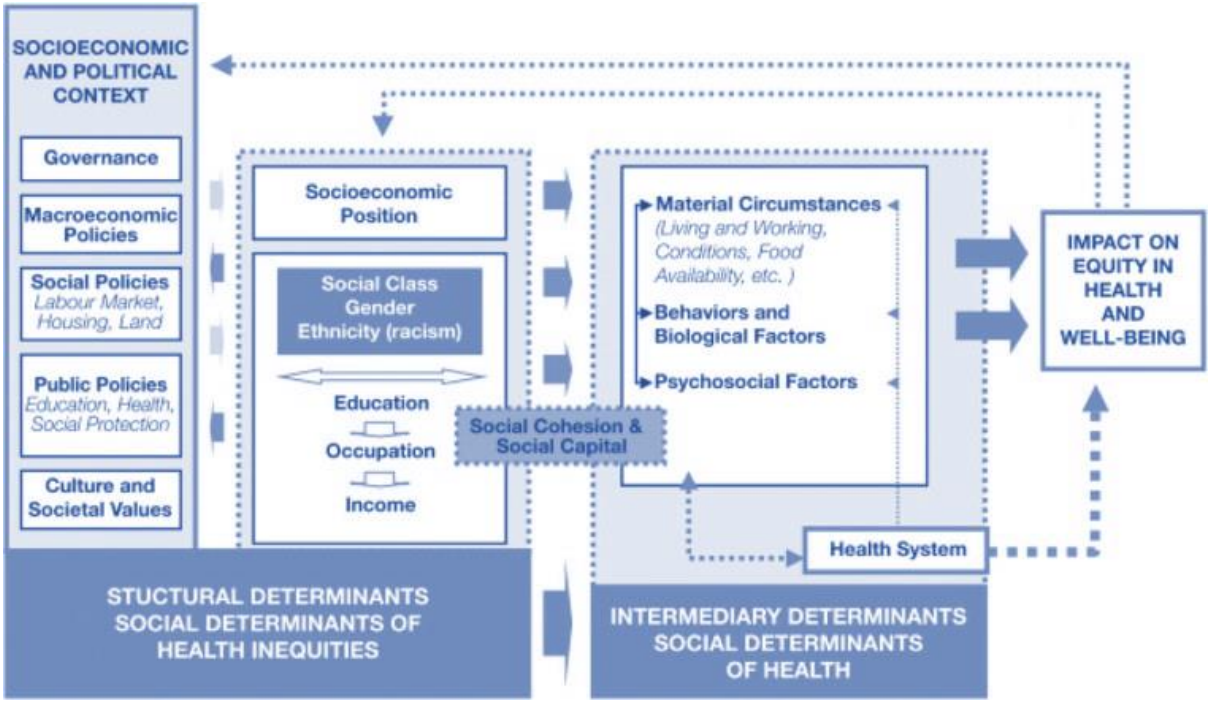

Figure 2: Conceptual Framework of the Commission on Social Determinants of Health

The social determinants of health are "the conditions in which people are born, grow, live, work and age, including the health system" (WHO 2007). These conditions are shaped by the distribution of resources – including both money and power – at local, national, and global

levels, which are in turn influenced by policy choices. The unequal distribution of these resources is what produces inequities in health (Graham 2000), such as the difference in stunting rate seen in children living in low-cost flats in Kuala Lumpur, which is more than twice the overall Kuala Lumpur rate (UNICEF 2018). The conceptual framework above (Figure 2) adopted by the World Health Organization's Commission on Social Determinants of Health (CSDH) provides an example of the different types of the social determinants of health, and the causal relationships between these determinants and health outcomes. The CSDH conceptual framework includes three interactive levels of dynamic influences: the wider socio-political context, individual socioeconomic position and intermediary socioeconomic influences.

Research that centres on social determinants of health would shift the focus on health as a direct outcome solely from individual lifestyle choices to a recognition that health is also influenced by social, political, economic, and political factors (Wuest et al 2002). This shift is crucial to avoid the increasingly common 'individualisation" of government policies which only emphasise individual effort and personal responsibility, particularly in the area of nutrition through the use of dietary guidelines, food labels, menu labelling, and clinical counselling (Mozaffarian et al 2018). This is despite substantial evidence that programmes that aim to change individual behaviour without taking into account social determinants have limited effectiveness (Baum 2002; Patil 2013). In fact, studies suggest that these programmes are especially less effective among lower socio-economic groups which paradoxically are the prime targets of such programmes (Attree 2005; Emmons 2000; Lynch 1996). While this individual approach has met with some success among people in more affluent socioeconomic positions, the net effect of this has been to increase inequities in health outcomes (Baum & Harris 2006).

This narrow approach in policymaking can inadvertently lead to the rise of a narrative that blames women – especially low-income mothers – for the unsatisfactory health outcomes in their families, whilst ignoring the influence of nearly insurmountable social determinants that can constrain the individual choices and decisions of caregivers. To quote Wuest et al (2002):

> "*Women, as principal family caregivers, were seen not only as responsible for their own health status but also for that of their children and partners. If women served the right meals, ensured that family members exercised, and surveyed their families for poor health habits, the nation would be healthy. The unfortunate consequence of such a philosophy is victim blaming, that is, blaming the person who becomes sick for her sickness*."

In addition to this, another unintended consequence of framing health outcomes in this individualistic manner, is that resources and efforts are disproportionately shifted towards policies and programmes that promote behaviour change instead of investing in structural interventions that can impact social determinants. As such, any mooted intervention ends up only addressing the symptoms of the cause rather than the causes themselves. Despite the follies of such an approach, this framing still dominates research and practice (Baum & Harris 2006; Raphael 2009). In India for example, the government identified ten strategies to address the country's high levels of child stunting and other forms of malnutrition, all of which merely addresses nutrition-specific, individual-focused determinants such as encouraging breastfeeding and complementary feeding practices (Haroon et al 2013; Lassi et al 2013; Bhutta

et al 2013). Programmes introduced under these strategies unsurprisingly focus on promoting individual behavioural change but ignore other determinants of stunting such as household poverty that may affect the food security of families and even women's ability to breastfeed (Subramanian et al 2016). Similarly in Malaysia, the strategies outlined in the National Plan of Action for Nutrition of Malaysia 2016-2025 take identical approaches in trying to shift behavioural changes at individual levels, while omitting the more structural and systemic social determinants – unsurprisingly the national stunting rates reflect the folly and failure of this approach.

Tackling child stunting in Malaysia therefore requires a paradigm shift in the government's playbook. By refining their understanding of stunting and adopting a more holistic lens, Malaysia can make meaningful strides towards reducing stunting rates and ensuring the well-being and development of its future generations. For our children to stand tall, we must start with ending stunting.

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