Advancing towards double-duty policies: intergenerational and life-course transmission of the double burden of malnutrition

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Introduction

Malnutrition is a major global health challenge, with the Sustainable Development Goal 2.2 calling for an end to malnutrition in all its forms by 2030. Malnutrition may include overnutrition, such as overweight, obesity, and non-communicable diseases, and undernutrition, such as energy and micronutrient deficiencies. These two types of malnutrition are often considered as two distinct issues. However, as food systems in low- and middle-income countries (LMICs) transition towards more globalised diets high in ultra-processed foods, overnutrition and undernutrition can simultaneously co-exist within populations, households, and individuals, giving rise to the emergent issue of double burden of malnutrition (Popkin, Corvalan and Grummer-Strawn, 2020).

The double burden of malnutrition is particularly evident in countries that have seen a rapid increase in overweight and obesity, while still facing an enduring undernutrition legacy. In Peru, obesity among women has almost doubled over the last decades (Santos, Turner and Chaparro, 2021). Undernutrition in the form of stunting, which refers to delayed growth in children resulting in low height for their age, has reduced significantly but remains in high and unequal levels, reaching a prevalence of 30% among the poorest Peruvian households (Huicho *et al.*, 2020). In order to tackle this double burden of malnutrition, there is a need for double-duty policies, i.e. policies that consider that overnutrition and undernutrition co-exist, often driven by common determinants.

The World Health Organization (WHO) recognises that double-duty actions can be implemented at three levels. First, by ensuring existing policies that target one form of malnutrition have no unintended side-effects on other forms of malnutrition; second, by retrofitting existing policies that target one form of malnutrition to also address other forms of malnutrition; and third, by designing de-novo double duty policies that simultaneously target multiple forms of malnutrition (WHO, 2017). Linear cause-and-effect approaches to policy design, implementation, and evaluation are unable to rigorously consider these unintended side-effects, which may be the result of complex interconnections between different forms of malnutrition that change over time and create causal feedback mechanisms (Galea, Riddle and Kaplan, 2010; Rutter *et al.*, 2017).

System dynamics provides a useful tool to design and evaluate double-duty actions, by identifying and visualising common causal drivers and feedback loops between overnutrition and undernutrition. The influential Lancet report on the Global Syndemic of Obesity, Undernutrition, and Climate Change calls for system dynamics approaches as the appropriate solution-oriented approach to address the double burden of malnutrition (Swinburn *et al.*, 2019). There are several system dynamics models that simulate overweight and obesity dynamics, but no previous work has concurrently investigated population dynamics of both overnutrition and undernutrition (Morshed *et al.*, 2019). This would allow to inform double-duty policies that avoid unintended consequences and effectively address multiple forms of malnutrition at the same time.

To inform the design and impact of double-duty policies on the double burden of malnutrition, we have to first understand the common drivers of overnutrition and undernutrition and how they interact over time. According to the WHO, these common drivers include biology, particularly related to poor maternal and early child nutrition interactions, as well as environmental, such as food systems, and socioeconomic drivers (WHO, 2017). Thus, we developed a research project that aims to build a system dynamics model that simulates interconnections between overweight and stunting, through their shared drivers in Peru. First, interactions through biological mechanisms of maternal and early life nutritional status will be informed by relevant epidemiological evidence on the intergenerational and over the life-course transmission of overweight and stunting. Second, interactions with local food system drivers will be informed by local stakeholders, through group model building exercises. Finally, socioeconomic drivers will be captured by investigating diversity of dynamics across two diverse regions in Peru.

This paper presents the first step of the research project. Its specific objective is to provide a preliminary conceptual simulation model of the intergenerational and life-course transmission of overweight and stunting through maternal and neonatal characteristics among the Peruvian population. It seeks to establish the basis for this structure, its potential uses, limitations, and areas for further elaboration, in order to expand the model boundary by incorporating food system drivers in future work, using group model building with policymaker and community stakeholders in Peru.

Methods

This model was developed based on standard formulations of population dynamics as described by Sterman (J. Sterman, 2000) and relevant epidemiological evidence on intergenerational and over the life-course dynamics of overweight and stunting, informed by rapid literature reviews and relevant observational data on the nutritional status of children and adults in Peru. The conceptual structure of the model (Figure 1) was refined through an online consultation session with two double burden of malnutrition experts, one with global and one with Peru-specific expertise. Here, we present the simulative interpretation of two of

the four feedback loops presented in this conceptual structure. The model was developed in Vensim PLE PLUS.

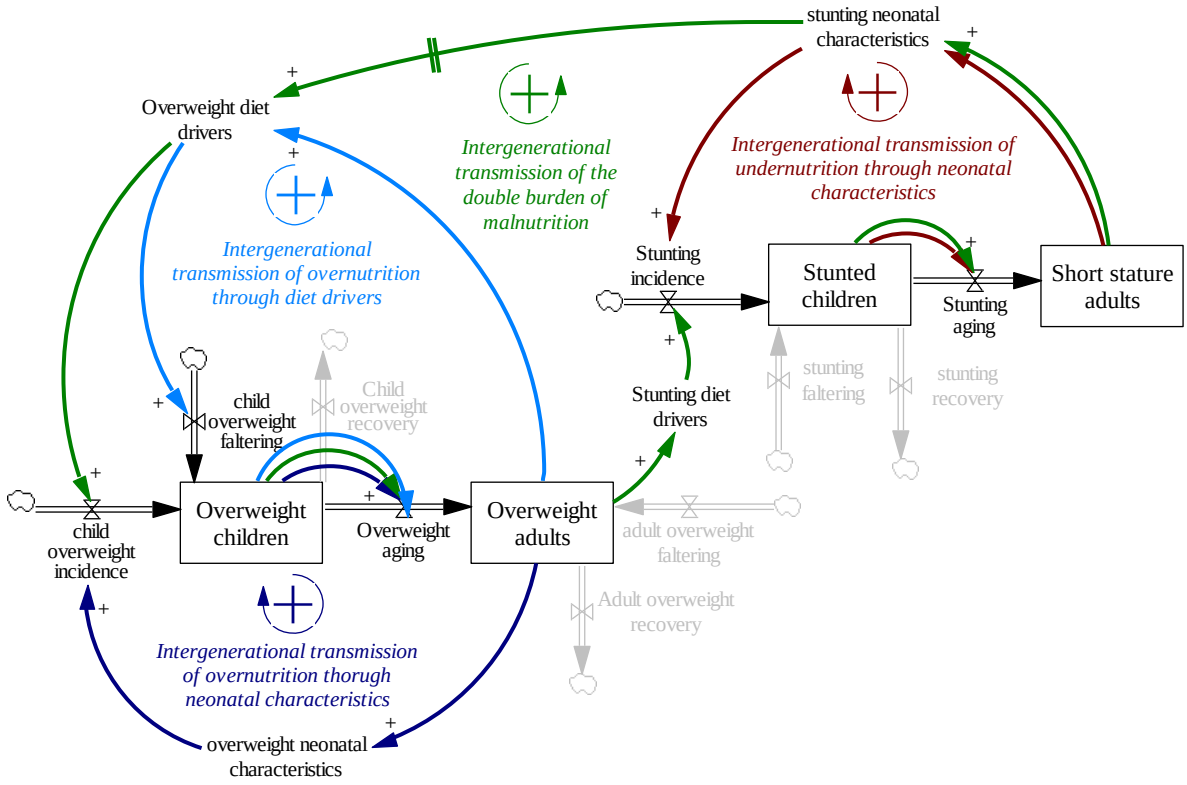

Figure 1. Conceptual representation of the intergenerational feedback loops of overweight and stunting

Population aging chain

We used aging chains and a co-flow structure to simulate a network of three stock and flow structures that represent: (1) the overall Peruvian population; (2) the stunted/short stature Peruvian population, and (3) the overweight Peruvian population, over a 100-year horizon. The three aging chains comprised four stocks each, representing the following age groups: newborns (age 0-12 months), children 1-5 years, children 5-15 years, and adults of reproductive age (15-49 years). The level of aggregation of the aging chains was decided in order to best represent relevant stunting and overweight dynamics. The first year of life is particularly important in the development of stunting and overweight later in life. For example, stunting in children accumulates until the age of two, although the most rapid growth faltering rate occurs during the first 12 months (Christian *et al.*, 2013; Victora *et al.*, 2021). Rapid weight gain in the first year of life has shown the strongest association with overweight later in life (Zheng *et al.*, 2018). The first five years of life are also of particular importance. For example, growth faltering in children continues up to the age of 5 (Victora *et al.*, 2021). Moreover, WHO's definition of child overweight is age group-specific, and is defined separately for children above and below the age of 5. Finally, we used a stock for

adults of reproductive age, defined by international organisations as the age between 15 and 49, in order to simulate intergenerational effects from parents to their children.

The population aging chain was informed by Sterman (J. D. Sterman, 2000b). The births inflow was defined as a function of the adult stock, the female percentage in the population, and the annual fertility rate. Aging flows were defined using the *DELAY FIXED* function in Vensim PLE PLUS, which allows all individuals to remain in the stock for a fixed amount of time, as the aging process is the same for all individuals and therefore all must remain in the stock for the same amount of time (J. D. Sterman, 2000a). Death outflows in each stock of the population aging chain simulated the number of people that die every year in that age group. The behaviour of this co-flow was validated using four tests: integration error, behaviour reproduction, extreme conditions, and sensitivity analysis (J. D. Sterman, 2000d).

Stunting and overweight coflows

Stunting and overweight aging chains were informed by the population aging chain using a co-flow structure, as described by Sterman (J. D. Sterman, 2000c). Co-flow structures allow for system attributes, in this case stunting and overweight, to travel through the stock and flow structure of the system, in this case overall population. We modelled aging of stunting and overweight from age group j (0-1, 1-5, 5-15, 15-49) to age group j+1 as the product of aging in the overall population and the average stunting or overweight prevalence in age group j (Figure 2). Average prevalence was estimated as the ratio of stunted or overweight people in age group j to the overall population in age group j. Faltering inflows, i.e. inflows of non-overweight/non-stunting people that enter the overweight/stunting stocks through mechanisms other than aging, were also modelled through the co-flow structure. They were defined as the product of aging in the overall population, the faltering rate for that age group, and the complement of average prevalence of stunting or overweight, representing the non-stunting and non-overweight population respectively, divided by the number of years in the stock.

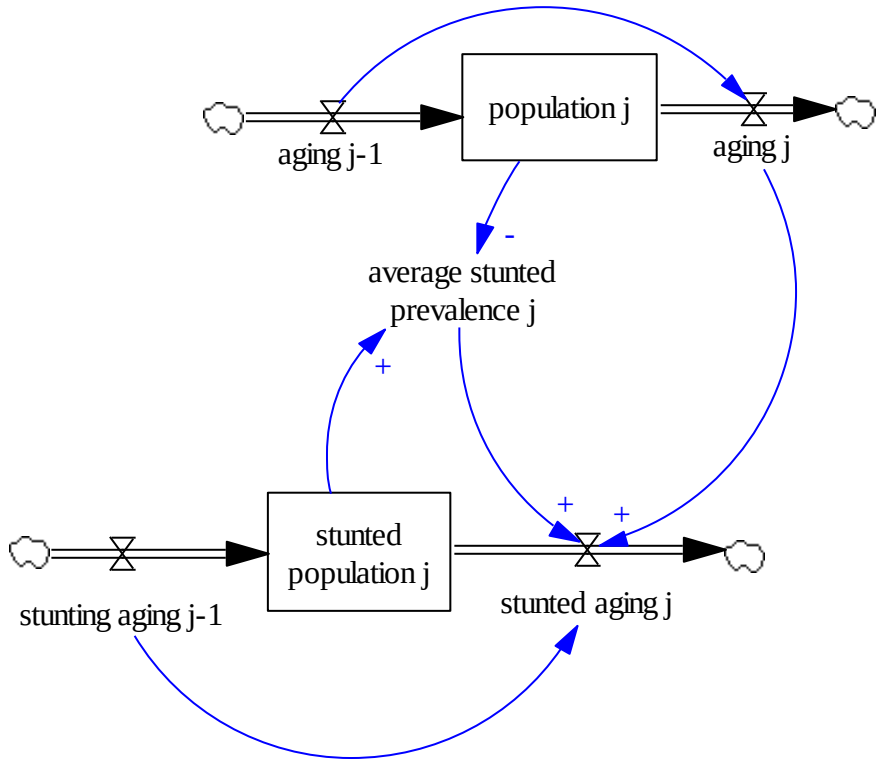

Figure 2. Example of co-flow structure for stunted population in age group j

We performed rapid umbrella reviews to inform modelling of the intergenerational transmission of stunting and overweight, i.e. from mothers to their children, through neonatal characteristics. Based on previous assessments of quality of published evidence on neonatal risk factors of stunting, we identified convincing evidence to support that maternal short stature is associated with child stunting through gestational age (preterm births), small size for gestational age (SGA) and their combinations (Danaei *et al.*, 2016). Moreover, there is consistent evidence to indicate that high birth weight or large size for gestational age (LGA), is associated with both maternal and childhood overweight (Woo Baidal *et al.*, 2016; Vats *et al.*, 2021). To quantitatively simulate these associations, we used relative risks (RR) from relevant meta-analyses of longitudinal studies (Schellong *et al.*, 2012; Christian *et al.*, 2013; Kozuki *et al.*, 2015; Vats *et al.*, 2021) (see Appendix 1.2). Relative risks are epidemiological measures that compare the risk of a health outcome (e.g. low birth weight) between one population group (e.g. overweight mothers) and another (e.g. non-overweight mothers), without explicitly revealing the causal mechanisms that may drive potential differences in risk. Although the aging chains model the intergenerational transmission of maternal short stature and overweight to infants aged 0-12 months, we used RR for the association between neonatal characteristics and stunting or overweight later in life (12-60 months and 1-72 years respectively), as age-specific RRs were not available.

Faltering and recovery of stunting and overweight

Although some children are born stunted or overweight and continue to be stunted or overweight throughout their life, recovery from or faltering to these conditions can also occur later in life. We modelled stunting and overweight recovery and faltering for children aged 1-5 and 5-15 using estimated transition probabilities between stunting/non-stunting and overweight/non-overweight status. Transition probabilities were estimated using data from the Young Lives study, a longitudinal cohort study of children in Peru, who were followed up for 15 years between the ages of 1 and 15. We estimated transition probabilities between stunted and non-stunted or overweight and non-overweight status for children between the ages of 1 and 5, and 5 and 15. We used the xttrans command in Stata and data from rounds one (2002), two (2006-7) and five (2016-17) of the cohort. These transition probabilities capture both aging effects (effects due to aging from younger age groups to older age groups) and period and cohort effects (effects due to overall changes in stunting and overweight prevalence in Peru between 2002 and 2016), as it was not possible to distinguish between the two.

We assumed that no recovery and faltering of stunting occurred for newborns aged 0-1 years and after the age of 15. Indeed, stunting recovery is very minimal in the first months of life (Victora *et al.*, 2021), whereas height is unlikely to change in adulthood. However, adults are likely to transition in and out of overweight throughout their life. Thus, we modelled recovery from and faltering to overweight for the stock of adults 15-49. As there are no longitudinal data for Peruvian adults available to estimate transition probabilities of overweight in adulthood, we used data from repeated cross-sectional surveys and the heuristic developed by Meisel et al. (Meisel *et al.*, 2018) to estimate transition rates between overweight and not overweight groups of adults.

We used data from ENDES (Encuesta Demográfica y de Salud Familiar) to estimate prevalence of overweight and non-overweight in 5-year age groups for adults aged 15-49, in 2015 and 2020. ENDES is a nationally representative annual survey of children 0-5 and adults over 15 years of age in Peru, which collects anthropometric data, including weight and height. As the survey is nationally representative, we assumed that individuals that were, for example, 15 years old in 2015 were the same individuals that were 20 years old in 2020, etc.

We expressed prevalence of non-overweight and overweight for people aged i in 2020, as a function of the prevalence of non-overweight and overweight for people aged i-1 in 2015, as follows:

$$\Pr[non\_ow]_{20,i} = \Pr[non\_ow]_{15,i-1} * a_1 - \Pr[non\_ow]_{15.i-1} * t_1 + \Pr[ow]_{15.i-1} * t_2 \quad (1)$$

$$\Pr[ow]_{20,i} = \Pr[ow]_{15,i-1} * a_2 - \Pr[ow]_{15.i-1} * t_2 + \Pr[ow]_{15.i-1} * t_1 \quad (2)$$

where $\Pr[non\_ow]_{20,i}$ and $\Pr[non\_ow]_{15,i}$ is a vector with prevalence of non-overweight for each single year of age within age group i in 2020 and 2015 respectively; $\Pr[ow]_{20}$ and $\Pr[ow]_{15}$ is a vector with prevalence of overweight for each single year of age within age group i in 2020 and 2015 respectively, i=1-7 is the 5-year age group (15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49); t_1 is the transition rate from non-overweight to overweight, t_2 is

the transition rate from overweight to non-overweight; and a_1 and a_2 are the retention rates for non-overweight and overweight respectively, i.e. the proportion of non-overweight and overweight individuals that remained within the same weight category between 2015 and 2020.

We then estimated the retention and transition rates for each age group i, so that the difference between the estimated prevalence rates from equations (1) and (2) and the prevalence rates observed by ENDES is minimised. In particular, we used the 'nloptr' package in R, to minimise the below equation:

$$(\Pr[non\_ow]_{20,i} - \Pr^*[non\_ow]_{20,i})^2 + (\Pr[ow]_{20,i} - \Pr^*[ow]_{20,i})^2 \quad (3)$$

where Pr are the estimated prevalence rates from equations (1) and (2) and Pr* are the observed prevalence rates from ENDES in 2020.

We used the Augmented Lagrangian algorithm, which is a local and derivative-free optimisation approach, as recommended by Meisel et al, and allows for equality constraints in the optimisation process. We employ the following equality constraints: $a_1 + t_1 = 1$ and $a_2 + t_2 = 1$, which assume that the retention and transmission rates for each weight group equal to 100%. We also set all retention and transmission rates to be between 0 and 1, and we initiated the optimisation process at 1 for retention rates and 0 for transmission rates. Validity of transition rates was tested by estimating the quadratic differences between observed prevalence rates from ENDES 2020 and the estimated 2020 prevalence rates using the heuristic, for each age group i. We used the mean of transition rates across all age groups to model recovery and faltering rates of overweight in adults aged 15-49.

Parameter estimation

We used age group specific data from Peru to inform baseline parameters of the model. Baseline characteristics on age-specific population, age-specific mortality rates, fertility rate, and female population percentage were informed by national data from Peru's National Institute of Statistics and Informatics (INEI: Instituto Nacional de Estadística e Informática), the World Bank, and the United Nations. Baseline prevalence of overweight and stunting for children 0-5 years and adults of reproductive age was informed by ENDES 2019. ENDES does not collect data for children over the age of 5, thus information on stunting and overweight prevalence for children aged 5-15 was obtained from published literature (Tarqui-Mamani, Alvarez-Dongo and Espinoza-Oriundo, 2018; Santos-Antonio *et al.*, 2020). Finally, baseline neonatal characteristics were obtained from published data from Peru's national birth registries (Carrillo-Larco *et al.*, 2021). More details on data inputs and their sources are presented in Appendix 1.

Results

Model structure and equations

Equations of stocks and flows in each co-flow structure are presented in Table 1. The model was characterised by three reinforcing feedback loops. In the population aging chain, a reinforcing feedback loop occurred between adults of reproductive age and births (Figure 3). In the stunting/short stature co-flow a reinforcing feedback loop simulates the intergenerational transmission of undernutrition in the Peruvian population, i.e. the transmission of stunting and short stature between mothers and their children through small gestational size and preterm births (Figure 4). Finally, a reinforcing feedback loop occurs in the overweight co-flow, depicting the intergenerational transmission of overnutrition in the Peruvian population, i.e. the transmission of overweight between mothers and their children through large gestational size (Figure 5).

Table 1. Model equations

	Equation
Population co-flow	
Stocks	
Newborns, i=0-1	$Newborns = \int_{t_0}^{t} \big(Births(s) - Aging_1(s) - Death\ rate_{0-1}(s)\big)ds + Population_{0-1,t=0}$
Population for age group i*	$Population_i = \int_{t_0}^{t} \big(Aging_{i-1}(s) - Aging_i(s) - Death\ rate_i(s)\big)ds + Population_{i,t=0}$
Flows	
Death rate for age group i	$Death\ rate_i = \frac{(Population_i * Mortality\ rate_i)}{Years_i}$
Aging flow for age group i	$Aging_i = DELAY\ FIXED(\ Aging_{i-1} * (1 - Mortality\ rate_i)\ , Years_i\ , Initial_i)$ Where $Initial_i$ is the amount of people that leave the stock before $Years_i$ and represents the aging process of those who were initially in the stock (at baseline until $Years_i$). We modelled this as a first order delay, where the initial population in the stock ages at equal intervals each year for the length of years in the stock: $Initial_i = \frac{Initial\ population\ at\ stage_{i,\ t=0} * (1 - Mortality\ rate_i)}{Years_i}$
Birth rate	$Birth\ rate = \frac{Adults\ 15-49 * Female\ percentage * Fertility\ rate}{Years_{15-49}}$

Stunting co-flow	
Stocks	
Stunted population for newborns 0-1 years	$$Stunted\ newborns = \int_{t_0}^{t} \big(stunting\ incidence(s) - stunted\ aging_{0-1}(s)\big)ds + (Population_{0-1,t=0} * stunting\ prevalence_{0-1,t=0})$$
Population for age group i**	$$stunted\ population_i = \int_{t_0}^{t} \big(stunted\ aging_{i-1}(s) - stunted\ aging_i(s) - stunting\ recovery_i(s) + stunting\ faltering_i(s)\big)ds + (Population_{i,t=0} * stunting\ prevalence_{i,t=0})$$
Population for short stature adults 15-49	$$short\ stature\ adults = \int_{t_0}^{t} \big(stunting\ aging_{5-15}(s) - short\ stature\ aging_{49+}(s)\big)ds + (Population_{15-49,t=0} * short\ stature\ prevalence_{15-49,t=0})$$
Flows	
Stunting incidence	$$stunting\ incidence = stunting\ prevalence_{0-1,t=0} * Birth\ rate * ((1 - AGA\ and\ preterm\ rate - SGA\ and\ term\ rate - SGA\ and\ preterm\ rate) + (AGA\ and\ preterm\ rate * AGA\ and\ preterm\ stunting\ RR) + (SGA\ and\ term\ rate * SGA\ and\ term\ stunting\ RR) + (SGA\ and\ preerm\ rate * SGA\ and\ preterm\ stunting\ RR))$$
Stunted/short stature aging	$$stunting\ aging_i = Aging_{i-1} * average\ stunting\ prevalence_i$$
Stunting recovery for age group i**	$$stunting\ recovery_i(s) = \frac{(stunted\ population_i * stunting\ recovery\ rate_i)}{Years_i}$$
Stunting faltering for age group i**	$$stunting\ faltering_i(s) = \frac{(Population_i * (1 - average\ stunting\ prevalence_i) * stunting\ faltering\ r}{Years_i}$$
Endogenous variables	
Average stunting/short stature prevalence	$$average\ stunting\ prevalence_i = \frac{stunted\ population_i}{Population_i}$$

Average female short stature prevalence	$$average\ female\ short\ stature\ prevalence_{15-49} = average\ short\ stature\ prevalence_{15-49} * ratio\ of\ female\ to\ male\ short\ stature\ prevalence$$
Neonatal characteristics (NC) rate	$$NCrate = NC\ prevalence_{t=0} * (1 - average\ short\ stature\ prevalence_{15-49}) + NC\ prevalence_{t=0} * average\ short\ stature\ prevalence_{15-49} * RR_{short\ stature,NC}$$
Overweight co-flow	
Stocks	
Overweight population for newborns 0-1 years	$$Overweight\ newborns = \int_{t_0}^{t} \big(overweight\ incidence(s) - overweight\ aging_{0-1}(s)\big)ds + (Population_{0-1,t=0} * overweight\ prevalence_{0-1,t=0})$$
Population for age group i*	$$overweight\ population_i = \int_{t_0}^{t} \big(overweight\ aging_{i-1}(s) - overweight\ aging_i(s) - overweight\ recovery_i(s) + overweight\ faltering_i(s)\big)ds + (Population_{i,t=0} * overweight\ prevalence_{i,t=0})$$
Flows	
Overweight incidence	$$overweight\ incidence = overweight\ prevalence_{0-1,t=0} * Birth\ rate * ((1 - LGA\ and\ preterm\ rate) + (LGA\ rate * LGA\ overweight\ RR))$$
Overweight aging	$$overweight\ aging_i = Aging_{i-1} * average\ overweight\ prevalence_i$$
Overweight recovery for age group i*	$$overweight\ recovery_i(s) = \frac{(overweight\ population_i * overweight\ recovery\ rate_i)}{Years_i}$$
Overweight faltering for age group i*	$$overweight\ faltering_i(s) = \frac{(population_i * (1 - average\ overweight\ prevalence_i) * overweight\ falterin}{Years_i}$$
Endogenous variables	
Average overweight prevalence	$$average\ overweight\ prevalence_i = \frac{overweight\ population_i}{Population_i}$$

Average female overweight prevalence	$$average\ female\ overweight\ prevalence_{15-49} = average\ overweight\ prevalence_{15-49} * ratio\ of\ female\ to\ male\ overweight\ prevalence$$
LGA rate	$$LGA\ rate = LGA\ prevalence_{t=0} * (1 - average\ female\ overweight\ prevalence_{15-49}) + LGA\ prevalence_{t=0} * average\ female\ overweight\ prevalence_{15-49} * RR_{overweight,LGA}$$

**excludes age group 0-1 years*
***excludes age group 0-1 years and 15-49 years*
i is age-groups: 0-1 years, 1-5 years, 5-15 years, 15-49 years
$Years_i$ is the number of years in each population stock: 1 year, 4 years, 10 years, 35 years respectively
SGA: small for gestational age
AGA: average for gestational age
LGA: large for gestational age
RR: relative risk
NC: Neonatal characteristics. They include: small for gestational age and preterm; small for gestational age and term; average for gestational age and preterm

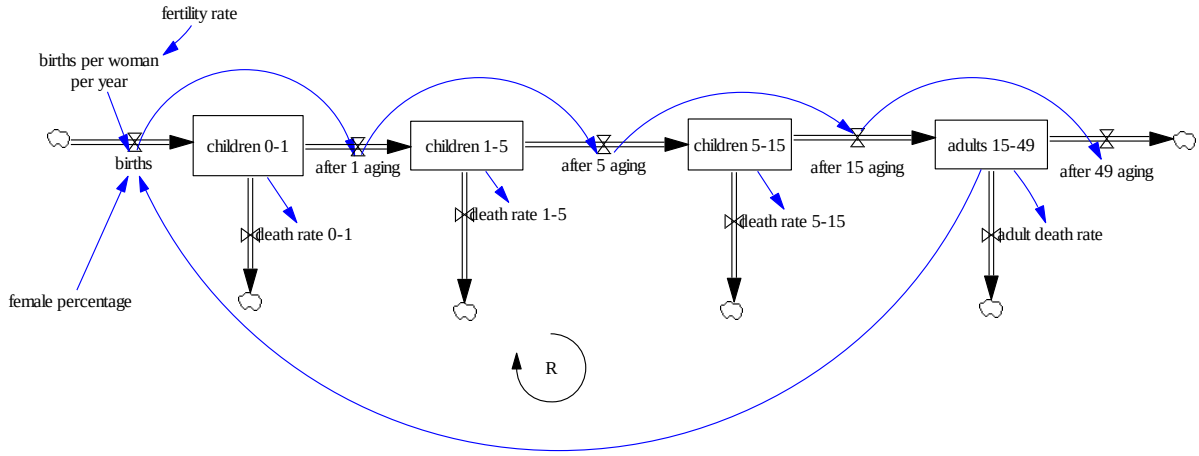

Figure 3. Structure of population co-flow

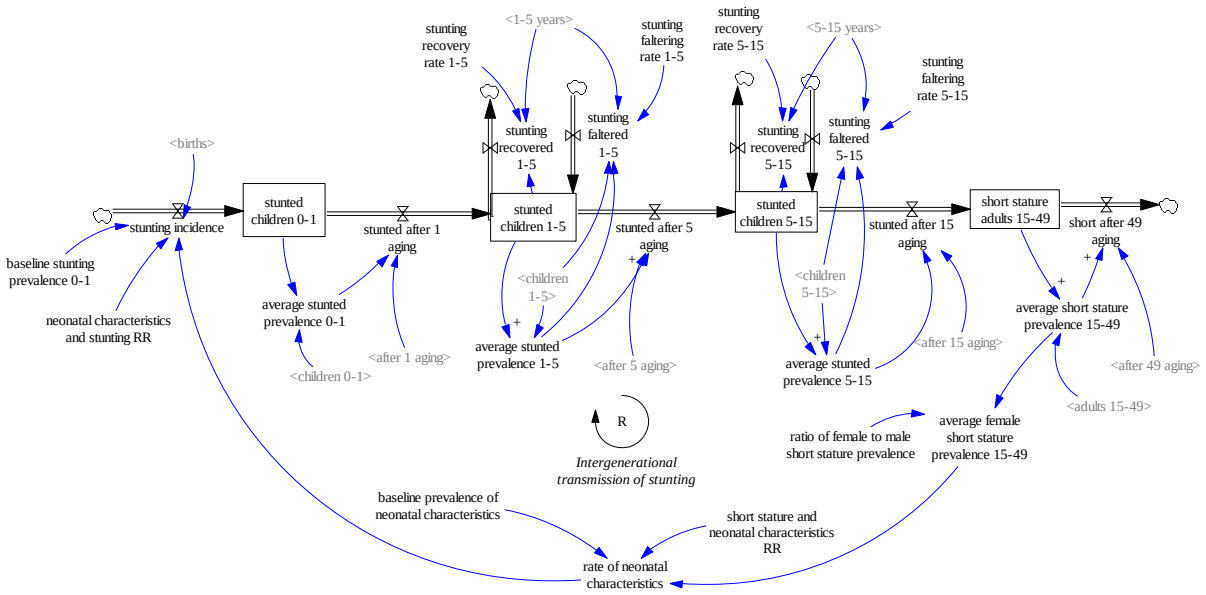

Figure 4. Structure of stunting co-flow

Neonatal characteristics include three distinct subgroups: small for gestational age and preterm; small for gestational age and term; average for gestational age and preterm; RR: relative risk

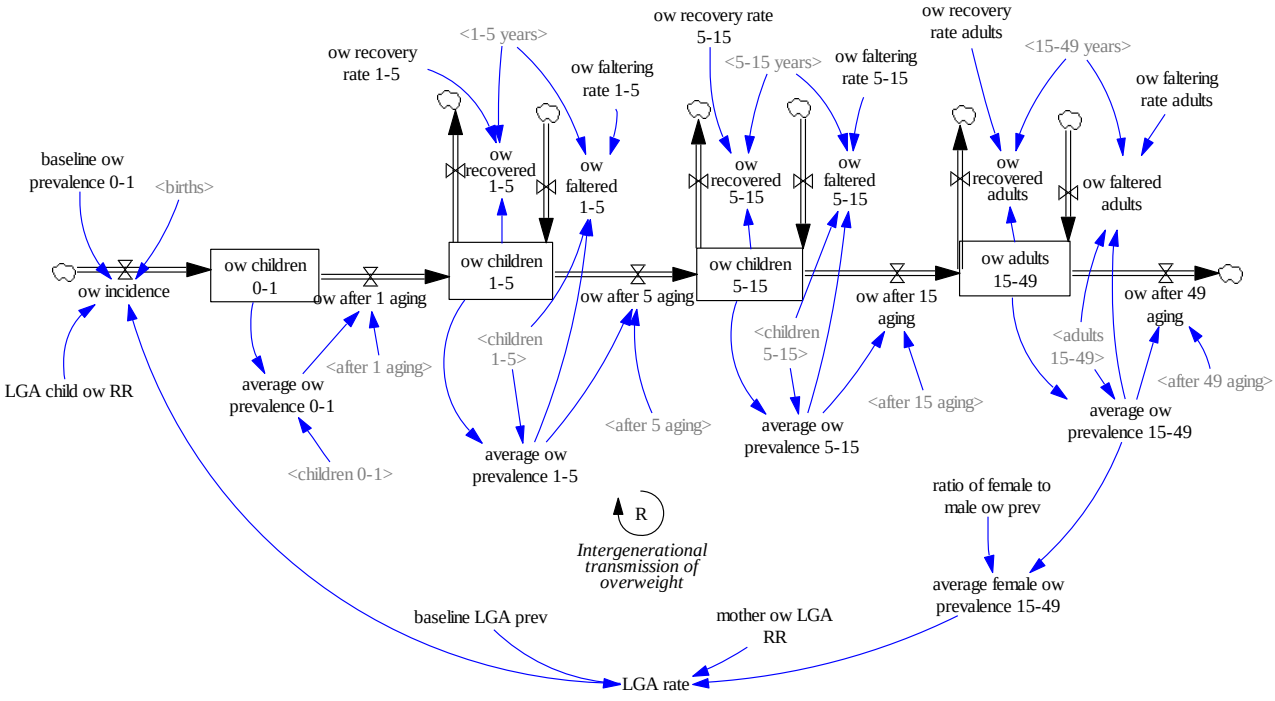

Figure 4. Structure of overweight co-flow

ow: overweight; LGA: large for gestational age; RR: relative risk

Validation

Validations tests showed that model results were not sensitive to changes in the integration method and the DT value; Euler´s integration method with a DT of 0.03125 was used. Simulation results of population stocks were consistent with behavioural patterns of historical data in Peru between 1950 and 2019 for all age groups. Model sensitivity related to fertility and death rates produce numerical sensitivity. For some variations of fertility rate and female percentage, simulations showed changes in behaviour patterns. Assessment of extreme conditions for exogenous variables, i.e. female population percentage, fertility rate, and age group-specific mortality rates for children, produced results consistent with expectations. However, simulation results using extreme conditions for adult mortality rate were not fully consistent with expectations. This was due to software limitations, as Vensim PLE PLUS does not allow the formulation of leakage functions for stocks with fixed delays. Thus, we used an alternative formulation that allows the stock to have outflows of both the aging process and of annual deaths that guarantees no-negativity of the stock. Moreover, the adult stock has a higher level of aggregation than the other stocks in the aging chain, conditioning the death outflow to a delay of 34 years. Although this allows the model to simulate acceptable population dynamics under 'normal' conditions, it might not provide expected results in extreme conditions. For example, having a death rate of 100% will not produce an immediate decrease of the adult population. Detailed descriptions of validation tests and conclusions are presented in Appendix 2. Validity of estimated transition rates was also confirmed, as quadratic differences between observed and estimated prevalence of overweight and non-overweight were below 1% for most 5-year age groups between 15 and 49 years of age (Appendix 3).

Discussion

This system dynamics model uses a co-flow structure to simulate the life-course and intergenerational transmission of overweight and stunting in the Peruvian population. It includes reinforcing feedback loops that model the intergenerational transmission of undernutrition, between maternal short stature and child stunting, and the intergenerational transmission of overnutrition, between maternal and child overweight, through neonatal characteristics. This model provides the foundation for future work to understand and simulate the over the life-course and intergenerational transmissions of the double burden of malnutrition, occurring through the interaction between overnutrition and undernutrition co-flows and dietary drivers.

Contributions to the literature

This model provides important contributions, both to system dynamics applications and to the understanding of the double burden of malnutrition. Previous system dynamics models that investigated population dynamics of overweight and obesity, did not take into account the intergenerational transmission between parents and their children (Chen *et al.*, 2018; Meisel

et al., 2018). The intergenerational transmission of overweight has been studied before only through the social transmission of weight loss behaviours (Frerichs, Araz and Huang, 2013), or through a single case study that does not take into account population dynamics (Sabounchi *et al.*, 2014). Our model quantified the intergenerational transmission of overweight and stunting through neonatal characteristics, using estimated associations derived from robust epidemiological evidence. Furthermore, this model provides a vehicle to further understand transmission of overweight and stunting through diverse dietary drivers. Contrary to previous work that limited overweight as a condition of excess energy intake (Fallah-Fini *et al.*, 2014), our model can use relative risks to inform changes in overweight and stunting through established dietary risk factors. This is important for the understanding of the double burden of malnutrition, as shared dietary drivers of overnutrition and undernutrition go beyond energy and nutrient intake and may include dietary risk factors such as dietary diversity and ultra-processed food consumption. Finally, there are no previous system dynamics models that concurrently investigate overnutrition and undernutrition dynamics. By incorporating these two types of malnutrition within the same system boundary, our model provides the basis for a novel understanding of the double burden of malnutrition, which is not possible using common epidemiological approaches, by recommending a model structure that facilitates the identification and study of feedback loops occurring over the life-course and across generations.

Limitations

This paper has some limitations. We only provided simulation results for the population co-flow. This is because this paper aims to describe a preliminary structure that will be used as a basis for future model development and does not reflect the overall system, conceptually represented in Figure 1. Thus, simulation results may lead to inaccurate conclusions. The model used the *DELAY FIXED* function in Vensim PLE PLUS to simulate population aging. This function is appropriate given that all individuals stay in each stock for the same amount of time. However, Vensim PLE PLUS does not allow the formulation of leakages, hence the aging outflow can only take into account inflows without considering other outflows in the same stock, such as deaths. The alternative formulation of death outflows used to address this software limitation may not respond accurately to extreme conditions, as indicated from our validation results, although it was proved appropriate under usual conditions, as it performed consistently with historical data.

We made several assumptions due to lack of available data. For example, RRs used to model associations between neonatal characteristics and stunting or overweight in infants 0-12 months represent associations with these conditions later in life (12-60 months for stunting and 1-72 years for overweight). Although evidence suggests that some of these associations may differ across age groups (Heslehurst *et al.*, 2019), no age-specific RRs from robust meta-analyses were available in the literature. Estimated transition probabilities between overnutrition and undernutrition groups do not only represent transition due to aging effects, i.e. effects due to people growing older, but also period and cohort effects, i.e. effects due to changes occurring at a particular period or affecting people born at a specific point in time.

For example, to estimate transition probabilities for children, we used data from the Young Lives cohort, which followed children born in 2001 until they were 15 years old in 2016. During this time, Peru showed a significant decrease in child stunting due to concerted efforts to improve undernutrition through welfare and social policies (Huicho *et al.*, 2017). These impacts might be represented in the transition probabilities used in this model and they cannot be separated from impacts solely attributed to aging. Finally, this model does not incorporate differences in mortality rates between the overall and stunted/overweight populations. It also does not consider the potential combined effect of maternal short stature and overweight on child malnutrition (Wells *et al.*, 2020). These dynamics will be incorporated in future versions of this model.

Opportunities for model elaboration

Our model captures the intergenerational transmission of stunting/short stature and overweight only through neonatal characteristics. However, these conditions are likely to be transmitted over the life-course and across generations through other mechanisms, including dietary behaviours, food environments, social norms, and socioeconomic characteristics. For example, overweight parents may have dietary habits that impact their children's diets by creating a more obesogenic household food environment. These children are more likely to become overweight and grow up to become overweight adults, creating a new intergenerational transmission loop through dietary risk factors (Figure 1). Some of these drivers might also intersect both overnutrition and undernutrition, creating a reinforcing feedback loop of the intergenerational transmission of the double burden of malnutrition, where undernutrition drives overnutrition and overnutrition drives undernutrition across generations (Figure 1). For example, evidence suggests that early-life undernutrition followed by rapid weight gain later in childhood can increase the risk of overweight in adulthood, while maternal overweight can lead to delayed growth in offspring, depending on setting and exposure to other malnutrition determinants such as infant feeding practices and food environments (Wells *et al.*, 2020).

Our future work involves group model building workshops in Peru to inform dynamics between overnutrition, undernutrition, and food system drivers, aiming to unravel some of these interconnections between overweight and stunting. The workshops will be region-specific, given the diversity of the Peruvian context. For example, previous evidence showed that stunting and adiposity were positively associated among children living at low altitude and negatively associated among children living in high altitude areas in Peru (Pomeroy *et al.*, 2014). Outputs from stakeholder workshops will be used to extend the described model to investigate the double burden of malnutrition in Peru and its interconnections with the local food system.

Conclusion

In our knowledge, this is the first system dynamics model that represents the intergenerational transmission of overnutrition and undernutrition within populations, individuals, and across generations, paving the way for understanding the complex and

dynamic phenomenon of the double burden of malnutrition. We identified that across different age groups, the prevalence of overweight and stunting may be impacted by the number of individuals aging with the condition, as well as the number of individuals that recover and falter during different periods of life. We also showed how maternal characteristics may further contribute to overweight and stunting across generations. Though this model represents a conceptual foundation for further elaboration through group model building, it showcases processes through which policies that only target overnutrition or undernutrition in specific age groups may fail to achieve population health impact. This insight highlights the importance of double-duty interventions that target overnutrition and undernutrition across generations. We hypothesise that although these interventions might not show an immediate improvement to the problem, they may be more effective in the long-term. Through our future work, we will consider the mechanisms through which overnutrition and undernutrition interact over time in order to identify double-duty policies that may effectively address the double burden of malnutrition.

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