

1 **Distinct clusters of stunted children in India: An observational study**

2

3 Green MA^{1*} PhD, Corsi DJ² PhD, Mejía-Guevara I³ PhD, Subramanian SV⁴ PhD.

4

5 ¹ Department of Geography & Planning, University of Liverpool, Liverpool, UK.

6 ² Ottawa Hospital Research Institute, Ottawa, Canada.

7 ³ Centre for Population Health Sciences, Stanford University, USA

8 ⁴ School of Public Health, Harvard University, Boston, USA.

9

10 * Corresponding autor. Address: Department of Geography & Planning, Roxby Building,

11 University of Liverpool, Liverpool, L69 7ZT, UK. Email: mark.green@liverpool.ac.uk. Tel:

12 +44 151 794 2854.

13

14 **Short title:** Distinct clusters of stunted children in India

15

16 **Source of funding:** None

17

18 **Conflict of interest statement:** None declared.

19

20 **Acknowledgements:** None.

21

22 **ABSTRACT**

23 Background: Childhood stunting is often conceptualised as a singular concept (i.e. stunted or
24 not) and such an approach implies similarity in the experiences of children who are stunted.
25 Furthermore, risk factors for stunting are often treated in isolation and limited research has
26 examined how multiple risk factors interact together. Our aim was to examine whether there
27 are subgroups among stunted children, and if parental characteristics influence the likelihood
28 of these subgroups among children.

29 Methods: Children who were stunted were identified from the 2005-06 Indian National
30 Family Health Survey (n=12 417). Latent class analysis was used to explore the existence of
31 subgroups among stunted children by their social, demographic and health characteristics.
32 We examined whether parental characteristics predicted the likelihood of a child belonging to
33 each latent class using a multinomial logit regression model.

34 Results: We found there to be five distinct groups of stunted children; ‘poor, older and poor
35 health-related outcomes’, ‘poor, young and poorest health-related outcomes’, ‘poor with
36 mixed health-related outcomes’, ‘wealthy and good health-related outcomes’, and ‘typical
37 traits’. Both mother and father’s educational attainment, body mass index and height were
38 important predictors of class membership.

39 Conclusions: Our findings demonstrate evidence that there is heterogeneity of the risk factors
40 and behaviours among children who are stunted. It suggests that stunting is not a singular
41 concept; rather there are multiple experiences represented by our ‘types’ of stunting.
42 Adopting a multi-dimensional approach to conceptualising stunting may be important for
43 improving the design and targeting of interventions for managing stunting.

44

45 **KEYWORDS**

46 Stunting; India; Latent Class Analysis; Children; Undernutrition; Socioeconomic Factors.

47 **INTRODUCTION**

48 Stunting is the chronic retardation of child growth as a result of nutritional inadequacies and
49 defined by low height for age. The World Health Organisation (WHO) estimated that in 2016
50 there were 155 million children aged under 5 years (23.8%) globally who were stunted
51 (WHO, 2017). While India has seen large improvements towards tackling stunting with the
52 estimated prevalence declining from 51% of all Indian children under 5 in 2005-06 (Black et
53 al., 2008) to 33% in 2015-16 (Corsi, Meija, & Subramanian, 2016a), India still has the largest
54 global burden of childhood undernutrition. While there has been progress in tackling stunting
55 both internationally and in India recently, aided by investment towards achieving Millennium
56 Development Goal 1, India is unlikely to achieve the goal set by the UN (Ministry of Women
57 and Child Development, 2014; United Nations, 2015) or future goals such as the Sustainable
58 Development Goals (de Onis et al., 2013).

59

60 Stunting reflects the accumulation of micronutrient deficiencies typically as a result of
61 undernutrition. A lack of specific nutrients can lead to stunted children having reduced
62 immunological capacity to fight against (infectious) disease(s) (Black et al., 2008; Schaible &
63 Kaufmann, 2007). Stunting itself can lead to longer term implications for child and
64 subsequent adult health (UNICEF, 2013). It can also produce social implications as well. For
65 example, children who are stunted are more likely to have poorer cognitive development
66 which can restrict their educational achievement and future employment prospects
67 (Crookston et al., 2011; Grantham-McGregor et al., 2007; Martorell et al., 2010). The impact
68 of stunting can be intergenerational with stunted mothers more likely to have premature
69 children who then typically suffer from retarded growth (Grantham-McGregor et al., 2007;
70 Subramanian, Ackerson, & Davey Smith, 2010; UNICEF, 2013). The combination of the

71 high prevalence of stunting alongside these associated health and social implications, makes
72 stunting an important policy consideration in India.

73

74 Current epidemiological and public health studies tend to focus on single risk factors of
75 stunting rather than explore the complex interplay between multiple factors. While there is
76 growing evidence that childhood stunting is influenced by multi-factorial drivers (Corsi,
77 Meija, & Subramanian, 2016b; Danaei et al., 2016; Fenske, Burns, Hothorn, & Rehfuess,
78 2013), there have been no studies to our knowledge that have sought to explore the existence
79 of heterogeneity among stunted children. Previous reviews on the effectiveness of nutritional
80 interventions to prevent stunting have only reported limited success in reducing the
81 prevalence of stunting (Bhutta et al., 2008, 2013; Dewey & Adu-Afarwuah, 2008). One
82 possible explanation for this is that policy interventions are often delivered to all children
83 who are stunted together, who are effectively treated as a single homogenous entity. This
84 approach may not be an efficient distribution of resources if individual characteristics (and
85 how individuals may respond to an intervention) are not similar. Failure to incorporate multi-
86 dimensional explanations for stunting characteristics will also miss out on understanding the
87 wider determinants of stunting and hence limit our ability to design effective interventions.

88

89 Our study presents an alternative approach to exploring stunting in children. Using latent
90 class analysis to explore similarities in multivariable associations across observations, we
91 identify five ‘types’ of stunted children in a representative survey of India (2005-06). We also
92 analyse the role of parental characteristics on latent class membership. Previous research has
93 demonstrated the importance of parental characteristics such as education (Bhutta et al.,
94 2013), body mass index (BMI) (Subramanian et al., 2010), height (Subramanian, Ackerson,

95 Smith, & John, 2009) and whether the mother was married as a child (Raj et al., 2010) on the
96 risk of their children being stunted (also see Corsi et al., 2016b; Danaei et al., 2016).

97

98 **KEY MESSAGES**

- 99 • The determinants of stunting are numerous, complex and interacting, however current
100 research fails to consider the multidimensional nature of stunting rather treating it as a
101 singular concept.
- 102 • Our paper demonstrates the existence of five distinct types of stunted individuals in
103 terms of health, social and parental characteristics.
- 104 • Applying our multidimensional approach will help improve our understanding of the
105 condition, as well as how to design effective interventions.

106

107 **METHODS**

108 **Data**

109 Data were taken from the 2005-06 Indian National Family Health Survey (NFHS). The
110 NFHS is the largest and most recent representative survey currently available that includes
111 objective data on child anthropometry. We focused on children aged between 6 months and 5
112 years. We selected all singleton children whose mothers (the survey does not allow for proxy
113 reporting i.e. by other caregivers) had fully completed a 24 hour dietary assessment (n =
114 32,360). Observations were excluded if children had missing data or values of height and
115 weight were implausible (defined as ± 6 standard deviations WHO growth standards; WHO,
116 2006) resulting in a sample size of 28,984. Ethical approval was not required due to the study
117 being secondary data analysis.

118

119 We identified whether children were stunted based on WHO guidelines. Height and weight
120 measurements were converted into age- and sex-specific z-scores based on WHO child
121 growth standards (WHO, 2006). Stunting was defined as any z-score below -2 standard
122 deviations. All children who were stunted were included in the analysis resulting in a final
123 sample size of 12,417 (43%).

124

125 The selection of risk factors related to stunting, to be included as variables in our analysis,
126 was based on the approach taken in a previous study (Corsi et al., 2016b). Each variable
127 selected below was adapted from the UNICEF conceptual framework on the determinants of
128 child undernutrition (Bhutta et al., 2008). We included variables to measure both social and
129 health characteristics of children to account for different aspects of stunting. Most of the
130 variables we used have standard definitions and have been described in more detail elsewhere
131 (Barros et al., 2012; Corsi et al., 2016b). Variables included were:

- 132 • Sex (male or female)
- 133 • Household wealth (split into quintiles)
- 134 • Life-stage (categorised as: 6 to 11 months, 12 to 23 months, 24 to 35 months, 36 to 47
135 months, and 48 to 59 months)
- 136 • Diet diversity based on a scoring system designed by Ruel and Menon (Ruel &
137 Menon, 2002) (categorised by quintile)
- 138 • Child was breastfed within one hour of birth or not
- 139 • Child had an infectious disease in previous two weeks or not
- 140 • Water source was through a piped connection to the dwelling or not
- 141 • Stools were safely disposed in the house or not

- 142 • Sanitation facility was improved (i.e. the hygienic separation of human excreta from
143 contact with individuals using facilities such as a latrine flushing to a sewer or septic
144 tank) or not
- 145 • House air quality (defined as; use of non-solid fuels, solid fuels in a separate kitchen,
146 and solid fuels in a non-separate kitchen) Iodized salt used in household or not
- 147 • Child was fully vaccinated or not
- 148 • Vitamin A supplements taken or not

149

150 Seven variables of parental characteristics were also included as predictors of latent class
151 membership. Mother's and father's education were included separately and divided into the
152 following categories; no schooling, primary education, secondary education and post-
153 secondary education. An issue with the inclusion of education is that household wealth is
154 included in the latent class input variables and education may be endogenous to household
155 wealth. While they are capturing slightly different concepts (i.e. material resources versus
156 parental cognition), the issue should be considered alongside the interpretation of our results.
157 The height of the mother was also included and divided into the following categories (cm);
158 greater than or equal to 160.0, 155.0-159.9, 150.0-154.9, 145.0-149.9, and less than 145.0.
159 For fathers' height the categories were: greater than or equal to 170.0, 165.0-169.9, 160.0-
160 164.9, 155.0-159.9, and less than 155.0. The height categories are different to reflect the
161 different distribution of values between males and females (i.e. men were taller on average).
162 Parental BMI was calculated separately by dividing body mass (kg) by height-squared (m^2)
163 and further split into groups based on WHO defined cut offs; underweight (<18.5), normal
164 ($18.5-24.9$) and overweight (≥ 25). Finally, we included whether the mother was married
165 before the age of 18 years.

166

167 **Statistical Analysis**

168 Latent class analysis (LCA) was used to explore the existence of homogenous groups within
169 children who are stunted. LCA is a finite mixture model which seeks to identify a latent
170 structure within data through a probabilistic model (Collins & Lanza, 2010). The aim is to
171 identify a categorical latent variable which is not directly measured but captured through
172 other observed variables. Groups are identified based on the multi-dimensional distribution of
173 variables.

174

175 As LCA is an exploratory method; a decision must be made on the number of latent classes
176 that best describes the underlying structure of the data. To identify the most appropriate
177 number of classes we ran several models for a range of solutions between 2 and 10. We did
178 not consider a larger number of latent classes since we wanted the chosen model to be
179 parsimonious. Model fit was assessed using the adjusted Bayesian Information Criterion
180 (BIC), consistent Akaike Information Criterion (AIC) and G-squared statistic (Collins &
181 Lanza, 2010).

182

183 One strength of LCA is that covariates can be included in the model to predict how factors
184 are associated with class membership (Collins & Lanza, 2010). Covariates were modelled
185 using a multinomial logit model. Mother's and father's education level, body mass index
186 category, and height were each included as covariates, as well as whether the mother was
187 married under the age of 18. We report odds ratios for the model and the 95% confidence
188 intervals for these estimates.

189

190 All analyses were undertaken using SAS v9.3 and the PROC LCA procedure (Lanza et al.,
191 2007). Sample weights were included in the analysis allowing our observations to be

192 representative and to account for the survey design (although PROC LCA cannot account for
 193 the stratified multi-stage cluster design for how the data were collected, limiting the
 194 representativeness of the analyses).

195

196 **RESULTS**

197 Table 1 presents summary statistics of the characteristics of children who were stunted and
 198 for the whole sample to help contextualise the data. There were slightly more males
 199 compared to females in our sample of stunted children, although this only differed slightly
 200 from all children. There was a greater proportion of stunted children from poorer households
 201 compared to the whole sample. Stunted children were on average older than the average for
 202 the entire sample. Stunted children had less diverse diets and lower prevalence of some
 203 health-related measures (e.g. vaccinations, safe disposal of stools) in comparison to the whole
 204 sample. The characteristics of parents also followed these patterns.

205

206 **Table 1: Sample characteristics of stunted children (sample weights were applied).**

207

	Stunted children (percentage)	All children (percentage)
<i>Panel A: Latent class analysis input variables</i>		
Males	51.9	51.6
Females	48.1	48.5
Household wealth quintile		
1 (Lowest)	34.7	28.6
2	24.9	21.6
3	19.1	18.9

	4	14.9	17.6
	5 (Highest)	6.9	13.4
<hr/>			
	6-11 months	8.7	14.9
	12-23 months	30.2	29.2
Life stage	24-35 months	30.7	28.1
	36-47 months	14.9	11.8
	48-59 months	15.5	9.3
<hr/>			
	1 (Lowest)	35.6	31.4
Diet	2	25.5	25.3
diversity	3	21.6	22.3
score	4	10.8	11.8
quintile	5 (Highest)	6.6	9.3
<hr/>			
	Breast fed within one hour of birth	28.1	23.4
	Infectious disease in past two weeks	12.9	28.5
	Water drinking source was through a piped connection	11.6	17.8
	Safe disposal of stools	11.6	15.5
	Improved sanitary facility	15.5	22.4
<hr/>			
	Non-solid fuels	11.9	18.3
Household	Solid fuels in separate kitchen	52.8	51.1
air quality	Solid fuels in non-separate kitchen	35.3	30.6
<hr/>			
	Iodized salt used	41.9	46.2
	Fully vaccinated	37.3	40.9
	Vitamin A supplement taken	18.5	20.1
<hr/>			

Panel B: Covariates for explaining class

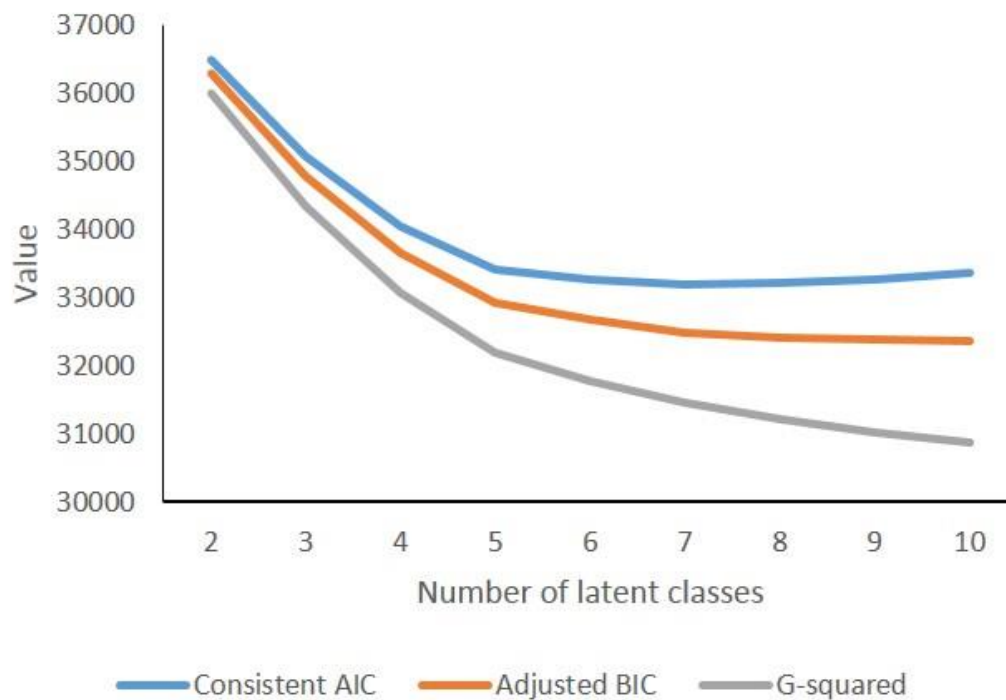
membership

	No schooling	59.3	50.0
Mothers	Primary	14.1	14.0
education	Secondary	22.2	26.6
	Post-secondary	4.5	9.4
Mothers	Underweight	44.8	41.1
body mass	Normal	51.5	53.1
index	Overweight	3.7	5.8
	<145	15.8	12.1
Mothers	145-149.99	31.6	27.0
height	150-154.99	32.4	33.5
(cm)	155-159.99	15.8	19.8
	>=160	4.5	7.6
Mother was married before 18		66.8	60.7
	No schooling	34.6	29.3
Fathers	Primary	16.8	15.2
education	Secondary	36.4	37.6
	Post-secondary	12.2	17.9
Fathers	Underweight	36.2	31.4
body mass	Normal	58.0	59.5
index	Overweight	5.8	9.1
Fathers	<155	9.0	7.0
height	155-159.99	20.8	17.5
(cm)	160-164.99	31.5	29.9

165-169.99	24.1	26.5
≥ 170	14.6	19.1

208

209 Figure 1 presents the analysis of the number of latent classes that best captures the underlying
 210 structure of the data. While an increasing number of groups typically resulted in an improved
 211 model across each measure of model fit, the rate of these improvements decreased with
 212 increasing number of solutions. Both the consistent AIC and adjusted BIC have a clear ‘knee
 213 point’ at 5 classes whereby increasing number of classes adds little additional information.
 214 The G-squared also has a kink at 5, but continues decreasing. However, the measure is less
 215 useful with large samples (Collins & Lanza, 2010). We selected a 5 class solution for our
 216 analysis.



217

218 **Figure 1: Examining model fit statistics by the number of latent classes.**

219

220 Table 2 presents the conditional probabilities of each latent class and the latent class
221 prevalences (Figure 2 presents the conditional probabilities using a radial plot to aid
222 interpretation). We named each latent class and described their characteristics below:

223

- 224 1. *Poor, older and poor health-related outcomes*: While the characteristics are mostly
225 similar to ‘poor, young and poorest health-related outcomes’, there are some key
226 differences. The children mainly differ based on life stage being older. The latent
227 class also have a higher prevalence of children fully vaccinated, fewer children
228 suffering from infectious diseases and a bimodal distribution for the diet variable.
- 229 2. *Poor, young and poorest health-related outcomes*: The latent class contained the
230 largest probability of the lowest two quintiles of household wealth. They are also the
231 youngest class compared to the other classes. Characteristics were largely the worst in
232 comparison to the other classes. Diet diversity was low and while ‘poor, older and
233 poor health-related outcomes’ had the largest probability for quintile 1, the class has
234 the largest combined probability for quintiles 1 and 2. The class also had some of the
235 poorest health-related characteristics especially for hygiene and sanitation, household
236 air quality and full vaccinations. It is the largest class.
- 237 3. *Poor with mixed health-related outcomes*: There are higher probabilities of children in
238 the lower quintiles of household wealth. The class displays relatively good health-
239 related characteristics, with the highest probability for vitamin A supplements, full
240 vaccinations and breast feeding. However, it also has the highest probability for
241 infectious disease and low probabilities for the hygiene, sanitation and household air
242 quality variables. The majority were aged between one and three years old. It is the
243 second smallest class as well.

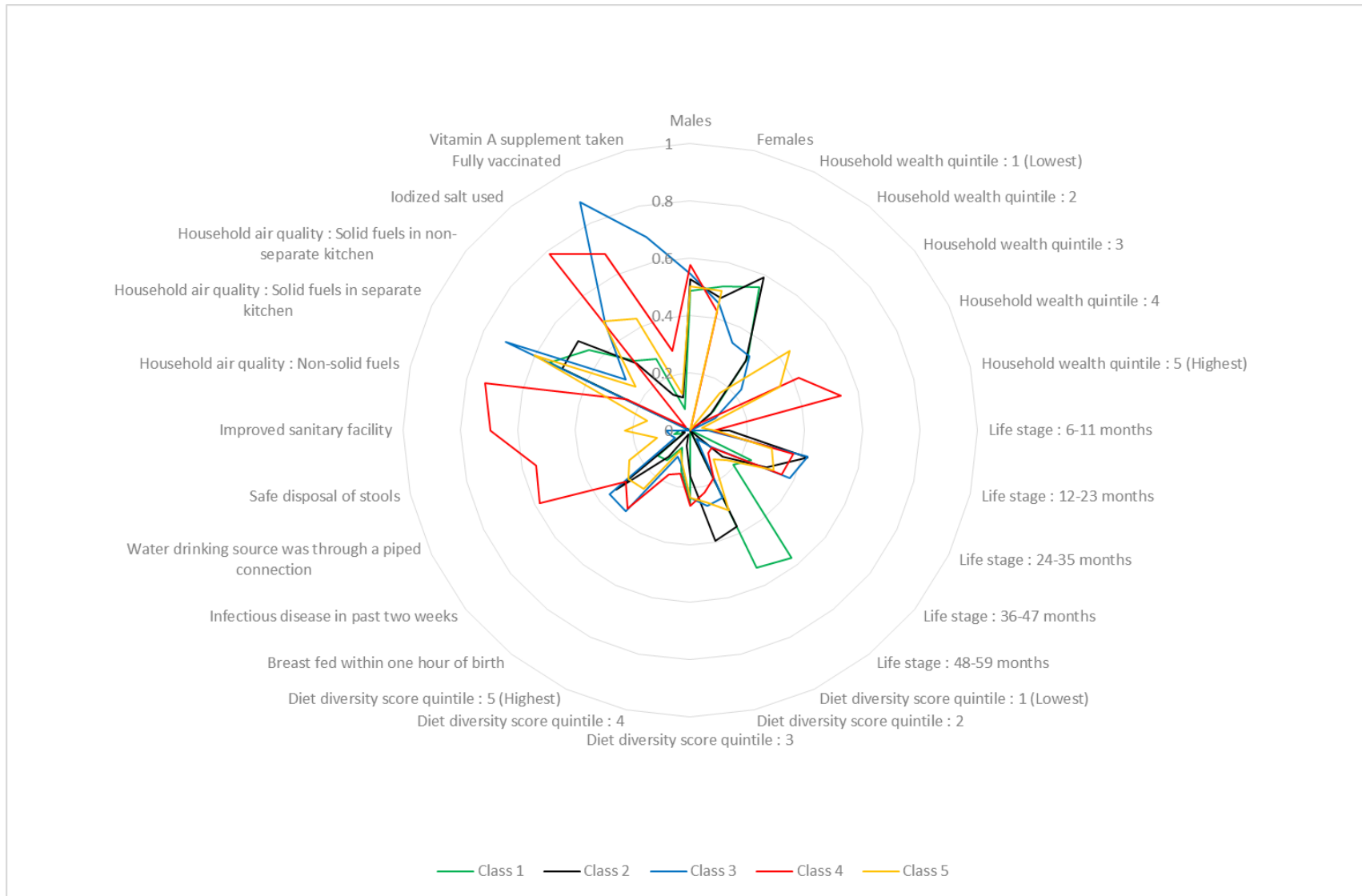
244 4. *Wealthy and good health-related outcomes*: Most children in the class are in the top
245 two quintiles of household wealth. They displayed the best health-related outcomes
246 characteristics in comparison to the other clusters. However, they did not perform the
247 best for every health-related outcome variable e.g. fully vaccinated or vitamin A
248 supplements. The majority were aged between one and three years old. It was the
249 smallest class.

250 5. *Typical traits*: The conditional probabilities largely fall in the middle in comparison to
251 the other latent classes. There are few features that make the class distinctive other
252 than this.

253

254 There was little variation in proportion of males and females in each latent class suggesting
255 that our latent classes are largely independent of sex.

256



257

258 **Figure 2: A radial plot of the conditional probabilities for each latent class.**

259 **Table 2: The characteristics of a 5 group latent class analysis of stunted children.**

260

		Class 1	Class 2	Class 3	Class 4	Class 5
<i>Prevalence (Y)</i>		0.190	0.349	0.103	0.107	0.251
<i>Conditional Probabilities (ρ)</i>						
Males		0.485	0.527	0.545	0.576	0.501
Females		0.515	0.473	0.455	0.424	0.499
1 (Lowest)		0.554	0.591	0.340	0.000	0.001
Household wealth quintile	2	0.321	0.309	0.331	0.001	0.167
	3	0.106	0.092	0.229	0.040	0.442
	4	0.019	0.009	0.099	0.420	0.347
	5 (Highest)	0.001	0.000	0.001	0.539	0.043
	6-11 months	0.000	0.139	0.065	0.081	0.091
Life stage	12-23 months	0.002	0.420	0.419	0.368	0.291
	24-35 months	0.237	0.296	0.385	0.355	0.322
	36-47 months	0.194	0.146	0.094	0.095	0.166
	48-59 months	0.567	0.000	0.036	0.101	0.131
	1 (Lowest)	0.533	0.372	0.261	0.186	0.310
Diet diversity score quintile	2	0.000	0.396	0.270	0.223	0.259
	3	0.255	0.160	0.237	0.264	0.235
	4	0.145	0.057	0.129	0.155	0.120
	5 (Highest)	0.067	0.015	0.103	0.171	0.076
	Breast fed within one hour of birth		0.132	0.118	0.362	0.348
Infectious disease in past two weeks		0.143	0.333	0.360	0.287	0.275

Water drinking source was through a piped connection	0.026	0.019	0.057	0.583	0.237
Safe disposal of stools	0.056	0.028	0.070	0.551	0.117
Improved sanitary facility	0.036	0.023	0.085	0.696	0.227
Non-solid fuels	0.005	0.003	0.000	0.735	0.154
Household Solid fuels in separate kitchen	0.544	0.497	0.713	0.247	0.604
air quality Solid fuels in non-separate kitchen	0.451	0.500	0.286	0.018	0.242
Iodized salt used	0.312	0.300	0.478	0.785	0.486
Fully vaccinated	0.275	0.139	0.882	0.682	0.432
Vitamin A supplement taken	0.077	0.118	0.690	0.283	0.131

261

262 Table 3 presents the results exploring the association between parental characteristics and
263 latent class membership. The group ‘typical traits’ (class 5) were selected as the comparator
264 class for interpreting the estimates. Mothers who were married before the age of 18, lower
265 educational attainment and underweight parents were positively associated with membership
266 of classes 1 to 3 compared to ‘typical traits’. Relationships were fairly consistent between
267 mothers and fathers, other than for ‘poor with mixed health-related outcomes’ (class 3) where
268 mother’s education was not important. The direction of these associations to ‘wealthy and
269 good health-related outcomes’ (class 4) were opposite (although there was little association
270 for underweight fathers).

271

272 Height displayed less certainty in the direction of estimates for both mothers and fathers with
273 most confidence intervals crossing a value of 1. While taller mothers were positively
274 associated with class membership of ‘wealthy and good health-related outcomes’ (class 4),

275 the result for father's height was contrary. Taller fathers were negatively associated with
 276 membership of 'poor, young and poorest health-related outcomes' (class 2) compared to
 277 'typical traits' (class 5).

278

279 **Table 3: A multinomial logit model of the association between characteristics of parents**
 280 **and class membership. Note: We present odds ratios with 95% confidence intervals in**
 281 **brackets.**

282

		Class 1	Class 2	Class 3	Class 4	Class 5 (Reference)
Mothers education	No schooling	2.99 (2.19-4.09)	3.02 (2.27-4.03)	0.67 (0.37-1.22)	0.56 (0.33-0.95)	
	Primary			Reference		
	Secondary	0.35 (0.23-0.54)	0.32 (0.22-0.48)	0.72 (0.36-1.46)	3.12 (2.13-4.56)	
	Post-secondary	0.17 (0.03-0.87)	0.11 (0.03-0.47)	0.44 (0.08-2.42)	10.29 (6.33-16.71)	
	Underweight	1.32 (1.08-1.61)	1.59 (1.34-1.90)	1.75 (1.23-2.50)	0.73 (0.58-0.93)	
	Normal			Reference		
Mothers body mass index	Overweight	0.53 (0.28-1.01)	0.38 (0.21-0.69)	0.46 (0.12-0.169)	3.40 (2.30-5.02)	
	<145	1.07 (0.78-1.48)	1.25 (0.94-1.66)	1.16 (0.70-1.91)	1.41 (0.98-2.02)	

	145-149.99	1.05 (0.83- 1.34)	1.02 (0.83- 1.27)	1.33 (0.91- 1.94)	0.83 (0.63- 1.09)
Mothers	150-154.99	Reference			
height (cm)	155-159.99	0.98 (0.73- 1.31)	0.84 (0.65- 1.09)	0.82 (0.53- 1.25)	1.60 (1.19- 2.33)
	>=160	0.70 (0.45- 1.08)	0.80 (0.54- 1.19)	0.78 (0.38- 1.58)	1.50 (0.96- 1.33)
Mother was married before 18		1.56 (1.25- 1.95)	1.72 (1.40- 2.11)	1.16 (0.83- 1.63)	0.52 (0.42- 0.65)
	No schooling	3.02 (2.26- 4.03)	3.14 (2.41- 4.11)	1.31 (0.77- 2.22)	0.51 (0.27- 0.98)
Fathers education	Primary	Reference			
	Secondary	0.69 (0.52- 0.91)	0.68 (0.53- 0.86)	0.48 (0.33- 0.70)	1.82 (1.23- 2.71)
	Post- secondary	0.58 (0.35- 0.94)	0.44 (0.27- 0.70)	0.34 (0.17- 0.70)	2.32 (1.46- 3.68)
Fathers body mass index	Underweight	1.84 (1.36- 2.49)	1.62 (1.21- 2.16)	1.47 (0.88- 2.47)	0.96 (0.65- 1.41)
	Normal	Reference			
	Overweight	0.30 (0.11- 0.83)	0.30 (0.12- 2.16)	0.25 (0.04- 1.50)	4.72 (2.89- 7.70)
Fathers height (cm)	<155	1.25 (0.73- 2.15)	1.19 (0.74- 1.92)	1.17 (0.56- 2.47)	0.18 (0.06- 0.54)
	155-159.99	0.90 (0.63- 1.28)	0.91 (0.67- 1.24)	0.64 (0.36- 1.14)	0.67 (0.44- 1.03)

160-164.99			Reference	
165-169.99	0.83 (0.60- 1.14)	0.66 (0.48- 0.89)	0.70 (0.43- 1.14)	1.03 (0.74- 1.43)
>=170	0.65 (0.42- 1.14)	0.61 (0.41- 0.93)	0.55 (0.26- 1.19)	0.65 (0.44- 0.98)

283

284

285 **DISCUSSION**

286 Our study revealed five distinct ‘types’ of stunted children in India demonstrating clear
287 heterogeneity of stunting. An important contribution of our study is the examination of
288 multiple risk factors and determinants within a mutually adjusted casual framework. A major
289 limitation of many high profile studies in this field is the continued examination of single risk
290 factors in isolation leading to biased effect estimates (Danaei et al., 2016). The high
291 prevalence of stunting in India (and globally), combined with the associated health and social
292 implications of stunting (Black et al., 2008; Schaible & Kaufmann, 2007; UNICEF, 2013),
293 has seen considerable interest in how to effectively design interventions to tackle stunting
294 (Bhutta et al., 2008, 2013). Treating childhood stunting as a singular concept may lead to a
295 false dichotomy of the determinants and experiences of stunting, limiting our understanding
296 of the issue and our ability to tackle it.

297

298 The multidimensional nature of childhood stunting identified in our study has important
299 implications for future policy. The distinct characteristics of each latent class suggest
300 differing strategies are required to tackle the issue. Ignoring the complex combination of
301 characteristics that constitute each class may restrict the effectiveness of policies or lead to
302 inefficient targeting of resources (Dewey & Adu-Afarwuah, 2008; Fenske et al., 2013). For

303 example, improving vaccination uptake among stunted children to protect against infectious
304 diseases would appear important given that only 37.3% of stunted children were fully
305 vaccinated. However, the policy would be less appropriate for ‘poor with mixed health-
306 related outcomes’ (class 3) who had a high prevalence of vaccination, where it may be better
307 to focus on other issues such as improving access to clean water. Future research should
308 explore the application of our latent classes within different interventions to assess how
309 useful they are for delivering policy more efficiently.

310

311 The discovery of a ‘poor but mixed health-related outcomes’ cluster was a particularly
312 important finding since it demonstrates the possibility to achieve good health-related
313 outcomes despite the imposing forces of a lack of wealth. Children in the class had the
314 highest prevalence of children fully vaccinated and vitamin A supplements taken, but low
315 prevalence of improved drinking water source or safe sanitation. The finding may suggest
316 that it is useful when implementing interventions aimed at improving health-related outcomes
317 in poor and stunted children to improve education and awareness about access to health
318 services as these represent potential successes. Wealth and neighbourhood conditions are less
319 modifiable, and therefore harder to address (Bhutta et al., 2008; Poel, Hosseinpoor,
320 Speybroeck, Ourti, & Vega, 2008). However, the finding may simply relate to the successful
321 delivery of programmes targeting low socioeconomic status areas or regions (e.g. to increase
322 vaccinations).

323

324 The associations found with the covariates (as well as the relative importance of household
325 wealth in determining the latent classes) are indicative of the importance of the social
326 gradient in understanding latent classes. Whilst most stunting related interventions are aimed
327 at improving nutrition (Bhutta et al., 2013), it is clear that for any intervention to be

328 successful they must be combined with the wider social context (Corsi et al., 2016b; Dewey
329 & Adu-Afarwuah, 2008). Tackling the social gradient will need to be more targeted than
330 simply encouraging economic growth, since economic growth alone is not associated with
331 improved child nutrition (Subramanyam, Kawachi, Berkman, & Subramanian, 2011). While
332 wealthy children have been previously shown to be less likely to be stunted (Corsi et al.,
333 2016b; Fenske et al., 2013; Poel et al., 2008), our findings indicate that they make up their
334 own latent class with more favourable health-related outcomes compared to the other groups.
335 They were, however, a small class but nonetheless it underscores that certain “better off”
336 children may be at risk for stunting despite relatively higher socioeconomic status.

337

338 Parental BMI was associated with class membership, although more consistently for mothers
339 compared to fathers. This association may partly reflect the social gradient as well, since
340 poorer individuals have been previously demonstrated to be more likely to be underweight
341 (Bhutta et al., 2008; Subramanian, Corsi, Subramanyam, & Smith, 2013). However, there
342 may also be an independent association. It is plausible that underweight parents pass on
343 similar habits to the children. Previous research has shown that stunting displays an
344 intergenerational aspect (Grantham-McGregor et al., 2007; Subramanian et al., 2009;
345 UNICEF, 2013). A similar interpretation may be derived for height as well although the
346 results were inconsistent. Our finding that fathers characteristics explain the type of stunting
347 supports calls to move away from a ‘maternal’ to a ‘household’ understanding of the
348 determinants of stunting (Corsi et al., 2016a).

349

350 There are several limitations to our approach. The data were cross-sectional and therefore our
351 ability to draw inferences about our groupings is limited. It will be important to replicate the
352 approach using longitudinal observations to explore how the groups develop and change over

353 time. We only included seven predictors of class membership and it will be important to
354 examine the association of additional factors that have been shown to be related to stunting
355 such as the socio-economic context of local regions (Subramanyam et al., 2011). LCA can
356 only make observations about how factors are co-associated within stunted children, and it
357 does not show the strength of an association to the risk of stunting. The data we used were
358 collected in 2005-06 and are therefore outdated. They are, however, the most recent data
359 available at the time of analysis. Irrespective of the date, it does not change the notion that the
360 strength of the paper is its conceptual approach of exploring a typology of stunting. The
361 NFHS is currently processing the data collected in the fourth survey wave (2015-16) and it
362 will be important to update our study using these newer data when they are released to
363 examine how the situation has changed. The risk factor data are self-reported and therefore
364 potentially subject to bias. There were no alternative large representative data sets which
365 included objective measures limiting the quality of our observations. The data used for the
366 characteristics of fathers correspond to the mother's partner at the time of the survey. While
367 the majority of partners are the biological father of the child, it may introduce a small amount
368 of bias in estimates. However, this should not distract from the importance of how adults
369 within the household may influence experiences.

370

371 In conclusion, the causes of childhood stunting are complex and multidimensional. Our paper
372 contributes to a literature that has largely examined stunting as a singular concept,
373 demonstrating heterogeneity among stunted children. We hope that the approach outlined in
374 this paper will help policy makers in designing effective interventions as opposed to more
375 simplistic approaches that do not differentiate in terms of the individuals they target.

376

377 **REFERENCES**

378 Barros, A. J. D., Carine, P., Drph, R., Axelson, H., Loaiza, E., Bertoldi, A. D., ... Victora, P.

379 C. G. (2012). Equity in maternal, newborn, and child health interventions in Countdown
380 to 2015: a retrospective review of survey data from 54 countries. *The Lancet*, *379*,
381 1225–1233.

382 Bhutta, Z. A., Ahmed, T., Black, R. E., Cousens, S., Dewey, K., Giugliani, E., ... Kirkwood,
383 B. (2008). What works? Interventions for maternal and child undernutrition and
384 survival. *The Lancet*, *371*, 417–440.

385 Bhutta, Z. A., Das, J. K., Rizvi, A., Gaff, M. F., Walker, N., Horton, S., ... Black, R. E.
386 (2013). Evidence-based interventions for improvement of maternal and child nutrition:
387 what can be done and at what cost? *The Lancet*, *382*, 452–477.

388 Black, R. E., Allen, L. H., Bhutta, Z. A., Caulfi, L. E., Onis, M. De, Ezzati, M., ... Rivera, J.
389 (2008). Maternal and child undernutrition: global and regional exposures and health
390 consequences. *The Lancet*, *371*, 243–260.

391 Collins, L. M., & Lanza, S. T. (2010). *Latent Class and Latent Transition Analysis*. Hoboken,
392 New Jersey: Wiley.

393 Corsi, D. J., Meija, I., & Subramanian, S. V. (2016a). Improving household-level nutrition-
394 specific and nutrition-sensitive conditions key to reducing child undernutrition in India.
395 *Social Science & Medicine*, *157*, 189–192.

396 Corsi, D. J., Meija, I., & Subramanian, S. V. (2016b). Risk factors for child chronic
397 undernutrition in India: Estimating relative importance, population attributable risk and
398 fractions. *Social Science & Medicine*, *157*, 165–185.

399 Crookston, B. T., Dearden, K. A., Alder, S. C., Porucznik, C. A., Stanford, J. B., Merrill, R.
400 M., ... Penny, M. E. (2011). Impact of early and concurrent stunting on cognition.
401 *Maternal & Child Nutrition*, *7*, 397–409.

402 Danaei, G., Andrews, K. G., Sudfeld, C. R., Mccoy, C., Peet, E., Sania, A., ... Fawzi, W. W.
403 (2016). Risk Factors for Childhood Stunting in 137 Developing Countries: A
404 Comparative Risk Assessment Analysis at Global, Regional, and Country Levels. *PLoS*
405 *Medicine*, *13*(11), e1002164.

406 de Onis, M., Dewey, K. G., Borghi, E., Onyango, A. W., Blössner, M., Daelmans, B., ...
407 Branca, F. (2013). The World Health Organization 's global target for reducing
408 childhood stunting by 2025 : rationale and proposed actions. *Maternal & Child*
409 *Nutrition*, *9*(S2), 6–26.

410 Dewey, K. G., & Adu-Afarwuah, S. (2008). Systematic review of the efficacy and
411 effectiveness of complementary feeding interventions in developing countries. *Maternal*
412 *& Child Nutrition*, *4*, 24–85.

413 Fenske, N., Burns, J., Hothorn, T., & Rehfuess, E. A. (2013). Understanding Child Stunting
414 in India: A Comprehensive Analysis of Socio-Economic, Nutritional and Environmental
415 Determinants Using Additive Quantile Regression. *PLoS One*, *8*(11), e78692.
416 <http://doi.org/10.1371/journal.pone.0078692>

417 Grantham-McGregor, S., Cheung, Y. B., Cueto, S., Glewwe, P., Richter, L., & Strupp, B.
418 (2007). Developmental potential in the first 5 years for children in developing countries.
419 *The Lancet*, *369*, 60–70.

420 Lanza, S. T., Collins, L. M., Lemmon, D. R., Schafer, J. L., Lanza, S. T., Collins, L. M., ...
421 Schafer, J. L. (2007). PROC LCA: A SAS Procedure for Latent Class Analysis.
422 *Structural Equation Modelling*, *14*(4), 671–694.

423 Martorell, R., Horta, B. L., Adair, L. S., Stein, A. D., Richter, L., Fall, C. H. D., ... Barros, F.
424 C. (2010). Weight gain in the first two years of life is an important predictor of
425 schooling outcomes in pooled analyses from five birth cohorts from low- and middle-
426 income countries. *Journal of Nutrition*, *140*(2), 348–354.
427 <http://doi.org/10.3945/jn.109.112300>.(SES)

428 Ministry of Women and Child Development. (2014). Rapid Survey on Children 2013-14.

429 Retrieved from [http://wcd.nic.in/sites/default/files/RSOC National Report 2013-14](http://wcd.nic.in/sites/default/files/RSOC_National_Report_2013-14_Final.pdf)
430 [Final.pdf](http://wcd.nic.in/sites/default/files/RSOC_National_Report_2013-14_Final.pdf)

431 Poel, E. Van De, Hosseinpoor, R., Speybroeck, N., Ourti, V., & Vega, J. (2008).
432 Socioeconomic inequality in malnutrition in developing countries. *Bulletin of the World*
433 *Health Organization*, 86, 282–291.

434 Raj, A., Saggurti, N., Winter, M., Labonte, A., Decker, M. R., Balaiah, D., & Silverman, J. G.
435 (2010). The effect of maternal child marriage on morbidity and mortality of children
436 under 5 in India: cross sectional study of a nationally representative sample. *BMJ*, 340,
437 b4258.

438 Ruel, M. T., & Menon, P. (2002). *Creating a Child Feeding Index Using the Demographic*
439 *and Health Surveys: an example from Latin America*. Washington, DC: International
440 Food Policy Research Institute.

441 Schaible, U. E., & Kaufmann, S. H. E. (2007). Malnutrition and Infection: Complex
442 Mechanisms and Global Impacts. *PLoS Medicine*, 4(5), e115.

443 Subramanian, S. V., Ackerson, L. K., & Davey Smith, G. (2010). Parental BMI and
444 Childhood Undernutrition in India: An Assessment of Intrauterine Influence. *Pediatrics*,
445 126(3), e663–e671.

446 Subramanian, S. V., Ackerson, L. K., Smith, G. D., & John, N. A. (2009). Association of
447 Maternal Height With Child Mortality, Anthropometric Failure, and Anemia in India.
448 *JAMA*, 301(16), 1691–1701.

449 Subramanian, S. V., Corsi, D. J., Subramanyam, M. A., & Smith, G. D. (2013). Jumping the
450 gun : the problematic discourse on socioeconomic status and cardiovascular health in
451 India. *International Journal of Epidemiology*, 42, 1410–1426.

452 Subramanyam, M. A., Kawachi, I., Berkman, L. F., & Subramanian, S. V. (2011). Is
453 Economic Growth Associated with Reduction in Child Undernutrition in India? *PLoS*
454 *Medicine*, 8(3), e1000424. <http://doi.org/10.1371/journal.pmed.1000424>

455 UNICEF. (2013). Improving child nutrition. Retrieved from
456 [http://www.unicef.org/gambia/Improving_Child_Nutrition_-](http://www.unicef.org/gambia/Improving_Child_Nutrition_-_the_achievable_imperative_for_global_progress.pdf)
457 [_the_achievable_imperative_for_global_progress.pdf](http://www.unicef.org/gambia/Improving_Child_Nutrition_-_the_achievable_imperative_for_global_progress.pdf)

458 United Nations. (2015). India and the MDGs Towards a sustainable future for all. Retrieved
459 from http://in.one.un.org/img/uploads/India_and_the_MDGs.pdf

460 WHO. (2006). *WHO Child Growth Standards: Length/height-for-age, Weight-for-age,*
461 *Weight-for-length, Weight-for-height and Body Mass Index-for-age: Methods and*
462 *Development*. Geneva: WHO.

463 WHO. (2017). Levels and trends in child malnutrition. Retrieved from
464 http://www.who.int/nutgrowthdb/jme_brochure2017.pdf
465