

Risk Factors and the Development of Competence in Children from Low-Income Countries: The Importance of Social-Emotional Outcomes and Multiple Process Models

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Abstract

A major focus of the second paper in *The Lancet* Child Development series was identification of biological and psychosocial risk factors that compromise the cognitive competence of young children from developing countries. Additional evidence is presented on two issues raised in the second paper: (a) the necessity to go beyond cognitive outcomes when defining children's competence, and include critical dimensions of social-emotional development such as attachment and temperament, and (b) the need to assess underlying mechanisms such as covariance among risk factors, and additive co-action and nonlinear interaction processes, through which exposure to multiple risks can compromise the development of infants and young children. Implications for the identification of children at risk and for designing interventions are presented.

The second paper in *The Lancet* Series identified nine commonly encountered biological and psychosocial risk factors which can compromise the cognitive and social-emotional competence of young children in developing countries (Walker et al., 2007). A major focus of the second *Lancet* paper was on cognitive outcomes, in good part due to the general lack of research evidence from developing countries on children's social-emotional development. In addition to identifying risk factors the paper also emphasized the covariance between developmental risk factors and the implications of such covariance for child development. A fundamental theme of the second *Lancet* paper was that child development is increasingly compromised as the number of encountered risk factors increases.

Using the second paper in *The Lancet* Series as a framework the present paper has two aims. The first is to document the importance of certain aspects of early social-emotional development as a means of highlighting the need to broaden the evidence base

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in developing countries in these areas. The second is to illustrate how the underlying nature of multiple risk factors translates into compromised developmental competence. To meet these aims evidence from prior research studies, as well as recently analyzed data from my own research in developing countries will be used.

The Importance of Social-Emotional Development

While the acquisition of culturally and age-appropriate cognitive skills plays an important role in the development of competence in young children, cognitive skills are not the only domain defining competence. Children who are viewed as highly competent by other members of their culture are those with a greater range of cognitive skills, who also have interpersonal characteristics that appeal to caregivers and peers in their culture, and who have personal characteristics that promote socially appropriate behaviors and that motivate the child to actively interact with their environment (Wachs, 1999). Supporting this statement, evidence indicates that school readiness is influenced not just by cognitive or pre-academic skills, but also by the level of a child's social-emotional development (Blair, 2002). However, the contributions of social-emotional development to child competence go well beyond just school readiness. This conclusion is illustrated by reference to two major domains of the social-emotional development of young children, attachment and temperament.

Attachment

Attachment is defined as an enduring emotional tie between infants and their main caregivers (Ainsworth, Blehar, Waters, & Wall, 1978). Attachment relationships provide a context in which infants and children explore and learn about the world, learn about regulating emotions, and learn about interacting with others (Bowlby, 1969; Cassidy, 1994; Lay, Waters, Posada, & Ridgeway, 1995). The quality of the attachment relationship is primarily a reflection of the quality of interactions between infants and their primary caregivers, with particular reference to caregiver availability, sensitivity and responsiveness to the infant's signals (Belsky, 2006). However, consistent with the emphasis in the *Lancet* series papers on multiple influences on development, there is a small body of evidence suggesting that the development of secure attachments may also be compromised by biomedical risk factors such as early neurological impairment (Brisch et al., 2005; Cox, Hopkins, & Hans, 2000) and undernutrition in infancy (Valenzuela, 1990; Waters & Valenzuela, 1999).

Direct consequences of an insecure attachment. Failure to develop a secure attachment in infancy has been linked to a variety of adverse developmental outcomes. These include impaired relationships with peers (Collins & Sroufe, 1999), a lower quality of problem-solving abilities, reduced resourcefulness in negotiating the environment (Matas, Arend, & Sroufe, 1978; Sroufe, 1983), and an increased risk of behavioral problems during the preschool and early school years (Cohn, 1990; Elicker, Englund, & Sroufe, 1992; Greenberg, Speltz, & DeKlyen, 1993).

Indirect consequences of an insecure attachment. In addition to these direct consequences differences in attachment status may also moderate the impact of children's exposure to developmental risk factors. Specifically, evidence indicates that a secure

attachment relationship may promote resilience in children who are exposed to family or psychosocial risk factors (Fergusson & Horwood, 2003; Masten & Obradovic, 2006). There is also evidence that children with an insecure attachment have a greater likelihood of encountering other developmental risk factors than do children with a secure attachment. For example, even though they have no knowledge of the child's attachment history, preschool teachers are more likely to react with anger, low warmth, or controlling-infantilizing behaviors towards children with insecure attachments (Sroufe & Egeland, 1991).

These findings indicate that insecure attachment can operate as a developmental risk factor, increase the probability of exposure to multiple risk factors, and identify which children are most likely to be affected when exposed to multiple developmental risks.

Temperament

Temperament is traditionally defined as: "Biologically rooted individual differences in behavior tendencies that are present early in life and are relatively stable across various kinds of situations and over the course of time" (Bates, 1989). There is a consistent body of evidence documenting that individual differences in temperament are strongly influenced by individual biological characteristics, such as genes (Goldsmith, Buss, & Lemery, 1997), and central nervous system structure and function (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). Again, consistent with the emphasis in the *Lancet* series papers on multiple influences on development, there is evidence indicating that individual variability in temperament is also influenced by environmental-contextual influences (Wachs, 2006a; Wachs & Kohnstamm, 2001).

Direct consequences of temperament characteristics. Individual differences in temperament have been directly linked to variability in children's competence in a variety of domains. For example, early inhibition has been linked to later internalizing behavior problems, while early resistant temperament is related to an increased risk of later externalizing behavior problems (Rothbart & Bates, 2006). Similarly, variability in early stimulus sensitivity, adaptability, mood, and persistence were found to predict later differences in school achievement (Guerin, Gottfried, Oliver, & Thomas, 2003), while more efficient self-regulation in infancy and early childhood has been linked to the development of better peer relations and lower risk for later antisocial behavior disorders (Grolnick, McMenamy, & Kurowski, 2006).

Indirect consequences of temperament characteristics. Paralleling the findings for attachment, individual differences in temperament also have the potential to moderate the impact of exposure to developmental risks. Results from a number of studies indicate that the negative effects of parental rejection, hostility, or alcoholism on children's adjustment are accentuated in children who are low in positive emotionality and self-regulation or high in impulsivity, irritability, or sensory sensitivity (Aron, Aron, & Davies, 2005; Lengua, Wolchik, Sandler, & West, 2000; Morris et al., 2004). In contrast, greater resilience when exposed to developmental risks is more likely to occur in children who are high in positive emotional reactivity, sociability, self-regulation, and task orientation (Wachs, 2006b).

As with attachment, individual differences in temperament also may influence the degree of a child's exposure to a variety of developmental risk factors. Not surprisingly children with temperaments characterized as "challenging" by a given culture are more

likely to encounter parental punishment and/or lower levels of parental stimulation and interaction (Guerin et al., 2003; Putnam, Sanson, & Rothbart, 2002). In addition, as emphasized in the second paper in *The Lancet Series*, maternal depression is a major developmental risk factor. Mothers of infants with fussy, difficult, hard-to-manage temperaments are at greater risk for depression than are mothers with more placid infants (Beck, 2001; Dudley, Roy, Kelk, & Bernard, 2001; Murray, Stanley, Hooper, King, & Fiori-Cowley, 1996; Wald, Yake, & Kelley, 2007).

The contribution of temperament to the likelihood of encountering developmental risk factors extends beyond the psychosocial into the realm of biological risks as well. One such biological risk involves nutrition, with evidence from a number of studies indicating that infants with fussy-intense temperaments are less likely to be breastfed or are breastfed for shorter durations of time than less fussy-intense infants (Field, Hernandez-Reif, & Feijo, 2002; Hagekull, Bohlin, & Rydell, 1997; Thome, Alder, & Ramel, 2006; Vandiver, 1997). Individual differences in temperament have also been associated with an increased risk of physical injury (Matheny, 1986; Morrongiello & Dawber, 1998) and an increased risk of parasitic infection in childhood (Kvalsvig & Becker, 1988).

As further documentation of relations between individual differences in child temperament and indices of biological risk, Table 1 illustrates how measures of temperament are related to subsequent indices of child nutrition. These recently analyzed findings come from a longitudinal study done in collaboration with colleagues at the Institute for Nutritional Investigation in Peru. The study involved over 240 low-income families from urban Lima. Infants in this study were followed from the prenatal period through 12 months of age. Individual difference characteristics included measures of infant temperament assessed via maternal report on a standardized questionnaire, coding of both neonatal and infant temperament during laboratory-based assessment procedures, and observer coding of neonatal and infant temperament during naturalistic home observations. Biological risk factors included indices of child diet based on 24-hour recall taken on two nonconsecutive day at 6 and 12 months, a maternal report measure of exclusivity of breastfeeding taken at 3 and 6 months, and measures of child anthropometry obtained at 3, 6, and 12 months.

In interpreting the findings from Table 1 it is important to emphasize that specific regressions were computed only after overall significance was established for sets of multiple predictors and multiple outcomes using canonical correlation procedures (Sherry & Henson, 2005). In addition all regressions were computed controlling for family economic resources. As can be seen in Table 1, infants with better nutritional status or diet are those who are more placid or those with higher levels of alertness or attention. Infants with less adequate nutritional status or diet are those who are more difficult to manage or more inhibited. The findings for inhibition are particularly relevant given evidence from earlier studies done in developing countries indicating lower food intake (Pollitt et al., 1974) and less adequate physical growth (Galler, Cervera, & Harrison, 1998) for more inhibited infants.

Table 1
Individual characteristics as predictors of infant nutrition

Age and temperament	Nutrition	Findings	Interpretation
Neonatal			
Alert	3–6 month change in	$R^2 = .042^*$	More alert, greater

Age and temperament	Nutrition	Findings	Interpretation
	length	$B = .179^*$	length gain.
3 months			
Attentive/involved	3–6 month change in breastfeeding	$R^2 = .091^{**}$ $B = .164^*$	More attentive, more likely continuation of exclusive breastfeeding.
Placid	3–6 month change in height	$R^2 = .048^*$ $B = .162^*$	More placid, greater increase in height.
6 months			
Hard to manage	12-month skinfold thickness	$R^2 = .05^*$ $B = -.181^{**}$	More difficult, less thick skinfold.
Inhibited	12-month weight	$R^2 = .044^{***}$ $B = -.195^*$	More inhibited, slower weight gain.
Placid	6–12 month change in dietary risk index	$R^2 = .041^*$ $B = .134^*$	More placid, less change in quality of dietary intake.

Note. Initial screening for overall significance level using canonical correlation before testing specific regressions and all analyses done controlling for family SES.

* $p < .05$. ** $p < .01$. *** $p = .072$.

The overall pattern of findings indicates that both quality of attachment and specific dimensions of temperament can operate as developmental risk factors, can increase the probability of a child's exposure to multiple risk factors, and can predict which children are most likely to be effected when exposed to multiple developmental risks. Taken together these findings emphasize the importance of integrating measures of social-emotional development, such as attachment and temperament, into studies on risk and development of children living in poverty in developing countries. Measures of social-emotional development can function not only as an important outcome measure, but also can provide valuable information on which children living in a context characterized by high levels of the developmental risks identified in the second paper in the *Lancet* series are most likely to both encounter and be adversely affected by exposure to such risks.

Multiple Risks and Developmental Competence

When considering what influences the development of competence in young children, researchers have preferred to focus on specific individual influences taken in isolation (e.g., genes, family, school, nutrition, or culture). Thus, behavioral geneticists have focused primarily on genetic influences upon behavior (e.g., DeFries, Plomin, & Fulker, 1994), developmental psychologists have emphasized the importance of the family environment (e.g., Bradley, 1999), nutrition researchers have focused on the role of nutritional deficiencies (e.g., Allen, 1995), anthropologists have promoted the importance of cultural influences (e.g., Super & Harkness, 1999) and evolutionary psychologists have looked for causes emanating from our species' historical past (e.g., Bjorklund & Pellegrini, 2002). However, particularly over the past decade, the inadequacies of this

type of main effect, single domain approach for understanding variability in individual human developmental competence has become increasingly clear (Gottlieb, 2002; Horowitz, 2000; Johnson & Edwards, 2002; Magnusson, 1995). Rather than isolated main effects, it is now clear that variability in human development is the result of “complex interactions among multiple influences that are each necessary but not sufficient contributors to behavioral development” (Wachs, 2000, p. 3). There are two important processes through which multiple influences can contribute to variability in child competence: covariance among multiple influences and functional linkages between multiple influences. Covariance describes a situation when the various biological and psychosocial risks that impact upon children tend to co-occur at a greater than chance probability. Functional linkages refer to situations when independent multiple influences converge to influence variability in a given outcome. Functional linkages between multiple influences typically can take two forms, additive co-action and nonlinear interaction (Rutter, 1983).

Covariance Among Multiple Influences

Passive covariance. This refers to developmental risk (or protective) influences tending to covary, even though in many cases the reasons leading to such covariance are unknown (Wachs, 2000). The degree of passive covariance is likely to depend in part on the number of risk (or protective) factors that are occurring. In contexts where multiple risk or protective factors naturally occur there is an increased chance that a child living in this context will encounter more than one risk or protective factor. The classic example of passive covariance is seen for children growing up under poverty conditions. Fundamentally, poverty is an umbrella term, describing the multiplicity of psychosocial and bio-ecological risks children growing up in poverty are likely to encounter, such as family turmoil or instability, less responsive parenting, less access to educational stimulation at home or in school, increased exposure to dangerous neighborhoods, and environmental pollution (Evans, 2004). Passive covariance also occurs between specific known developmental risks. There is ample evidence that malnourished or chronically undernourished children are more likely to be exposed to inadequate housing, developmentally inhibiting parental rearing styles, inadequate schooling, and biological risk factors such as low birthweight and a compromised immune system (Grantham-McGregor & Ani, 2001; Keusch, 1990; Pollitt, 2000; Ricciuti, 1993). Similarly, children with higher levels of lead exposure are more likely to have greater exposure to other developmental risk factors including child abuse, lower levels of parental involvement, and increased exposure to other environmental toxins including parental smoking (Bithoney, Vandeven, & Ryan, 1993; Fuggle & Graham, 1991; Lewis, Worobey, Ramsay, & McCormack, 1992; McMichael et al., 1992).

Causal covariance. Covariance among risk factors also may be the result of underlying causal processes through which exposure to one developmental risk factor produces an increased risk of exposure to other developmental risks (Wachs, 2000). One such causal mechanism is reactive covariance, wherein the behaviors of children with highly intense, negative reactive temperament are more likely to elicit parental rejection or physical punishment than children with less reactive or intense negative temperament (Guerin et al., 2003; Wills & Dishion, 2004). Reactive covariance can also occur between

bio-ecological and psychosocial risk factors, as seen in situations where the signals displayed by undernourished or iron deficient infants (e.g., increased proximity seeking or wariness, or fewer displays of positive affect) may adversely influence the nature of caregiver-child transactions (Lozoff et al., 1998; Pollitt, 2000).

Reactive covariance is not the only type of causal covariance that has been identified. The negative feedback loop between child gastrointestinal illness and child nutritional status is a well-documented example of a mechanism through which one biological risk factor increases the risk of encountering a second biological risk factor (Keusch, 1990). Similarly, high levels of family stress can adversely affect the child's immune system, thus increasing the child's susceptibility to infectious agents (Barr, Boyce, & Zeltzer, 1994). In some cases passive covariance can lead to causal covariance. For example inadequately nourished children are more likely to have inadequately nourished parents (passive covariance); poorly nourished parents have less energy available to allow them to stimulate their children appropriately (causal covariance; Grantham-McGregor, 1984). Similarly, pregnant women who use cocaine are less likely to be adequate parents to their young infants (passive covariance); grossly inadequate parenting is likely to result in loss of custody and their child being placed in alternative caregiving arrangements, such as foster homes (causal covariance; Singer et al., 1997).

Consequences of covariance. There are two important implications of covariance between risk factors. The first implication involves research methodology. In studies where the emphasis is on the role of a single, specific risk factor, the traditional response, when there is covariance between the risk factor of interest and other risk factors, is to statistically partial out the covariates. However, there are multiple conceptual and statistical pitfalls associated with such partialling strategies (Evans, 1999). When organism-environment covariance is operating, the correct unit of analysis is the covariance, rather than the covarying elements considered in isolation from each other (Bronfenbrenner, 1999).

The second implication involves the cumulative effects of exposure to multiple risk factors, either at a given time or over time. Evidence presented both in *Lancet* paper 2 (Walker et al., 2007) and multiple other sources (e.g., Fergusson & Horwood, 2003; Sameroff, Gutman, & Peck, 2003) clearly documents that as the number of risks increase child competence decreases. Further, evidence from longitudinal studies indicates that covariance among multiple risk factors occurs not just at a given point in time but across time as well. For example, correlations of $r = .77$ and higher were found for the number of environmental risks encountered by children between 4 and 13 years and between 13 and 18 years (Sameroff & Rosenblum, 2006). These results suggest that a critical marker for identifying those children who are most at risk for compromised development is the number of developmental risk factors the child is exposed to, either concurrently or cumulatively.

Functional Linkages Between Multiple Influences

Additive co-action. This occurs when an outcome is the result of the summed (additive or subtractive) contribution of independent multiple influences. A common example of additive co-action would occur when a child's environment contains both protective and risk factors, as in the case when the child's parents use authoritative

childrearing techniques (protective factor) but are having major marital problems (risk factor) (Hetherington & Elmore, 2003). In this type of situation the child's level of competence varies, depending on the balance between risk and protective factors. Additive co-action processes operate even when the risk and protective factors come from different domains, as occurs when the detrimental impact of chronic otitis media upon preschool children's language development is attenuated when children with this disorder attend high-quality daycare (Vernon-Feagans, Emmanuel, & Blood, 1997), or when previously malnourished children are subsequently reared in more adequate psychosocial environments (Colombo, de la Parra, & Lopez, 1992; Paine, Dorea, Pasquali, & Monteior, 1992).

Within an additive co-action framework an obvious strategy for intervention is to increase the number of protective factors for children who are exposed to developmental risk. The positive effects of this type of strategy are seen in the studies reviewed in *Lancet* paper 2 (Walker et al., 2007, Table 2), where providing environmental stimulation to children at biological risk due to stunting or preterm birth resulted in higher levels of cognitive or social-emotional competence compared to at-risk children who did not receive this stimulation. Providing specific protective factors can have a greater impact when there are naturally occurring protective factors as well, as in the case where medical treatment of parasitic-infected children was more efficacious for those children living in more stimulating home environments, in terms of their subsequent level of cognitive functioning (Boivin, Giordani, Ndanga, & Maky, 1993).

Nonlinear interaction. This occurs when the influence of a specific risk (or protective) factor varies depending on individual characteristics, individual history, or aspects of the child's environment (Wachs, 2000). Most of our evidence in this area involves individual characteristics. As described earlier the impact of exposure to developmental risk factors can vary depending on differences in individual characteristics such as child attachment (Fergusson & Horwood, 2003) and child temperament (Rothbart & Bates, 2006; Wachs, 2006b). There is also an increasing body of research documenting that the developmental consequences of risk factors such as insensitive maternal rearing (Bakermans-Kranenburg & van Ijzendoorn, 2006) and child abuse (Caspi et al., 2002), or protective factors such as social support (Kaufman et al., 2004), vary depending upon the child's individual genetic makeup. Nonlinear interactions can also occur as a function of nongenetic biological risk factors. One such example is seen in our findings from a study done in Jamaica, indicating that term, low birthweight infants are either less sensitive to facilitative aspects of maternal behaviors and/or more sensitive to developmentally inhibitory aspects of maternal behaviors than are term, normal birthweight infants (Wachs, Chang-Lopez, Walker, & Meeks-Gardner, in press).

While fewer examples exist, evidence also supports the hypothesis that individuals' reactivity to stress can vary depending on their developmental histories. For example, there is evidence indicating that children with a history involving higher levels of biosocial risk conditions will be more sensitive to low-level lead exposure than children without such a history (Bellinger, 1995). There is also evidence documenting that early nutritional deficits may increase children's sensitivity to later nutritional deficits (Grantham-McGregor, Chang, Walker, & Powell, 1998; Pollitt, Cueto, & Jacoby, 1998).

Similarly, although evidence is again relatively limited, variability in children's exposure to environmental risk or protective factors may also vary depending on contextual characteristics. A excellent example is seen in results from a study done in Jamaica, documenting that in orderly school environments breakfast feeding facilitated children's on-task behavior in the classroom, whereas in chaotic school environments supplementary breakfast feeding led to reduced on-task classroom behavior (Grantham-McGregor et al., 1998). A similar pattern of findings came from a study done in Guatemala, indicating that the social behavior patterns displayed by poorly nourished children who received nutritional supplementation varied depending upon the harshness of ecological conditions in their villages (Barrett, Radke-Yarrow, & Klein, 1982).

Distinguishing Between Covariance and Interaction

When multiple influences are involved in individual variability, additive co-action and/or nonlinear interaction processes can be operating. Particularly with regard to intervention it is essential to distinguish whether relations between multiple influences and outcomes involve additive co-action or nonlinear interaction. All too often nonlinear interaction processes are inferred when a closer look at the evidence supports the operation of additive co-action. In order to make this distinction it is necessary to test for statistical interactions. If there is a statistical interaction between two or more predictors of a given outcome then nonlinear interaction processes are occurring. If two or more variables predict a given outcome but there is no statistical interaction then additive co-action is involved (Rutter, 1983).

An example of this distinction is shown in Table 2, describing the results from two different studies, each of which involved prediction of variability in child competence by adequacy of child nutrition and characteristics of the child's rearing environment. The first set of results comes from a previously reported study done in Egypt involving toddlers assessed between 18 and 30 months (Wachs et al., 1993). Our nutrition measure was child diet, based on monthly 24-hour food recall taken over two nonconsecutive days. Maternal child-rearing patterns were coded based on two home observations taken each month for 30 minutes per observation. Our outcome measure was extent of child's symbolic play behavior coded during a 10-minute toy play assessment done in the child's home. The second data set comes from our recently analyzed data from Peru described above. In this case our predictors were the child's iron status and two measures of environmental chaos in the child's environment: rooms/people ratio and level of noise in the home that is controllable by adults. Both measures were assessed via 30-minute home observations done twice in a given month. In the Peru study the outcome measure was child temperament. In both studies we tested for both additive co-action and nonlinear interactions. The findings (after establishing overall significance by means of canonical correlations) are shown in Table 2.

As can be seen from Table 2 both child intake of animal source calories and the nature of parental verbal interactions with the child are related to the amount of symbolic play shown by the child. However, the effects are not additive. There is no change in variance accounted for when we add the effects of verbal interactions to the effects of intake of animal source calories. Rather, the effects are interactive, in that the contributions of animal source intake vary depending on the level and type of verbal interactions between child and parent, or alternatively, the contributions of verbal

interactions vary depending on level of animal source intake. In contrast, for temperament the results shown in Table 2 indicate only additive co-action, in that both iron status and chaos indices add unique variance to the prediction of infant temperament, but their contributions are independent of each other.

As noted earlier this distinction has implications for interventions. When either additive co-action or nonlinear interactions are occurring the interventions must be multidimensional in nature. However, it will be easier to develop and carry out intervention strategies when dealing with additive co-action processes. Specifically, in the case of Egypt, an intervention would need to include changing both verbal interaction patterns between caregivers and children and intake of animal source food. However, intervening to influence animal source intake and level of verbal stimulation from caregivers could not be done independently of each other. Rather, interventions would need to deal with the issue of how changing animal source intake would influence the impact of changes in verbal interactions, and how changing patterns of verbal interactions would influence the impact of changes in animal source intake. In contrast, in the case of Peru, an intervention would need to include changing both iron status and crowding or noise level in the home. However, such changes could be done independently since the influence of iron status does not depend on the level of chaos in the home, and the influence of chaos does not depend on the child's iron status.

Table 2
Examples of additive co-action and non-linear interaction

Outcome	Predictors	Analysis	Results (change in R^2)
	Egypt	study	
Symbolic play	(1) Intake of animal source Kilo-calories [An-Kcal]; (2) Non-verbal response by caregiver to child vocalization. [Nvroc].	Increase in R^2 when prediction is based on An-Kal versus the sum of An-Kal + Nvroc.	R^2 change = .005 <i>ns</i>
		Increase in R^2 when prediction is based on the sum of An-Kal + Nvroc versus AnKal x Nvroc.	R^2 change = .118*
Symbolic play	(1) An-Kcal; (2) Level of caregiver verbal stimulation of child [Voc].	Increase in R^2 when prediction is based on An-Kal versus the sum of An-Kal + Voc.	R^2 change = .041 <i>ns</i>
		Increase in R^2 when prediction is based on the sum of An-Kal + Nvroc versus AnKal x Voc.	R^2 change = .136**
	Peru	Study	
Temperament factor: Involved/Manageable	(1) Child hemoglobin [Hgb]; (2) Rooms/people	Overall model R^2 ; Beta for Hgb, Beta for r/p, Beta for Hgb x r/p.	R^2 = .103* Beta Hgb = -.169* Beta r/p = -.223**

Outcome	Predictors	Analysis	Results (change in R^2)
	ratio [r/p].		Beta Hgb x r/p = -.111, <i>ns</i>
Temperament factor: Involved/Manageable	(1) Child hemoglobin [Hgb]; (2) Controllable home noise level [cn].	Overall model R^2 ; Beta for Hgb, Beta for cn, Beta for Hgb x cn.	$R^2 = .280^{**}$ Beta Hgb = -.134* Beta nc = .131* Beta Hgb x cn = -.050, <i>ns</i> .

Note. *x* refers to a statistical interaction term.

* $p < .05$; ** $p < .01$.

Summary and Conclusions

The major contributions of *The Lancet* Series of papers were to quantify the number of young children in developing countries at risk for impaired cognitive and social-emotional competence, identify the major biological and psychosocial risk factors that act to reduce competence, and present empirically validated intervention strategies that have the potential to promote competence in young children at risk. Paper 2 of this series noted the lack of research from developing countries on social-emotional competence and emphasized not only that developmental risk factors covary, but also that development is increasingly compromised as exposure to risk factors cumulate. This paper provides additional evidence emphasizing the importance of social-emotional development as a major domain defining child competence. In addition, this paper provides evidence documenting not only that multiple risk factors covary, but also that the nature of functional relations between multiple risk factors has implications for how developmental risks translate into impairments in child competence. These findings illustrate not only the truism that development is complex, but also the necessity of taking such complexity into account when trying to understand the nature of developmental risks and design interventions to prevent or reduce the impact of such risks.

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