Vulnerable children in Ukraine : impact of institutional care and HIV on the development of preschoolers

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Chapter 2

Physical Growth Delays and Stress Dysregulation in Stunted and Non-Stunted Ukrainian Institution-Reared Children

Dobrova-Krol, N.A., Van IJzendoorn, M.H., Bakermans-Kranenburg, M.J., Cyr, C., & Juffer, F. (2008).
Infant Behavior and Development, 31, 539-553.

Abstract

To study the effect of institutional rearing on physical growth and stress regulation we examined 16 institution-reared children (3 to 6 years old) in Ukraine and compared them with 18 native family-reared children pair-matched on age and gender. Physical growth trajectories were examined on the basis of archival medical records and current measurements of height, weight, and head circumference. Stress regulation was studied on the basis of diurnal salivary cortisol sampled 6 times during one day. 31% of institution-reared children were stunted at 48 months whereas none of the family-reared children were. Substantial delays in physical growth were observed in institution-reared children especially during the first year of life. From 24 months onwards a tendency for improvement in physical growth was evident among the temporarily stunted institution-reared children, with complete catch-up in weight and partial catch-up in height by the time of assessment. Chronically stunted institution-reared children demonstrated persistent severe growth delays. Institution-reared and family-reared children showed similar patterns of diurnal cortisol production with decreases over the day. However, temporarily stunted institution-reared children had a significantly higher total daily cortisol production than both chronically stunted institution-reared children and family-reared children. These data confirm previous findings regarding physical growth delays and stress dysregulation associated with institutional care, but also point to differences in cortisol production between stunted and non-stunted institution-reared children.

Introduction

During the second half of the 20th century empirical research produced overwhelming evidence that institutional care has adverse influence on the development of children. Johnson and colleagues analyzed more than 40 studies, covering the period from 1940 until recently. They addressed the development of children who experienced institutional care varying in quality from a number of countries. The authors concluded that, regardless of differences in quality, institutional care not only failed to support optimal development but was fundamentally damaging to children (Johnson, Browne, & Hamilton-Giachritsis, 2006).

In the meantime, brought to the attention of the public, the conclusions as to the adverse effect of institutional care on the development of children have already contributed to a decline in its use throughout the so called developed countries; elsewhere, institutions have remained as a main alternative for children deprived of parental care (Browne, 2005). Thus, Ukraine, previously a republic of the Soviet Union, impelled by economic needs and former ideological convictions to maintain a collective form of child rearing, until now relies mainly on institutional care rather than family-based care for abandoned and orphaned children (Ball, 1994; Bronfenbrenner, 1970; Dunstan, 1980; Ransel, 1988). As a consequence, out of the 52 countries in the WHO European region, Ukraine at this moment takes the third place as to the absolute number and the sixth place as to the relative number of institution-reared children under 3 years of age (Browne, Hamilton-Giachritsis, Johnson, & Ostergren, 2006). Currently the total number of orphans in Ukraine is 112,000 or 1.11% of the total number of children (State Institute for Family and Youth Development, 2007). Also, since 1999 Ukraine has been in the list of the top 10 source countries for international adoption to the United States (Data from U.S. Department of State; Miller, 2005). However, little is known yet about the quality of institutional care in Ukraine, its comparability to institutional care in other countries, and its impact on its young residents.

The Context of the Study: Institutional Care in Ukraine

The majority of the child-care institutions in Ukraine are state-run, with a standardized structure and functioning across the country. They are organized in such a way as to maintain children who are deprived of parental care from birth to young adulthood. Institutions are differentiated according to children's age (for the age groups from 0 to 3 years; 3 to 7 years, and 7 to 18 years); they are also specialized depending on children's physical condition (there are special boarding schools for children with various developmental and physical impairments).

While in institutional care, children are frequently transferred within and between institutions (Ukrainian Institute of Social Studies, 2001).

Child-care institutions for young children in Ukraine may house up to 200 young residents and are usually characterized by high child-to-caregiver ratios, multiple shifts and frequent change of caregivers, which, as research reveals, are common to institutional care across different countries (see Table 1).

Table 1
Composition of residential institutional care in Ukraine, Russia, Romania and Greece

Country	Study	Children in one institution *n*	Children in one group *n*	Caregivers in one group[a] *n*	Child-to-caregiver ratio[b]
Ukraine	Present study	60 - 200	10 - 17	6 - 9	3 - 7 : 1
Russia	The St. Petersburg – USA Orphanage research Team, 2005; Sloutsky, 1997	60 - 200	9 - 20	8.7	4.5 - 7 : 1
Romania	Smyke, Dumitrescu, & Zeanah, 2002; Zeanah et al., 2003; Kaler & Freeman, 1994	120 - 200	30 - 35	9	10 - 12 : 1
Greece	Vorria et al., 2003	100	12	12	4 - 6 : 1

Note: [a]Specialists and pediatricians who are assigned to several groups are not included. [b]Child-to-caregiver ratios during a day shift are reported here, there are usually fewer caregivers during a night shift.

The daily schedule across Ukrainian institutions is strictly regimented. Apart from routines around sleeping, meals, and hygiene it usually includes group learning activities adjusted to age, and indoor and outdoor play activities. All children are expected to participate in the daily routine and may be exempt from it only if they are ill or as a form of punishment. Most institutions provide fairly clean environments, good medical care and adequate nutrition, with limited cognitive and social stimulation, especially during the first year of life.

Despite the established standards of functioning, during the last decade a growing tendency for divergence in the standards of care, living conditions and rearing beliefs can be observed among Ukrainian child-care institutions.

Heterogeneity of Institutional Care

A common feature of child-care institutions, evident from studies conducted in different countries, is the lack of stable, long-term relationships with consistent caregivers (Bowlby, 1951; Frank, Klass, Earls, & Eisenberg, 1996; Sloutsky, 1997; The St. Petersburg - USA Orphanage research Team, 2005; Zeanah, Smyke, & Settles, 2006). Indeed, the regimented nature of institutional care and a high child-to-caregiver ratio almost inevitably deprive institution-reared children of continuous and reciprocal interactions with stable caregivers, necessary to respond to their developmental needs. However, Gunnar (2001) emphasized that institutional settings can not be encompassed only by reference to the lack of stable child-caregiver relationships. Child-care institutions are widely used in countries with different ethnic, cultural and economic backgrounds and may vary not only between but also within countries. In response to the heterogeneity of institutional settings, Gunnar (2001) identified three levels of privation of the child's needs that should be considered in the examination of developmental outcomes: (1) institutions with global privation of health, nutrition, stimulation, and relationship needs; (2) institutions with adequate health and nutrition support, but privation of stimulation and relationship needs; and (3) institutions that meet all needs except for stable, long-term relationships with consistent caregivers. In the light of this classification most Ukrainian child-care institutions are best described by the second category. In addition to existing differences between child-care institutions, empirical studies also demonstrate that children reared in the same institutions, and therefore presumably subject to the same caregiving circumstances, do not show the same developmental outcomes (Smyke et al., 2007; Vorria et al., 2003; Zeanah et al., 2005). Besides, as evident from the comparisons with native family-reared children, not all developmental domains of a child are equally affected by institutional care (e.g., Smyke et al., 2007; Van IJzendoorn & Juffer, 2006). Such heterogeneity suggests the presence of certain protective and/or risk factors, which may be related to individual caregiving experiences as well as child characteristics. Identification of these factors may be highly valuable for the development of future intervention programs in child-care institutions. Therefore careful examination of the rearing environment as well as child characteristics against adequate native comparison groups is required. However, such studies are still scarce (e.g., Smyke et al., 2007; Zeanah et al., 2005; Kaler & Freeman, 1994; Vorria et al., 2003; Vorria, Rutter, Pickles, Wolkind, & Hobsbaum, 1998).

In the present study we focus on the development of Ukrainian institution-reared children who all experienced about the same level of institutional privation. To examine how institutional rearing in interplay with child characteristics affects physical growth and stress regulation of institution-reared children we compared them to native family-reared peers.

Physical Growth

The majority of studies addressing the influence of institutional care on physical growth were based on the population of formerly institutionalized international adoptees. Johnson and colleagues (1992) examined 65 Romanian adoptees and found that these children lost approximately 1 months of linear growth for every 3 months they spent in institutional care. Albers and colleagues (1997) analyzed preadoptive medical records of 56 adoptees from the Former Soviet Union and Eastern Europe and established that children had 1 month of linear growth delay for every 5 months spent in an orphanage. A meta-analysis of studies addressing the physical growth of adopted children with early institutional experience confirmed that institutional care has a dramatic negative effect on growth, especially evident in the development of height and head circumference. It was also confirmed that the longer children spent in institutional care the more they lagged behind in physical growth (Van IJzendoorn, Bakermans-Kranenburg, & Juffer, 2007).

Whereas body weight and subcutaneous fat reflect more recent nutritional condition, faltering of the linear growth reflects long term chronic adversities (Espo et al., 2002; Grantham-McGregor, Walker & Chang, 2000; Miller, 2005). Head circumference growth indicating brain growth appears to be most vulnerable for the combined negative effects of the rearing environment and the least subject to catch-up after adoption, which may be explained by experience-expectant maturational process of the brain, meaning that the absence of specific experiences during critical periods facilitated in the early stage of life by a caregiver prevent the brain from normal growth (Glaser, 2000; Greenough, & Black, 1992; Rutter, O´Connor, & the English and Romanian Adoptees Study Team, 2004; Van IJzendoorn et al., 2007).

Although the etiology of physical growth delay is multifactorial, it could be brought down to three major causes: malnutrition, child morbidity, and maltreatment or neglect, with the latter two often being the cause of the failure of absorption or utilization of nutrients, leading to secondary malnutrition (Blizzard & Bulatovic, 1992; Grantham-McGregor, Fernald, & Sethuraman, 1999, Miller, 2005; Skuse, Reilly, & Wolke, 1994). Even in the presence of adequate nutritional provision, institution-reared children may suffer from poor absorption of nutrients due to ill-health, apathy, and lack of response-contingent stimulation (Frank et al., 1996; Gunnar, 2001; Spitz, 1945). Besides, psychosocial deprivation may cause inhibition of the growth hormone production and cell resistance to growth factors, usually reversible upon removing from the depriving environment (Blizzard & Bulatovic, 1992; Khadilkar, Frazer, Skuse, & Stanhope, 1998).

The individual contribution and interplay of these etiological factors in the physical growth delay of institution-reared children remains underresearched. Johnson (2000) suggests that psychosocial deprivation may be a predominant cause

of the growth delay in institution-reared children. In the absence of longitudinal prospective research, one way to test this hypothesis is to study physical growth dynamics in institutions that provide adequate health and nutrition support. Besides, to exclude possible influence of ethnic differences comparisons with native family-reared children are necessary. The present study describes the course of physical development of institution-reared children in institutions with adequate health and nutrition support in comparison to native family-reared peers from their birth onwards, basing on archival data and current assessments of physical growth.

Regulation of Stress

Recent advances in the field of developmental neuroscience have opened up new avenues for examination of the impact of early unfavorable experiences on the development of the child. A growing body of research points to neurophysiological sequelae of early adversity that are related to the changes in the limbic-hypothalamic-pituitary-adrenocortical axis (LHPA) functioning (Gunnar, 2000).

LHPA is one of the stress regulation systems, with cortisol as its end product. LHPA is engaged in a range of basal metabolic as well as stress-sensitive responses in the body. Under non-stress or basal conditions production of cortisol follows a circadian rhythm and promotes the sleep-awake cycle of the body: It rises near the end of the night sleep, reaches its highest peak about 30 min after awakening, afterwards it drops throughout the day with some surges related to eating and nap, and reaches its nadir 30-60 min after the night sleep has began (Kirschbaum & Hellhammer, 1989; Watamura, Donzella, Kertes, & Gunnar, 2004).

In human infants the LHPA system is highly labile and responsive; it continues to mature throughout infancy and childhood (De Weerth, Zijl, & Buitelaar, 2003; Watamura et al., 2004). In this maturational process the caregiver plays an essential role. By helping an infant to regulate his or her affective state, the caregiver is regulating the release of neurohormones in the infant's brain. If an infant is distressed, the caregiver's tactile and emotional soothing reduces the levels of cortisol and related stress hormones, at the same time, the frontal cortex develops a greater concentration of glucocorticoid receptors that can modulate stress responses (Gunnar, 1998). When comforting interaction with a caregiver is absent or when the caregiver is abusive, neglectful or continually mis-attuned, infants may remain in chronically negative states. Such chronic negative states or chronic stress may lead to dysregulation of circadian cortisol production resulting in some individuals in an elevated pattern and in others in a flat pattern of cortisol production, which in turn may have deleterious consequences for emotional and physical development (Gunnar, 2000; Gunnar & Vazquez, 2006).

Because the institutional environment confronts a child with multiple stressors on the one hand and with highly limited or absent comforting interactions with a caregiver on the other, we may expect that such rearing circumstances will lead to a LHPA functioning dysregulation with respect to the diurnal pattern of cortisol production in children subjected to institutional care. However, the number of studies testing this hypothesis is highly limited. Carlson and Earls (1997) measured the diurnal pattern of salivary cortisol production in institutionalized Romanian children compared with that of home-reared children at 2 years of age. While home-reared children demonstrated a normal decline of cortisol production during the day with its peak in the morning, institutionalized children had relatively low wake-up levels, a slight peak at noon and an overall blunted pattern of diurnal cortisol production. Another study conducted in a Russian Baby Home with 11 children at 3 to 5 months of age produced similar results of blunted rhythms of diurnal cortisol production (Kroupina, Gunnar, & Johnson, 1997, cited in Gunnar, 2000).

Gunnar and Vazquez (2001), commenting on these studies, suggested that the altered dynamics of the normal circadian rhythm may be caused by the neglectful institutional environment and repeated daily intermittent stress. However, it is also possible that this alteration is related to the child's characteristics, such as prenatal substance exposure, perinatal complications, or untoward health condition which are often observed in institution-reared children (e.g., Johnson et al., 1992, 1996; Judge, 2003; Miller, 2005), and were also found to be related to LHPA functioning (e.g., Cianfarani, Geremia, Scott, & Germani, 2002; Hng, Cheung, & McLean, 2005; Zhang, Sliwowska, & Weinberg, 2005). Besides, stunted growth caused by perinatal complications, undernourishment or psychosocial adversities appears to be related to altered LHPA functioning (Fernald & Grantham-McGregor, 1998; Fernald & Grantham-McGregor, 2002; Fernald, Grantham-McGregor, Costello, & Manadhar, 2003; Vazquez, Watson, & Lopez, 2000, cited in Gunnar &Vazquez, 2001).

Thus, in order to examine the influence of institutional rearing on the LHPA functioning of the child it is not sufficient to have a comparison group of native family-reared children, but we also have to take into consideration the background characteristics of the children that might influence cortisol production.

Hypotheses

In the present study we examined whether children reared in institutional care that provides adequate nutrition and health support, showed delays in their physical development and dysregulation of their LHPA functioning as compared to native family-reared peers. We hypothesize that even in the presence of adequate nutrition and health provision institution-reared children show physical growth delays especially evident in height and head circumference (cf. Van IJzendoorn et

al., 2007). We also hypothesize that institutional rearing leads to dysregulation of LHPA functioning with respect to the diurnal pattern of cortisol production and the overall daily production of cortisol (e.g., Carlson & Earls, 1997). Finally, we expect that stunted children are different from non-stunted children in that they show an altered pattern of diurnal cortisol production (e.g., Fernald et al., 2003).

Method

Participants
Participants were 16 institution-reared children and 19 family-reared children living with their biological parents, matched for gender and age.

Institution-reared children. Institution-reared children were recruited from four Children's Homes located in Odessa and Belgorod-Dnestrovsky, Ukraine. The following selection criteria were applied: a) age between 3 and 6 years old; b) admission to institutional care within the first 6 months of age; c) no genetic syndromes (e.g., Down syndrome); d) no evidence of fetal alcohol syndrome in the medical records; e) no HIV infection; f) permanent residence in residential care institutions since admission. Eighteen children were selected, but examination of the case records of these children revealed that 16 of them were admitted to institutional care within the first three months of life. Two other children, although left without parental care within the first six months after the birth, were initially cared for by relatives and admitted to institutional care at 37 and 55 months respectively. These two children were not included in our sample.

The data on the history of institutionalization show that only one child in the institution-reared group was an orphan, whereas the rest were admitted to institutional care because of poverty (n = 9), family disruption (n = 2), or because one or both parents were in prison (n = 4). All mothers of the institution-reared children were abusing alcohol or drugs. Although almost all institution-reared children had parents and/or relatives, only one child remained in contact with his birth family on a regular basis, 6 had sporadic contacts, and 9 children had no contacts with their parents or family members. Two children were living in the same institution with their siblings who did not participate in this study. Since admission to institutional care 8 children remained in the same institution, whereas 7 children had been transferred to another institution once and 1 child had been transferred twice. Three children were born in prison and immediately upon their birth admitted to a prison orphanage where they spent on average 38.01 months (SD = 3.35; range: 35.44 – 41.80); afterwards they were transferred to a regular orphanage. We tested whether this sub-group of children of incarcerated mothers differed from the other institution-reared children on all outcome measures, but

no significant differences emerged (.08 < p < 1.00). Since admission to their current institution, all children had experienced a change of primary caregivers, with 56% having experienced more than three changes. On average, children had been living in institutional care for 47.14 months (*SD* = 9.50; *range* 35.11 – 64.73).

Family-reared children. For the comparison group, family-reared children were recruited in the same geographical area as the Children's Homes from kindergartens, schools and clinics where routine health checks take place. Children were selected according to the following criteria: a) age between 3 and 6 years old; b) living in two-parent biological families; c) no genetic syndromes (e.g., Down syndrome); d) no fetal alcohol syndrome; e) no HIV infection; f) no previous history of institutionalization, hospitalization or prolonged separation (more than 2 weeks) from a primary caregiver.

Background characteristics inspection. Each child from the comparison group was pair-matched on age and gender with a child from institutional care. Mean age of institution-reared children was 48.14 months (*SD* = 9.72; *range* 35.11 - 66.73), and mean age of family-reared children was 51.44 months (*SD* = 9.80; *range* 37.48 - 67.06). There were 8 boys in the institutional care group and 9 in the comparison group.

At the time of assessment there were 5 chronically stunted children in the institution-reared group (four of latest assessment at 48 months, one at 36 months), i.e., from their first birthday onwards they had height-for-age z-scores below -2 SD of the reference population (World Health Organization (WHO), 1995) on all time points. There were no chronically stunted children in the family-reared group. Temporarily stunted children at some point achieved height scores below -2 SD of the reference population, but not persistently so.

Further sample inspection revealed that all chronically stunted children had perinatal hypoxic neurological conditions (PHNC), whereas only one child in the temporarily stunted institution-reared group had PHNC. There were no cases of PHNC in the family-reared group (see Table 2). Although by the time of the assessment all institution-reared children had been declared recovered and healthy by the institutional paediatricians, we decided to set apart the group of chronically stunted children in our further analysis because of their perinatal conditions and unfavorable growth development.

Results of univariate ANOVAs and chi-square test on available demographical data, presented in Table 2, showed no significant differences between the family-reared group and temporarily or chronically stunted institution-reared groups on age of biological mother, child gender, or child age. However, the biological mothers of all institution-reared children were current substance users, while none of the comparison group mothers were.

Table 2
Descriptive statistics for family-reared vs. institution-reared children

	Family-reared children		Institution-reared children					
			Temporarily stunted		Chronically stunted		Total[1]	
	n	*M (SD)*	*n*	*M (SD)*	*n*	*M (SD)*	*n*	*M (SD)*
Parental characteristics								
Age of mother in years	17	32.12 (5.93)	8	28.00 (8.14)	3	36.00 (8.89)	11	30.18 (8.73)
Mothers' substance use	18	0[a]	10	10[b]	3	3[b]	13	13
Child characteristics								
Gender (male)	19	9	11	5	5	3	16	8
Age in months	19	51.44 (9.80)	11	45.12 (7.80)	5	54.78 (11.05)	16	48.14 (9.72)
Prenatal substance exposure								
Drugs	19	0[a]	3	2[b]	2	1[b]	5	3
Alcohol	19	0[a]	5	4[b]	3	3[b]	8	7
Tobacco	19	1[a]	5	5[b]	2	2[b]	7	7
Child condition at birth								
Perinatal hypoxic conditions	19	0[a]	11	1[a]	5	5[b]	16	6
Low birth weight (< 2.5 kg)	17	3	11	0	4	2	15	2
Child medical condition in infancy and early childhood								
Total morbidity score	18	0.02[a] (0.02)	11	0.08[b] (0.05)	5	0.05[b] (0.03)	16	0.07 (0.04)
Medication intake on the day of saliva sampling	19	5	11	1	5	0	16	1
Cortisol								
Diurnal cortisol production[2]	16	0.45[a] (0.17)	11	0.63[b] (0.15)	5	0.40[a] (0.03)	16	--

Note: Means in the same row that do not share superscripts differ at $p < .05$.
[1] No statistical comparisons were made with the total institution-reared group.
[2] Diurnal production of cortisol computed with AUCg formula.

To examine possible differences in the medical background between temporarily and chronically stunted institution-reared children and family-reared children we conducted a series of chi-square tests and univariate ANOVAs with respect to child condition at birth and medical condition in infancy and early childhood. Results presented in Table 2 show that a higher number of both temporarily and chronically stunted institution-reared children suffered from prenatal substance exposure.

No significant difference was found between both institution-reared groups and the family-reared group on the number of children with low birth weight (less than 2.5 kg), (temporarily stunted versus family-reared: $\chi^2 = 2.17$, $p = .26$, chronically stunted versus family-reared: $\chi^2 = 0.64$, $p = .56$). In infancy and early childhood both groups of institution-reared children suffered more often from various diseases compared to family-reared children which was reflected by their higher total morbidity score, $F(2, 31) = 12.79$, $p < .01$ (see Table 2).

Procedure

For all children enrolled in the study, informed consent was obtained: for the children in the Children's Homes from the local department of the Ministry of Health, and for the children in the family-reared group from their biological parents. All children were invited for a laboratory assessment procedure. Institution-reared children were accompanied by their "favorite" caregiver, as determined through preliminary informal interviews with children and caregivers. If a favorite caregiver was difficult to identify, the person who spent most of the time with a child and knew him or her best was invited. Family-reared children were accompanied by their primary caregiver who was also the biological parent.

Laboratory assessment. During the laboratory assessment procedure the children underwent a physical examination (height, weight, and head circumference) and were administered some other tests that will be reported on elsewhere.

Measures

Medical background. A Medical Background Checklist composed for this study was used to collect information about the health of the children. The checklist concerned the children's prenatal risks (prenatal exposure to substances), as well as health condition and medical history at birth, during infancy and during early childhood. Institutional pediatricians were asked to fill out the Medical Background Checklist, basing their answers on the children's medical records. In

case of family-reared children, parents were asked to obtain the medical records from the pediatric clinics and fill out the Medical Background Checklist in consultation with their pediatricians, when possible.

On the basis of these reports a total morbidity score was calculated. Total morbidity score (TMS) was defined as the total number of diseases requiring medical intervention that the child had experienced during infancy and early childhood until the day of assessment. In the total morbidity score we did not include conditions such as light forms of upper respiratory tract infections or common childhood diseases, like chickenpox, measles and mumps.

To control for age differences among the children, TMS was obtained by dividing the number of reported diseases by the current age of a child in months.

Physical growth. Data on physical growth through the course of the child's development were collected on the basis of the children's medical records. Data on weight, height, and head circumference were obtained for the following ages: birth, 3, 6, 9, 12, 24, 36, and 48 months, depending on the child's current age. Not all medical records were complete and different children had missing data at different time points (see Table 3). Current height, weight and head circumference of all children was measured during the laboratory visit. Anthropometric indices (weight-for-age = WAZ, height-for-age = HAZ, and head circumference-for-age = CAZ) were calculated with the software program, Epi Info™, Version 3.3.2 using the sex specific 2000 CDC reference database (Dean et al., 2002). Epi Info™ calculates HAZ scores for children up to 36 months, however we did not have sufficient data on HAZ between 12 and 36 months for the family-reared children to make group comparisons. Two other growth indices were calculated from birth until the day of assessment.

Diurnal salivary cortisol sampling. To study diurnal cortisol on a typical day a six-sample protocol was followed: 1) awakening, 2) 45 minutes after awakening, 3) 2.5 hours after awakening, 4) 8 hours after awakening, 5) 12 hours after awakening, and 6) bedtime. Saliva samples were collected from the institution-reared children by an institutional nurse and from the family-reared children by their parent. The saliva collection procedure was explained and demonstrated to the parents and institutional nurses and they received the saliva-sampling kits with written instructions for the sampling. Parents and nurses were asked to select a day when children did not attend day-care or school and when nothing unusual, exciting or particularly stressful was scheduled. They were informed that children were not allowed to eat, brush their teeth, or drink liquids (juice or milk) before taking a sample. No stimulation of saliva flow was employed in the sampling procedure. After rinsing the mouth with plain water participants took a roll of cotton into the mouth, chewed on it for approximately 30 seconds or until it became saturated,

and placed it in a salivette with a corresponding label including the time of the sampling. Saliva samples were frozen immediately upon the sampling until they were collected by the research assistant. Nurses and parents registered the exact time of sampling and provided data on activities and experiences that might influence the child's cortisol production during the day of sampling, including time of awakening, stressful daily events, food and medications intake, the child's mood and health condition. The records were screened for intake of psychotropic or corticosteroid medications and for being in a poor health condition at the day of saliva sampling, as both circumstances can potential alter the salivary cortisol production. There were no children who took psychotropic or corticosteroid medications. However, one comparison group child had become ill at the day of saliva sampling and was excluded from the analyses involving diurnal cortisol.

Assay procedure for cortisol. In order to determine the cortisol concentration in the saliva sample we used a time-resolved fluorescence immunoassay. The saliva samples were stored at -20 °C until analysis. After thawing, saliva samples were centrifuged at 2000 g for 10 minutes, which resulted in a clear supernatant of low viscosity. 100 ul of saliva were used for duplicate analysis. Cortisol levels were determined employing a competitive solid phase time-resolved fluorescence immunoassay with fluorometric end point detection (DELFIA). 96-well-Maxisorb microtiterplates (Nunc) were coated with rabbit-anti-ovine immunoglobulin. After an incubation period of 48 h at 4° C, plates were washed three times with washbuffer (pH = 7,4; containing sodium phosphate and the Tween-40). In the next step the plates were coated with an ovine anti-cortisol antibody and incubated for 48 h at 4° C. Synthetic saliva mixed with cortisol in a range from 0 - 100 nmol/l served standards. Standards, controls (saliva pools) and samples were given in duplicate wells. 50 µl of biotin-conjugated cortisol was added and after 30 minutes of incubation the non-binding cortisol/biotin-conjugated cortisol was removed by washing (3x). 200 µl europium-streptavidin (Wallac, Turku, Finland) was added to each well and after 30 minutes and 6 times of washing 200 µl enhancement solution was added (Pharmacia, Freiburg, Germany). Within 15 minutes on a shaker the enhancement solution induced the fluorescence which can be detected with a DELFIA-Fluorometer (Wallac, Turku, Finland). With a computer-controlled program a standard curve was generated and the cortisol concentration of the samples was calculated. The intra-assay coefficient of variation was between 4.0% and 6.7%, and the corresponding inter-assay coefficients of variation were between 7.1% - 9.0%.

A preliminary examination of the obtained cortisol values demonstrated that the distribution of the diurnal cortisol scores was positively skewed. Therefore, diurnal cortisol scores were log 10 transformed prior to analyses (Azar et al., 2004; Oosterlaan et al., 2005). Due to the low concentration of saliva within the cotton

swabs, 6 out of the 19 family-reared children had missing data: 1 child at all six time points; 1 child at awakening and 45 minutes after awakening; and 4 children either at awakening, 12 hours after awakening or before going to bed. Log curve estimation analyses, using individual sampling times as the independent variable, were undertaken to generate missing cortisol values for all except the one child who had missing data at all six time points.

In order to assess the overall production of cortisol from awakening until bed time the computation of the 'Area under the curve with respect to ground' (AUCg) derived from the trapezoid formula was employed (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). Since the AUCg was related to the total time that the children were awake (from awakening till bed time) and the institution-reared children were awake somewhat longer we corrected the AUCg for children's total time of being awake.

Results

Preliminary Analyses

Preliminary analyses were performed to examine whether child characteristics, such as gender, age, and low birth weight (less than 2500 g), as well as morbidity during infancy and early childhood should be included as control variables in the analyses of physical growth and diurnal cortisol production. Gender, age, and low birth weight were not associated with any of the outcome variables. Univariate ANOVA on the total morbidity score with group membership (family-reared children, temporarily stunted institution-reared children, and chronically stunted institution-reared children) as an independent variable revealed that both temporarily and chronically stunted institution-reared children suffered more often from various diseases and had higher total morbidity score compared to the family-reared children, $F(2, 31) = 12.79$, $p < .01$ (see Table 1). No significant difference was found between the temporarily and chronically stunted institution-reared children. Correlation analyses of the total morbidity score with the outcome variables revealed that morbidity during infancy and early childhood was not related to physical growth, however, higher morbidity score was associated with higher diurnal cortisol production, $r = .35$, $p = .05$.

We examined whether mood, not feeling well (excluding one more seriously ill case) or medication intake on the day of saliva sampling were related to the child's overall diurnal production of cortisol. No significant relation was found between the mood of the child or not feeling well on the day of saliva sampling and overall diurnal production of cortisol; but medication intake on the day of saliva sampling was related to decreased overall diurnal production of cortisol, $t(31) = 2.22$, $p = .03$.

Table 3

Means and standard deviations for physical growth variables as a function of rearing environment and stunting

	Family-reared children				Institution-reared children								
					Temporarily stunted				Chronically stunted				
	n	*M*	*SD*	%< -2SD[1]	*n*	*M*	*SD*	%< -2SD[1]	*n*	*M*	*SD*	%< -2SD[1]	F
Height-for-age z score													
Birth	17	0.63	1.10	6	10	0.70	0.75	0	4	-0.23	0.27	0	1.57
3 months	12	0.33	0.78	0	7	-1.30	1.15	43	2	-2.13	2.52	50	7.73*
6 months	12	0.55	0.76	0	8	-1.79	0.80	25	2	-2.21	0.78	50	26.78*
9 months	12	0.66	0.73	0	7	-1.49	0.70	43	2	-1.92	1.24	50	22.94*
12 months	11	0.74	0.65	0	9	-1.71	0.95	33	3	-3.04	0.67	100	39.16*
24 months	4	0.59	1.08	0	7	-2.09	1.16	71	3	-3.01	0.25	100	12.61*
36 months	8	0.87	1.20	0	10	-1.50	1.50	30	3	-3.45	0.60	100	13.97*
48 months	9	0.61	0.88	0	7	-0.75	0.68	0	4	-2.29	0.65	100	20.27*
Weight-for-age z-score													
Birth	17	-0.33	0.96	0	11	-0.58	0.48	0	4	-1.42	0.50	0	3.15
3 months	11	0.22	1.11	0	7	-1.44	0.54	14	2	-2.71	0.62	100	12.43*
6 months	11	0.38	0.88	0	8	-1.55	0.61	25	2	-2.52	0.62	100	21.12*
9 months	11	0.42	0.60	0	7	-2.18	0.84	57	2	-3.16	0.43	100	43.52*
12 months	12	0.61	1.04	0	9	-1.88	0.84	44	3	-3.48	0.60	100	32.13*
24 months	6	0.64	0.56	0	7	-2.15	1.68	57	3	-3.60	1.34	100	12.71*
36 months	8	0.62	0.94	0	10	-0.56	1.00	10	3	-1.98	1.53	33	7.17*
48 months	9	0.24	0.88	0	7	0.07	0.89	0	4	-1.79	1.51	25	5.79*
Head circumference-for-age z -score													
Birth	8	-0.07	1.53	14	6	-0.55	0.60	0	2	-1.58	0.36	0	1.33
3 months	9	-0.68	1.20	25	7	-2.10	1.36	71	2	-3.68	2.36	100	4.83
6 months	9	-0.05	1.34	13	8	-1.91	1.43	38	2	-4.61	2.66	100	8.71*
9 months	8	0.14	1.37	13	7	-1.51	1.16	29	2	-4.52	1.02	100	11.54*
12 months	8	0.70	0.55		7	-0.76	0.91	14	3	-3.12	1.39	100	22.32*

Note. [1]Percentage of children with anthropometric values of less than 2 SD from the reference population. *p-level remaining significant after adjusting for multiple comparisons (for weight and height: $p \leq .0063$; for head circumference: $p \leq .01$).

Thus, in the analysis on diurnal cortisol production we controlled for morbidity and medication intake on the day of saliva sampling.

Physical Growth

To examine children's physical growth across infancy and early childhood as a function of rearing environment and stunting, we conducted a series of ANOVAs comparing family-reared children, temporarily and chronically stunted institution-reared children at different time points from birth to 48 months of age for weight and height measures, and from birth to 12 months of age for head circumference measures. Because of the varying numbers of missing data on growth at the various times of assessments, we were not able to conduct a repeated measures analysis of variance. As multiple comparisons were performed, probability values were Bonferroni adjusted (within each growth parameter) to prevent Type I errors. Figures 1, 2, and 3 illustrate changes in weight, height and head circumference (with standard errors) of the three groups across infancy and early childhood. In Table 3 the means and standard deviations of the growth assessments, the ANOVA results, and a priori contrasts between the family-reared children and temporarily and chronically stunted institution-reared children are presented.

Height. There was no significant group difference in children's supine length at birth (see Table 3). As Figure 1 shows, the family-reared children demonstrated a normal pattern of growth in comparison to the reference population from birth through 48 months of age, whereas the growth of both temporarily and chronically stunted institution-reared children deviated markedly from the family-reared children and the reference population.

Most temporarily stunted children demonstrated stunted growth at some point in their life (see Table 3). At 3 months of age the supine lengths of the temporarily and chronically stunted institution-reared children were significantly lower than the supine length of the family-reared children (see Table 3). The height faltering in the temporarily stunted institution-reared children persisted and reached its peak at 24 months of age, when they were over 3 SD behind the family-reared group and over 2 SD behind the reference population. From 24 months through 48 months of age a relative improvement of growth could be observed and by 48 months temporarily stunted institution-reared children lagged 1.63 SD behind the family-reared group and 0.75 SD behind the reference population.

The height faltering in chronically stunted institution-reared children continued throughout infancy and early childhood and became most pronounced between 12 and 36 months, when they lagged more than 3 SD behind the family-reared group and the reference population. From 36 months to 48 months the gap between the family-reared and the chronically stunted institution-reared children had decreased by a little more than 1 SD and by 48 months the chronically stunted

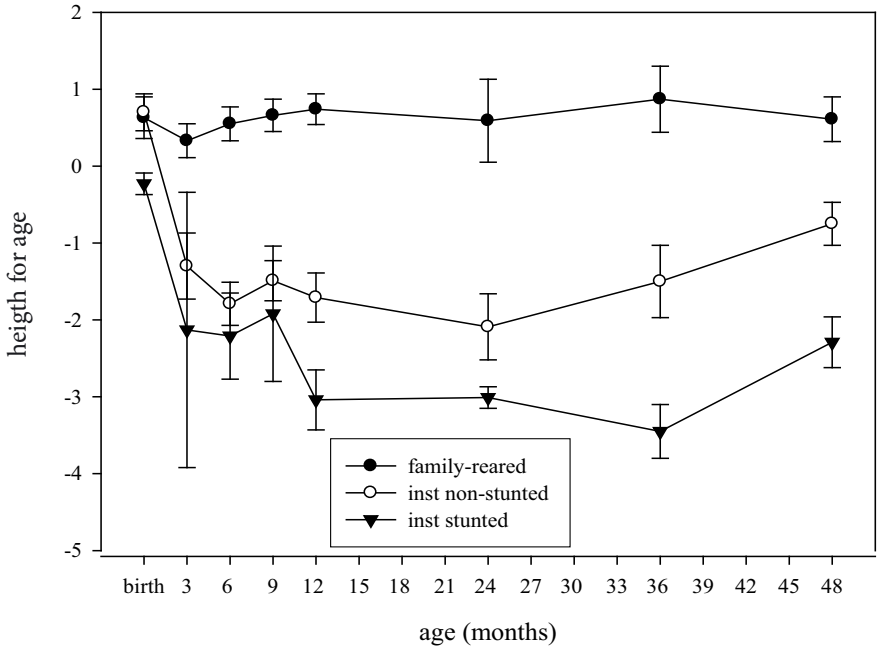

Figure 1. Height-for-age of institution- and family-reared children from 0 to 48 months

institution-reared children were lagging over 3 SD behind the family-reared group and over 2 SD behind the reference population (see Table 3).

Weight. There were no significant group differences in children's weight at birth. However, as Figure 2 shows, chronically stunted institution-reared children were slightly lighter than temporarily stunted institution-reared and family-reared children.

While family-reared children showed a normal weight gain pattern in comparison to the reference population from birth through 48 months of age, the faltering of weight gain in institution-reared children became apparent already at 3 months of age, when both groups of institution-reared children weighed significantly less than the family-reared children (see Table 3 and Figure 2), and became most pronounced between 9 and 24 months, when the temporarily stunted institution-reared group lagged more than 2 SD behind the reference population and more than 2.5 SD behind the family-reared group (see Table 3).

The chronically stunted institution-reared group lagged near 4 SD behind the family-reared group and more than 3 SD behind the reference population, and over 1 SD behind the temporarily stunted institution-reared group. From 24 months of age the difference between the temporarily stunted institution-reared

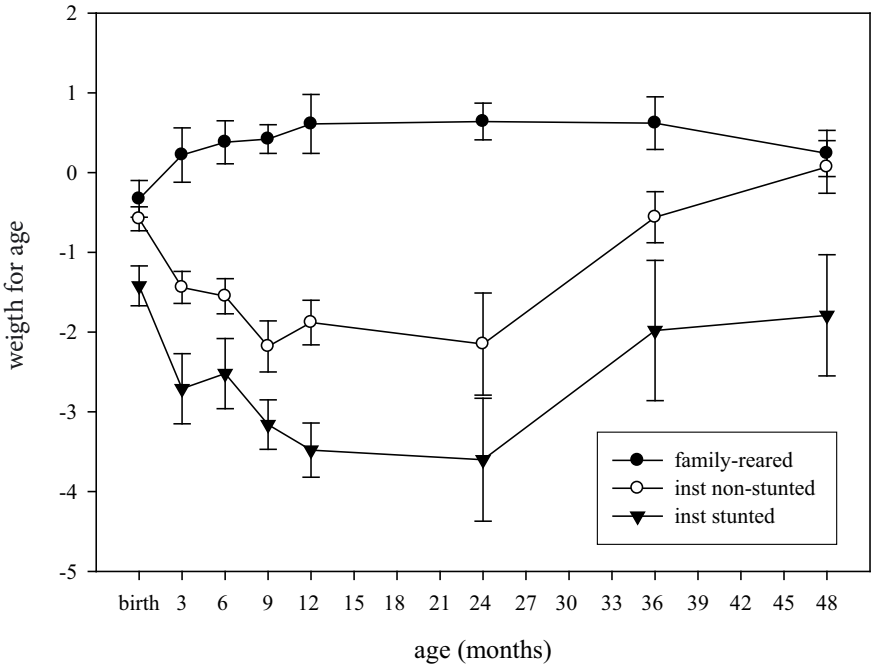

Figure 2. Weight-for-age of institution- and family-reared children from 0-48 months

group and the family-reared group began to level off and by 48 months of age catch-up in weight could be observed in the temporarily stunted institution-reared group, when they reached the normal weight range. The WAZ scores of the chronically stunted institution-reared children at 48 months as compared to both family-reared and temporarily stunted institution-reared children remained significantly lower (see Table 3 and Figure 2).

Head circumference. There was no significant group difference in children's head circumference at birth; however, as Figure 3 demonstrates, the chronically stunted institution-reared children had the lowest CAZ scores among the three groups at birth. From birth until 12 months of age the head circumference growth of the family-reared children remained within the normal range as compared to the reference population. In contrast, as Figure 3 shows, both the currently temporarily and the chronically stunted institution-reared children showed marked retardation in their head circumference growth from birth to 3 months, lagging over 2 SD and over 3 SD, respectively, behind the family-reared group and the reference population at 3 months of age (see Table 3). Whereas in the temporarily stunted institution-reared children there was a tendency for improvement of the head circumference growth from - 2.10 SD at 3 months to -0.76 SD at 12

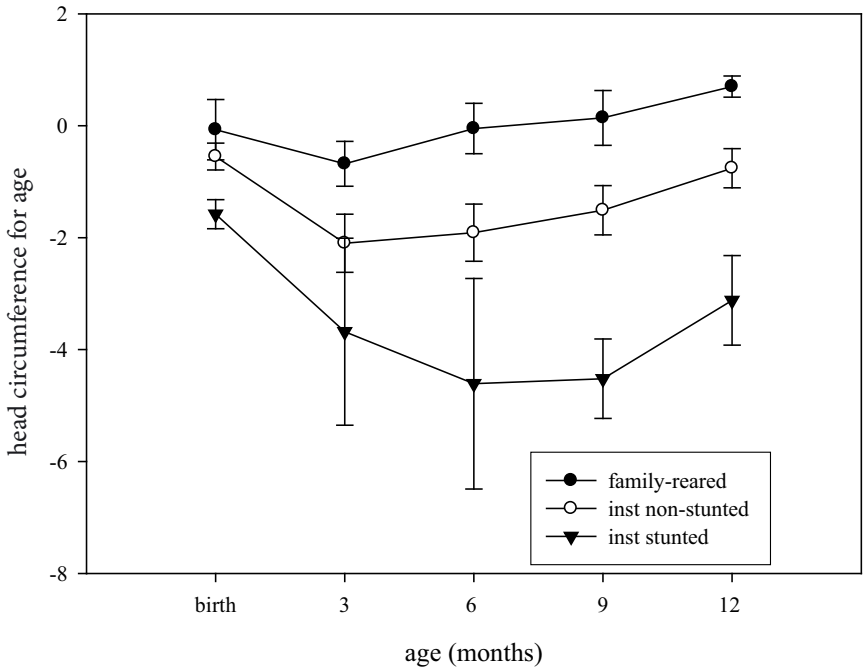

Figure 3. Head circumference-for-age of institution- and family-reared children from 0-12 months

months, the chronically stunted institution-reared group demonstrated sustained severe retardation of head circumference growth up till 9 months of age, when they lagged more than 4 SD behind the family-reared group and the reference population. There was improvement in head circumference growth at 12 months when the chronically stunted institution-reared children decreased the gap with the family reared-children and the reference population with more than 1 SD (see Table 3). Epi Info™ calculates HAZ scores for children up to 36 months; however we did not have sufficient data on HAZ between 12 and 36 months for the family-reared children to make group comparisons.

Diurnal Cortisol Production

To examine diurnal cortisol production (AUCg) as a function of rearing environment and stunting, we conducted a 3 Group X 6 Times repeated measures ANCOVA with groups of children (family-reared, temporarily stunted institution-reared, chronically stunted institution-reared) as the between-subjects factor and the time of sampling (awakening, 45 minutes after awakening, 2.5, 8, and 12 hours after awakening, and bed time) as the within-subjects factor. Child total morbidity score and medication intake on the day of saliva sampling were included as covariates. Results revealed a significant main effect of time, F (5, 135) = 36.48,

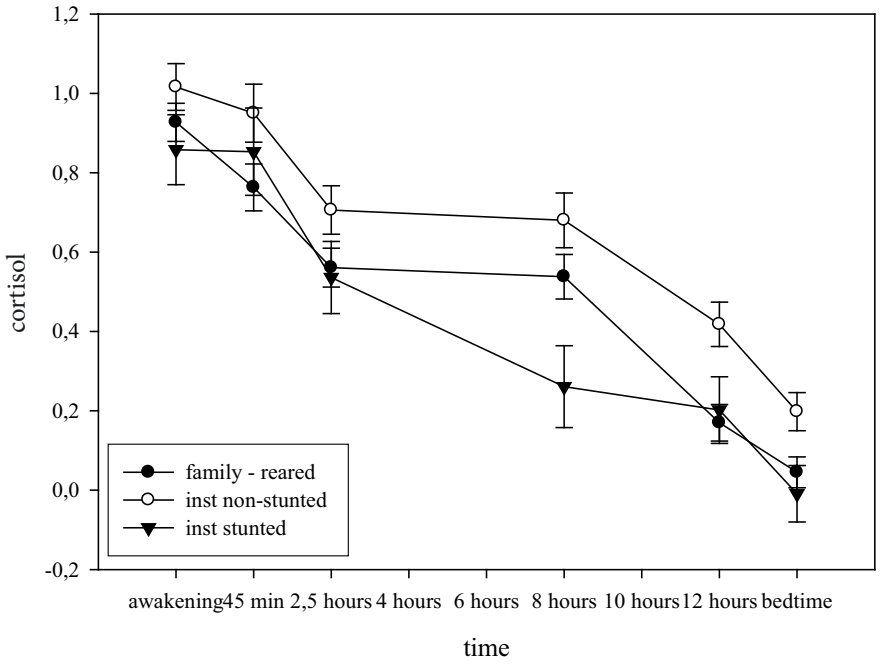

Figure 4. Diurnal cortisol values of institution- and family-reared children

$p < .01$, partial $\eta^2 = .58$, indicating a decrease in diurnal cortisol over the day for the sample as a whole. Results also revealed a significant main effect of group, F (2, 27) = 5.28, $p < .01$, partial $\eta^2 = .28$, indicating that daily average cortisol was higher for temporarily stunted institution-reared children than that of chronically stunted institution-reared children and family-reared children. No significant Group X Time effect was found, F (10, 135) = 1.21, $p = .31$, partial $\eta^2 = .08$, indicating similar decreases over the day for the three groups. Diurnal cortisol values for the three groups are graphically presented in Figure 4.

In order to assess whether the overall diurnal production of cortisol (from awakening until bed time) was related to rearing environment and stunting we conducted a one-way ANCOVA on the overall diurnal production of cortisol with group membership (family-reared children, temporarily stunted institution-reared children and chronically stunted institution-reared children) as an independent variable and total morbidity score and medication intake on the day of saliva sampling as covariates. A significant effect of the group membership was found, F (2, 27) = 5.15, $p = .01$, partial $\eta^2 = .28$. The overall diurnal cortisol production of institution-reared children was higher than in the family-reared group but only for the temporarily stunted institution-reared group ($p = .03$, see Table 2). No significant difference was found in the overall diurnal cortisol production between the family-reared group and chronically stunted institution-reared group.

Discussion

The current study provided a unique opportunity to examine physical growth and stress regulation of children in institutional care. Because the institutions in our study were characterized by the second level of institutional privation according to Gunnar's (2001) classification, providing adequate nutrition and health care, we were able to examine the influence of stimulation and relationship privation on institution-reared children's physical development and stress regulation. The contribution of various child characteristics to physical development and stress regulation were also examined. Finally, comparison with native family-reared peers allowed controlling for possible ethnic differences in developmental outcomes. We found severe delays among institution-reared children in physical growth, especially during the first two years of life. Afterwards, a tendency for improvement in physical growth was evident among most institution-reared children with complete catch-up in weight and partial catch-up in height by 48 months of age. Chronically stunted institution-reared children demonstrated persistent severe growth delays from the first months of their life onwards. Institution-reared and family-reared children showed similar patterns of diurnal cortisol production. However, temporarily stunted institution-reared children had a significantly higher total daily cortisol production than both chronically stunted institution-reared children and family-reared children.

Physical Growth

Archival data showed that there was no significant difference between institution-reared and family-reared children at birth with respect to height, weight and head circumference, but substantial delays in these growth domains were already evident at three months of age. Even in the presence of adequate nutrition and health provision and controlling for the child's morbidity, institution-reared children showed substantial delays in physical growth. Examination of current measurements of physical growth revealed that in our sample about one third of the institution-reared children were stunted. These findings support the hypothesis that stimulation and relationship privation is a predominant cause of physical growth delay in institution-reared children.

After the second birthday a tendency for improvement in physical growth emerged among the institution-reared children, resulting in the group of temporarily stunted children in complete catch-up in weight and partial catch-up in height by 48 months of age. Chronically stunted institution-reared children suffered from more severe delays which persisted from the first months of their life onwards. These findings raise two additional questions: What triggers growth improvement in children who remain in a presumably unchanged caregiving environment? Why do many institution-reared children show temporary (severe)

delays in growth but improve after their second birthday, whereas chronically stunted children show persistent severe delays in physical growth?

Catch-up or improvement in physical growth is usually associated with the removal of growth-inhibiting conditions (Boersma & Witt, 1997; Gafni & Baron, 2000). However, institution-reared children in our sample were born at different times and reared from the first months of their life onwards in the institutions; still all of them showed a tendency for improvement at about the same age. This may point to the emergence of certain protective factors allowing a child to cope with the growth inhibiting condition. The fact that a similar tendency is also observed in stunted malnourished family-reared children who after pronounced faltering of linear growth in the first two years of life seem to experience some catch-up around 40 months (Grantham-McGregor et al., 2007) points to the child rather than its environment in the search for such protective mechanisms.

Observed improvement in physical growth may be related to increasing capacity for adaptation of the somewhat older child. In fact, the younger the child is, the greater the risks for growth retardation are. The growth velocities during the first year of life are the highest. At the same time, this is the period when children are totally dependent on others for their care and, therefore, most vulnerable to poor caregiving. The older the child is, the broader the nutritional and behavioral repertoires are, the easier it may be to make its needs known, and procure more food in case of malnourished children and attention and stimulation in case of institution-reared children. Older children may more actively shape their environment (and this environment may be somewhat more stimulating when they are moved to more "educational" institutions in Ukraine at 36 months) and take care of their own needs for food or stimulation. Complete catch-up in weight in temporarily stunted institution-reared children supports this explanation; it also confirms that weight gain is more easily subject to improvement. The incomplete catch-up in height, on the other hand, may be explained by the presence of a critical period of bone growth in the first years of life which, unlike weight gain, when compromised may result in permanent alteration of the growth trajectory (Cooper et al., 1997), and/or influence of ongoing adversities of institutional rearing for which height appears to be more susceptible than weight.

But why did chronically stunted institution-reared children not improve in the same fashion? Figures 1, 2, and 3 show that chronically and temporarily stunted institution-reared children's growth trajectories are remarkably parallel for all three physical growth parameters. This may indicate that the same mechanisms govern growth in both groups, however, the somewhat less favorable start of the chronically stunted institution-reared children at birth reflected by all three growth measures suggests the presence of a certain risk factor emerging before or at birth, which may exacerbate the influence of institutional rearing on physical growth. All the mothers of institution-reared children were abusing alcohol. Although we

did not include children with fetal alcohol syndrome in our sample, we can not rule out that chronically stunted children may have suffered from this condition, which, as research reveals, is often associated with physical growth failure (e.g., Miller et al., 2006). The fact that all chronically stunted institution-reared children had perinatal hypoxic conditions seems to be an alternative explanation. This assumption is supported by other studies, pointing to the negative effect of perinatal hypoxic conditions on the subsequent development of the child (e.g., Ellis et al., 2001; Hankins & Speer, 2003; Maslova et al., 2003). In our sample, 5 out of 6 institution-reared children with perinatal hypoxic conditions showed severe delays in physical growth, suggesting that either the condition itself, the way it was treated, or a combination of both may be a serious risk factor. Importantly, perinatal hypoxic conditions are not unique to our sample. Other authors also report that neurological diagnoses including perinatal hypoxic conditions are widely spread among international adoptees formerly reared in institutions (Albers et al., 1997, Landgren et al., 2006, Pomerleau et al., 2005). Miller (2005) reports that nearly 50% of medical records of children adopted from Eastern Europe contain the diagnosis of perinatal encephalopathy. Therefore, further examination of this group is important.

Stress Regulation

Temporarily stunted institution-reared children had significantly higher total daily cortisol production compared to family-reared children, which, as we hypothesized, may reflect the dysregulation of the LHPA functioning caused by a stressful institutional environment and limited or absent comforting interactions with a caregiver. Surprisingly, no difference in total daily cortisol production was found between chronically stunted institution-reared children and family-reared children coming from hardly comparable rearing environments. On the other hand, chronically stunted and temporarily stunted institution-reared children differed significantly on the total daily cortisol production, whereas they lived in the same institutional environment. These findings point to certain factors related to the child rather than the rearing environment. From the medical records of institution-reared children we know that all chronically stunted children who also suffered from perinatal hypoxic conditions underwent a treatment to stabilize the functioning of the nervous system. Depending on the condition, such treatment among other medications and procedures may also involve the use of diazepam as well as corticosteroids, such as hydrocortisone and prednisone (Edelstein, Bondarenko, & Bykova, n.d.), which could have lasting effect on the LHPA functioning of these children.

Other than we expected, no differences in the diurnal pattern of cortisol production between chronically stunted and temporarily stunted institution-reared children and family-reared children were found: All groups demonstrated

a normal pattern with elevated morning cortisol values and subsequent decline during the day. We did not confirm the findings of Carlson and Earls (1997) and Kroupina and colleagues (1997), who reported a marked difference in the diurnal pattern of cortisol production between institution-reared and family-reared children. The discrepancy may be explained by the fact that Carlson and Earls (1997) did not differentiate between potential chronically and temporarily stunted children in their study. Besides, the children in their sample were two years younger than the children in the present study. Because diurnal pattern of cortisol production in early childhood is related to age (e.g., Watamura et al., 2004), it is possible that the difference in diurnal cortisol patterns can be explained by the age difference between the children in the two studies. Moreover, the children in their sample were at the peak of the period (24 mos) when significant growth delays were found in the present sample, which may have resulted in divergent patterns of cortisol production.

Limitations

The current study is limited in several aspects. Due to scarce information on perinatal experiences of institution-reared children we cannot disentangle the influence of prematurity and physical condition at birth from the influence of institutional care on physical development and stress regulation in this sample. However, there were no significant differences between the institution-reared and family-reared children on their anthropometric parameters at birth; besides the children with perinatal hypoxic conditions were set apart in the analysis. Therefore we may assume that the physical condition of the temporarily stunted institution-reared children and family-reared children was not much different at birth. In our sample of institution-reared children prenatal substance exposure was reported in all cases when information was available, whereas, as far as we know, none of the family reared-children was exposed to substances during prenatal development. Unfortunately, we did not have sufficient information to examine the influence of prenatal substance exposure on physical development and stress regulation of institution-reared children. However, exclusion of children with fetal alcohol syndrome from our sample allowed ruling out the more severe cases. As there were no cases of perinatal hypoxic conditions among family-reared children, we could not disentangle the role of this set of conditions from the role of institutional care in the persistent growth delays of the chronically stunted institution-reared group. Furthermore, the institutions where we conducted our study were evaluated as adequate in terms of nutrition provision; however, we may not exclude that nutritional needs of the children were still compromised if not in quantity, then in quality, and did not provide children with the necessary range of nutrients required for normal development, especially in the first year of life, when children are dependent primarily on breast-milk substitutes. As to the cortisol measurement,

obviously, single day assessment that was employed in our study may be affected by day-to-day variations. However, we controlled for possible activities and experiences that could influence the child's cortisol production during the day of sampling, including time of awakening, stressful daily events, medications intake, and the child's mood and health condition. Besides, we used a six sample protocol to obtain a more accurate picture of the area under the curve. Concerning the diurnal pattern, the lack of an awakening response observed in the family-reared children may be explained by a poor adherence to the protocol. Although parents were explicitly asked to register any deviations from the protocol and only in three cases delays around the awakening time ranging from 5 to 20 minutes were reported, more deviations from protocol might have occurred. Finally, our small sample size reduced the power of the statistical analysis. At the same time it should be noted that data had to be collected in rather difficult circumstances.

Further research is needed to test our findings related to the growth trajectory of institution-reared children and the relation between LHPA functioning and stunted growth. As children are not admitted to institutional care at random and often suffer from various disadvantageous conditions including poor physical health, we need to extend our understanding of the contribution of the individual child characteristics to the developmental outcomes and their interplay with different aspects of the rearing environment. Children appear to be differentially susceptible to adverse rearing experiences (Belsky, 2005; Belsky, Hsieh, & Crnic, 1998), and genetic differences may play a part in this respect (e.g., Bakermans-Kranenburg & Van IJzendoorn, 2006; 2007; Caspi et al., 2002), therefore, further research examining the influence of gene by environment interactions may shed light on how inheritance contributes to both the dynamics and the outcome of development of institution-reared children (Rutter, 2006). This will contribute to the exploration of possible risk and protective factors, the identification of which is indispensable in the development of targeted and effective intervention programs.