Effects of Home Environment, Socioeconomic Status, and Health Status on Cognitive Functioning in Children With HIV-1 Infection

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Objective: To investigate the effects of the home environment, socioeconomic status (SES), and health status on cognitive functioning in a sample of children with HIV-1 infection in a cross-sectional study.

Methods: Forty-three caregivers and their children (2.5 to 12 years) participated. Caregivers completed two self-report measures of the home environment that included questions regarding the organization of the environment, play materials, parental involvement, variety of stimulation, and parental attitudes toward the provision of a cognitively stimulating environment. Cognitive functioning was assessed using a standardized intelligence (IQ) test. Children’s medical charts were reviewed for HIV-1 classification status (CDC, 1994), CD4 cell counts, and current medication.

Results: This study revealed two primary findings. First, measures of the home environment mediated the association between SES and child IQ. Second, measures of the home environment had a stronger association with child IQ during the advanced stages of disease than earlier stages of disease.

Conclusions: The home environment is associated with cognitive functioning among children with HIV-1 infection. Moreover, interventions aimed at enhancing the quality of the home environment may have a positive impact on these children’s cognitive development.

Key words: HIV-1 infection; children; cognitive development; family.

Studies of the development of children infected with HIV-1 have documented a wide range of outcomes from normal development to varying degrees of cognitive, motor, and behavioral impairments (e.g., Drotar et al., 1999; Fishkin et al., 2000; Wachtel et al., 1993; Wolters, Brouwers, Moss, & Pizzo, 1994). Cognitive deficits may be directly related to the effects of HIV-1 on the central nervous system (CNS), or to the effects of CNS infections or neoplasms secondary to immunocompromise (Budka, 1993). Neuroimaging studies have demonstrated a high incidence of structural brain abnormalities in children with symptomatic HIV-1 infection, including cortical atrophy, white matter abnormalities and calcifications (DeCarli, Civitello, Brouwers, & Pizzo, 1993), and researchers have found that children with white matter abnormali-
ties were more impaired on measures of cognitive functioning (Brouwers, van der Vlugt, Moss, Wolters, & Pizzo, 1995). Other factors that may contribute to cognitive outcomes include those associated with the disease (e.g., prenatal drug exposure, prematurity, poverty, home environment).

Developmental research has clearly established that both socioeconomic status (SES) and aspects of the home environment account for a significant proportion of the variance in cognitive functioning of both healthy and preterm children (e.g., Bradley et al., 1989; Brooks-Gunn, Klebanov, & Duncan, 1996). In addition, researchers have also established that the home environment may serve as a protective factor for children. For example, Bradley et al. (1989) found that when children had low intelligence test scores (IQ), less responsive parenting, and fewer stimulating play materials, they were at greater risk for low IQ scores later in life than children with low IQ scores, more responsive parenting, and more stimulating play materials in the home. Furthermore, preliminary evidence from studies of both low birth weight children and children with traumatic brain injury suggests that the strength of the association between the home environment and cognitive functioning may vary as a function of the child's CNS integrity (Bradley et al., 1993; Yeates et al., 1997).

Aspects of the child's home environment and their associations with CNS factors may explain some of the variability in the cognitive functioning of children with HIV-1 infection. That is, despite CNS pathology, protective mechanisms may promote cognitive development in children with HIV-1 infection; or conversely, risk factors may result in greater vulnerability to cognitive dysfunction. Biological parents of children with HIV-1 infection face numerous daily stressors (e.g., alcohol and drug use, low income and education, psychological and physical manifestations of their own terminal illness) that place their child at risk for poor cognitive development (Brown, Lourie, & Pao, 2000; Ickovics & Rodin, 1992; Sherwen & Boland, 1994). However, no studies have attempted to identify potential protective or risk factors in the home environment that may influence cognitive functioning in children with HIV-1 infection. Identification of such factors would help elucidate the mechanisms of the cognitive deficits associated with pediatric HIV-1 infection and is crucial for developing appropriate interventions for these children.

The purpose of this study was to investigate the influence of SES and the home environment on cognitive functioning in children with HIV-1 infection. The first hypothesis was that the home environment mediates the association between SES and child cognitive functioning. Although this mediational relationship has been established among healthy and preterm children (e.g., Bradley et al., 1989), it has not been examined among children with HIV-1 infection. The second hypothesis was that the influence of the home environment on child cognitive functioning is moderated by disease severity. In particular, we hypothesized that the home environment is more strongly associated with child cognitive functioning at earlier stages of disease versus advanced stages of disease when the child is at risk for greater CNS involvement. Little research has addressed the impact of health or CNS status on the relationship between the home environment and cognitive functioning.

**Method**

**Participants**

Forty-three children with HIV-1 infection (19 boys, 24 girls) and their caregivers participated in this study. Participants were recruited from three major medical centers: two in the southeast (n = 6) and one in the northeast (n = 37) between 1995 and 1997.

**Characteristics of Children.** Thirty-four children were African American, 6 were Caucasian, 2 were mixed race, and 1 was Latino. Children were predominantly school age (M = 6 years, SD = 2 years, range = 2.5–12 years) and 70% were attending school. Of those children in school, 21% were receiving special education services.

All of the children in our sample had perinatally acquired HIV-1 infection. HIV-1 infection status was confirmed for all children using viral detection tests (i.e., polymerase chain reaction). Children's HIV-1 symptoms were categorized by their physician according to Centers for Disease Control and Prevention Classification Criteria (CDC, 1994). Twenty-three percent (n = 10) of the children had no clinical symptoms (Category N), 21% (n = 9) had mild clinical symptoms (Category A), 26% (n = 11) had moderate clinical symptoms (Category B), and 30% (n = 13) had severe symptoms (Category C). Twenty-six percent (n = 11) of the children had no immune suppression (Category 1), 44% (n = 19) had moderate immune suppression (Category 2),
and 30% (n = 13) had severe immune suppression (Category 3). Chi-square analyses revealed no significant differences between the number of children across the four clinical and three immunologic categories. Sixty-five percent of the children (n = 28) were taking antiretroviral medication such as zidovudine (AZT) or didanosine (ddI). Twelve percent (n = 5) were taking immunoglobulin (IgG) only or a combination of IgG and AZT.

**Characteristics of Caregivers.** Twenty-three percent (n = 10) of the caregivers were extended family members and 33% (n = 14) were either foster or adoptive parents who were not related to the child. Forty-four percent (n = 19) of the caregivers were the biologic parents of the child, all of whom were infected with HIV-1. No caregivers in either the extended family members or foster/adoptive group had HIV-1 infection. Caregivers were generally middle-aged (M = 40 years, SD = 14 years, range = 20–75 years) women with at least a high school education (M = 13 years, SD = 3 years, range = 8–19 years). Forty-two percent (n = 18) of the caregivers were employed at least part time. The mean Hollingshead Index for the caregivers was Class III (range = I–V; Hollingshead, 1975) and the mean monthly income was $1,674 (SD = $1,252). Only 25% (n = 12) of the caregivers reported having the support of a spouse or partner in the home. Forty-seven percent (n = 20) of the caregivers reported being the sole caregiver for the child. Twenty-six percent (n = 11) of the caregivers had additional forms of caregiving support available (e.g., support from a relative or respite care).

**Home Environment Measures**

*Parent as a Teacher Inventory (PAAT).* The PAAT (Strom, 1984) is a 50-item, self-report measure that assesses attitudes toward the provision of cognitive stimulation or education in the home. Items are grouped into subsets related to five areas of parenting: (1) creativity—parental acceptance of creativity in their child; (2) frustration—frustrations around parenting and locus of frustrations; (3) control—parental feelings about and need for control of child behavior; (4) play—parental understanding of play and its influence in child development; and (5) teaching-learning—parental perceptions of their own ability to facilitate the teaching-learning process for their child. The total score of these five subtests was used in analyses. Strom reports coefficient alphas for PAAT total scores between .75 and .88 using separate samples. Concurrent validity was established through correlations with observations of parental behavior in the home (Johnson, 1975; Panetta, 1981).

*Home Screening Questionnaire (HSQ).* The HSQ (Coons, Gay, Fandahl, Ker, & Frankenburg, 1981) is a self-report questionnaire based on items from the Home Measurement of the Environment (HOME) Inventory (Caldwell & Bradley, 1978). The HOME is one of the most widely used observation measures in child development research and is associated with intellectual ability in children. The items on the HSQ focus on the organization of the environment, play materials, parental involvement, and variety of stimulation. In addition to a 34-item questionnaire, parents are asked to complete a toy checklist that asks about presence of various toys in the home. The total score is the sum of the questionnaire and toy checklist. The alpha coefficient is .80 and the test-retest coefficient is .86. Adequate concurrent validity was established utilizing the HOME as the criteria.

**Children’s Cognitive Functioning**

Children were administered one of three standardized intelligence tests. The McCarthy Scales of Children’s Abilities (MSCA; McCarthy, 1975) are created for children ages 2.5 to 8.5 years and provide an overall index of cognitive functioning (GCI). Mean test-retest reliability for the GCI is .90. Adequate predictive validity has been demonstrated via associations with performances on a variety of achievement measures. The Wechsler Preschool and Primary Scale of Intelligence–Revised (WPPSI-R; Wechsler, 1989) is designed for children ages 3 to 7.3 years. Average internal consistency reliability for the Full Scale IQ is .96 and adequate concurrent validity has been established. The Wechsler Intelligence Scale for Children–3rd Edition (WISC-III; Wechsler, 1990) is designed for children ages 6 to 17 years. Excellent reliability and concurrent validity have been established.

**Child HIV-1 Health Status**

To classify disease severity, children were grouped into one of three HIV-1 health categories according to their CDC classification at the time of their cognitive evaluation. The three health categories were created according to a model developed and empirically validated by Turner et al. (1993). Turner and
colleagues attempted to define predictors of survival in 789 children with HIV-1 infection using the child’s CDC classification status. An expert panel divided their sample into three groups based upon their estimation of survival. They then tested their model using the Cox proportional hazards models. Group 1 children (CDC classification = N1, N2, A1, A2, B1, and B2) were in the earliest stages of disease and with the highest survival rate (median survival time = 66 months); group 3 children (CDC classification = C2 and C3) were those at the end stages of disease and with the lowest survival rate (median survival time = 9 months); and group 2 children (CDC classification = N3, A3, B3, and C1) fell between groups 1 and 3 (median survival time = 48 months). We utilized this model when dividing this sample into three groups. Table I shows the CDC diagnostic categories included in each group, the number of children in each group and the mean absolute and percentage CD4 counts for each group.

Procedure

The following procedures were approved by the institutional review boards at each of the three medical centers. After providing written consent to participate, caregivers completed a demographic/medical questionnaire and a battery of self-report psychological measures. Fifty-five caregivers were approached and asked to participate in the study, and 50 caregivers agreed to participate. Seven caregivers who originally agreed to participate did not have sufficient time during their appointment to complete the battery. Therefore, complete data on 43 caregivers and their children were analyzed.

Children were administered one of three standardized IQ measures by a licensed psychologist or graduate student examiner. Selection of the type of IQ measure was based on two factors: (1) chronological and developmental age of the child and (2) the child’s participation in a clinical drug trial that required administration of a specific IQ measure. Eleven children were administered the McCarthy Scales, 18 children were administered the WPPSI-R and 14 children were administered the WISC-III. Children’s medical records were also reviewed following their evaluation and the following data were collected: CDC classification, CD4 cell counts (absolute and percentage), and current medications. Efforts were made to obtain CD4 counts most proximal to the time of evaluation, and the majority of children had immunological tests on the day of their evaluation (range = 0–30 days).

Results

Preliminary Analyses

Before the main analyses, missing measures (HSQ [n = 4]) and missing test items (n = 20 across all items) were replaced by the mean value for that measure or item. Raw data were examined for univariate and multivariate outliers, normality, linearity, homoscedasticity, and multicollinearity (Tabachnick & Fidell, 1989) and no violations were detected.

Because children completed one of three different cognitive measures, IQ scores were compared across tests to explore the possibility of test measure by IQ score interaction. Analysis of variance (ANOVA) revealed no significant differences between IQ scores across measures, F(2, 40) = .45; p = .64. IQ scores were then transformed to Z scores based on this study sample to establish a common metric.

Mean scores on psychological measures were examined and were within normal ranges when compared to normative samples or demographically similar samples (Table II). The association between

<table>
<thead>
<tr>
<th>Health status group</th>
<th>CDC classification categories</th>
<th>n</th>
<th>Mean (SD)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>N1, N2, A1, A2, B1, B2</td>
<td>25</td>
<td>888 (451)</td>
<td>28 (9)</td>
</tr>
<tr>
<td>2</td>
<td>N3</td>
<td>7</td>
<td>753 (694)</td>
<td>25 (11)</td>
</tr>
<tr>
<td>3</td>
<td>C2, C3</td>
<td>11</td>
<td>367 (310)</td>
<td>12 (10)</td>
</tr>
</tbody>
</table>

CDC = Centers for Disease Control; N = no clinical symptoms; A = mild clinical symptoms; B = moderate clinical symptoms; C = severe clinical symptoms; 1 = no immune suppression; 2 = moderate immune suppression; 3 = severe immune suppression; Chi-square analysis revealed a significant difference across health status groups, χ²(2, 43) = 12.4, p < .01. ANOVA indicated a significant absolute CD4 effect, F(2, 40) = 4.7, p < .05. THSD revealed significant differences between health status group numbers 3 and 1. ANOVA indicated a significant percent CD4 effect, F(2, 40) = 11.8, p < .001. THSD revealed significant differences between health status group numbers 3 and 1 and between group numbers 3 and 2.
Baron and Kenny (1986) do not stipulate a statistically significant difference between the associations, rather that the associations are reduced to nonsignificance after controlling for the mediational variable. Thus, results from this analysis satisfied all four conditions (Baron & Kenny, 1986) and demonstrate the mediating role of home environment in the association between SES and cognitive outcome among these children (see Figure 1A).

**Moderator Hypothesis: Association Between Home Environment Child HIV-1 Health Status and Child Cognitive Functioning**

To test the hypothesis that the association between home environment and child cognitive functioning is moderated by the HIV-1 health status of the child, we used hierarchical multiple regression analysis (Baron & Kenny, 1986). The predictor variables and their order of entry were (1) SES (HHI; covariate), (2) HIV-1 health status and home environment, and (3) the interaction between HIV-1 health status and home environment (i.e., Health × Home). To demonstrate that HIV-1 health status acts as a moderator variable, the interaction term must account for a significant and unique proportion of the variance in IQ scores, after controlling for SES, health status, and home environment. Results of the final regression analyses are summarized in Table III and Figure 1B.

Table II. Descriptive Statistics of the Study Measures for the Entire Sample (n = 43)

<table>
<thead>
<tr>
<th>Variable</th>
<th>M (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Home environment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HSQ</td>
<td>40.1 (7.5)</td>
<td>21–56</td>
</tr>
<tr>
<td>PAAT</td>
<td>142.6 (12.5)</td>
<td>125–173</td>
</tr>
<tr>
<td>Child IQ score</td>
<td>90.2 (15.4)</td>
<td>50–129</td>
</tr>
</tbody>
</table>

HSQ = Home Screening Questionnaire; PAAT = Parent As A Teacher Inventory.

Child IQ scores are presented as standard scores (M = 100, SD = 15) to facilitate the reader’s interpretation of the level of cognitive functioning. However, all analyses were calculated using Z scores.

the two measures of the home environment (PAAT and HSQ) was .65 (p < .001). To create an index of the home environment, we transformed scores from the PAAT and HSQ to Z scores based on this study sample and summed them.

**Mediator Hypothesis: Association Between Socioeconomic Status, Home Environment, and Child Cognitive Functioning**

To establish that the home environment mediates the association between SES and child cognitive functioning, we performed a series of hierarchical regressions (Baron & Kenny, 1986). Home environment can be designated as a mediating variable if the following four conditions are met: (1) SES significantly predicts home environment; (2) SES significantly predicts child cognitive functioning; (3) home environment significantly predicts child cognitive functioning after controlling for SES; and (4) after controlling for home environment, the previously significant association between SES and child cognitive functioning is no longer significant.

Conditions 1 and 2 were met. SES was significantly correlated with home environment, β = .56, R² square = .32, F(1, 41) = 18.9, p < .001, and with child IQ, β = .42, R² = .18, F(1, 41) = 8.9, p < .005. Condition 3 was met. Home environment was significantly associated with IQ after controlling for SES, β = .43, R² change = .13, F change = 7.3, p < .001. Finally, condition 4 was also met. The association between SES and IQ was reduced to non-significant levels after controlling for home environment, β = .18, R² change = .02, F change = 1.31, p = .26. We tested to see if the difference between these two associations (SES and IQ, β = .42; SES and IQ controlling for home environment, β = .18) was statistically significant and found that there was a trend for significance (t = 1.56, p < .10).
1B. SES (HHI) accounted for 18% of the variance in IQ scores, $R^2 = .18$, $F(1, 41) = 8.9, p < .01$. Health status and home environment variables explained 13% of the variance in IQ scores when entered on Step 2, $R^2 = .31$, $F(3, 39) = 5.7, p < .005$. The Health × Home Environment interaction term accounted for an additional 9% of the variance in child IQ scores, $R^2 = .39$, $F(4, 38) = 6.2, p < .005$. Taken as a whole, these variables accounted for 63% of the variance in IQ scores. These results support the hypothesis that HIV-1 health status acts as a moderator variable in the association between home environment and child IQ score.

Review of zero-order correlations between home environment and child IQ revealed a stronger association for children in more advanced stages of disease than for children in earlier stages of disease (HIV-1 health status group 1 [$n = 25$]: $r = .39, p = .05$; HIV-1 health status group 2 [$n = 7$]: $r = .71, p = .07$; HIV-1 health status group 3 [$n = 11$], $r = .76, p = .007$). These data suggest that among children in more severe stages of disease, the effects of the home environment on cognitive functioning is stronger, as compared to those children who are in earlier and healthier stages of disease.

## Discussion

This study investigated the association among home environment, SES, cognitive functioning, and health status in a group of children with HIV-1 infection. Home environment was found to mediate the association between SES and child IQ. This finding is consistent with the literature on the development of both healthy and preterm children (e.g., Bradley et al., 1989), and provides support for the hypothesis that the home environment can either serve as a protective factor against or risk factor for the negative effects of poverty on cognitive functioning. Furthermore, review of associations between SES and the home environment suggest that children with HIV-1 infection and living in poverty also live in less stimulating and supportive home environments. The resultant milieu is likely a major factor for developmental problems in this population. Support was also found for the moderating role of health status in the association between home environment and child cognitive functioning. That is, the association between home environment and child cognitive functioning varied as a function of the health status of the child. However, contrary to a priori hypotheses, the association between home environment and IQ was stronger for children in more severe stages of disease than for those who were in healthier disease stages. Thus, disease severity appeared to magnify or enhance the effects of the environment on child cognitive functioning.

One possible explanation for this latter finding is based on the transactional model of development. In the transactional model, the child is viewed as a product of a continuous dynamic interaction between the child and the family and social context (Sameroff & Chandler, 1975). According to this model, aspects of the child have a strong role in determining his or her experiences. Perhaps within this population of children, becoming severely ill will alter caregivers’ perceptions. This change in perception may translate into changes in the way the parent cares for and interacts with the child. These behavioral changes, in turn, may have greater impact on the child’s development.

The association between home environment and cognitive functioning varied as a function of disease severity. Specific aspects of disease severity, such as CNS integrity, may account for the stronger association between the home environment and child cognitive functioning in children at end stages of disease. Thus, children who have greater CNS impairment may also be at greater risk for more negative cognitive outcome secondary to a
less stimulating environment. Our findings supported this hypothesis. Of those children in our study who were referred for CT/MRI brain imaging by their physician for clinical purposes \( n = 31 \), a greater percentage of the children in group 3 (27%) had an abnormal CT/MRI brain scan, as compared to children in Groups 1 and 2 (.03%). Also noteworthy is that all of the children in group 3 children were taking AZT or other antiretroviral medications. Future studies should attempt to understand the relative unique contribution of various health or disease factors (e.g., CNS integrity, antiretroviral medication), as well as their additive or synergistic effects on the relationship between home environment and cognitive outcome.

Results of this study should be interpreted cautiously for a number of reasons, including small sample size and the resultant decrease in statistical power, limited number of measures of similar constructs, and selection bias. Reliance upon self-report measures is another limitation. Effort was made to assess for potential bias in reporting by including a measure of positive impression management in the battery of measures that the caregivers completed. All caregivers scored within normal limits on this scale, suggesting that they all answered questions in a seemingly valid manner. Other limitations include the use of a single IQ score in measuring child cognitive functioning. This may have masked any subtle, but meaningful, impairment in specific areas of cognitive functioning. In addition, researchers have found improved cognitive and behavioral functioning in children following initiation of antiretroviral regimens (e.g., Brouwers et al., 1990; Raskino et al., 1999). The potential effect of medications on child cognitive functioning was not explored in this study. Furthermore, although this was not a focus of this study, it is reasonable to assume that the variability in family structures (e.g., biological parent vs. extended family vs. foster/adoptive care) may differentially affect cognitive functioning in this population of children. Future studies should attempt to examine this important issue. Finally, whereas the analyses suggest that the quality of care in the home predicts child cognitive functioning, these findings are based on correlational data and causality cannot be inferred. The predictive power of the caregiving construct would be strengthened through the use of longitudinal designs or through statistical procedures utilizing path analyses or structural equation modeling. Furthermore, analysis of alternative interpretations for the data—for example, that developmental disability among children with HIV-1 infection affects the quality of caregiving—is certainly plausible, given the reciprocal nature of child and family influences (Sameroff & Fiese, 1993).

Despite the limitations of this study, the findings have important implications for clinical treatment of these families. First, this study demonstrated that SES and the home environment are associated with cognitive development in this population. This finding supports the creation of environmentally focused interventions (e.g., provision of stimulating toys for the home, parenting education classes) to enhance cognitive skills. Second, home environment was found to mediate the association between SES and child cognitive functioning. Thus, interventions aimed at parenting skills may circumvent environmental risk factors such as poverty. Finally, the association between home environment and cognitive functioning is strongest among children who were severely ill. This suggests that children who are in advanced stages of disease may experience greater positive effects from a cognitively stimulating environment and, conversely, may experience greater negative effects from a less cognitively stimulating environment.

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References


