

# Unintentional Injuries in a Twin Study of Preschool Children: Environmental, Not Genetic, Risk Factors

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**Objective** To analyze the relative contribution of latent genetic and environmental factors to differences in the injury liability of children, and to examine the association between measured socio-economic, family, and child-behavior variables and unintentional injury risk. **Methods** Unintentional injuries from birth to age 5, together with information regarding measured risk variables, were reported by mothers in a sample of 1027 same-sex twin pairs from a nationally representative 1994–1995 birth cohort. **Results** Child-specific environmental factors accounted for most of the variance (86.4%) in the likelihood of ever having an injury. When considering the risk of two or more injuries child-specific environmental factors explained 60.2% of the variance and family-wide environmental influence 39.8%. Measured socio-economic, family, and child-behavior factors predicted frequent injury. **Conclusions** Results give little support to the concept of a heritable injury-prone trait in preschool children; environmental influences accounted for most of the injury variance in this sample. However, behavioral variables, especially the child's externalizing problem behaviors, are also important in explaining unintentional injuries.

**Key words** environmental factors; genetic predisposition; injury-prone; twins; unintentional injuries

Injury is the commonest cause of mortality in children under 14 years of age in developed countries (UNICEF, 2001). Injury mortality has fallen by more than 40% in the last quarter of the 20th century, and is still decreasing in industrialized areas (Arias, MacDorman, Strobino, & Guyer, 2003; Rivara, 1999; Taft, Paul, Consunji, & Miller, 2002). However, a large proportion of childhood deaths continue to occur as a result of preventable injuries. Of these, unintentional injuries account for nearly half of all deaths to children and adolescents in the US (Martin, Kochanek, Strobino, Guyer, & MacDorman, 2005), and it is estimated that 30% of all child mortality in Europe is due to unintentional injuries (Morrison, Stone, & EURORISC Working Group, 1999). Such injuries are also a major contributor to morbidity and long-term disability (Grossman 2000).

The magnitude of this problem varies according to several factors, including age, sex, region, income group, cultural background, and kind of injury (Chishti & Stone, 2004; Ramsay et al., 2003; Vaughan, Anderson, Agran, &

Winn, 2004). Behavioral factors are also implied, as injuries are the result of specific behavior–environment transactions (Bradbury, Janicke, Riley, & Finnney, 1999). Hence, both child factors and environmental factors need to be taken into account in order to understand why these events take place (Bradbury et al., 1999; Dal Santo, Goodman, Glick, & Jackson, 2004).

## Children's Injury Liability

In an attempt to analyze and prevent the causes of unintentional injury, an effort has been made to identify risk factors and groups of children which would predict higher injury frequency (Jaquess & Finney, 1994; Ramsay et al., 2003; Schwebel & Plumert, 1999). This search has led, occasionally, to the concept of “accident proneness”, which reflects the fact that some children seem to have an increased risk of injury compared to others within the same environmental conditions (Vollrath, Landolt, & Ribi, 2003). This idea has been criticized in the pediatric

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literature for, at least, two important reasons. First, accident proneness can only be diagnosed after repeated injury, which has little benefit for prevention. Second, invoking accident proneness is viewed as blaming the victim; and consequently ignoring environmental risk factors that could be responsible for the increased risk (Grossman, 2000; Rivara, 1995).

Even taking into account the accuracy of these criticisms, there is substantial correlational evidence that children's psychological characteristics could influence their injury liability (Bradbury et al., 1999; Matheny, 1987; Rivara, 1995; Schwebel, 2004; Schwebel & Barton, 2005; Vollrath et al., 2003). Aggressive behavior, high level of activity, presence of behavioral disorders, low adaptability, inattention, impulsivity, extraversion, sleep habits, sensation seeking, or developmental status have been mentioned as factors which may affect the probability of unintentional injury (Bradbury et al., 1999; Brehaut, Miller, Raina, & McGrail, 2003; DiScala, Lescohier, Barthel & Guhoua, 1998; Jaquess & Finney, 1994; Morrongiello, Carbett, McCourt, & Johnston, 2006a; Phillips & Matheny, 1995; Rowe, Maughan, & Goodman, 2004; Wazana, 1997). All these child behaviors are known to be under moderate to strong genetic influence, and thus, underlying genetic factors have also been suggested as playing a role in individual differences in liability to childhood injuries (Matheny, 1991; Phillips & Matheny, 1995). Evidence for, or against, a genetic influence on individual differences in injury proneness could help to support, or challenge, the concept of the injury prone child.

### **Do Genetic Factors Contribute to Unintentional Injuries in Young Children?**

Individual differences in injury liability may result from environmental factors, but also because some groups of children expose themselves to more risk than others. In response to this question, Matheny's (1986) early work linked the difference within twin pairs for histories of injuries with differences reported for the twins' behaviors, and found an association between temperamental characteristics and injury liability. Unfortunately, this method only allowed for comparison within pairs, and the effect of variables external to the child was not established. A decade later, another study applied a threshold model of latent liability to infant and toddler twin data (Phillips & Matheny, 1995). They found strong dominance variance in the absence of detectable additive genetic variance, a feature that could

only be explained by appealing to low-order epistasis or other complex interaction effects. The analysis did not detect family-wide environmental effects. These unclear results led the authors to call for additional studies on this topic. More recently, Rowe, Simonoff and Silberg (2007) studied an older sample of twins aged 8–17 years and found significant genetic effects on unintentional injuries, but only for girls. They also found a strong effect for family-wide environment for both sexes.

The presence of genetic effects, if confirmed, would highlight the importance of intermediate phenotypes between genes and risk of injury (e.g., temperamental or behavioral characteristics under genetic influence). However, we are still far from understanding the relative contribution of underlying genetic and environmental factors to unintentional injuries, or whether those factors interact in some way to produce a higher risk level in specific groups of individuals.

The study reported here attempts to address some of those problems by applying quantitative genetic models to injury data in a sample of 5-year-old same-sex twins. By comparing phenotypic resemblances of monozygotic (MZ) and dizygotic (DZ) twins, it is possible to estimate the relative contribution of genetic variation to the phenotypic differences found in a trait (Boomsma, Bushjan, & Peltonen, 2002). If there is an accident proneness trait, then MZ twins should be more similar than DZ twins in the number of injuries they experience. We examined a sample of twins participating in a longitudinal study focused on children's development. Their mothers reported the number and type of unintentional injuries from birth to age 5, together with information regarding environmental and behavioral variables. We report the relative contribution of genetic and environmental factors to differences in children's injury liability.

### **Method** **Sample**

Participants are twins who are members of the Environmental Risk (E-risk) Longitudinal Twin Study. The E-risk sampling frame was two consecutive birth cohorts (1994 and 1995) from a birth register of twins born in England and Wales (Trouton, Spinath, & Plomin, 2002). The E-risk Study was constructed in 1999–2000, when 1116 families with same-sex 5-year-old twins (93% of those eligible) participated in a home-visit assessment, forming the base cohort. Findings from the twin cohort can be generalized to the population

of British families with children born in the 1990s (for further details see Moffitt et al., 2002). Zygosity was determined by a questionnaire administered to the parent about physical similarities, differences, and confusion between the twins. This questionnaire has been found to classify the zygosity of 95% of twins accurately (Price et al., 2000). Unclear zygosity was resolved by DNA testing. The cohort includes 55% MZ and 45% DZ twin pairs. Sex is evenly distributed within zygosity (49% male). The E-risk study received ethical approval from the Maudsley Hospital Ethics Committee. Parents gave informed consent.

### **Data Collection**

Data were collected within 2 months of the twins' fifth birthday. Research workers visited each home for 2.5–3 h. Mother interviews about children were administered twice, to obtain information about each twin separately. Families were given shopping vouchers for their participation, and children were given coloring books and stickers. All research workers had university degrees in behavioral science, and experience in psychology, anthropology, or nursing, and received 2 weeks of training in data collection.

### **Unintentional Injuries**

Injury data were obtained retrospectively from the mother. The interview was guided by using the Life History Calendar (Caspi et al., 1996), a visual grid of a family's life events on a month-to-month basis that has been proven to enhance the validity of retrospective reports. Parents reported whether the children had ever experienced any injury that required medical or surgical attention, and described each injury episode. Later, another examiner encoded the episodes according to the International Classification of Diseases, Tenth Revision (ICD-10). Another independent examiner reviewed the data to refine the list. Following Matheny's methodology (Matheny, 1987), cases where children's behavior was not implied in the injury event were excluded from the analyses here (e.g., car accidents if the child was not a pedestrian, animal bites or stings, or injuries clearly resulting from adult maltreatment or neglect).

### **Child Behavior Variables**

Child variables were assessed through interviews with the mothers using the Child Behavior Checklist (CBCL) (Achenbach, 1991). The externalizing scale reported in this article was the sum of items in the Delinquent Behavior and Aggressive Behavior subscales; the internal

consistency reliability was .89. The internalizing scale was the sum of items in the Withdrawn, Somatic Complaints, and Anxious/Depressed subscales; the internal consistency reliability was .85. The ADHD scale measured symptoms of hyperactivity, inattention, and impulsivity (McGee, Williams, & Silva, 1985; Sclare, 1997). Internal consistency was .88.

### **Socioeconomic Disadvantage**

Socioeconomic status of the children's families was measured at age 5 by counting the following disadvantages (Moffitt et al., 2002): (a) head of household has no educational qualifications; (b) head of household is employed in an unskilled occupation or is not in the labor force; (c) total household gross annual income is less than £10,000; (d) family receives at least one government benefit, excluding disability benefit; (e) family housing is government subsidized; (f) family has no access to a vehicle, and (g) family lives in the poorest of six neighborhood categories, in an area dominated by government-subsidized housing, low incomes, high unemployment, and single-parent families. We report the percentage of homes with one or more of these indicators of disadvantage.

### **Family Variables**

Family structure and instability risks were assessed using the Life History Calendar to date the occurrence, number, duration and sequence of parents' marriages, separations, cohabitations, births of children, and family size. Spells of unemployment for adults in the home were also reported on the calendar.

### **Statistical Analysis**

Data are presented in three sections. First, we provide a description of the sample and the incidence of unintentional injuries. Second, we examine the relative latent genetic and environmental (family-wide and child-specific) effects upon population variation in injury. Finally, we present the results of logistic regressions, from which the odds ratio (OR) was used to test the relationship between unintentional injuries and social, family and behavioral measures. Analyzing two children in the same family creates dependence in the data, and thus these regression analyses were based on the sandwich or Huber/White variance estimator (Gould & Scribney, 1999), a method available in STATA 9.0 (StataCorp, 2005), which adjusts estimated standard errors to account for that dependence and provides statistical tests that are robust to model assumptions (Lumley, Diehr, Emerson, & Chen, 2002).

Tetrachoric correlations were computed for MZ and DZ twins. We used maximum likelihood estimation of model parameters in univariate genetic models of children's injury (Neale & Maes, 2005; Plomin, DeFries, McClearn, & McGuffin, 2001). When a phenotypic measure is analyzed, models decompose variance in children's injury into latent additive genetic (A; i.e., the sum of the average effects of individual alleles at all loci), latent family-wide environmental (C), and latent child-specific environmental (E) factors. In comparing the fit of different models, we used the  $\chi^2$  goodness-of-fit statistic. Large values compared to model degrees of freedom indicate poor model fit to the observed covariance structure. When two models are nested, the difference in fit between them can be evaluated with the  $\chi^2$  difference, using as its degrees of freedom the *df* difference from the two models. When the  $\chi^2$  difference is not statistically significant, the more parsimonious model is selected. The second model-selection statistic was Akaike's Information Criterion (AIC), which has a negative value if the fit is good; the model that minimizes AIC is selected as the best-fitting model (Akaike, 1987). The third was the RMSEA statistic, which should be .05 or less for very good fit, or between .05 and .10 for good fit (Neale, Boker, Xie, & Maes, 2005). Structural equation modeling program Mx was used to test genetic models (Neale et al, 2005). Univariate and multivariate tests were performed with the SPSS 11.0 and STATA 9.0 statistical packages.

## Results

### *Description of Sample and Prevalence of Unintentional Injuries*

Data on history of unintentional injuries were available for 2054 children (1027 twin pairs; MZ = 567; DZ = 460). Mean number of UIs was .49 per child (*SD* = .84) with a range between 0 and 16 injury episodes. Data were collapsed into three categories (no injuries; one injury; and two or more injuries). More than one third of the children (35.2%) had experienced at least one unintentional injury. Almost one in ten children had experienced two or more injury episodes by the age of five years (9.6%). Taking into account the objectives of the study, data were analyzed according to two categorical variables: presence or absence of any injury (None vs. one or more) and frequency of unintentional injuries (One or none vs. two or more). Table I summarizes means and percentages of the main social, familial, and behavioral variables taken into account, distributed by sex and number of unintentional injuries.

### *Relative Genetic and Environmental (Family-wide and Child-specific) Effects upon Variation in Unintentional Injuries*

Injuries were distributed evenly across zygosity (MZ: 64.2% none, 26.7% one, 9.1% two or more; DZ: 65.4% none, 24.3% one, 10.3% two or more). Table II shows the MZ and DZ tetrachoric correlations, for the total sample and by sex.

When we analyzed the measure reflecting children who had had one or more injuries, the MZ twin correlation (.14) was double that of DZ twins (.06), pointing to some effect of additive genetic variance in the probability of suffering at least one injury. The correlations also pointed to the importance of child-specific environmental effects on having any injury, reflected in the difference between the MZ correlation and unity. Thus, having any injury in the first 5 years of life was modestly influenced by children's genetic background, but child-specific environmental circumstances played the greatest part. Table II also shows the results of model fitting for the total sample. As can be seen, a model containing all three parameters (ACE) adequately fit the MZ and DZ correlation matrices. Three reduced models were tested to establish the most parsimonious model for these data. An AE model was the best fit when considering the probability of having at least one injury, according to the fit statistics in the table. The proportion of variance accounted for was 13.6% for additive genetic effects [95% confidence interval (CI): 1.3, 25.7] and 86.4% for child-specific environment (95% CI: 74.3, 98.7). An examination of the ACE models for males and females separately showed that the best fit for both genders was also an AE model with very similar proportions of explained variance across the sexes.

In contrast, when we analyzed the measure reflecting frequency of children's unintentional injuries, the DZ twin correlation (.49) was higher than that for MZ twins (.29) indicating no effect of genetic factors on the probability of having two or more injury episodes in the first 5 years of life. Model-fitting confirmed that the genetic parameter was not needed, and a CE model showed the best fit. According to this model, 39.8% of the variance could be accounted for by family-wide environmental influences (95% CI: 24.9, 53.3), while 60.2% relates to child-specific environmental factors (95% CI: 46.7, 75.0). Again, when examining the models separately for males and females, the best fit for both genders on frequent injuries was this CE model.

**Table I.** Per cent Data or Means (SD) on Selected Socioeconomic, Familial and Behavioral Variables as Related to Sex and Number of Unintentional Injuries (UI)

	Total sample	Total			Boys			Girls		
		No UI	1 UI	≥ 2 UIs	No UI	1 UI	≥ 2 UIs	No UI	1 UI	≥ 2 UIs
N (2054)	100.0	64.8	25.6	9.6	60.5	26.7	12.8	69.1	24.4	6.5
Socioeconomic										
Social disadvantage, 1 or more	46.3	43.6	43.3	64.2	44.5	41.4	69.2	42.9	45.3	54.4
Total family income										
>£41000/year	20.4	21.3	22.4	11.3	20.9	22.6	12.5	21.7	22.3	10.1
26000–40999/year	27.6	29.2	26.2	27.2	28.4	24.9	25.0	29.8	27.5	31.9
12000–25999/year	35.2	34.7	34.6	42.1	35.4	33.7	42.2	34.2	35.6	40.6
<£11999/year	16.8	14.8	16.7	19.5	15.4	18.8	20.3	14.4	14.6	17.4
Unemployed parents	6.7	6.2	5.8	10.0	5.7	7.2	10.6	6.6	3.9	7.5
Family										
Age of mother at first birth	25.6 (5.8)	26.1 (5.8)	25.6 (5.6)	24.6 (5.1)	25.9 (5.6)	25.8 (5.9)	24.5 (5.3)	26.2 (6.0)	25.3 (5.6)	24.8 (4.9)
Age of mother when had twins	29.1 (5.4)	29.5 (5.3)	29.1 (5.3)	27.9 (5.3)	29.4 (5.3)	29.2 (5.4)	27.5 (5.3)	29.6 (5.4)	28.8 (5.3)	28.8 (5.2)
Older siblings (Yes)	58	58.2	56.0	57.2	61.5	55.1	53.4	55.3	57.0	64.7
Partner status (% not with dad)	17.8	17.1	15.7	24.9	17.6	16.5	25.6	16.6	14.8	23.5
Child behavior										
CBCL Externalizing Scale	11.8 (8.6)	11.2 (8.4)	12.2 (8.4)	13.6 (9.4)	12.2 (9.1)	13.1 (8.9)	14.8 (10.1)	10.3 (7.6)	11.1 (7.5)	11.2 (7.2)
ADHD Subscale	10.4 (7.5)	10.0 (7.3)	10.3 (7.5)	11.8 (8.2)	11.1 (7.7)	10.9 (8.0)	12.7 (8.5)	9.0 (6.9)	9.7 (6.8)	10.0 (7.1)
CBCL Internalizing Scale	8.4 (6.7)	8.1 (6.6)	8.2 (6.4)	8.6 (6.8)	7.6 (6.6)	7.9 (6.4)	9.3 (6.9)	8.5 (6.6)	8.5 (6.3)	7.4 (6.3)

**Table II.** Tetrachoric Correlations (for the Total Sample and by Sex) and Results of Model Fitting (Total Sample) for Ever Having an Unintentional Injury (UI) and Frequent UIs

MZ correlation (95% CI)	DZ correlation (95% CI)	Model	$\chi^2$	df	p	AIC	RMSEA
Unintentional injury ever (≥1)							
		ACE	2.77	3	.43	– 3.22	.011
Total: .14 (.00, .27)	Total: .06 (–.08, .21)	<b>AE</b>	<b>2.77</b>	<b>4</b>	<b>.59</b>	– <b>5.22</b>	<b>.000</b>
Boys: .13 (–.06, .32)	Boys: .09 (–.11, .28)	CE	3.34	4	.50	– 4.65	.008
Girls: .13 (–.06, .32)	Girls: .03 (–.19, .24)	E	7.46	5	.19	– 2.54	.019
Unintentional injuries frequent (≥2)							
		ACE	3.21	3	.36	– 2.79	.000
Total: .29 (.06, .49)	Total: .49 (.30, .66)	AE	10.14	4	.04	2.14	.034
Boys: .36 (.12, .56)	Boys: .29 (.05, .51)	<b>CE</b>	<b>3.21</b>	<b>4</b>	<b>.52</b>	– <b>4.79</b>	<b>.000</b>
Girls: .15 (–.12, .41)	Girls: .14 (–.18, .44)	E	28.97	5	.00	18.97	.081

A = Additive Genetic Effect; C = Family-wide Environmental Effect; E = Child-specific Environmental Effect. Bold values indicate best-fitting models

### Measured Environmental Factors Predicting Occurrence of Unintentional Injuries

Given that we did not find any significant genetic effects in this sample, we proceeded to examine the impact of some measured environmental factors on the risk for unintentional injuries. All categories of

variables (sex of the children, socioeconomic, family, and behavioral) showed an influence on the probability of being injured (Table III). Being male was the main risk factor for injuries, ever or frequent. Therefore the rest of the analyses were adjusted by sex. Living in a socially disadvantaged family, having a younger mother,

**Table III.** Demographic, Socioeconomic, Familial, and Behavioral Variables Related to Unintentional Injuries (UI) Ever and Frequent, Adjusted by Sex

	UI Ever OR (95% CI)	Two or more UIs OR (95% CI)
Child sex (Male)	1.48 (1.23, 1.78)***	2.20 (1.60, 3.04)***
Socioeconomic		
Social disadvantage, 1 or more	1.26 (1.04, 1.52)*	1.96 (1.39, 2.74)***
Total family income		
>£41000/year	1.00	1.00
26000–40999/year	1.00 (.72, 1.39)	1.85 (.95, 3.59)
12000–25999/year	1.15 (.84, 1.57)	2.27 (1.20, 4.32)*
<£11999/year	1.27 (.90, 1.79)	2.34 (1.18, 4.64)*
Unemployed parents	1.03 (.73, 1.45)	1.46 (.91, 2.32)
Family		
Age of mother at first child	0.98 (.96, .99)**	0.97 (.94, .99)*
Age of mother when had twins	0.97 (.96, .99)**	0.96 (.93, .99)**
Older siblings (Yes)	0.90 (.74, 1.09)	0.87 (.63, 1.20)
Partner status (not living with dad)	1.12 (.89, 1.40)	1.55 (1.10, 2.18)*
Child behavior		
CBCL Externalizing Scale	1.02 (1.01, 1.03)***	1.02 (1.01, 1.04)***
ADHD Subscale	1.02 (1.00, 1.03)**	1.02 (1.01, 1.04)*
CBCL Internalizing Scale	1.01 (.99, 1.02)	1.02 (.99, 1.04)

The reference category for the multi-category variable is denoted by an odds ratio of 1.00. The *ns* for the analyses involving all variables ranged from 1962 to 2054. \**p* < .05; \*\**p* < .01; \*\*\**p* < .001

and showing higher levels of externalizing problems and overactivity (as measured by the ADHD scale) increased the child's risk of being injured.

When we analyzed the measure reflecting frequency of injury, virtually all the aforementioned variables showed a stronger effect. Also, additional risk factors emerged for frequent injury (i.e., low family income and absence of the father). Multiple logistic regression analyses indicated that the main factors uniquely explaining the occurrence of unintentional injuries were male sex of the child, young age of mother, and externalizing problem behaviors,  $\chi^2(3) = 36.3$ ,  $p < .001$ . The main factors uniquely explaining frequent injuries were male sex of the child, social disadvantage, and externalizing behaviours,  $\chi^2(3) = 41.8$ ,  $p < .001$ .

Since male sex of the child seemed to be one of the most important factors affecting probability of being injured, both ever and frequent, interaction terms to test for gender differences were calculated for each of the social, family and child measures, but these yielded no statistically significant sex differences.

## Discussion

The concept of an injury-prone trait in young children receives little support from our data. If injury proneness were a trait, then we would have expected to observe a strong genetic influence on children's injury, particularly for frequent, repeated injuries. In contrast, evidence from the twin models for genetically influenced accident proneness was very modest for any injury, and nil for frequent injury. Although the literature has suggested that some specific behavioral variables, thought to be under moderate genetic influence, are related to the probability of unintentional injuries, a direct test of genetic influence in this twin sample showed that the impact of environmental factors on injury liability is much stronger and accounts for most of the variance in this variable.

The importance of child-specific environmental effects is reflected in the difference between the MZ twins' correlation and unity. Thus, the twin models suggested that the experience of an injury episode is largely influenced by child-specific factors that are not genetic. Examples of such factors might be each child's idiosyncratic choice of play equipment or active games, interactions with other children, spontaneous behavioral decisions, or just chance.

Regarding the probability of suffering unintentional injuries more frequently, the twin models suggested that this liability was influenced by family-wide risk factors (examples of these could be physical hazards in the home or garden, lack of parental supervision, or inappropriate parenting skills). This finding at the latent level from the twin model was consistent with our analyses of measured risk factors, which showed that frequent injury was strongly associated with family-wide social disadvantage. Variables such as family income or absence of the father appeared in our study as relevant factors and they have also been mentioned in previous research (Brehaut et al., 2003; Ramsay et al., 2003; Reimers & Laflamme, 2005).

There are few studies analyzing the relative contribution of genetic factors to unintentional injury with which to compare our work. As stated before, an early report with a sample of 3-year-old children (Phillips & Matheny, 1995) was not able to detect additive genetic or family-wide environmental effects on unintentional injuries. Our data confirm the small effect of additive genetic factors on injury, for preschool children, found by these authors. More recently, other researchers (Rowe et al., 2007) have found significant genetic effects, but only for girls, in an older age range (8–17), and for an injury measure that included minor incidents such as bumps and bruises. Since age may be an important factor related to the kind

and frequency of injuries, our results should not be considered incompatible with theirs. Moreover, in agreement with these last authors, we did find a relevant effect for measured family-wide environment variables (household and parental characteristics), especially for frequent injury events. This finding fits well with theoretical explanations of the distribution of risk factors for unintentional injuries, as well as with the research literature in the field.

Nonetheless, it ought to be stressed that our conclusions should be applied only to children of this age (0–5 years old). All young children, like those in this sample, share characteristics such as the need to explore and interact with the physical environment, limited understanding of the implications of their actions, and the tendency to learn through physical manipulation, all of which increase their injury exposure. For example, all young children attempt to taste things that could be dangerous for them. As children grow, more individual differences develop and children diverge in motor coordination, cognitive abilities, their personal estimation of risks, their interest in sport and other activities, and expression of their temperamental characteristics. Thus, genetically-influenced traits could become more salient in exerting their effects on injury risk with increasing age (Matheny, 1987). Additionally, other factors external to the child also change with age. For instance, parental supervision, which has been reported to serve as a protective factor, tends to be more intense for younger children (Aken, Junger, Verhoeven, Aken, & Dekovic, 2007; Morrongiello, Carbett, McCourt, & Johnston, 2006b; Munro, van Niekerk, & Seedat, 2006; Schwebel, Hodgens, & Sterling, 2006). Hence, the relative weight of genetic and environmental factors in the liability for unintentional injury may well change during child development (Matheny, 1986, 1987).

The pre-eminence of latent family-wide and child-specific environmental influences in this study should not lead us to discard the effect of behavioral styles on risk for unintentional injury. First, children's behavior should not be equated with genetic traits; it has many causes. Additionally, childhood injury is a complex phenomenon predicted by a set of sociodemographic, cognitive, behavioral, and child-related factors, and there is a common conclusion in the literature that aggressive, oppositional, overactive, impulsive, and undercontrolled behavioral styles predict an increased risk of subsequent and concurrent unintentional injury (DaSanto et al., 2004; Schwebel & Barton, 2005). Our data confirm this idea, showing that externalizing behaviors and

hyperactivity contribute significantly to both any injury and frequent injury. But these behaviors do not occur in a vacuum; they are expressed in response to particular stimuli from the environment, and they increase the risk of injury especially when hazards are present in the environmental context (Schwebel, 2004). Thus, unsafe households and playground areas, less frequent adult supervision or a lower level of preventive education by parents in socially disadvantaged families, might make the environment more hazardous and potentiate the contribution of behavioral styles to unintentional injuries.

### **Limitations of the Study**

Methodological concerns that could limit our conclusions must be mentioned. Most of these are related to data collection. First of all, our data relied on retrospective information that might be affected by maternal recall bias. We tried to reduce bias by using the Life History Calendar which has been shown to improve the validity of retrospective reports (Caspi et al., 1996). Additionally, the present study dealt with occurrence of unintentional injuries in general and we were unable to analyze different kinds of accidents. Different factors are related to different types of injury (Grossman, 2000; Powell & Tanz, 2002; Rowe et al., 2004). For example, burns and poisonings may be more related to the family home environment than fractures, which could be more related to children's activity level. Elsewhere we have reported on intentional injury by maltreatment of twins in this sample, which also showed no genetic influence (Jaffe et al., 2004). Future research should address this question of injury types in greater depth, and provide separate examination of genetic factors for each category. Another flaw related to the data collection is that our study was not designed specifically as a study of injury; rather injury data were collected as one part of a broader investigation of children's health and development. Thus, possibly relevant information about child or social factors that could help to explain part of the variance in unintentional injuries was not gathered systematically (e.g., we did not have measures of child social development or direct measures of risks in the physical environment). Moreover, developing measures aimed at distinguishing psychological antecedents to injury (e.g., risk-taking vs. error-proneness) may also be necessary in future attempts to study this question (Rowe et al., 2007).

Another problem is that we could not rigorously test for possible sex differences regarding underlying factors on injury liability. Boys have an increased injury risk,

but no sex differences were observed regarding the relative impact of genetic and environmental factors on injury liability. In fact, the confidence intervals for boys and girls correlations overlap completely, and the models for boys and girls showed virtually identical parameters. Also, we did not find any significant interaction between sex of the child and the variables measured. However, latent genetic or family-wide factors could be of a different nature in boys and girls. Opposite-sex twin pairs are needed to test this notion decisively, but our sample included same-sex twins only (Neale & Maes, 2005).

Finally, we cannot establish the temporal order of the risk factors. We assessed injuries in the first 5 years of life, and risk factors were evaluated at the age-5 home visit. Some injuries, in particular severe traumatic brain injury, occasionally may lead a child to develop externalizing behavior problems (Bloom et al., 2001). Nonetheless, Rowe et al. (2007) reported, in a longitudinal study, that previous injury did not predict later measures of conduct disorder, oppositional defiant disorder, attention deficit/hyperactivity disorder, or impulsivity. Besides, our sample comprised a much wider range of injuries. All in all, although a reverse casual effect could be possible in specific cases, it is not likely to have a confusing effect on our results. In any case, future longitudinal analyses will be of importance to draw firm conclusions about the direction of effects.

Despite these limitations, this study constitutes an important step in the research on unintentional childhood injuries. It is based on a large representative sample of same-sex twins and allows for an analysis of the relative contribution of genetic and environmental factors to one of the leading causes of morbidity and mortality in children of all ages.

### **Implications for Preventive Interventions**

Bearing in mind that these results need to be replicated, they point to some relevant questions regarding preventive interventions. Because we did not find evidence for injury proneness as a behavioral trait, our results would not indicate focusing on screening or intervention at the level of the injury-prone individual. Instead, these results lead us to stress that injury prevention for preschool children should rely heavily on making their environment safer. From this point of view, injury prevention interventions might work along several complementary lines to make their environment safer (Aken et al., 2007; Brehaut et al., 2003; Garry et al., 2007; Munro et al., 2006; Powell & Tanz, 2002; Ramsay et al., 2003): (a) Generalized public health interventions,

such as strong media messages or promotion of safety devices; (b) specific actions to improve hazardous environments, such as increasing the safety of playground areas or implementing interventions in low-income contexts; and (c) injury-prevention information and teaching for children and families, especially those with increased risk because they live in a hazardous area, lack of adequate parenting behaviors, or the child has serious behavioral problems. Such health-promotion interventions directed primarily toward modifying environmental factors would possibly have the best impact on the global prevalence of this problem.

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