

In developmental psychopathology it almost goes without saying that contextual risk factors do not occur in isolation and that it is the combination of various risk factors that portends numerous negative child outcomes. Despite this, the body of literature that examines the relation between multiple risk exposure and child psychopathology using a cumulative risk approach is still relatively small. Even when studies use a cumulative risk approach they rarely test properly whether the relation between cumulative risk and child psychopathology is linear or nonlinear, with consequences for both theory development and intervention design: if cumulative risk impacts problem behavior in a positively accelerated exponential manner, for instance, it means that exposure to multiple risk is especially difficult to manage as problem behavior accelerates at a critical level of risk. Furthermore, few studies have actually examined factors that protect from negative outcomes in those exposed to cumulative risk and even fewer have explored cumulative protection in relation to cumulative risk. On the other hand, there is the view that a cumulative risk approach at least implicitly assumes that risk factors are, in essence, interchangeable. According to this view, the importance of testing for specificity should not be underestimated. Finally, the renewed interest in the role of neighborhood risk in child development has initiated a lively debate as to whether contextual risk should be operationalized at the family or the area level. In this letter I discuss these issues, and offer some suggestions as to how future research can address them.

### **Risk accumulation**

In the classic approach to cumulative contextual risk in child psychiatry (Rutter, 1979), organismic characteristics as well as proximal and distal qualities of the environment are modeled collectively. For each environmental construct a dichotomous classification of risk exposure is determined, typically by a statistical cutoff (e.g., greater than one standard deviation above the mean, upper quartile, etc.) or on the basis of a conceptual categorization (e.g., being below the poverty line). Cumulative risk is then calculated by a simple summation of the multiple risk categories. These risk categories are not weighted. This is for two reasons (Evans, 2003). First, the foundation of cumulative risk theory is that the confluence of risk factors rather than any singular risk, regardless of its context, is what leads to dysfunction because it overwhelms the adaptive capacities of the organism. In this framework no one risk factor is seen as more important than another. Second, weighted models do not outperform unweighted models over repeated applications. These models,

however, necessarily treat risks as if they are independent events when in fact they may not be.

In this approach, therefore, risk is viewed as an accumulation of stressors, and the number of risks that children experience carries more gravity than the experience of any particular risk. Indeed, cumulative risk indexes have been noted for their potential to capture the natural covariation of risk factors. For example, physical risk factors such as poor housing quality, noise and pollution are strongly interrelated as are psychosocial risk parameters such as family turmoil and violence (Evans, 2003). Furthermore, aggregate variables of risk are more stable than any individual measure, and there is increased power to detect effects because errors of measurement decrease as scores are summed and degrees of freedom are preserved (Burchinal, Roberts, Hooper, & Zeisel, 2000). Cumulative risk measures are consistently found to explain more variance in children's outcomes than single factors (Atzaba-Poria, Pike, & Deater-Deckard, 2004; Deater-Deckard, Dodge, Bates, & Pettit, 1998; Sameroff, Seifer, Baldwin, & Baldwin, 1993).

Despite this, the relationship between multiple risk exposure and child psychopathology using a cumulative risk approach has been little explored (Appleyard, Egeland, & van Dulmen, 2005; Gerard & Buehler, 2002, for reviews). It is encouraging, however, that recent developments in the field include theoretical developments (Schoon, Sacker, & Bartley, 2003, for a discussion). For example, a model of cumulative risk exposure was recently merged with new developments in neurobiology and allostasis theory (Evans, 2003).

Furthermore, it is not clear if the relationship between cumulative risk and children's problem behavior is linear or nonlinear, which is significant. If cumulative risk impacts problem behavior in a positively accelerated exponential manner, for instance, it means that exposure to multiple risk is especially difficult to manage as problem behavior accelerates at a critical level of risk. Although there is evidence for a linear relationship whereby increments in risk factors have a steady, additive effect on psychopathology in children (Deater-Deckard et al., 1998), few researchers (Flouri & Kallis, 2007; Gerard & Buehler, 2002; Simmons, Burgeson, Carlton-Ford, & Blyth, 1987) actually report whether their investigations included appropriate tests for nonlinear patterns of cumulative risk.

Finally, although recent studies have paid attention to the phenomenon of resilience (Masten, 2001), meaning a degree of resistance to adversities, few studies in child psychology and psychiatry have actually examined factors that protect children against

cumulative risk (Ackerman, Izard, Schoff, Youngstrom, & Kogos, 1999; Flouri & Kallis, 2007; Tiet et al., 1998). Even fewer have explored cumulative protection in relation to cumulative risk, despite calls for research in this area (Yoshikawa, 1994). Two teams of investigators have found evidence, analogous to that from the cumulative risk literature, indicating that resources can also accumulate providing better protection than single beneficial factors to children with high cumulative risk exposure (Dunst & Trivette, 1994; Zhao, Brooks-Gunn, McLanahan, & Singer, 2000).

### **Risk specificity**

A drawback of the cumulative risk approach is, however, the assumption that each risk factor carries the same weight in children's lives, and that risk factors are interchangeable. Risk factors do vary in their respective impacts (Ackerman et al., 1999). Therefore, one needs to also assess the influence of individual risk factors so that they could be examined simultaneously without losing their particular salience. Furthermore, risk indicators underlying the development of an outcome in one child adjustment domain might not be the same as those underlying the development of an outcome in another child adjustment domain. In other words, the importance of testing for full specificity should, equally, not be underestimated (McMahon, Grant, & Compas, 2003). A stressor-specific outcome-specific (full specificity) model includes a heterogeneous sample of stressors (i.e., single risk factors) and a range of child outcomes therefore allowing for specificity of both stressor and outcome to be determined (Sandler, Reynolds, Kliwer, & Ramirez, 1992). Studies finding that the number is more important than the type of risk factors in predicting a child outcome, for instance, would highlight the importance of having as broad a picture as possible of environmental components, as this would suggest that by identifying solely "extreme" risk on the basis of single risk factors children who may be at higher risk due to experience of multiple medium-level risks are neglected.

### **Risk levels**

Increasingly researchers recognize that neighborhood or area risk can, via the resources, services or role models that deprived areas provide, strongly influence children's mental health outcomes (Leventhal & Brooks-Gunn, 2000, for a review; Xue Leventhal, Brooks-Gunn, & Earls, 2005). Despite the numerous studies exploring family-level risk in child psychopathology and despite the renewed research interest in the role of neighborhood-level

risk in child psychopathology, studies do not usually compare the effects of these two levels of contextual risk. A recent exception is McCulloch's (2006) study which, using data from the second generation in the 1991 sweep of the 1958 British birth cohort and comparing 10 neighborhood types with family-level conditions, found that, although internalizing behavior problems were not related to neighborhood type, in predicting levels of externalizing behavior problems residence in a deprived area was as significant as the family factors. Within family research designs in studies of neighborhood effects (i.e., of children nested in families which were themselves nested in neighborhoods) are particularly useful as they allow the exploration of both within-family and within-neighborhood effects. However, it is not enough to outline future research questions without discussing some important analytical issues that research should take into account when answering these questions.

### **Analytical considerations**

To test for the effect of risk specificity vs. accumulation on a child mental health outcome one needs to compare the cumulative risk model with the specific risks model, and then identify the most parsimonious specification. The comparison of the goodness of fit between these two models must be carried out using an appropriate statistic that takes into account that the models are not nested, such as the Bayesian Information Criterion which is a function of the likelihood, the number of observations and the number of free parameters of each model (Schwarz, 1978). Assuming that it is found that the cumulative risk model should be preferred over the specific risks model then the functional form of the cumulative risk gradient can be tested by including polynomial terms. Testing the functional form of the risk gradient might sometimes be done more appropriately with exponential models. In exponential regression models the change in the mean of  $Y$  for a one-unit increase in  $X$  is multiplicative. In other words, for the linear model  $[E(Y)=a+\beta X]$   $E(Y)$  changes by the same quantity for each 1-unit increase in  $X$ , whereas for the exponential model  $[E(Y)=a\beta^X]$   $E(Y)$  changes by the same percentage for each 1-unit increase.

When logistic regression is used to model the effect of contextual risk on child mental health and the incidence of the mental health outcome of interest is common in the study population researchers should also remember that the adjusted odds ratio derived can no longer approximate the risk ratio. The more frequent the outcome, the more the odds ratio overestimates the risk ratio when it is more than 1 or underestimates it when it is less than 1.

Therefore, in these situations some form of correction of the odds ratio is necessary (Zhang & Yu, 1998). Furthermore, studies offering suggestions for interventions on the basis of their findings that the higher the number of risks experienced the worse the outcome should note that, because the largest cumulative risk is experienced by only a small portion of the population, prevention programs targeting that proportion of the population will not always have the greatest public health benefit to reduce the incidence of the outcome in the population (Tu, 2003). Studies comparing the use of relative risk and population attributable fraction (i.e., the proportion of cases in the study population that is attributable to the exposure (and potentially could be eliminated if exposure were eliminated)) are very informative in this respect (e.g., Davis, MacKinnon, Schultz, & Sandler, 2003). Obviously, since its formula is *Prevalence of exposure in the population\*(Risk Ratio-1)/1+[Prevalence of exposure in the population\*(Risk Ratio-1)]*, the population attributable fraction depends on the prevalence of the risk factor in the population, and so as the prevalence of exposure declines, the attributable fraction declines as well.

If the data are hierarchical data then multilevel modeling can be used (Goldstein, 1995). For example, in a three-level data structure (e.g., a dataset of children clustered within families which are clustered within neighborhoods), the simplest three-level multilevel model is a three-level random intercepts model. Multilevel models have several advantages over conventional fixed effects models. Firstly, when many neighborhoods and/or families exist in the dataset the practicality of fixed effects analysis is limited since estimation requires the construction of many dummy variables. In contrast, with multilevel models the number of clusters does not pose a problem. Secondly, a fixed effects analysis will only be efficient provided that the neighborhood and family-specific sample sizes are large enough. This is not a problem with multilevel analysis since estimation for multilevel models is based on the concept of borrowing strength. Thirdly, unlike fixed effects models multilevel models allow for the inclusion of both contextual explanatory variables and random effects.

Having established the modeling framework, the modeling process can be operationalized as follows: To start with variables that control for complex survey designs with multilevel data, if appropriate, can be entered first. Then a basic regression model that relates children's mental health to child-level explanatory variables can be used as a baseline model. Having selected the baseline model, the next step can add the family-level random effect. This model can quantify family effects through the computation of an intra-cluster correlation coefficients at family-level. This can be interpreted as the correlation, after

controlling for individual-level effects, between the psychopathology scores of two siblings in the same family. Next, family-level explanatory variables can be included in the model. This can determine how much of the family-level variation can be explained by fixed family-level variables. The neighborhood-level random effect can be included next, and this can determine how much of the existing variation can be attributed to the clustering of the sample in geographical areas. Entering neighborhood-level fixed variables in the next step of the model can show how much of the variation can be explained by fixed neighborhood-level variables. Interactions with family-level fixed effects and neighborhood-level fixed effects, as well as between family-level fixed effects and neighborhood-level fixed effects can be entered in the final step of the model. Therefore, within this modeling framework one can 1) explore the relationship between family and neighborhood contextual risk and child mental health, 2) compare family-level with neighborhood-level risk effects, 3) investigate factors that moderate the effect of contextual risk, and 4) investigate within level effects on child mental health. To further elaborate the structure of the random part of the model one can also investigate random coefficients models. As a complementary approach to multilevel models one can also consider novel alternatives, such as M-quantile models (Chambers & Tzavidis, 2006) which do not depend on strong distributional assumptions and can also serve as diagnostic tools for specifying the structure of multilevel models and for selecting contextual variables.

### **Future directions**

To develop the field future research should tackle several substantive issues as well. First, to echo others' calls for future research (Grant, Compas, Stuhlmacher, Thurm, & McMahon, 2003), studies should aim to develop a taxonomy of stressors similar to the taxonomies developed for child and adolescent psychopathology. This is because the variability in cumulative stressor measurement is often such that makes comparisons of studies almost meaningless. Second, studies looking at family contextual risk particularly should disentangle the family-wide from the child-specific risk factors. With few exceptions (Ackerman et al., 1999), previous studies group in the same cumulative risk index both behaviors which can be child-specific and family-wide factors such as household dysfunction or poverty. For example, the well-known Adverse Childhood Experiences (ACE) Study (e.g., Anda et al., 1999; Chapman, Whitfield, & Felitti, 2004; Dube et al., 2001; Whitfield, Dube, & Felitti, 2005), a retrospective longitudinal study of adult respondents in the United States,

operationalized cumulative contextual risk on the basis of the presence/absence of eight adverse childhood experiences: emotional, physical, and sexual abuse; a battered mother; parental separation or divorce; and growing up with a substance-abusing, mentally ill, or incarcerated household member. Third, even studies on family contextual risk that disentangle the family-wide from the child-specific risk factors often group together factors from theoretically dissimilar domains without justifying the decision behind their particular choice. Although this might seem to be in line with the cumulative index approach that the number is more important than the type of risks, if the selection of risk factors is not carefully justified but instead represents little more than a haphazard choice of variables then neither can the field move theoretically nor can prevention or intervention be discussed. Smaller indexes representing theoretically derived risk clusters might be useful in this regard (Ackerman et al., 1999). Fourth, they should explore if there is evidence for specificity or accumulation (Yoshikawa, 1994) of protection from contextual risk. Finally, they should use experimental and prospective longitudinal designs. Although retrospective or cross-sectional designs may suggest possible risk factors for outcomes, the test of the validity of these hypothetical relationships lies in prospective designs (Widom, Raphael, & DuMont, 2004) and experiments (Costello, Compton, & Keeler, 2003).

## References

- Ackerman, B.P., Izard, C.E., Schoff, K., Youngstrom, E.A., & Kogos, J. (1999). Contextual risk, caregiver emotionality, and the problem behaviors of six- and seven-year-old children from economically disadvantaged families. *Child Development, 70*, 1415-1427.
- Anda, R.F., Croft, J.B., Felitti, V.J., Nordenberg, D., Giles, W.H., Williamson, D.F., & Giovino, G.A. (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *Journal of the American Medical Association, 282*, 1652-1658.
- Appleyard, K., Egeland, B., & van Dulmen, M.H.M. (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry, 46*, 235-245.
- Atzaba-Poria, N., Pike, A., & Deater-Deckard, K. (2004). Do risk factors for problem behaviour act in a cumulative manner? An examination of ethnic minority and majority children through an ecological perspective. *Journal of Child Psychology and Psychiatry 45*, 707-718.
- Burchinal, M., Roberts, J., Hooper, S., & Zeisel, S. (2000). Cumulative risk and early cognitive development: A comparison of statistical risk models. *Developmental Psychology, 36*, 793–807.
- Chambers, R., & Tzavidis, N. (2006). M-quantile models for small area estimation. *Biometrika, 93*, 255-268.
- Chapman, D.P., Whitfield, C.L., & Felitti, V.J. (2004). Adverse childhood experiences and the risk of depressive disorders in adulthood. *Journal of Affective Disorders, 82*, 217-225.
- Costello, E.J., Compton, S.N., & Keeler, G. (2003). Relationships between poverty and psychopathology: A natural experiment. *Journal of the American Medical Association, 290*, 2023-2029.
- Davis, C.H., MacKinnon, D.P., Schultz, A., & Sandler, I. (2003). Cumulative risk and population attributable fraction in prevention. *Journal of Clinical Child and Adolescent Psychology, 32*, 228-235.
- Deater-Deckard, K., Dodge, K.A., Bates, J.E., & Pettit, G.S. (1998). Multiple risk factors in the development of externalizing behavior problems: Group and individual differences. *Development and Psychopathology, 10*, 469–493.
- Dube, S.R. Anda, R.F., Felitti, V.J., Chapman, D.P., Williamson, D.F., & Giles, W.H. (2001). Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life



span: Findings from the Adverse Childhood Experiences study. *Journal of the American Medical Association*, 286, 3089-3096.

Dunst, C.J., & Trivette, C.M. (1994). Methodological considerations and strategies for studying the long term effects of early interventions. In S.L. Friedman & H.C. Haywood (Eds.), *Developmental follow-up* (pp. 277–313). New York: Academic Press.

Evans, G.W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology*, 39, 924-933.

Flouri, E., & Kallis, C. (2007). Adverse life events and psychopathology and prosocial behavior in late adolescence: Testing the timing, specificity, accumulation, gradient, and moderation of contextual risk. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46, 1651-1659.

Gerard, J.M., & Buehler, C. (2004). Cumulative environmental risk and youth problem behavior. *Journal of Marriage and Family* 66, 702-720.

Goldstein, H. (1995). *Multilevel statistical models*. London: Edward Arnold.

Grant, K.E., Compas, B.E., Stuhlmacher, A.F., Thurm, A.E., & McMahon, S.D. (2003). Stress and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin*, 129, 447-466.

Leventhal, T., & Brooks-Gunn, J. (2000). The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin*, 126, 309-337.

Masten, A.S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist*, 56, 227-238.

McCulloch, A. (2006). Variation in children's cognitive and behavioural adjustment between different types of place in the British National Child Development Study. *Social Science & Medicine*, 62, 1865-1879.

McMahon, S.D., Grant, K.E., & Compas, B.E. (2003). Stress and psychopathology in children and adolescents: Is there evidence of specificity? *Journal of Child Psychology and Psychiatry*, 44, 107-133.

Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. In M.W. Kent & J.E. Rolf (Eds.), *Primary prevention of psychopathology: III. Promoting social competence and coping in children* (pp. 49-74). Hanover, NH: University Press of New England.

- Sameroff, A.J., Seifer, R., Baldwin, A., & Baldwin, C. (1993): Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development*, 64, 80-97.
- Sandler, I.N., Reynolds, K.D., Kliwer, W., & Ramirez, R. (1992). Specificity of the relation between life events and psychological symptomatology. *Journal of Clinical Child Psychology*, 21, 240-248.
- Schoon, I., Sacker, A., & Bartley, M. (2003). Socio-economic adversity and psychosocial adjustment: A developmental-contextual perspective. *Social Science & Medicine*, 57, 1001-1015.
- Schwarz, G. (1978). Estimating the dimension of a model. *Annals of Statistics*, 6, 461-464.
- Simmons, R. G., Burgeson, R., Carlton-Ford, S., & Blyth, D.A. (1987). The impact of cumulative change in early adolescence. *Child Development*, 58, 1220–1234.
- Tiet, Q.Q., Bird, H.R., Davies, M., Hoven, C., Cohen, P., Jensen, P.S., & Goodman, S. (1998). Adverse life events and resilience. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37, 1191-1200.
- Tu, S. (2003). Developmental epidemiology: A review of three key measures of effect. *Journal of Clinical Child and Adolescent Psychology*, 32, 187-192.
- Whitfield, C.L., Dube, S.R., & Felitti, V.J. (2005). Adverse childhood experiences and hallucinations. *Child Abuse & Neglect*, 29, 797-810.
- Widom, C.S., Raphael, K.G., & DuMont, K.A. (2004). The case for prospective longitudinal studies in child maltreatment research: Commentary on Dube, Williamson, Thompson, Felitti, and Anda (2004). *Child Abuse & Neglect*, 28, 715-722.
- Xue, Y.G., Leventhal, T., Brooks-Gunn, J., & Earls, F.J. (2005). Neighborhood residence and mental health problems of 5-to 11-year-olds. *Archives of General Psychiatry*, 62, 554-563.
- Yoshikawa, H. (1994). Prevention as cumulative protection: Effects of early family support and education on chronic delinquency and its risks. *Psychological Bulletin*, 115, 28-54.
- Zhang, J., & Yu, K.F. (1998). What's the relative risk? A method of correcting the odds ratio in cohort studies of common outcomes. *Journal of the American Medical Association*, 280, 1690-1691.
- Zhao, H., Brooks-Gunn, J., McLanahan, S., & Singer, B.H. (2000). Study the real child rather than the ideal child: Bringing the person into developmental studies. In L. Bergman, R.B.

Cairns, L.G. Nilsson, & L. Nystedt (Eds.), *Developmental science and the holistic approach* (pp. 393–419). Mahwah, NJ: Erlbaum.