probiotics are being aggressively marketed in these areas. Our study failed to show any practical benefit of lactobacillus rhomnosus: gorbach goldin in this setting. Even in industrialized countries, cost-benefit analysis of expensive probiotic therapy is warranted.

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References


Hispanic youth: violence and aggression ecological influences

To the Editor:

The focus on social influences of Hispanic youth is intriguing because they are an underrepresented ethnic group in family therapy research literature.1 Although Ferguson et al report that their results cannot be generalized across ethnic groups, Szapocznik et al has facilitated 25 years of research specifically with antisocial delinquent Hispanic youth.1,2

I wondered why the authors eliminated a review of school influence, considering that numerous research-based models of delinquency have concluded that poor school performance is a strong causal factor for antisocial behaviors in youth.3 Research has examined numerous risk factors associated with antisocial behavior of youth, supporting a view that the interaction of the youth and his or her social ecology, including the family, peers, school, neighborhood, and other community systems has the strongest relationships influencing antisocial delinquent behavior.3

Although the authors multivariate analysis included an equal number of female and male subjects, their conclusion ultimately left out any variance in male and female subjects, such as the influence of aggressive and violent behavior in their own dating relationships. Self-reports may not control for victim fear factors relevant to partner and guardian relationships; this is somewhat concerning. Reportedly, 33% of teens in the United States report some kind of abuse, and 12% of teens report physical abuse associated with intimate relationships.3 Demographic data of intimate relationship abuse in teens, school discipline reports, and juvenile offense statistics should be reviewed to control for minimizing of behaviors within self-reports.

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References


Reply

To the Editor:

Brooks comments on risk factors that we did not explore that also might play some role in youth violence. Naturally, in any single analysis, it is difficult to consider all risk factors, although we explored a number that had been identified as important in past research.

Looking into the influence of schools may bear some fruit, although we caution that it is too early to consider the influence of schools as “causal” as opposed to a correlational, predictive relationship. Although youth in low-functioning schools indeed have higher rates of violent behavior, this may reflect wider social influences of poverty, neighborhoods, and families.

Similarly, we agree that further explorations of dating violence risk factors are important. Our current sample ranged in age from 10 to 14 years; although we do not doubt that some of these youth were dating, there would not have been sufficient variance among such a young cohort to fully elucidate risk factors for dating violence. However, because it is our intention to follow this cohort longitudinally, examining dating violence will be a facet of future evaluations.

Finally, we fully agree that interactions among youth and their social ecology can have a strong influence on antisocial outcomes—indeed, this was a premise of our original article. We do make two cautionary remarks, however. First, the influence of any single risk factor tends to be very small. It is important to focus on the “big picture,” examining multiple
risk factors in concurrence. Small influences by single risk factors may turn out to be explained fully by other larger risk factors. Indeed, that appears to be the case for such variables as media violence exposure. Second, we note the paucity of research examining genetic influences and social influences in well-designed multivariate analyses. Given the strength of research linking genetics to violent behavior, it is crucially important that future studies incorporate genetic and social variables together. Only then will we have a full picture of the influences on youth violence.

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Reference


Exhaled nitric oxide and childhood asthma

To the Editor:

Sivan et al investigated the use of exhaled nitric oxide (FeNO) in the diagnosis of asthma in school-age children. They found a remarkable high diagnostic yield of FeNO and concluded that the test should be considered in the evaluation of children suspected of having asthma.

Earlier studies on the matter have led to inconclusive results. Unfortunately, neither Sivan et al nor other authors evaluated the additional value of FeNO compared with readily available information, such as a simple patient history. The authors did compare the diagnostic yield of FeNO with that of sputum eosinophils. It is not surprising that a combination of these 2 measurements did not improve the area under the receiver operating characteristic curve for the diagnosis of asthma, because both were highly correlated.

The clinically relevant question remains: What is the added value of FeNO compared with available information in clinical practice? We would be interested to see this analysis performed on the study material of Sivan et al. Besides a clinical history, it would be useful to take specific immunoglobulin E into account. It has been suggested that a large part of the association between FeNO and asthma may be explained by the correlation between FeNO and atopy.

Second, we want to express our concern about the exclusion criteria. Although not explicitly stated, it seems from the footnote of Table II that more than one-third of the children with asthma (n = 37; Table I) were excluded from analysis because of steroid use before the study inclusion. Information on earlier steroid use in children without asthma is not provided. Exclusion of steroid users selectively from the asthma group and not from the non-asthma group leads to biased results, with overestimation of the diagnostic yield of FeNO.

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References


Reply

To the Editor:

We thank Caudri and de Jongste for their interest in our article and thoughtful comments. Their first comment has been addressed at length in the discussion of our article and also in the meticulous debate presented by Bush and Eber. In brief, there is no question that asthma may be diagnosed without difficulty by the primary physician on the basis of typical history, response to therapy, and, when needed, additional tests. However, even though this statement holds for many or even most children in the community with clinical symptoms suggesting asthma, it may not be true for a relatively small percentage of children with less-specific complaints or who ignore mild to moderate symptoms or do not respond characteristically to treatment. Because of the high incidence of asthma, this group still includes a substantial number of children referred to special clinics. It is this minority, albeit a considerable load, that consumes more healthcare resources and can benefit the most from early diagnosis, with the emphasis on "early." We agree that the diagnostic yield of asthma also will increase in this minority population by adding IgE levels and other tests, such as skin tests for allergy, adenosine, exercise, and methacholine challenge tests. However, when the patient arrives for the first