Rethinking What Is Important

Biologic Versus Social Predictors of Childhood Health and Educational Outcomes

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Background: Social risk factors are often less vigorously pursued in clinical assessments of infant risk than are biologic risk factors. We examined the relative importance of early social and biologic risk factors in predicting poor health and educational outcomes in children.

Methods: The study was composed of all infants born in Winnipeg, Canada, during April–December 1984, who were followed up until age 19 years (n = 4667). Predictors were 3 routinely assessed biologic risks (birth weight, gestational age, and Apgar score) and 3 prominent social factors (mother's age, parent marital status, and socioeconomic status). Outcomes were childhood hospitalization and passage of a required high school examination. Analyses included logistic regression, measures of accuracy, and population attributable risk percent (PAR%).

Results: Biologic and social risk factors were associated with similarly steep poor outcomes gradients. Social risk factors had similar, and in some cases stronger, measures of association and accuracy. Using biologic risk criteria alone misclassified as low-risk 65% of cohort children who had high rates of later hospitalization and examination failure. PAR% associated with social risk factors exceeded biologic risk factors in most cases (eg, hospitalization PAR% = 4.4 for offspring of teen mothers vs. 1.7 for low birth weight).

Conclusions: In a population-based sample of infants followed-up through adolescence, early social risk factors were as threatening as, and more common than, routinely documented biologic risks—frequently identifying otherwise-unrecognized at-risk children. These findings together suggest that rigorous evaluation of social factors should be made a routine part of clinical assessment to more comprehensively and accurately identify infants at risk for later serious health problems and academic failure.

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When evaluating newborns or young infants for future health or developmental risk, physicians routinely consider a number of key biologic characteristics, including birth weight, prematurity, delivery complications, Apgar scores, maternal prenatal laboratory tests and health history, and important components of the physical examination. Although social characteristics, such as maternal history of drug or alcohol use, domestic abuse, or teen parenthood, are considered when known, such nonbiologic risk factors often do not receive the same level of systematic evaluation or chart documentation. For example, the American Academy of Pediatrics guidelines for preventive pediatric health care for newborns and infants lean heavily toward biologic measures such as height/weight monitoring and screens for metabolic disease, lead exposure, and anemia.[1]

A broader approach to child health risk factors, however, is well supported by the scientific literature. Biologic risk factors like low birth weight and prematurity carry well-known risks for negative long-term health and developmental consequences, but a rich literature also links early social risk factors to later health and illness in both children and adults. Adler et al[2] and Chen et al,[3] for example, provide extensive documentation of socioeconomic effects on adult and child health. Teen motherhood,[4] parental marital status,[5] and neighborhood of residence[6] also strongly influence child health and well-being.

Most studies examining how early biologic risks are related to later health either ignore common social risk factors or statistically remove them from analyses, rather than comparing their relative associations directly. Furthermore, child outcomes are frequently constrained to measures of single biomedical disorders, without placing those findings within the context of the child's overall functioning. In contrast to this trend, the 2004 Institute of Medicine (IOM) report,

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"Children's Health, the Nation's Wealth," calls for both a broader definition of children's well-being and an expanded view of the determinants of health and disease.[7]

This study responds to these calls and also addresses the recommendation of the IOM report to increase the use of regional administrative data on behalf of efforts to improve children's health.[7] Taking advantage of an unusually rich population database, we examined the relative importance of early social and biologic risk factors in predicting poor health and education outcomes in a large birth cohort of children followed up for 19 years.

METHODS

Population and Source of Data

The study examined all children born in Winnipeg, Canada (population 620,000) from April through December 1984, who continued to reside in the city until age 19 (in 2003).

All data were derived from the Manitoba Population Health Research Data Repository, which obtains province-wide data from the Ministries of Health, Family Services, and Education. Individual-level data and small-area census information are linked across data sets and over time using anonymized identification numbers. The validity and utility of the information in the repository have been well documented.[8–11]

The repository does not collect individual-level race and ethnicity information; however, the 2001 Canadian Census reported the Winnipeg population as 78% white, 9% Aboriginal, and 13% "visible minorities" (including individuals of Latino, African, or Asian/Pacific Islander descent). Canada uses a single-payer healthcare system, providing all residents access to routine health services, including emergency and specialty care.[12]

Variables Used in Analyses

Independent Variables

We selected 3 routinely documented biologic risk factors: gestational age, birth weight, and 5-minute Apgar score. These were compared with 3 social risk factors also available at birth: maternal age, parent marital status, and socioeconomic status (SES). Maternal age, although not a social variable per se, is used here as a marker for the higher-risk social settings in which young mothers often live. SES was determined using both individual- and group-level data. Because early analyses found that families with a history of receiving government income assistance made up a separate and higher-risk group than any SES level, children in families receiving income assistance for 2 or more consecutive months from 1995 through 2002 (the period of time these data were available) were placed in the lowest SES category. Families who had never received income assistance were divided into 4 SES groups on the basis of their neighborhood of residence using the Socioeconomic Factor Index. This validated measure, developed using principal components analysis, uses public Canadian Census data reported at the level of the dissemination area (average about 400 people), assessing unemployment rate, rate of high school completion, percentage of single-parent households, and female workforce participation.[13] Because SES remained stable for 85% of neighborhoods during the study period and because the 2001 census had greatly improved data quality, the Socioeconomic Factor Index score based on the 2001 Census was assigned to the family residence postal code at the time of the child's birth in 1984. The dual approach to defining SES—using both income assistance status and the index—had the benefit of a binary split to identify the highest risk children (those receiving income assistance) without losing the ability to further subdivide the large population of lower risk children using the less precise, but more widely available, neighborhood-based SES measure.

Dependent Variables

Two outcomes in different domains were used to represent long-term, cumulative assessments of child well-being: physical health and educational success (both assessed at the end of adolescence). Childhood hospitalization for any cause before age 19 years was used as the marker for child health. Hospitalization represents a relatively serious threat to child health and a form of healthcare utilization less influenced by access issues.[12] Use of "all-cause" rather than disease-specific hospitalization allowed for normal developmental change in age-specific causes of illness. Neonatal hospitalizations were excluded to reduce the effect of short-term, birth-related events, and provide a longer-term picture of health. Cumulative educational success was assessed by on-time passage of a 12th grade achievement examination required for graduation. Failure to graduate represents a serious threat to well-being in young adulthood and serves here also as a marker for a broad range of previous scholastic difficulties.

Grade 12 exams were coded as "pass" or "no-pass." Previous work has demonstrated that children whose examination scores were "missing" (eg, children absent the day of the examination, retained 1 or more grades, or withdrawn from school) were substantially less likely to graduate than those who took the examination on time and passed.[10] These children with missing scores (but known to live in Winnipeg) were all coded as no-pass. Earlier-than-expected examination scores due to grade acceleration were captured in the analysis and coded as pass. Previous work has shown that few children are electively held back a year at school entry (<2%); thus limiting the effect of any resulting misclassification.[10] Children in all types of schools were included.

Analysis

The biologic and social predictors were compared in several ways. We used logistic regression to examine relationships between predictor variables and C statistics to evaluate the explanatory power of the 2 sets of independent

variables (biologic and social), individually and combined, for the outcomes. Population attributable risk percent (PAR%) was used descriptively to compare the population-level relationships between biologic and social factors and the outcomes. PAR% was calculated using the formula PAR% = $[(Px \times (RR - 1))/(1 + (Px \times (RR - 1))] \times 100$.[14] PAR% confidence intervals (CIs) were calculated using the standard deviation of a bootstrapped mean PAR% derived from 100 samples of the population. Finally, we calculated specificity, sensitivity, positive predictive value, and negative predictive value.

The study protocol was approved by the University of Manitoba Health Research Ethics Board, the Manitoba Health Information Privacy Committee, Manitoba Education, Citizenship, and Youth, Manitoba Family Services and Housing, and the University of California, Berkeley. All analyses were performed using SAS version 9.0 (SAS Inc, Cary, NC).[15]

RESULTS

The study sample was composed of 4667 children. An additional 1754 children were not included in the analytic sample because they moved away or died before reaching age 19 years (27.0% and 0.3%, respectively). Using χ^2 and t tests, we compared birth parameters of the included and not-included children. Small differences were noted for gestational age (39.0 weeks [95% CI = 39.0–39.1] and 38.6 weeks [38.4–38.8] for the included and not-included populations, respectively), maternal age (26.9 years [26.7–27.0] and 26.7 [26.3–26.8], respectively), and SES designation (mean Index level, 2.43 [2.41–2.46] and 2.36 [2.31–2.40], respectively). However, mean birth weight and Apgar score, as well as the percentage of children with married parents, were the same in both groups. Comparing the analytic sample with children still living in Manitoba but no longer in Winnipeg and thus excluded (n = 1380), the rate of one or more childhood hospitalizations through age 19 years was 385 per 1000 (95% CI = 371–399) and 374 per 1000 (348–400), respectively.

Population Characteristics and Overall Outcomes

Table 1 presents the population distributions for sex and each of the 6 predictor variables. The on-time pass rate for the 12th grade examination was 62%. One or more postneonatal hospitalizations occurred for 37% of the children in the study sample. The most common International Classification of Diseases, 9th Revision categories for cause of admission were diseases of the respiratory (43%), digestive (11%), nervous (5%), or genitourinary (3%) systems; injury or poisoning (12%); infectious or parasitic diseases (5%); and "other" (13%), which included cellulitis or abscess, impetigo, contact dermatitis, chemotherapy, "abdominal or pelvic swelling," and "other aftercare." Details on the top 10 ICD-9 diagnoses at various ages are provided in Table A1.

TABLE 1. Distribution of Study Population Across Key Variables[a] (n = 4467)

Variables	No. (%)	Mean (SD)	Missing No.
Sex			0
Women	2248 (48.2)		
Men	2419 (51.8)		
Birth weight (g)		3437 (545)	57
<1500	22 (0.5)		
1500–2499	171 (3.7)		
2500–4000	3835 (83.2)		
>4000	582 (12.6)		
Gestational age (wk)		39.0 (2.6)	632
25–27	11 (0.3)		
28–30	22 (0.6)		
31–33	62 (1.5)		
34–36	221 (5.5)		
37–41	2868 (71.1)		
>41	851 (21.1)		
5-minute Apgar score		8.9 (0.7)	57
0–3	3 (0.1)		
4–5	15 (0.3)		
6–7	137 (3.0)		
8–10	4455 (96.6)		
Maternal age (yr)		27.4 (5.0)	0
<18	96 (2.1)		
18–19	185 (4.0)		
20–24	1036 (22.2)		
25–29	2113 (45.3)		
≥30	1237 (26.5)		
Socioeconomic status			12
On income assistance	433 (9.3)		
No income assistance			
Lowest SES	443 (9.5)		
Low-middle SES	768 (16.5)		
High-middle SES	2560 (55.0)		
Highest SES	451 (9.7)		
Parent marital status			4
Not married	779 (16.7)		
Married	3884 (83.3)		
Postneonatal hospitalizations			0
0	2936 (62.9)		
1–3	1544 (33.1)		
>3	187 (4.0)		
12th grade exam			0
Passed	2880 (61.7)		
Failed	316 (6.8)		
Absent	526 (11.3)		
11th grade or lower	783 (16.8)		
Not enrolled	162 (3.5)		

[a]Due to rounding, percentages may not sum to 100%.

Income assistance refers to family receipt of income assistance. SES is based on the Socioeconomic Factor Index for neighborhood of residence.

Relationships Between Predictors and Outcomes

Figure 1 presents the proportion of poor health and educational outcomes associated with the examined biologic and social predictors. Steep gradients in hospitalization and examination no-pass were associated with both the biologic risk factors (Fig. 1, left column) and social risk factors (right column).

Because the unadjusted graphs in Figure 1 allow children to be classified into >1 risk category, the data were evaluated to determine the degree of overlap between at-risk populations (Table 2, bottom). Data were available on all 6 risk predictors for 4026 (86%) of the cohort children. Most children (65%) had no risk factors, 8% had only biologic risk factors, and 23% had only social risk factors. A few (5%) had both biologic and social risk factors. Of those with some elevated risk (n = 1392, from Table 2: 300 + 905 + 187), only 35% had biologic risk (Table 2: (300 + 187)/1392). Thus, use of biologic risk criteria alone misclassified as low risk 65% of the children with an increased likelihood of subsequent serious health or scholastic problems.

Regression Analyses

Table 3 presents odds ratios from logistic regression analysis for postneonatal hospitalization and examination no-pass. Model 1 includes each predictor alone, model 2 combines the 3 biologic or social predictors and sex, and model 3 includes all 6 predictors and sex. Individually, each of the predictors was associated with postneonatal hospitalization, and all but the Apgar score were associated with examination no-pass.

Among biologic markers, only gestational age was associated with hospitalization, after adding the remaining biologic measures (model 2: OR = 0.95 [95% CI = 0.92–0.98]) or combined social and biologic factors (model 3: 0.96 [0.93–0.99]). In the full model, each additional week of gestation was associated with a 4% decrease in odds of postneonatal hospitalization. For the examination outcome, only birth weight was associated with no-pass in the full model (model 3: 0.86 [0.74–0.99]), with a 14% decrease in odds of examination failure for each additional kilogram at birth.

In contrast, the social predictors maintained a robust, independent relationship with both hospitalization and examination success after the addition of further variables. In the full model, odds ratios for postneonatal hospitalization were as follows: for maternal age, 0.94 (0.94–0.97), a 6% reduction in odds for every additional year of maternal age; for married parents, 0.75 (0.62–0.92), a 25% decrease in odds of hospitalization over that of children of unmarried parents; and for receipt of income assistance, 1.98 (1.43–2.75), a doubling of the odds when compared with the highest SES group. The odds ratios for examination no-pass were also pronounced, for example, a 50% reduction in odds of not passing for children of married parents (model 3: 0.48 [0.39–0.59]) and an 18-fold increase in odds for children in families on income assistance (model 3: 18 [12–28]). Each level of lower SES had a graded increase in risk of examination no-pass over the reference group (the highest SES category).

The explanatory power, using C statistics, of each individual social predictor was higher than the biologic predictors in every case (Table 3, model 1). The combined social predictors (model 2) had an explanatory power higher than that of the combined biologic predictors for both hospitalization (C statistic = 0.621 and 0.540, respectively) and examination no-pass (C statistic = 0.744 and 0.589, respectively). Combining all 6 predictors did not further increase the model explanatory power for either outcome.

Risk Prevalence and Population Attributable Risk

Table 2 lists the cut-points for defining "exposed" versus "not exposed" in each of the 6 risk categories and the prevalence of that risk category in the study population. These risk exposure cut-points were then used to calculate the PAR% associated with hospitalization and examination no-pass (presented in Fig. 2).

Figure 2 shows that for childhood hospitalization, the social risks of low SES, unmarried parents, or having a young mother were associated with a PAR% of 2%–8%, whereas the routinely documented biologic risks of prematurity, low birth weight or, poor Apgar scores were associated with a PAR% of 0.5%–3%. The examination no-pass PAR% associated with early biologic and social risk factors also varied widely with the PAR% higher for social than biologic factors (3%–15% vs. 0.6%–3%, respectively).

Measures of Accuracy

Table 4 presents calculations for sensitivity, specificity, positive predictive value, and negative predictive value, relating each predictor to each outcome variable using the same cut-points as the PAR% in Figure 2. Relatively similar measures of accuracy were found across biologic and social risk predictors. For postneonatal hospitalization, social and biologic predictors had similarly high specificities (most >90%) and generally low sensitivities, although sensitivities were substantially higher for most social predictors. Negative predictive values were similar for both groups of predictors (~65%). Positive predictive values for the social predictors were lower than those seen for conservative biologic cut-points (eg, very low birth weight), but higher than those seen with less-restrictive definitions of biologic risk (eg, low birth weight).

For the examination no-pass outcome, similar patterns were noted for social and biologic predictors, all with high specificity and low sensitivity. Negative predictive values were similar for both types of predictors, but the social predictors, overall, had higher positive predictive values.

DISCUSSION

In a large birth cohort followed up through adolescence, this study found strong associations between early

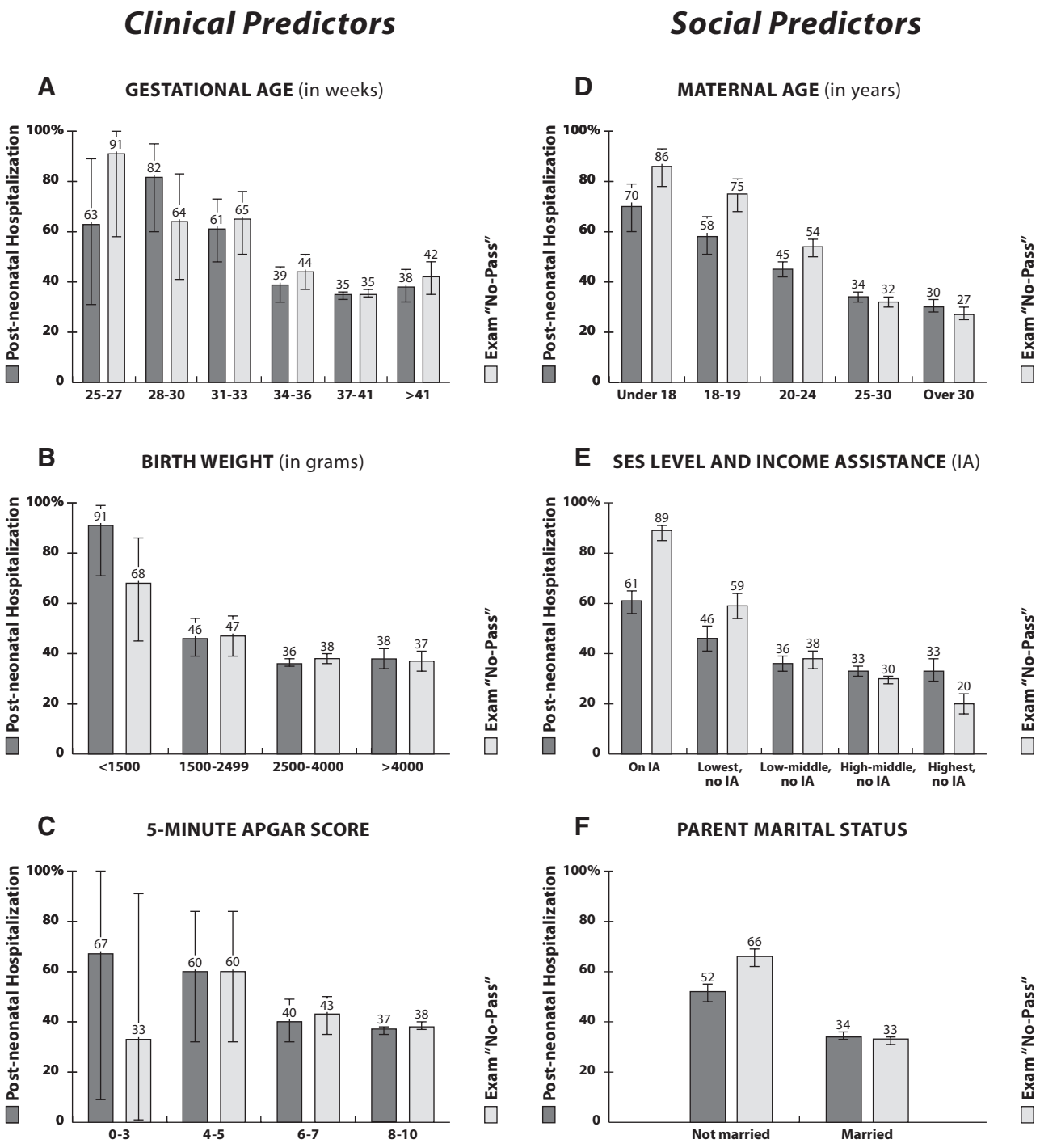

FIGURE 1. Proportion of poor health and educational outcomes associated with selected, early life biologic and social predictors. Results are presented with biologic predictors in the left column of the graphs: gestational age (**A**), birth weight (**B**), and 5-minute Apgar score (**C**); and social predictors in the right column of the graphs: maternal age at birth of cohort child (**D**), family socioeconomic status or receipt of income assistance (**E**), and parent marital status (**F**). Dark and light gray bars represent the proportion of children (with exact 95% confidence intervals) who experienced one or more postneonatal hospitalizations before age 19 years (overall mean, 37%) or who were defined as "no-pass" on a compulsory 12th grade academic achievement examination (overall mean, 38%), respectively.

TABLE 2. Prevalence of the 6 Biologic and Social Risk Factors Examined in the Population and the Prevalence of None or Multiples of These Risk Factors[a]

	Prevalence (%[a])
Predictor variables for elevated risk definition	
Birth weight	
VLBW (<1500 g)	22 (0.5)
LBW (<2500 g)	193 (4.2)
Gestational age (wk)	
<30	27 (0.7)
<34	95 (2.4)
<37	316 (7.8)
5-minute Apgar score	
<6	18 (0.4)
<8	155 (3.4)
Maternal age (yr)	
<18	96 (2.1)
<20	281 (6.0)
Socioeconomic status	
Family received income assistance	433 (9.3)
Lowest SES neighborhood, no family receipt of income assistance	443 (9.5)
Marital status (unmarried)	779 (16.7)
Multiple risks (n = 4026)[b]	
Zero risk factors	2634 (65.4)
Only clinical risks (1 or more)	300 (7.5)
Only social risks (1 or more)	905 (22.5)
Both clinical and social risks	187 (4.6)

Cut-points listed here were used to calculate PAR% in Figure 2.
[a]Percentage excludes those with missing data (see Table 1).
[b]Sample size with data available for all 6 predictor variables.
VLBW indicates very low birth weight; LBW, low birth weight.

social and biologic risk factors and later poor health and educational outcomes. Social risk factors (poverty, unmarried parents, or having a teenage mother) were stronger predictors of both outcomes, and, because social risk factors were far more common, they played a larger role in the overall population. Further, in terms of risk assessment, use of biologic criteria alone (prematurity, low birth weight, and low Apgar score) misclassified 65% of children who subsequently experienced high rates of poor health and educational outcomes as children with low risk. Few studies have compared the predictive accuracy of early biologic and social risks or used multiple methods to do so, and to our knowledge, none has simultaneously examined both health and educational outcomes within the same cohort. Our findings suggest that children's exposure to social risk is a medical issue meriting more serious and systematic attention and assessment.

Although the role of early social risk factors in the development of negative health trajectories has received increasing attention in the literature,[16] many healthcare providers still consider nonclinical risks as outside their domain. However, a growing body of work has demonstrated that early exposure to social factors can directly influence biologic systems through changes in neuroendocrine, immune, and autonomic nervous system function and thereby affect health far into adulthood.[17–19]

By comparing biologic and social risks, our findings provide vivid examples of the important relationship between early social risks and later health. For example, in the study population, infants born to mothers aged <18 years were more likely to experience postneonatal hospitalizations than low birth weight infants (70% vs. 46%, respectively) or infants born 2 months prematurely (70% vs. 61%, respectively). A low birth weight infant or one born 2 months early would appropriately be considered highly vulnerable medically both in the neonatal period and well past that first month. Beyond the intensive neonatal care provided early in life to these biologically fragile children, they would be followed up clinically much more closely and would likely receive a spectrum of additional neurodevelopmental, social, and financial supports. One need only consider the lack of medical or social resources routinely provided to infants born to 16- or 17-year-old mothers to recognize the different conception of risk applied to these socially high-risk infants by current medical and health policy standards.

One limitation of this study is that other important risk factors (eg, mother's education, child's IQ) were not examined. However, the selected risk factors represent some of those most routinely documented in newborn and infant medical records. This analysis also did not attempt to test a causal hypothesis or adjust for all possible confounding variables. Rather, we were examining the selected predictors as markers of underlying risk. Thus, used as a descriptive measure, the PAR% values presented may be illustrating upper bounds that have been inadequately controlled for the effect of other unexamined variables. Given the dichotomous nature of the examined predictors and the substantial passage of time between their measurement and the outcomes, it is likely that any causal relationships are far more complicated than the simple predictive associations described here.[20,21]

The measure of SES used here as well as the population examined, merit consideration. Although certain limitations are inherent in census-based socioeconomic information, previous research has established the validity and utility of census-based data in the United States,[22] and work in Canada has found that small-area socioeconomic data are well correlated with individual-level SES measures.[23] Potentially limiting the generalizability of the findings are the large numbers of First Nation indigenous residents, comprising at least 9% of the Manitoba population, making it somewhat atypical among North American provinces and states. However, in many ways, these families fill the same sociodemographic niche as impoverished, ethnic, and racial minority populations seen in all large North American cities. Importantly, previous epidemiologic work has

TABLE 3. Odds Ratios for Biologic and Social Predictors of Postneonatal Hospitalization and Examination No-Pass for Each Predictor Alone (Model 1), Combined With Remaining Social or Biologic Predictors (Model 2), and All Combined (Model 3)

	Postneonatal Hospitalization						Examination No-Pass					
Variables	Model 1 OR (95% CI)	C Statistic	Model 2[a] OR (95% CI)	C Statistic	Model 3[b] OR (95% CI)	C Statistic	Model 1 OR (95% CI)	C Statistic	Model 2[a] OR (95% CI)	C Statistic	Model 3[b] OR (95% CI)	C Statistic
Biologic				0.540						0.589		
Gestation age (wk)	0.94 (0.91–0.96)	0.518	0.95 (0.92–0.98)		0.96 (0.93–0.99)		0.95 (0.93–0.98)	0.518	0.97 (0.94–0.99)		0.99 (0.96–1.02)	
Birth weight (kg)	0.84 (0.75–0.93)	0.517	0.95 (0.83–1.09)		0.95 (0.83–1.09)		0.84 (0.75–0.93)	0.520	0.87 (0.76–0.99)		0.86 (0.74–0.99)	
5-minute Apgar score		0.516						0.510				
0–3	3.53 (0.32–38.9)		Nonconvergent		Nonconvergent		0.82 (0.08–9.09)		Nonconvergent		Nonconvergent	
4–5	2.65 (0.94–7.45)		3.28 (0.98–11.0)		2.91 (0.82–10.4)		2.47 (0.88–6.95)		3.06 (0.91–10.4)		2.34 (0.56–9.71)	
6–7	1.25 (1.06–1.48)		1.15 (0.96–1.39)		1.15 (0.95–1.39)		1.15 (0.97–1.37)		1.03 (0.85–1.25)		1.04 (0.84–1.28)	
8–10[c]	1.0		1.0		1.0		1.0		1.0		1.0	
Social				0.621						0.744		
Maternal age (yr)	0.94 (0.92–0.95)	0.593	0.96 (0.94–0.97)		0.94 (0.94–0.97)		0.89 (0.88–0.90)	0.654	0.93 (0.92–0.95)		0.93 (0.92–0.95)	
Married	0.48 (0.41–0.57)	0.553	0.71 (0.59–0.84)		0.75 (0.62–0.92)		0.25 (0.21–0.30)	0.598	0.50 (0.41–0.61)		0.48 (0.39–0.59)	
Socioeconomic status		0.576						0.679				
On income assistance	3.07 (2.34–4.05)		1.99 (1.48–2.67)		1.98 (1.43–2.75)		31.2 (21.4–45.3)		17.9 (12.1–26.5)		18.4 (11.8–28.4)	
Not on income assistance												
Lowest neighborhood SES	1.71 (1.31–2.25)		1.37 (1.04–1.82)		1.30 (0.96–1.75)		5.83 (4.32–7.87)		4.60 (3.36–6.30)		4.26 (3.05–5.95)	
Low-middle neighborhood SES	1.12 (0.88–1.43)		0.99 (0.77–1.27)		0.98 (0.75–1.28)		2.48 (1.89–3.26)		2.13 (1.61–2.83)		2.03 (1.50–2.75)	
High-middle neighborhood SES	0.97 (0.78–1.20)		0.92 (0.74–1.14)		0.94 (0.74–1.18)		1.70 (1.33–2.18)		1.58 (1.22–2.03)		1.56 (1.19–2.04)	
Highest neighborhood SES[c]	1.0		1.0		1.0		1.0		1.0		1.0	
Combined biologic and social						0.620						0.742

[a]Add sex and remaining 2 biologic or social predictors.
[b]Full model includes sex and all 6 biologic and social predictors.
[c]Reference category.

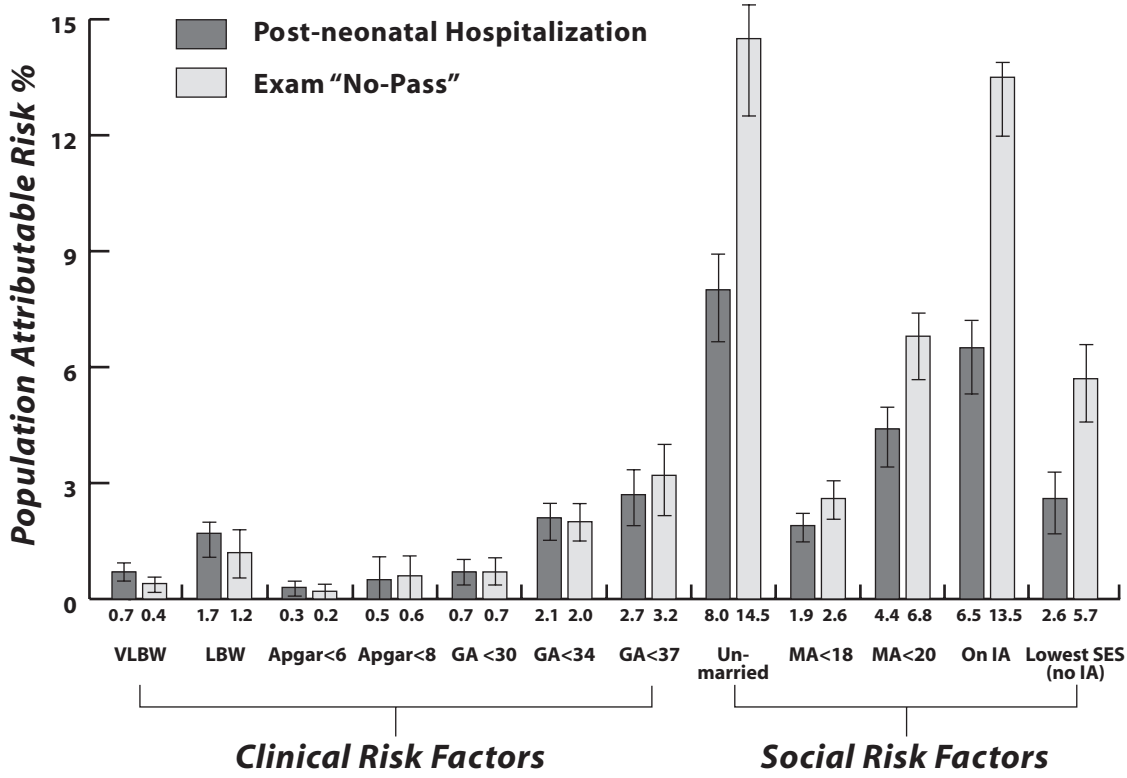

FIGURE 2. PAR% with 95% confidence intervals relating 3 biologic and 3 social risk factors to subsequent likelihood of postneonatal hospitalization through age 19 years or "no-pass" on a compulsory 12th grade scholastic achievement examination. No-pass was defined as failing score, absent on day of examination, retained one or more grades, or not enrolled. Table 2 provides the prevalence for each risk factor and binary cut-point used here. GA indicates gestational age in weeks; MA, maternal age in years.

found that minority status and ethnicity are inconsistently related to poor child health outcomes,[24] particularly when taking SES factors into account.[21]

The results presented here may underestimate the strength of the associations in a United States population. Low-income Manitoba children have higher rates of physician contact than do residents of more affluent neighborhoods, the opposite of findings in the United States.[25] Educational funding is also relatively equal across areas in Canada, with extra funding going to schools with more students from low-income families.[10] This is in direct contrast to most US school systems in which schools in less wealthy communities are likely to have lower per pupil spending, less-qualified teachers, and substandard facilities.[26,27]

Access to a population registry and a complete birth cohort reduced sampling bias and offered the inference advantages of longitudinal, rather than cross-sectional data. The Repository allowed the simultaneous analysis of 2 domains of child well-being over time, using complete records of hospitalization and scholastic success, in a large cohort of children—a combination of attributes unusual in the risk literature. This complete accounting of all those alive and residing in Winnipeg at age 19 compares favorably to longitudinal studies such as the 1958 British Birth Cohort, which reported a 71% follow-up among those still alive and residing in Britain at age 23.[28] Another strength of this study was the individual-level history of income support, which identified the highest risk families; the remainder could be subdivided using neighborhood-level data. Finally, no reliance was placed on parent- or self-report of health or academic achievement, a limitation common to other studies of this kind.[21,29]

A key notion of population attributable risk posits that reducing PAR requires that risk factors themselves be modified. Although once considered relatively unmodifiable, gestational age, birth weight, and Apgar scores are now routinely altered through advances in perinatal care and neonatal resuscitation. Social risk factors, similarly, are not cast in stone. Programmatic intervention and social policies exist, which have successfully reduced teen pregnancies in recent decades.[30] Social policies, arguably, could be used to increase the frequency of married or "partnered" mothers, and SES itself is potentially modifiable; witness Mexican and other Latin American efforts to augment household income and the attendant evidence of improvements in child health and development.[31] Although PAR% calculations inherently imply causation, in this study, they were used solely for the purposes of description.

TABLE 4. Measures of Accuracy for Selected Biologic and Social Predictor Variables

Selected Predictor Variables	Ever Hospitalized (Postneonatal to Age 19)				No-Pass on 12th Grade Exam			
	Positive Predictive Value	Negative Predictive Value	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value	Sensitivity	Specificity
Biologic								
VLBW (<1500 g)	90.9	63.3	1.2	99.9	68.2	61.8	0.9	99.8
LBW (<2500 g)	51.3	63.6	5.8	96.8	49.2	62.2	5.4	96.6
Gestational age (wk)								
<30	70.4	64.4	1.3	99.7	74.1	63.3	1.3	99.7
<34	66.3	64.9	4.4	98.8	67.4	63.8	4.3	98.8
<37	47.2	65.2	10.3	93.6	51.0	64.2	10.8	93.9
5-minute Apgar score								
<6	61.1	63.1	0.6	99.8	55.6	61.7	0.6	99.7
<8	42.6	63.2	3.9	96.9	44.5	61.9	3.9	97.0
Social								
Unmarried parents	52.0	65.9	23.4	87.2	66.0	67.3	28.8	90.8
Maternal age (yr)								
<18	69.8	63.6	3.9	99.0	86.5	62.7	4.6	99.6
<20	62.3	64.5	10.1	96.4	78.7	64.3	12.4	97.9
Family received income assistance	60.5	65.4	15.2	94.2	88.5	66.9	21.5	98.3
Lowest SES neighborhood, no family receipt of income assistance	46.1	63.9	11.8	91.8	58.9	64.0	14.7	93.7

Positive predictive value, negative predictive value, sensitivity, and specificity were computed using different cut-points for both biologic and social predictors. Predictor variables and cut-points match those presented for PAR% in Figure 2.

However, given the limits of prevention or modification of social risks, it is also important to consider options for reducing the likelihood of negative sequelae using, for example, regular nurse-home visits, improved support for breastfeeding and nutrition, routine developmental screening, parenting classes, or automatic referral to high-quality daycare or early educational settings. Large-scale interventions, such as the Chicago Child-Parent Centers[32] and other programs addressing child and parent health,[33] show that such interventions can have substantial positive effects and long-term societal cost savings.[34,35] These are exactly the types of interventions often used with biologically high-risk patients.

Social risk factors for subsequent hospitalization and scholastic failure are as threatening as, and more common than, routinely documented biologic risks. Because biologic and social risk factors often do not overlap, increased use of both is warranted to identify infants with greater likelihood of serious health and academic problems. Future work might usefully examine a broader range of risk variables and apply a functional, life course model such as that proposed by Hertzman et al.[36] Ultimately, however, the most effective interventions will require a deeper understanding of how common social risks "get under the skin" to affect human biology and influence the causal pathways to disease.[37]

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APPENDIX

TABLE A1. Top 10 Age-specific Hospitalizations in the Study Population According to the International Classification of Diseases, 9th Revision Diagnoses

Age			
1 mo to 1 yr	**1.1–5 yr**	**5.1–10 yr**	**10.1–15 yr**
Bronchiolitis	Asthma	Tonsillitis	Appendicitis
Pneumonia	Pneumonia	Asthma	Tonsillitis
Gastroenteritis	Tonsillitis	Pneumonia	Asthma
Acute upper respiratory tract infection	Bronchiolitis	Appendicitis	Abdominal pain
Bronchitis	Bronchitis	Arm fracture	Depression
Asthma	Croup	Abdominal pain	Pneumonia
Otitis media	Acute upper respiratory tract infection	Chemotherapy	Poisoning
Croup	Convulsions	Dental caries	Chemotherapy
Fever	Otitis media	Fever	Noninfectious gastroenteritis or colitis
Urinary tract infection	Fever	Urinary tract infection	Arm fracture