was associated with a reduction of head injuries up to 60% among children and adults. It is unlikely that this would have occurred if risk compensation due to ski helmet use generally results in an increase in individual risk-taking behavior. In addition, Hagel et al[6] found no evidence that ski helmet use increased the risk of severe injury or high-energy crash circumstances due to a higher speed. Their results suggest that helmet use in skiing and snowboarding is not associated with riskier activities leading to non-head–neck injuries.[6] Additionally, although ski helmet use has steadily increased worldwide over the past 10 years (up to 70% in Austria and Switzerland in the 2010 winter season),[7] the overall injury rate in alpine skiing remained constant at below 2 injuries per 1000 skier visits.[8]

In conclusion, available data do not support the risk compensation theory in this special field, and the risky behavior in some ski helmet wearers is no argument against the protective effect of ski helmet use.

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Estimation with Vanishing Baseline Risk

To the Editor:

Diseases associated with specific exposures may have little or no observable background rate in the absence of the exposure. Examples include mesothelioma (environmental asbestos), aplastic anemia (benzene), bronchiolitis obliterans (artificial butter flavorings), Reye's syndrome (aspirin in children), and angiosarcoma of the liver (vinyl chloride). Relative-rate models of exposure-response produce unstable near-zero baseline risk and unbounded coefficients, especially when age confounding requires baseline age dependence. The same problem arises in a proportional-hazards context. Baseline risk volatility also threatens meta-analyses, a procedure that assumes uniformity.

Using Poisson regression,[1] we investigated two methods: (1) fixing the intercept at a small value corresponding to 1% of attributable cases and (2) generating random sets of new cases across observation time independent of any predictor, possibly preempting true cases. Although models can be reliably fit using randomly generated cases, repetition would reduce variability in parameter estimates. We performed simulations with fixed intercepts (1,000) and with simulated populations (100) each with 100 random baselines. Hypothetical populations, constructed iteratively, consisted of 500 subjects with an exposure that could extend up to 200 time units. Exposure duration was random, favoring shorter durations to represent typical environmental or occupational exposures. Individual average exposure levels were randomly assigned and then randomly varied across time. We generated attributable cases with probability proportional to cumulative exposure, at which time follow-up ceased. Numbers of attributable or baseline cases averaged ~60–70. The analyses were implemented using an R algorithm[2] that called specific FORTRAN and EPICURE[3] steps with an indexing seed for random number generation. Additional information is included in the eAppendix (http://links.lww.com/EDE/A619).

The model specification was as follows:

$$\text{rate} = [\exp(\alpha)] \times [1 + \beta\text{cumX}] \text{ or}$$
$$\text{rate ratio} = 1 + \beta\text{cumX},$$

where cumX is an exposure metric, α is the intercept defining baseline risk, βcumX is the excess rate ratio, and β is the excess rate ratio coefficient.

Analyses were conducted as follows:

1. Attributable cases only
2. Attributable cases only, analyzed with fixed intercept
3. With added nonattributable cases
4. With added nonattributable cases analyzed with intercept fixed at known baseline risk (number of baseline cases/person-years of observation).

With the standard model, the excess rate ratio coefficient, β, varied widely across 1000 populations: mean = 13.4 (SD = 94.5) and range = 0.1-2834; with constrained intercept, the mean = 5.9 (0.76); range = 3.7-8.8. The mean of log(excess rate ratio coefficient) was 1.54 (SD =1.3)

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TABLE. Summary Comparisons of Estimation Performance With and Without Fixed Intercept or Random Baseline for Large and Small Population Simulations

	Large Sample (500)		Small Sample (50)	
	Mean	**(SD)**	**Mean**	**(SD)**
Log(excess rate ratio coefficient), log(β)				
Estimated intercept/no baseline (n = 1,000)	1.54	(1.34)	0.63	(1.86)
Fixed intercept/no baseline (n = 1,000)	1.77	(0.13)	1.69	(0.44)
Excess rate coefficient, [exp(α)] × β ($\times 10^5$), nominal value = 6.000				
Estimated intercept/no baseline (n = 1,000)	5.095	(0.90)	4.082	(2.19)
Fixed intercept/no baseline (n = 1,000)	5.981	(0.77)	5.989	(2.40)
Fixed intercept/random baseline, avg (n = 100[a])	5.964	(0.67)	6.278	(2.71)
Squared deviation: (excess rate coefficient − 6.0×10^{-5})^2 ($\times 10^{10}$)				
Estimated intercept/no baseline (n = 1,000)	1.62	(1.77)	8.48	(9.63)
Fixed intercept/no baseline (n = 1,000)	0.59	(0.87)	5.76	(9.33)
Fixed intercept/random baseline, avg (n = 100)	0.45	(0.46)	7.34	(14.5)

SD indicates standard deviation.
[a]Based on 100 iterations of study population each analyzed with 100 random baselines; average for each study population across the set of its 100 random baselines.

versus 1.77 (0.13) with fixed intercept (Table). The mean excess rate coefficient, exp(α)] × β, nominally 0.00006 in the simulation, was close to nominal with fixed intercepts (0.00005981), but biased downward in standard models (0.00005095) by 15%. The mean-squared deviation of the excess rate coefficient was substantially smaller with fixed intercepts (0.59×10^{-10}) versus standard model (1.62×10^{-10}), a 63% reduction.

In 100 simulated populations, each with 100 iterations of added baseline cases, estimates of excess rate ratio coefficient were much less variable than with standard models, especially with intercept fixed at the known baseline risk. The mean excess rate coefficient was now close to nominal with or without the fixed intercept (0.00005964 and 0.00006009, respectively). When the average squared deviation of the estimated excess rate coefficient was calculated within each set of 100 baseline iterations, the mean of those averages across the 100 simulated populations with intercepts fixed (0.45×10^{-10}), was comparable to that without baseline enhancements but with fixed intercepts (0.59×10^{-10}).

Simulations with small populations (n = 50) demonstrated greater bias (Table). The excess rate coefficient bias was 15% and 32% in the populations with 500 and 50 subjects, respectively. The two treatments for vanishing baseline yield equivalent results demonstrating that simply fixing the intercept is entirely adequate.

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Candidates at Risk for Postnatal Outcomes

To the Editor:

The recent extension of the fetuses-at-risk approach to outcomes originating in pregnancy and diagnosed postnatally remains controversial. Auger and colleagues[1] advocate a discussion on extending the fetuses-at-risk approach to neonatal and postneonatal outcomes, whereas Paneth[2] argues that such a formulation ignores the "transitions that permit live birth."

Whether fetuses or live births constitute the appropriate denominator for gestational age–specific neonatal mortality or gestational age–specific cerebral palsy hinges on how one conceptualizes the candidates for these outcomes. If the pathogenic process that causes neonatal death or cerebral palsy becomes established in utero, then fetuses constitute the appropriate denominator for these outcomes. Using the fetuses-at-risk approach for neonatal outcomes but not for postneonatal outcomes is a heuristic decision; for most (but not all) neonatal deaths and cerebral palsy, pathogenesis (neurologic injury in the case of cerebral palsy) occurs during pregnancy, whereas for many (but not all) postneonatal deaths, the causes arise postnatally. Thus, it would be appropriate to use the fetuses-at-risk approach for most neonatal outcomes and for cerebral palsy and sudden-infant-death syndrome but not for neonatal tetanus and most postneonatal outcomes.

If fetuses are the appropriate candidates for fetal death, neonatal death, and cerebral palsy, then fetuses (not live births) should be sampled for control selection in case–control studies, even if noncase status is ascertained subsequently. The Etude Epidémiologique sur les Petits Ages Gestationnels (EPIPAGE),[3] which followed a cohort of preterm infants (<33 weeks), illustrates this issue; the EPIPAGE cohort

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