To the Editors: We read with interest the recent article by Bray et al. (1) who studied age-cohort-period effects of incidence time trends of testicular cancer. They hypothesize “that similar temporal patterns in the cohort dimension imply that the etiologies of seminoma and nonseminoma are largely similar if not identical.” With the exception of Italy, they estimated similar birth cohort effects for seminoma and nonseminoma in several European countries.

We think that, from an epistemologic point of view, one cannot conclude that similar birth cohort effects by histologic subgroup imply similar etiologies. However, divergent birth cohort effects (given that classification errors and other biases did not occur) may imply different etiologies by subgroup. For example, smoking follows a strong birth cohort pattern in European populations. If cigarette smoke, a mixture of carcinogens, would contain a carcinogen A that specifically induced seminoma and carcinogen B that specifically induced nonseminoma, we would observe similar birth cohort effects, although the etiology differs by subgroup.

Bray et al. note that no etiologic difference “has been established with consistency.” They add that “difficulties in achieving sufficient statistical power to detect truly significant effects” make analytic studies problematic. However, Bray et al.’s own analyses do not provide information on the precision of estimates. For example, what is the precision of the incidence rate ratios by birth cohorts in the Czech Republic (Fig. 3)? Was the precision of their study high enough to conclude that these estimates are similar? In addition, we recalculated the incidence rate ratios (seminoma/nonseminoma) of the period 1994 to 1996 from Fig. 2. The ratios show a range of ~1.14 (France) up to 1.75 (Italy). The scatter plot does not imply a linear relationship between the incidence of seminoma and nonseminoma.

One cannot ignore the available literature that gives clues to different etiologies of seminoma and nonseminoma, although risk factors of seminoma and nonseminoma may overlap in some instances (2-7). Incidence analyses of germ cell cancers among children (excluded by Bray et al.) indicate that, up to the age of 15 years, nonseminoma is almost the only gonadal germ cell tumors and shows an early peak among boys ages 0 to 4 years (8).

In conclusion, the observation of apparently similar subgroup-specific birth cohort effects estimated with unknown precision can neither logically nor empirically (as based on several published reports) lead to the conclusion that the etiologies of seminoma and nonseminoma are “largely similar if not identical.”

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References
Etiologic Conclusions from Similar Birth Cohort Effects

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