To the editor:

Toppari and co-authors recently published a review on “the environmental connection” of cryptorchidism and hypospadias, including the potential adverse effect of alcohol (Toppari et al., 2010). Based on an article by Damgaard et al., 2007, they conclude that: “mothers’ alcohol consumption caused a dose-dependent increase in the risk of cryptorchidism” (Damgaard et al., 2007). We are not convinced that the article by Damgaard and co-authors actually present data that indicate a dose-response relation, and furthermore, it is not the only paper published on this topic (Jensen et al., 2007; Mongraw-Chaffin et al., 2008). None of these studies corroborate an increased risk of cryptorchidism with average weekly alcohol consumption. Damgaard’s findings included transient cryptorchidism, which could explain the incongruences if transient and persisting cryptorchidism do not share this risk factor. This is to our knowledge not supported by experimental or epidemiologic data. There is evidence to indicate that maternal and environmental risk factors do contribute to the occurrence of persisting cryptorchidism (Jensen et al., 2010). A large cohort study (n = 41,268) on both maternally reported cryptorchidism (which will include both transient and persisting cases) and persisting cases from treatment reports showed no association with average weekly prenatal alcohol exposure (Strandberg-Larsen et al., 2009). In our view, the combined body of evidence does not support the conclusion by Toppari et al., (2010) on maternal alcohol consumption and cryptorchidism.

REFERENCES


