cancer (Cvek and Dvorak, 2008). In comparison to bortezomib, disulfiram does not have several of the significant side effects, like peripheral neuropathy. In addition, since endometriosis usually is not treated as a potentially fatal disease, the reflections towards side effects may vary among patients (Guo, 2008). Consequently, there is a stronger demand for endometriosis drugs with a lower side-effect profile than anticancer drugs. This may be achieved by extensive preclinical testing and by exploiting the differential in sensitivity to the compound of interest or duration of treatment between target and normal cells in order to minimize any collateral damage to normal tissues (Guo, 2008). Given the lack of long-term efficacious medical therapy for endometriosis-associated pelvic pains and subfertility or for minimizing risk of recurrence, there is a clear and pressing need for novel medical therapies with more tolerable side effects and cost profiles. Therefore, in line with the advice of Dr Cvek, it is worth first trying disulfiram for the treatment of animals with endometriosis, and once we have achieved a considerable efficiency and safety, we will then perform clinical trials in humans.

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A new method for testing a hypothesis on a cause of polycystic ovary syndrome

Sir,

It is generally thought that hyperandrogenicity is becoming more common in women: moreover, the cause of this condition (or set of conditions) is not established (Azziz et al., 2006). Homburg and colleagues (2009), following Demissie et al. (2008) and Dumesic et al. (2007), has hypothesized that one cause of polycystic ovary syndrome (PCOS) is in utero exposure to high levels of androgens. The point of this note is to suggest a new method for testing this hypothesis.

There can be no reasonable doubt that high testosterone concentrations in women around the time of conception are associated with subsequent births of sons (James, 2004, 2008a). The point has recently been exploited in testing the analogous hypothesis of Baron-Cohen et al. (2003), namely that features of the autism spectrum syndrome are also caused by in utero exposure to high levels of androgens. I suggested that if my hypothesis and Baron-Cohen’s were both correct, then autistic probands should have a statistical excess of brothers (James, 2008b). In that paper, I documented such an excess; moreover, that finding has been replicated (Mouridsen et al., in press). These data provide strong support for Baron-Cohen’s hypothesis.

Accordingly, I suggest that if it were correct that one cause of PCOS is in utero exposure to high concentrations of androgens, then probands with PCOS should also have a statistically significant excess of brothers among their sibs (as contrasted with a population live birth sex ratio). The point could be tested by examining the sexes of the sibs of probands. How many brothers and how many sisters do they have?

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