FURTHER EVIDENCE ON THE HIGH PREVALENCE OF MALE FACTOR INFERTILITY DIAGNOSIS IN ISRAEL

To the Editor:

We read with great interest the recent report by Farhi and Ben-Haroush [1] on infertility causes in 2515 couples treated in two large primary infertility clinics in Israel during 1999–2007. This important update of local epidemiology which had not been thoroughly updated since 1977 [2] identified a remarkable nearly two-fold increase in male factor infertility diagnosis during the 30-year interim.

In Maccabi Health Services (MHS), the second largest health management organization in Israel, all members’ health-related interactions are automatically documented in a central computerized database. During the last several years MHS has established an automated registry of women diagnosed or treated for infertility. We used this database-driven registry to identify couples with physician-ascribed infertility diagnoses (ICD-9).

Out of a cohort of 87,400 suspected infertility cases between 1997 and 2010, we identified 29,282 with definitive diagnoses of infertility. Table 1 presents the distributions of causes of infertility in MHS compared to those described by Farhi et al. Similarly, male factor infertility was the most common cause, responsible for 48.80% of cases. The corroborations of this finding in a much larger population sample further emphasizes the high prevalence of male factor infertility diagnosis in Israel. It should be noted that most diagnoses of male factor infertility are based on semen analysis. A recent paper by Steeg et al. [3] concluded that the current World Health Organization criteria [4] for semen quality do not discriminate between fertile and sub-fertile men. Further studies are warranted to assess these seemingly secular trends in male reproduction function.

Table. Causes of infertility, MHS cohort, and Farhi et al.

<table>
<thead>
<tr>
<th>Definitive diagnoses</th>
<th>MHS (n=29517)</th>
<th>Farhi et al. (n=1953)</th>
</tr>
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<tbody>
<tr>
<td>Anovulation including PCOS</td>
<td>31.90% *</td>
<td>29.30% *</td>
</tr>
<tr>
<td>Mechanical</td>
<td>3.90%</td>
<td>10.00% **</td>
</tr>
<tr>
<td>Male</td>
<td>48.80%</td>
<td>37.50%</td>
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<tr>
<td>Combined</td>
<td>15.40%</td>
<td>23.10%</td>
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</table>

*Oligo-ovulation, **Tubal factor
PCOS = polycystic ovarian syndrome

References

Capsule

Genetic analysis of basophil function in vivo

Contributions by basophils to allergic and helminth immunity remain incompletely defined. Using sensitive interleukin 4 (IL4) reporter alleles, Sullivan et al. demonstrated that basophil IL-4 production occurs by a CD4+ T cell-dependent process restricted to the peripheral tissues affected. The authors genetically marked and achieved specific deletion of basophils and found that basophils did not mediate T helper type 2 (Th2) priming in vivo. Two-photon imaging confirmed that basophils did not interact with antigen-specific T cells in lymph nodes but engaged in prolonged serial interactions with T cells in lung tissues. Although targeted deletion of IL-4 and IL-13 in either CD4+ T cells or basophils had a minimal effect on worm clearance, deletion from both lineages demonstrated a non-redundant role for basophil cytokines in primary helminth immunity.

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Eitan Israeli

Capsule

How smoking effects body weight

Smokers are on average thinner than non-smokers, and many smokers gain weight when they quit. However, the specific cellular mechanisms of nicotinic receptors responsible for the effects of nicotinic agents on feeding are unclear. Mineur and co-scientists show that nicotine acts through α3β4-containing nicotinic acetylcholine receptors to increase hypothalamic pro-opiomelanocortin neuron activity, which then decrease feeding and body weight. Thus, nicotinic agonists may be useful for limiting weight gain after smoking cessation, and nicotinic drugs could also help control obesity and related metabolic disorders.

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Eitan Israeli