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LETTER TO THE EDITOR

Dietary fat consumption and endometriosis risk

Sir,

We welcome the recently published data evaluating the relationship between dietary fat consumption and endometriosis (Missmer *et al.*, 2010). However, we also feel some comments on the paper are appropriate.

First, we have some concern that the abstract may be misconstrued; readers may take the message that fish oil consumption might be beneficial in preventing endometriosis. The initial statement of the abstract indeed reads 'Fish oil consumption has been associated with symptom improvement in studies of women with primary dysmenorrhea and decreased endometriosis risk in autotransplantation animal studies'. On careful examination of the paper we eventually understand that pain improvement was shown for primary dysmenorrhea only (Deutch, 1995) but without a link to endometriosis-associated pain. The second half of the sentence suggests a therapeutic effect in preventing endometriosis based upon animal data (Covens *et al.*, 1988). The article only describes slightly smaller implants without any signs of apoptosis or cellular death, without evidence for prevention. We consider that the effect upon the transplanted endometrium is so limited that it might equally well be a consequence of a reduced inflammatory reaction masking the implant instead of evidence for regression.

The authors' assertion that 'These relations may indicate a modifiable risk' is speculation and that 'This evidence additionally provides another disease association that supports efforts to remove trans fat from hydrogenated oils from the food supply' is a premature conclusion. Indeed, an association cannot prove a cause and effect relationship, and in this article the effect is so weak (with an OR of 1.26) that these may thus be spurious correlations. From the data and elaborate analysis, we would conclude that the effect of dietary fat upon the incidence or severity of endometriosis, if any, is marginal and unlikely to be clinically relevant. The data seem not to support the conclusion that fish oil consumption is beneficial for the prevention of endometriosis.

The diagnosis of endometriosis was made by laparoscopy in women with pain or infertility. Since the reported incidence of endometriosis in these women is over 70% (Koninckx *et al.*, 1991), the association between the risk of undergoing a laparoscopy and fatty acid intake will therefore probably be as significant as the association between endometriosis and fatty acid intake. To us, this would rather suggest the conclusion that women with a high fatty acid intake are less likely to undergo a laparoscopy since a high fatty acid intake can reduce menstrual pain. Finally, laparoscopy as a diagnostic tool will pick up also subtle endometriosis and since these lesions can be found in almost all women with pain or infertility, all these women will finally get the diagnosis of endometriosis. Since it remains debated whether subtle endometriosis is a pathology, we would be interested to know whether more severe forms such as cystic and deep endometriosis would be affected by diet.

In conclusion, notwithstanding our appreciation for the meticulous analysis and the nice data, we do not consider that these data permit the conclusion that diet might affect the risk of developing endometriosis, certainly not of severe endometriosis.

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