A comparison of the probability of pregnancy can be performed by the use of the log rank test (Kaplan–Meier).

The authors, however, have used, according to the statistical methods described in their manuscript, the Fisher’s Exact test. It should be noted though that, this test requires that the observations are independent from each other, which, is not the case here, since some patients performed two cycles.

What is more important is that even by accepting the authors (inappropriate) choice of statistical methods (Fisher’s Exact test), the numbers they present in Table IV, do not support their claims of an improved probability of live birth, as shown by a \( P \)-level of 0.05. The application of a Fisher’s Exact test with the numbers provided in Table IV leads to a \( P \)-level of 0.099 and not 0.05 as the authors report.

Thus, even when analyzed by using inappropriate statistical methods, the data provided in this report cannot support the conclusion that administration of DHEA is associated with a higher probability of live birth.

Adopting DHEA as a beneficial intervention for the management of poor ovarian response should be guided by appropriately analyzed data originating from rigorously designed studies.

Reference


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Advanced Access publication on January 17, 2011

Reply: DHEA administration in poor responders

Sir,

Kolibianakis et al. raised important questions in their letter regarding the methods of data analysis used in our study.

They suggested analyzing the data by Kaplan–Meier survival analysis. This analysis estimates the survival function from life-time data. It might be used to measure the fraction of patients living for a certain amount of time after treatment. In our study, the time between the first and second treatments was short (1 month) and was for only two trials. The women in the two study groups had no large differences between them that would justify calculating a survival analysis. However, for further and larger studies with longer exposure to DHEA, this analysis could be done, also.

On the basis of previous retrospective studies where DHEA has a beneficial effect, we assumed the same tendency in this prospective study, and felt it was reasonable to perform a one-tailed test. When we summarized both cycles of the two groups, we found a higher live birth rate among the DHEA group, 6 (23.1%) versus 1 (4.0%), respectively \( P = 0.05 \). We erred in writing ‘two-tailed’ and not ‘one-tailed’ test in the Methods section.

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Advanced Access publication on January 17, 2011

Dietary fat consumption and endometriosis risk

Sir,

We welcome the recently published data evaluating the relationship between dietary fat consumption and endometriosis (Misser 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...