giving too many false negatives. Once we get a ‘truly normal’ population, we certainly need then to challenge it to a reference group of ‘sick patients’, in order to construct an ROC curve. So there was no other way than using a ‘certified’ PCOS group defined by the NIH classification, not using ultrasound parameters. As emphasized by Dr Perales-Puchalt, this gives the impression of having deliberately chosen the most extreme groups for obtaining an ROC curve with the highest performance, thus biasing the data in a more attractive way. Actually, this approach was detrimental to the sensitivity since it yielded a diagnostic threshold quite high, with the risk of getting a significant number of false negatives among patients with less severe forms of PCOS. This drawback is quite apparent in our Group 2 (suspected PCOS) for which the sensitivity of an AMH threshold >35 pmol/l or a number of follicles >19 is only 78 and 59%, respectively. On the other hand, a high threshold guarantees a good specificity and we believe that this conservative approach, i.e. favoring specificity at the expense of sensitivity, is preferable. Indeed, we believe it is more important to avoid overdiagnosis of POCOM or PCOS in a normal woman and diagnose instead idiopathic HA or idiopathic oligoovulation, which would be supported with the same care as a certified PCOS anyway.

We completely agree with Dr Perales-Puchalt who suggests that increasing the threshold of the follicle count from 12 to 19 is related to the inclusion of follicles <2 mm. This raises the question of the appropriateness of this threshold in relation to the US quality. However, proposing to count only follicles >2 mm, regardless of the machine, raises the concern of how to be sure that a follicle is 1.9 rather than 2.1 mm? This is awfully time consuming and inaccurate. For the echographer, it is much easier and realistic to count ‘every visible’ follicle. This emphasizes the concern about follicle count that cannot be accounted the women’s age. We selected all files where both AMH and blood type had been determined in the same blood sample between 2005 and 2010, n = 1020 dossiers. We analysed both blood type O and Rhesus factor. We performed the analysis both on crude results and by taking into account the women’s age.

Proportions of group O, A, B and AB, were 42.0, 41.7, 12.7, and 3.6%, respectively. Proportions of rhesus negative and positive were 12.9 and 87.1%, respectively. Mean AMH level was not different (P = 0.61) in groups O, A, B and AB (Table I). The percentages of cases with AMH <1.5 pg/ml were at the same level (between 26.5 and 31.0%, P = 0.36).

Neither was there any relationship between AMH and rhesus group for mean AMH (Table II, P = 0.41), nor for the percentage of low AMH (<1.5 pg/ml, P = 0.49).

Since AMH was strongly related with women’s age (from 6.8 ± 5.0 pg/ml below 30 years to 1.7 ± 1.8 above 40 years, P < 0.001), multivariate analyses of variance were conducted. If the effect of age was strong (P < 0.001 for both models on ABO and rhesus), the effect of ABO group and rhesus group on AMH remained far from significant (P = 0.81 and P = 0.28, respectively).

The analysis was finally restricted to a sub-sample of women aged 30–35 years, which did not alter the relationship between AMH and any of the blood group studied.

Thus contrary to Nejat et al., we were unable to find any relationship between blood groups and ovarian reserve. It must be stressed that AMH is supposedly a better marker of ovarian reserve than FSH. Moreover, authors acknowledged that they had no information on the exact timing of serum sampling in relation to the menstrual cycle, and FSH has huge variations during the cycle. They attempted to decrease the impact by restricting the analysis to women with E2 < 80 pg/ml, but this may not be totally adequate and, on the other hand, AMH has been demonstrated to be more stable during the cycle (Bungum et al., 2011). The authors analysed the data from two centres, with a view to minimize any potential race effect, but this also may add a confounding factor, even if a multivariate model was used.

**Blood type and ovarian reserve**

Sir,

Nejat et al. (2011) recently published a paper in *Human Reproduction* entitled ‘Implications of blood type for ovarian reserve’. They found...
In conclusion, our results differ from those of the authors, with different approaches. This means that further studies are needed, prospectively and with a more controlled approach in order to solve the question.

References


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Reply: Blood type and ovarian reserve

Sir,

We thank the authors for their interest in our work (Nejat et al., 2011). We concur that anti-Müllerian hormone (AMH) is indeed a more ‘reliable’ marker of quantitative ovarian reserve than FSH, and thus the null relationship observed by the authors is of particular interest to our group. We acknowledge that information on the timing of blood draw for estimation of FSH levels would have strengthened our work. The dichotomy in the observed relationships between the two studied populations may indeed, on the one hand, reflect an absence of any relationship between blood type and ovarian reserve (as the authors suggest); alternatively, the author’s null findings may relate to inherent population-related differences. Information on racial and ethnic makeup of their population sample would be relevant as ethnic differences in the studied populations may indeed, on the one hand, reflect an absence of any relationship between blood type and ovarian reserve (as the authors suggest); alternatively, the author’s null findings may relate to inherent population-related differences. Information on racial and ethnic makeup of their population sample would be relevant as ethnic differences in the studied populations may underlie the disparate findings. Alternatively, the association between FSH and ABO that we had observed may relate to FSH signalling and have nothing to do with quantitative oocyte pool (AMH being a more reliable reflector of this parameter than FSH). The relationship between ABO and FSH (as observed by our group) may thus reflect processes that are inherently distinct and unrelated to AMH (as studied by the authors). We would be particularly interested in hearing whether the authors observed any association between FSH level and blood type in their sample. We