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INVITED COMMENTARY

Sperm Biology

Is number of chiasmata an etiological factor of male infertility?

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Azoospermia and/or severe oligozoospermia can be treated as principal factors of male infertility. Specifically, azoospermia that stands for 1% of males and 10% of infertile male population has been mostly unexplained (idiopathic), however, it is suspected for its genetic and/or molecular background.[1] Pertaining to molecular background of severe oligo-astheno-teratozoospermia (OAT) – the meiotic abnormalities are suspected to reach almost 20%.[2] Therefore, this potentially addresses a significant number of cases that could find a possible etiological factor. Meiotic abnormalities as the authors point out encompass combination of anomalies in the process of pairing, synapsis, recombination, chromosome segregation and failures in controlling mechanisms – number of chiasma (diminished) could be one of the reasons.

After the long silence in this mechanism explanation, we have at last a report in which authors attempt to return to the hypothesis of number of chiasma studying metaphase I spermatocytes in a series of 31 infertile individuals.[3] A group consists of infertile males in whom only 10% has revealed karyotype polymorphisms (but not abnormalities) and five of them presented normozoospermia (infertility idiopathic background). The numbers of chromosomal units per metaphase I and chiasmata count per bivalent were established and according to statistical analysis two subgroups were subsequently formed (A and B) differing in number of chiasma counts. Chiasmata counts were subsequently correlated with defined karyotype (46, XY *vs* 46, XY polymorphism) and three categories of seminological parameters – normal, "abnormal non-OAT" and "abnormal OAT."

Several issues should be raised critically reading this manuscript. First of all, we may envy Local Bioethical Committee, which kindly granted permission for testicular biopsies that are aimed toward the patients (although infertile) who were far from azoospermic. In lot of countries, such invasive diagnostics could be forbidden apart of azoospermic cases. Nonetheless, due to this relaxed attitude at least we have access to the testis of rarely studied group of infertile males. A second issue is a lack of control group (retrospective comparison with the other data is not particularly convincing), specifically on the ground that the mean of number of chiasmata counts, in fact, provided similar results in infertile versus control groups (50.4 *vs* 50.7, respectively, see, Table 2).[3] As a matter of fact a distinction of these two subgroups analyzed in this particular study (Group A *vs* Group B) seems to have its origin in heterogeneity (range of data) within infertility cases than in objective statistical figures – particularly that meta-analysis (of results provided in Table 2) has not been performed.[3] Looking more closely into data provided we can be also surprised that out of 481 metaphases found only 412 were informative – that is, 14% was omitted, and this might have affected data produced exceeding the level of normal distribution. Taking seriously the significance between Groups A and B we could expect high percentage of individuals (approximately 48%) out of analyzed 31 infertile males that could potentially have revealed meiotic abnormalities etiology. However, it has not been directly addressed how many OAT cases have been contained within each group, we have only found an indirect evidence in the statement (results) of fewer number of chiasmata between "abnormal OAT" versus "abnormal non-OAT" category – however, this was not statistically significant (6 *vs* 10 OAT cases in cluster A *vs* cluster B; Figure 2). This puts into question abnormal meiotic background as etiological factor for OAT as the authors are trying to say. Hence, although hypothesis on activation of checkpoints and its consequence in the reduction in number of differentiating cells (finally leading to severe oligozoospermia or even azoospermia) seems to be sound, in our view, it may still wait for its proof. Yet, another intriguing question posed by the authors on the random event or repetitive event towards particular chromosome may also wait for its solution. First, the number of cases presented has not been sufficient, second we would like to know whether two cases including chromosome nine polymorphisms have been equally spread between Groups A and B - which, in fact, has been a case. Therefore, results generated do not put an additional light on ongoing debate concerning chromosome nine controversy in male infertility and its possible involvement in interchromosomal effect.[4] Overall, however, a paper sounds more than only intriguing.

COMPETING INTERESTS

The authors declare no competing interests.

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