Sperm Tail would Cause of Fertilization Failure due to the Genetic Problem

Editorial
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There is perfectly fascinating article which was published in Proceedings of National Academy of Sciences of United States of America (PNAS) in 2013 [1]. I had interview with the author, Dr. Lishko last year by e-mail. They show that the principal, progesterone-activated sperm calcium ion channel was disrupted in a CatSper2-deficient infertile patient. I strongly believe that their findings could be related to human male infertility and fertilization failure after in vitro fertilization in human.

Looking first at the genetic problem of infertility patient, they suggested that human CatSper mutations with infertility phenotype are quite rare because the patient must have homozygous mutations (the identical mutation in both alleles) for CatSper-less phenotype. Therefore, they only have seen two patients like this. The patient from PNAS paper had homozygous microdeletion in chromosome 15 which also affected CatSper2 gene, two of his brothers had the same deletions and all three brothers were infertile. They only examined sperm cells from one of the brothers. The men with affected CatSper, just like CatSper knockout mice and they theoretically should have normal sperm count and normal motility. However, sperm hyperactivation will be affected. Their patients had severe oligo-therato-asthenozoospermia, but probably due to the removal of other genes, adjacent to CatSper 2 locus. They never ever seen fertile men with CatSper locuses affected on both alleles. If only one is affected, the other one will rescue the phenotype. Heterozygous mice are perfectly normal, hence they assumed that humans should have similar trend. Additionally, three brothers indeed have very similar genetics with identical microdeletion [2].

Turning to the role of progesterone in sperm, progesterone is definitely required for fertilization process. In general, progesterone, which is present throughout the female genital tract with peaks of levels in the cumulus matrix surrounding the oocyte, stimulates several sperm functions, including hyperactivation and acrosome reaction. However, from recent findings, there are the similar trends at progesterone concentration in insemination media of human IVF used was detected in both patients with or without fertilization failure, while the patient exhibiting fertilization failure shows the induction of hyperactivation. Therefore, this demonstrated that fertilization failure is not due to hyperactivation and progesterone concentration producing by cumulus cells in human [3]. After I told about that, they said that it is very interesting. This means that other factors are involved, such as an ability to undergo acrosome reaction and so on. In addition, I did not have a case when sperm did not respond to progesterone and did not hyperactivate, I am not sure that the sperm still can fertilize an egg.

In conclusion, while lack of CatSper gene in patient could cause male infertility, semen quality of the patient might normal. Furthermore, progesterone can boost sperm motility at fertilization, however there was no clinical evidence that the lack of progesterone cause fertilization failure in human IVF.

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