

Vitamin A triggers meiosis

09 August 2006 | News



Vitamin A triggers meiosis

An Australian research team has solved one of biology's most fundamental questions – what triggers the germ cells to produce sperms in males and eggs in females. The finding is a breakthrough that could lead to improved infertility treatment, cancer therapy and pest management.

The team, led by Dr Josephine Bowles and Prof. Peter Koopman from the Institute for Molecular Bioscience at The University of Queensland, has discovered that derivatives of Vitamin A trigger the beginning of egg and sperm production, a process known as meiosis.

The cells that eventually turn into either eggs or sperm-known as germ cells-are identical in male and female embryos. According to Prof. Koopman, the timing of the reduction of cell division or meiosis determines whether the germ cell will develop into an egg or a sperm. In females, meiosis begins before birth and eggs are produced, whereas in males, meiosis begins after birth and the result is a sperm.

Prof. Koopman and his team found that retinoic acid, a derivative of Vitamin A, causes germ cells in female embryos to begin meiosis, leading to the production of eggs.

They also discovered an enzyme present in male embryos that wipes out retinoic acid and so suppresses meiosis until after birth, resulting in sperm production.

Nobody had been able to figure this out till now. It is expected that this discovery will provide the basis for a number of practical applications. It may allow researchers to improve fertility, for example in the case of an infertile couple wanting a baby, or suppress it, in the case of pest management.

Prof. Koopman has also suggested that an inappropriate retinoid signal might give the wrong instructions to germ cells, which could lead to the formation of germ cell tumors.

Novel approach to tackle avian flu

Chinese researchers have devised an innovative way to tackle the deadly avian flu. According to a pilot study conducted by Jiahai Lu of the School of Public Health at Sun Yat-sen University, Guangdong Province, China, passive immunotherapy is the best way to treat highly pathogenic avian influenza in humans.

Passive immunotherapy uses antibodies from horses. Purified antibodies to the H5N1 strain of avian influenza derived from horses were used against mice and it proved effective in preventing the disease. The researchers reported their findings in the journal *Respiratory Research*. Passive immunotherapy is an alternative strategy used to treat H5N1 infection.

The researchers reported two experiments, both using highly purified Fragment Antigen Binding (FAB) molecules derived from equine antibodies to the H5N1 hemagglutinin glycoprotein. In the first experiment, they showed that the equine (horse) antibodies prevented the infection of dog kidney cells. In the second one, the researchers showed that the equine antibodies prevented death from H5N1 flu in infected mice, which are known to be susceptible to the H5N1 flu. The mice were divided into four groups—each group containing 10 mice. The mice were infected with the H5N1 virus and after 24 hours three groups of mice were injected intraperitoneally with 50, 100, or 200 mcg of the antibody preparation. The fourth group, used as controls, received 200 mcg of normal horse sera. The H5N1 infection was fatal to all the control mice.

But seven of the 10 mice which were given 50 mcg of the antibody preparation and the mice that were given higher dosages survived. Dr Lu and colleagues reported that this method can be used until an efficacious vaccine, specific anti-H5N1 agents, and effective epidemiologic control measures for H5N1 virus infection was discovered. Dr Lu and colleagues concluded that H5N1-specific passive immunotherapy can be used for the early treatment of avian influenza patients to reduce the severity of illness and the likelihood of H5N1 transmission to others. But they also said that there is a major disadvantage as in these procedure polyclonal antibodies derived from horses has the potential to cause a strong host immune response, which might inhibit its use in the clinic.