

# Turkey Breeding Industry Sees Possible Breakthrough

Virgin birth in the turkey has been confirmed at The Pennsylvania State University to stem from eggs whose germ cells are normal.

The findings, which emerge after 12 years of cooperative research by Dr. Edward Buss and Dr. M. W. Olsen (USDA), clear the way for an intensive effort to determine if a virus is the trigger of the phenomenon, known as parthenogenesis.

Parthenogenesis, like cancer, is a form of unexpected cell proliferation. The term means virgin birth: female germ cells begin to divide and multiply without benefit of male sperm. Parthenogenesis is known in insects but the turkey is the highest animal in which it has been observed.

In that bird it is an extremely fragile mechanism, less than half of one percent of all eggs that are self-fertilized result in mature individuals.

Aborted embryos, spots of blood, or mere fragments of membrane are the issue of rule.

However, parthenogenesis could throw new light on the role of the virus in abnormal cell growth. It also has virtue that could prove a bonanza to the half-billion-dollar turkey breeding industry.

Until now, scientists have believed that some error in the process by which chromosomes separate (meiosis) was responsible for parthenogenesis.

But Buss and Olsen proved that no such errors take place. Eggs of parthenogenic mothers, they found, begin as haploids, that is, they contain one set of chromosomes, which is normal.

In view of this evidence, Buss and Olsen concluded that some outside agency is very likely the trigger of virgin birth in the turkey. That agency might be a virus.

In joint but deliberately separated experiments concluded in 1966, the two men had demonstrated that the fowl pox (a virus) seems to enhance parthenogenesis.

In a family of birds kept in a supposed virus-free environment at University Park, Buss, by selective mating, observed an 18 per cent increase in parthenogenesis but no embryos.

At Beltsville, when females were vaccinated with virus, the same 18 per cent increase was noted but, along with it, some embryos were produced. This was encouraging, but not conclusive evidence that a virus is involved.

Now, Dr. Buss has just embarked on a new experiment in which he will mate birds in the presumed absence of the virus, then remate the same birds after the male has been vaccinated with fowl pox.

"What we are after," says Buss, "is evidence as to whether the outside agent causing parthenogenesis is passed on—piggyback—from one generation to another. This would mean if had not only stimulated virgin birth but had infiltrated the cell nucleus and caused a genetic change. Positive results from the experiments—which are expected to take five years—would constitute strong evidence for the theory that a virus can trigger abnormal cell proliferation."

It has long been known that parthenogenesis may be triggered by mechanical means (one scientist got reproduction going in a frog by poking into its eggs with a needle), but it is not known if the virus works by mechanical or chemical means, or both.

If mechanical, the virus is presumed to act somewhat like a

speck of sand in an oyster: a foreign body, the sand stimulates growth that can result in a pearl. So the virus: its presence sometimes irritates the genetic material into activity.

The process, if it proves controllable, could lead to a pearl of spectacular dimensions for the turkey breeding industry.

For, if virgin birth can be deliberately induced in turkeys of top pedigree, by viral inoculation, a sort of "super-bird" might be achieved, one in which undesirable characteristics have been eliminated by parthenogenesis because of the fragility associated with it.

That fragility is due to inequities in one of nature's most delicate balancing acts: the "juggling" of genetic material (chromosomes) so that the contributions of both a male and female are intermixed.

In parthenogenesis, because no male is represented, this balance is impossible. Thus, "bad" genes from the mother have no chance of being tempered by "good" genes donated by a father.

The difficulty becomes manifest when cells undergo a genetic change known as diploidy. A diploid cell is one that has two sets of chromosomes. In normal germ cells, after

diploidy, the crucial balance is maintained between male and female donations.

In parthenogenic cells, however, when bad genes are doubled the balance is lost: their influence becomes lethal. This is why most parthenogenic offspring never see the light of day.

By the same token, those few who do survive (all males) are likely to be genetically clean: they must have been purged of bad genes or they would not have survived. If these "super-birds" can be mated to carefully selected females, considerable improvement might be made in a particular stock of turkeys.

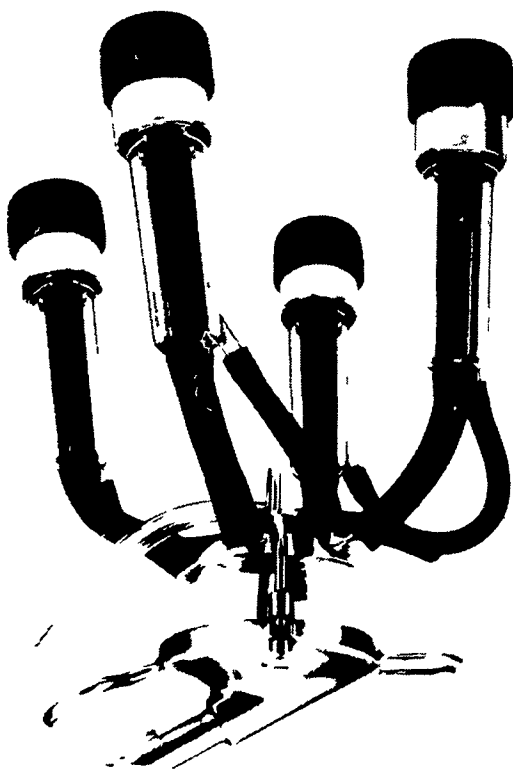
Dr. Buss' experiments are funded in part by the Agricultural Experiment Station at The Pennsylvania State University. Graduate Research Assistant Mr. L. J. Gaffney, of Australia, assists in one phase of the project.

The Buss-Olsen research into parthenogenesis was originally stimulated by the turkey breeding industry, which annually suffers millions of dollars of losses due to infertile eggs. These are thought to be the result of parthenogenesis which, as stated in the theory, usually results in abortions of only

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