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Optimal Phosphorus Management Technologies on Dairy Farms

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Phosphorus (P) management is a national issue with respect to water quality and the sustainability of animal agriculture. A major problem facing dairy farms is the surplus of phosphorus resulting from the excess quantity of the element in feeds and minerals. The cow can only utilize a small fraction of the nutrient, excess phosphorus is excreted in manure. It accumulates in soils, increasing the potential for phosphorus losses in runoff, which in turn contributes to accelerated water quality deterioration.

The traditional approach to reduce phosphorus problems is focused on the management of manure at the end of the production cycle, i.e. after manure is excreted. A more efficient and cost-effective approach of managing phosphorus on dairy farms is to eliminate excess of it in diets with optimal nutrient balances in the rations. This front-end approach minimizes phosphorus excretion in manure without impairing cow performance and farm profitability. This approach saves feed cost for the farmer.

In fact, phosphorus is an essential nutrient, needed by all plants and living species. The problem is that in many areas soils already have phosphorus buildup to levels far exceeding crop needs. Manure added as fertilizer compounds the problem. Run-offs from these high-phosphorus soils to rivers, streams, and the Chesapeake Bay impede the water quality and interfere with balance of plant and marine life in the watershed.

To help dairy farmers better manage phosphorus for enhanced farm profitability and environmental quality, a multi-state, multi-disciplinary project ($1,797,000 for year 2002-2005) supported by the USDA-IFAFS Program was recently initiated to develop optimal phosphorus management technologies on dairy farms. The long-term goal is to develop and deliver practical, scientifically sound, and economically viable phosphorus source control measures and management tools that sustain dairy farming and protect the environment.

Penn is the lead institution with **Dr. Zhengxia Dou** as the project director and **Dr. James Ferguson** the co-director. Researchers from five other institutions are collaborating on the project. They visit farms in their areas and collect samples, then send farm information and samples to Penn for processing and analyses. Drs. Dou and Ferguson are in charge of project planning, monitoring, implementation, evaluation, supervising, and reporting findings to USDA. They are also working with 20 dairy farms in Southeast Pennsylvania and Delaware to collect farm information and samples, just like the collaborators do for this project.

The specific objectives of the project are:

(A) Determine the dietary P range adequate for optimal cow performance but not in excess of animal needs by combining farm data across five states (PA, NY, DE, MD, VA) with research findings.

(B) Develop easy-to-use management tools including a fecal P testing procedure for assessing diet P adequacy vs. overfeeding and a modified ration formulation software program for balancing diet P and nitrogen.

(C) Establish quantitative relationships between dairy diets, fecal P, and P loss in runoff through laboratory and field-scale experiments.

(D) Provide training and education to veterinarians, nutritionists, producers, nutrient management personnel.

(E) Prepare a "white paper" on best management practices for P in dairy operations and present it to state/regional nutrient management commissions.

The five states participating in the project make up much of the Chesapeake Bay watershed.

The results of the study will be disseminated rapidly through existing multiple outreach channels. Project findings will be equally applicable on small, medium, or large farms. Project impact will be large-scale and long-lasting beyond the project period.

The principal investigators are **Dr. Zhengzia Dou** and **Dr. James D. Ferguson** at the School. The collaborating scientists are: Dr. L.E. Chase, Cornell University, Dr. K.F. Knowlton, Virginia Polytechnic Institute, Dr. R.A. Kohn, University of Maryland, Dr. J.T. Sims, University of Delaware, Dr. Z. Wu, Penn State University.

Clinic to bench

Christopher Hunter's lab on the second floor of Rosenthal is overflowing with equipment and people. In the broadest sense, the work here focuses on the role of cytokines in triggering immune responses. Cytokines, soluble messenger proteins, tell cells when to mount an immune response or when to stop such response. Cytokines are indispensable for the ability to fight diseases.

A major aspect of the basic research work here involves *Toxoplasma gondii*, a protozoan parasite that infects most warmblooded animals, including birds and man. Cats, domestic and wild, are the only known definitive host of the organism and serve as the main reservoir. Toxoplasmosis occurs world-wide. It is a major concern for pregnant women because the disease causes birth defects in fetuses. The disease is also a major concern for people with immune system dysfunction; here it causes meningoencephalitis.

Hunter's laboratory is home not only to bench scientists but also to clinicians who are pursuing basic research to enhance their clinical work. **Dr. Lillian Aronson, V'92**, assistant professor of surgery and head of the feline kidney transplant program at VHUP, and **Dr. Nicola Mason**, on leave from the section of medicine, and in the process of earning a Ph.D., are both working with Dr. Hunter's group to learn more about the immune complex.

Dr. Aronson came to the lab to evaluate the effects of a drug CTLA4-Ig on feline lymphocyte function. The theory behind this drug is that it is more specific in its mechanism of action, i.e. it can hopefully still prevent rejection (suppress T lymphocytes that are specifically involved with rejection), but also allow a patient to fight off an infection (not have an affect on memory T cells).

Some of her patients, after a kidney transplant when they received regular doses of cyclosporine and prednisolone as immunosuppressants to prevent rejection of the new kidney, suddenly developed acute generalized toxoplasmosis (because of a reactivation of a latent