Transmission of Avian Influenza Virus in an Urban California Central Valley Mallard Population
Alicia Bruce, SVM, UC Class of 2013, STAR STUDENT
University of California, Davis

Hypothesis:
Original: Transmission of avian influenza virus peaks during the summer months in the UCD Arboretum mallard population due to exposure of juvenile, immune-susceptible birds to virus shedding adults.
Revised: Avian influenza viruses found in urban ducks at the UCD Arboretum are related to viruses from wild waterfowl in the Pacific Flyway, and seasonal transmission of viruses can be identified by testing sera for the presence of antibodies.

Proposed Specific Aims:
1. Use a serological assay (bELISA) to detect seasonal prevalence of antibodies to avian influenza virus by testing UCD Arboretum mallard serum samples collected at 3 month intervals since March 2010.
2. Collect serum samples from adult and juvenile (hatch year) mallards in early summer 2011 to detect new viral infections based on AI antibody prevalence.

Results:
I performed the avian influenza ELISA on 350 mallard serum samples and found that antibodies to AI were circulating in the Arboretum mallard population in 2010 and 2011. Antibodies to AI were found in 4/31 (13%) of hatch year (HY) juvenile mallards, 156/257 (61%) of adult mallards, and 17/62 (27%) of unknown age mallards. Thus, antibodies were found at a higher frequency in adult mallards (61%) vs. juvenile mallards (13%) in 2010. The high prevalence in adults is likely due in part to an outbreak that occurred in 2008 (as found by the Boyce lab) and antibody persistence. The presence of antibodies in HY birds could only result from exposure in 2010 since these birds were not present in 2008. The presence of antibodies in recaptured individuals also provides strong evidence that AI transmission occurred in 2010, since 8 ducks seroconverted between sampling periods. AI antibody prevalence was highest in adults during June of 2010, and highest in HY mallards during March of 2010. These seasonality results differ slightly with literature showing AI transmission peaks in late summer. This disparity could be due to the small HY mallard sample size and the fact that we are looking at antibodies that can persist in birds for up to 12-15 months.

Serology revealed that AI infections occurred after 2008, and these results contrasted with environmental fecal sampling of the same population by the Boyce lab. AI virus was isolated from feces in 2008 only, though monthly sampling has continued through 2011. This disparity could be explained by the birds’ intermittent shedding of the virus. Infected individuals shed AI virus for 4-16 days post exposure and monthly sampling would have had to occur during this narrow window for positive results to have been obtained. Thus serology served as a better measure of exposure than previous fecal sampling in these populations.

Due to time constraints and lack of personnel availability, we did not collect new serum samples at the beginning of the summer 2011. Instead, I created a new objective with my mentor as follows:

New Specific Aim: 1. Evaluate UCD Arboretum AI sequences isolated from 2008 for phylogenetic relationships to other North American AI sequences

Results: Phylogenetic analyses using MEGA5 showed that H5N3 Arboretum sequences were most closely related to H5N2 mallard sequences from British Columbia in 2005. These sequences clustered more distantly with H5N3 from Guatemalan blue-winged teals in 2010 and Sacramento and Suisun mallard and teal sequences from 2007-2009. The H1N8 Arboretum sequence was near identical to a H2N8 sequence in a wintering California turkey from 2008. There was also a very close association with H3N8 sequences from a teal from Oregon in 2007 and an environmental sample taken in 2008 from an urban pond in Huntington Beach. This shows that close viral sequence relationships exist between urban and wild waterfowl of the Pacific Flyway. The findings could indicate that transmission is occurring between wild migrants and urban waterfowl in the Arboretum.