Research Week Recap: April 2012

Center for Persons With Disabilities

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RESEARCH WEEK RECAP: APRIL 2012

April 12, 2012 by Jolyne Lyon

The CPD salutes the undergraduates who introduced their work during Research Week. Here’s a look at what they’re doing:

Access to Education

Karen Hart, one of the students involved in the project, stands with her poster the the undergraduate research presentation.

A team of students from the Interdisciplinary Disability Awareness Class participated in a project to examine accessibility on the Utah State University campus. They looked for physical, technical and social barriers.

They worked with Utah State University to examine floor plans. They were encouraged to report their findings back so that they could help identify areas that needed improvement.

“We discovered that many existing accessibility features were not on floor plans, and some marked ‘accessible’ did not meet ADA (Americans with Disabilities Act) requirements,” the group reported. "We recommend that the project of mapping campus accessibility become a part of the IDASL class curriculum, making the surveying of campus buildings a service learning activity."

Student team members included Bemadette Caldwell, Karen Hart and Jennifer Maughn, with Macedonia and Lupita Damian as consultants. They were mentored by Jeanie Peck and Alma Burgess of the CPD. The team collaborated with Disability Resource Center Director Diane Baum and used WebAIM’s WAVE tool in some of its evaluations.

Copy Number Variation of C4 Genes in Autism

Elizabeth Robertson was one of the students involved in the research.

Students from the Biomedical Laboratory at the CPD continued to work on research investigating autism and its possible link to genes involved in immunity. Their conclusion was that the causes of autism are many, and more study needs to be done.

Undergraduates Curtis Steinfeldt and Elizabeth Robertson worked with Michal Benson and Dr. Jonna Westover, and with Dr. Anthony Torres, their research mentor.

“The C4 protein component of the complement system is made up of the protein products of the C4A ad C4B genes, which play an important role in innate immunity. An abnormal production level of C4A or C4B proteins can be attributed to deletion or duplication of C4 genes and is associated with several autoimmune diseases,” the team reported. Past research out of the CPD’s biomedical laboratory suggested that people with severe autism had lower copy numbers of C4B genes, while C4A gene copy numbers were higher.

This current study did not find a similar correlation.
"ASD has many causing factors," the researchers concluded. "This group does not seem to be affected by abnormal C4A or C4B gene copy numbers. Future studies of C4A and C4B genes in ASD subjects will need to be done in order to confirm the role of genetics and immune function in autism."

The researchers used DNA from newborn blood spots in their studies. The samples were from people identified with autism spectrum disorder and from control subjects.

The research was done in cooperation with the UC Davis MIND Institute, the CPD and the Utah State University Biological Engineering Department.

**Traumatic Brain Injury Training Module**

Deborah Blanchard was one of the team that worked on TBI.

Students from the Interdisciplinary Disability Awareness and Service Learning class took on Traumatic Brain Injury, with the objective of updating information and training regarding TBI, then making it available to the public online.

They incorporated new information about veterans and athletes. Eventually this training will be converted into a pair of modules that can be accessed on the CPD website.

Among the facts the team reported:

TBI hospitalization charges in Utah totaled over $91 million in 2009.

That same year, 2,395 Utahns sustained TBIs that resulted in hospitalization.

Another 471 Utahns died from a TBI.

The leading causes of hospitalizations included falls, motor vehicle traffic crashes and suicide attempts.

Students Katie Lovendale, Deborah Blanchard and Stefany Susanas worked on this project. Their mentors were the CPD’s Jeannie Peck and Alma Burgess.

**Toxoplasma gondii cyst formation in the female mouse brain**

Lori Stettler, a student who works at the CPD’s Biomedical Laboratory, was involved in a project at Johns Hopkins University involving parasites, mouse brains and schizophrenia. She worked on the research during an internship set up through Dr. Anthony Torres, who mentors her work at the CPD.

Toxoplasma gondii is a protozoan parasite known to cause birth defects in a fetus if a mother becomes infected during pregnancy. Cats can be carriers of the parasite; humans can carry it, too.

The researchers report the protozoan is also suspected to cause schizophrenia for these reasons: "1) individuals with schizophrenia have higher antibody titers to T. gondii than controls, 2) certain adult individuals with Toxoplasmosis have symptoms similar to schizophrenia, 3) Toxoplasma infections induce elevated levels of dopamine as commonly seen in schizophrenia, 4) individuals with schizophrenia have a
higher exposure to cats (Toxoplasma host) as children than control subjects, 5) mice infected with Toxoplasma have a lessened fear response towards cats and therefore are more susceptible to being eaten by cats."

The study involved three infected and three control female mice. Cysts formed in the brains of all three infected mice, and in none of the brains of the control mice. Microglia, or cells involved in an immune response in neural tissue, were also more prevalent around those cysts.

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