**Research Activity Report**
**Supported by "Leading Graduate Program in Primatology and Wildlife Science"**
(Please be sure to submit this report after the trip that supported by PWS.)

| | |
|---|---|
| | 2016. 04. 29 |
| **Affiliation/Positionn** | Primate Research Institute/D2 |
| **Name** | Duncan Wilson |

**1. Country/location of visit**

Kyoto University Yoshida Izumidono, Japan

**2. Research project**

Asura International Seminar

**3. Date (departing from/returning to Japan)**

2016. 04. 25 - 2015. 04. 25 (16:30 - 18:00)

**4. Main host researcher and affiliation**

Prof. Tetsuro Matsuzawa (Kyoto University Institute for Advanced Study)

**5. Progress and results of your research/activity** (You can attach extra pages if needed)

Please insert one or more pictures (to be publicly released). Below each picture, please provide a brief description.

**Title: The neuroscience of human social cognition.**
**Speaker: Prof. Ralph Adolphs (California Institute of Technology).**

*Introduction*

First, the concept of the neuroscience of social cognition was introduced. Prof. Adolphs argued that social cognition should be approached from a neuroscientific perspective, i.e. perception facilitates social attribution (by activating specific brain areas) which leads to social behaviour. He also suggested there are three kinds of knowledge; about the physical world (sensory or physiological), our own mind (metacognition) and the mind of others (social cognition). His goal is to develop computational models (e.g. single cell, networks) which can explain social behavior.

The main part of his talk focused on comparative neuroscience and social cognition studies involving autistic people, amygdala lesion patients, normal people, and monkeys.

*Autism studies*

In one study brain activity was compared between normal and autistic people when viewing video clips of semi-realistic social situations. Whilst visual cortex activity was the same in normal and autistic individuals (i.e. perception of low level features such as shapes and brightness), during socially embarrassing moments the ventromedial prefrontal cortex (responsible for processing social emotion) was less activated in autistic individuals.

In another study, normal and autistic people were shown pictures which were either non-social, facial expressions, or depicted social interactions. Although both groups showed increased activation in brain areas responsible for social emotion in response to the social stimuli, normal individuals showed much greater activation.

*Amygdala lesion studies*

In a famous study by Fritz Heider & Marianne Simmel (1944) people were asked to describe how simple shapes are interacting in a video. Normal people tend to describe the interaction using social words or phrases (even though the shapes and situation is non-social) - known as social attribution. However, recent research with patients with amygdala damage found that although they are able to describe how the shapes interact, they do not use social attributions. Therefore, the amygdala seems important for social cognition.

Some interesting case studies were also presented. One female patient with damage to her amygdala showed a loss of fear when presented with innately fear-inducing stimuli such as snakes, spiders and horror movies. However, her emotions of happiness and sadness remained intact. In addition, monkeys with experimentally induced amygdala lesions showed no fear when the experimenter approached their cage. This suggests the amygdala is important for processing fear in particular.

*Eye-tracking studies*

In an eye tracking study, pictures of human faces were presented to normal people, autistic people, and amygdala lesion patients. Normal people were found to look more at the eyes of the face, autistic people looked more at the area between the eyes, and amygdala lesion patients looked more at the center of the face (nose area). Deficits in brain areas responsible for different aspects of social cognition may be responsible for these variations in eye gaze patterns.

In another eye tracking study, humans with electrodes inserted into their brains (for epilepsy treatment) and monkeys with experimentally implanted electrodes were presented with a visual array of photos including human, monkey, natural and random pictures. Eye tracking revealed that the humans looked at human faces first, and the monkeys looked at monkey faces first. Brain activity recordings also showed the same pattern. However, humans had a delayed fixation on the stimuli compared to monkeys. This delay could be explained by greater semantic interpretation and social processing involved when humans view stimuli, resulting from differences in brain structure and size between humans and monkeys.

*Summary*

Social cognition depends on brain structures e.g. amygdala, ventromedial prefrontal cortex.
These structures communicate with each another as components of a network.
Implementation of computational models is necessary to understand these networks.
We need to understand at what point functional networks become dysfunctional networks (i.e. not due to individual differences or natural variation).

Overall, I found the seminar very interesting and informative. I was particularly interested in the extent to which human and non-human primates may differ in their ability to understand the social/semantic/emotional vs. low-level features of pictorial stimuli. This is an area I need to understand in greater depth, so that I can more accurately interpret my research findings.

**6. Others**

I would like to thank Prof. Matsuzawa (PWS Coordinator) and Prof. Tomonaga (PWS Mentor) for the opportunity to attend this valuable seminar.