Abstract Guidelines
These guidelines are very important. Please follow them exactly. See also sample abstracts below and on the following pages

Margins:
1 inch on the top, bottom, and right; but 1.25 inches on the left

Project Title:
Start it at 1 inch from the top
Bold and centered, Times New Roman, 14 pt font
Capitalize important words in the title (see samples below)

Names and Support:
Times New Roman, 12 pt font
Bold your name and your faculty mentor’s name, listed as shown in sample below
(Student Presenter, Faculty Mentor)
Type of Award: Bold, type of award you received (i.e., UROP, SURF, Beckman)

Text:
Use Times New Roman, 12 pt font
Limit: ~400 words… document must not be longer than one page.
Indent at beginning of each paragraph (0.5 inch).
After each period, leave ONE space only.

Sample of a partial abstract:

Promoter Methylation of the DRD4 Receptor Gene as a Factor in Schizophrenia

Student Presenter: Sara Vernam
Faculty Mentor: Melissa Christine, CAS Biology
UROP Award

Schizophrenia is a common psychiatric disorder (with a 1% frequency of occurrence worldwide) with various symptoms, including the impairment of reality perception and hallucinations. Schizophrenia is a complex disease that is linked to both genetic and environmental factors. Many of the genes linked to schizophrenia and many of the drugs used to treat this disease target proteins involved in dopamine metabolism.

Dopamine is a neurotransmitter that is released into the somatic cleft between neurons. Dopamine is synthesized in presynaptic neuron by tyrosine hydroxylase (TH), and taken up in the postsynaptic neurons……..
Global Remapping in the Rodent Entorhinal Cortex and Hippocampus during Exploration of Spatial Tasks

Student Presenter: Andrea Abi-Karam
Faculty Mentor: Michael Hasselmo, CAS Psychology
UROP Award

The hippocampus is essential for encoding memory; most of the information enters the hippocampus by way of the entorhinal cortex, a parahippocampal structure that connects the hippocampus with associated cortices. Research suggests that the medial entorhinal cortex functions as a neural map of the rodent’s spatial environment as represented by a hexagonal grid pattern. The basic unit of this map, the grid cell, generates action potentials at the apexes of a hexagonal grid that spans the surface of the rat’s environment. Such cells exist most prominently in layer two. The intersection of positional, directional, and translational information within layer two may allow the grid to update continuously during the rat’s self-motion-based navigation. The resonance frequency of firing differs depending on the location of the grid cell along the dorsal-ventral axis. Global hippocampal remapping occurs concomitantly with a coordinate shift in the firing vertices of the grid cells. Hippocampal remapping and grid cell realignment have been shown to occur simultaneously.

The use of two neural implant drives in the in vivo-electrophysiological recording paradigm will allow for simultaneous recording of hippocampal and medial entorhinal activity. This allows us to see if hippocampal remapping, grid cell realignment, or global remapping occurs during the rodent's exploration of two different contexts, and ultimately what remapping occurs during intra-trial context switches. The grid maps and hippocampal maps will be compared simultaneously to assess the role of remapping when a rat changes context.

Post-recording analysis focuses on theta-phase analysis, specifically on how it signifies memory encoding and decoding with the induction of LTP at the peak of theta and LTD induction at the trough of theta. By looking at the timeline on which this occurs in conjunction with histology, it may be possible to gain a better understanding of the mechanism underlying when global remapping occurs and when it does not.
Proteins in the myocyte enhancer factor 2 (MEF2) family of transcription factors are ubiquitously expressed in most mammalian tissues but are primarily active in striated muscle (cardiac and skeletal) and the brain. There are four transcription factors in the MEF2 family (MEF2 A-D) that help to regulate the function of muscle cells. It is not known how the different MEF2 proteins are able to bind particular promoters to start the transcription of a certain gene. For example, all MEF2 transcription factors bind the same consensus sequence in target gene promoters and in vitro they have been shown to act similarly. However, MEF2 factors likely activate different target genes in vivo based on the different phenotypes of mice with individual MEF2 genes “knocked out”. MEF2C knockout mice have shown that MEF2C is critical in embryonic heart development. Heart ventricles in MEF2C mice fail to form (embryonic day 7.5) making the knockout phenotype embryonic lethal. Two experiments were conducted 1) to determine whether there are cofactors that interact with the C-terminal sequences of the MEF2C protein, and 2) to identify the DNA-binding sequence specificity of MEF2C. To determine if there are any cofactors that interact with the C-terminal residues of MEF2C, cDNA sequences encoding the C-terminal residues of MEF2C were subcloned into the vector pGEX-2T which contains the GST fusion tag. This was done so that the GST-MEF2C protein could be used in a GST pull down assay to identify proteins that specifically interacted with the C terminus of MEF2C. To determine the DNA-binding specificity of MEF2C, a chimeric protein of MEF2C and MEF2A, which has one amino acid difference between the two, was constructed by using site-directed mutagenesis to create the relevant MEF2A point mutation in the MADS and MEF2 domain of MEF2C. This chimeric construct will be used in transfections in muscle cells to determine whether the target genes activated by the MEF2C/2A chimeric protein are more similar to those activated by MEF2A or MEF2C. This research will enable us to better understand the in vivo roles of MEF2C in striated muscle development because it is currently unknown how individual MEF2 factors activate specific target genes. These studies will help us to better understand how individual MEF2 factors regulate distinct transcriptional programs in vivo.
Dissecting the Interaction between STAT1, MEF2A and Angiotensin II on the myomaxin Promoter

Student Presenter: Katharine Davidoff
Faculty Mentor: Frank Naya, CAS Biology
UROP Award - Clare Boothe Luce Summer Undergraduate Research Fellow

Myomaxin is a gene that encodes a large actin-binding protein expressed specifically in cardiac and skeletal muscle cells. Previous studies have shown that expression of the myomaxin gene is upregulated in response to angiotensin II (AngII), a hormone known to cause hypertension (high blood pressure) and cardiac hypertrophy. Results from our lab have demonstrated that myomaxin expression is regulated by the transcription factor Myocyte Enhancer Factor 2A (MEF2A), one member of the MEF2 protein family. MEF2A regulation of myomaxin expression occurs through a MEF2A DNA-binding site located 75 bases upstream of the myomaxin transcription start site. Furthermore, recent research from our lab has demonstrated that an interaction between MEF2A and a second transcription factor, STAT1, is important in regulating myomaxin gene expression. STAT1 is a member of the STAT family of transcription factors, known downstream targets of AngII signaling. Intriguingly, the myomaxin gene contains a STAT binding site which overlaps the -75 MEF2A binding site. In previous experiments, we have shown that STAT1 is able to substantially activate the myomaxin promoter in the presence of MEF2A. This activation of the myomaxin promoter is further enhanced by the addition of AngII, demonstrating synergistic activity between STAT1 and MEF2A on the myomaxin promoter.

My previous research focused on analyzing the mechanisms by which AngII influenced MEF2A activity on the myomaxin promoter. Given these recent findings linking STAT1 and MEF2A, I sought to continue dissecting the interaction between AngII and MEF2A, but in addition, to investigate the role of STAT1 in enhancing or enabling this interaction. Luciferase reporter gene assays have demonstrated that the cooperation between STAT1 and MEF2A is conserved within the context of the most proximal 0.3 kb region of the myomaxin promoter. Interestingly, inspection of the myomaxin promoter DNA sequence revealed a second STAT1 binding site located 260 bases upstream of the transcription start site, within the 0.3 kb region. Because this site is a better match for STAT consensus sequence binding sites than the -75 site, I chose to focus on analyzing this region for evidence of STAT binding via electrophoretic mobility shift assays (EMSA). In addition to examining the promoter for evidence of STAT DNA binding, I am attempting to determine whether STAT and MEF2A interact via a protein-protein interaction to cause enhanced activation of the myomaxin gene. Therefore, I am generating a Myc-epitope tagged vector containing STAT1 in order to perform co-immunoprecipitation experiments with MEF2A. Through these experiments, I hope to determine the mechanism by which STAT1 interacts with AngII and MEF2A to increase activation of the myomaxin promoter. Taken together, these experiments may provide further insights into the cellular and molecular interactions that contribute to human cardiac development and disease.
The Influence of Age, Experience and Neurochemistry on the Development of Brood Care in the Ant *Pheidole dentate*

Student Presenter: **Anisa Djermoun**
Faculty Mentor: **James Traniello, CAS Biology**
NSF-REU Award (to Traniello)

Temporal polyethism, or age-related task performance, plays an important role in the organization of labor for many social insects. In most ant species, young workers perform tasks within the nest and progress to performing tasks outside of the nest as they age. One such task is the tending of brood. As ants age while tending brood, brain component sizes, biogenic amine titres and mandibular musculature increase. The physical and neural development that workers undergo as they age, while gaining brood tending experience, all support and influence age-related nursing capabilities. Because multiple causal associations among these factors could positively influence an ant’s ability to tend brood, an investigation of each would uncouple the relative roles of age, experience and neurochemistry in the development of brood-tending efficiency in *Pheidole dentata* minor workers. While studies show that minor worker age is correlated with increased nursing efficiency, the importance of age and brood-care experience, which may covary with maturation, is not well known.

The purpose of the first part of this study was to determine how well 10-day-old workers can brood tend if they lack prior experience. To differentiate between the relative contributions of age and experience, we compared brood-deprived minors to minors that gained experience tending brood for 10 days. For both treatment groups, brood-tending behavior was not significantly different and independent of worker rearing environment. Ten day-old *P. dentata* minor workers’ nursing abilities were not influenced by prior experience and development of task proficiencies gained during age-related repertoire expansion, which implies that the ability to nurse is innate. In the second study, we measured levels of serotonin and dopamine in both treatment groups of 10-day-old ants, and found that there was no significant difference in biogenic amine levels between control and deprived workers. In the final part of our study, we examined the abilities of control and deprived 10-day old minor workers to feed brood over a 24-hour period; this experiment measured the amount of weight gained in brood fed by control and deprived workers. Preliminary results suggest that there is no significant difference in weight gain of brood fed by both control and focal workers over the period of 1 day. Together with our first two studies, this implies that the ability to competently brood tend in 10-day-old *P. dentata* minor workers is an innate ability that does not require experience to be performed with high efficiency.
Investigations of the Religious Belief Systems and Thinking Traits of Individuals with Autism Spectrum Disorder through the Use of Online Discussion Boards

Student Presenter: Michaela Gaffley
Facult Mentor: Catherine Caldwell-Harris, CAS Psychology
SURF Award - NSF-AGEP

Autism Spectrum Disorder (ASD) is a disorder in which individuals display a triad of impairments. Individuals with the disorder range from low-functioning to high-functioning. These impairments are visible in social interactions (i.e., poor eye contact), social communication where there could be a delay in acquiring language, no language use at all (low-functioning), or an awkward use of language, and in their thinking and behavior characteristics. For example some individuals with ASD exhibit repetitive body movements such as head banging and rocking back and forth. Asperger’s (AS) is a form of autism, but there is no language delay and individuals have a normal or above average IQ. AS individuals have a high resemblance to high-functioning autism and some have savant like abilities as well as being able to communicate proficiently and have an understanding of linguistics. People with autism are high systemizers: they try to emphasize logic and reasoning and methodically work through things rather than use intuition. Neurotypical people that are religious tend to use intuition and feelings to make decisions and have faith.

Online discussion boards were used as the forum for the first phase of the research project. We hypothesized that the autistic individuals’ cognitive strengths and social-emotional deficits would influence their belief systems. We found that more individuals with ASD would classify themselves as Atheist (24 percent) versus neurotypicals (NT)—the general human population that was the control in the study—where only 17 percent classified themselves as Atheist. Also, more neurotypicals classified themselves as Christian (38 percent) in comparison to 17 percent of the ASD population. These findings supported our hypothesis.

We are analyzing the thinking traits of those individuals with ASD juxtaposed with thinking traits of neurotypicals. For the former these thinking traits include the following: emphasis on rationality, social disinterest, social discomfort, literal-mindedness, need for structure and focus on detail. For the neurotypicals, they include emphasis on intuition, oriented towards social rewards, empathizing, symbolic fluidity/gestalt thinking, and openness to experience. These will give us insight into ASD individuals reasoning behind religious affiliations.

This research impacts how individuals with ASD and their families interact and understand one another. Religious communities often provide support and encouragement for families and it can be especially difficult if a child or spouse does not have faith or has issues with the social aspect. Next there is an online survey with reputable measures in progress and finally we will have individuals physically come into the lab to continue our studies.