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On the Cover: Allison Barlows, Senior in Fisheries and Wildlife

McNair Staff
Vicki M. Curby, PhD
Director
Jeremy Bloss, BS
Student Services Advisor
Darlene Dixon
Program Assistant

Journal Designer
Karen Schmidt, Graphic Designer, Continuing Medical Education

Cover Photo
Allison Barlows

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As I end my eighteen year career with the McNair Scholars Program, it is my pleasure to introduce this outstanding collection of articles from the 2006-07 participants. The program is named for astronaut and scientist, Ronald E. McNair, PhD, who died in the Challenger explosion in 1986. Dr. McNair would be proud of the scholars who bear his name and appreciative of the faculty mentors who guided their research internships. McNair Scholars come from a variety of disciplines and their diverse reporting styles are reflected in the six papers that appear here in their entirety. The titles of the remaining research projects are also noted. All scholars are to be commended for their persistence through a rigorous undergraduate research experience that will serve them well in their graduate studies.

The fourteen of us who implemented this national program in 1989 did not fully comprehend the success and impact that it would have on the academy. The wholehearted support of so many staff, faculty, and administrative members of the MU community for the program over the years made my job a pleasure. I am honored and humbled to have been associated with so many exceptional scholars who are now shaping the future of higher education, our nation, and the world. My wish is for all who have been associated with the program to realize their dreams.

Farewell,

Vicki M. Curby, PhD
Director
Background

College students who are considering study beyond the baccalaureate level realize their dreams through the McNair Scholars Program at the University of Missouri-Columbia (MU). MU was one of the original fourteen universities selected to develop a program established by the U.S. Department of Education and named for astronaut and Challenger crew member Ronald E. McNair. The purpose of the program is to provide enriching experiences that prepare eligible students for doctoral study.

Program Elements

One of the most exciting aspects of the McNair Scholars Program is the opportunity for junior or senior undergraduate students to participate in research experiences. McNair Scholars receive stipends to conduct research and engage in other scholarly activities with faculty mentors from the areas in which they hope to pursue graduate study. These research internships are either for the academic year or for the summer session and are under the supervision of faculty mentors. For academic year internships, students work a minimum of ten hours per week during the fall and winter semesters. Summer interns work full-time for eight weeks.

McNair Scholars also attend professional conferences with their mentors, go to graduate school fairs, prepare for graduate school entrance exams, receive guidance through the graduate school application process and obtain information on securing fellowships, graduate assistantships, and loans. Participants learn about graduate school life, advanced library skills, and effective ways to present their work. At the completion of the research internships at MU, McNair Scholars make formal presentations of their research to faculty and peers at the McNair Scholars Conference and submit papers summarizing their work. Students who participated as juniors the previous year continue in the program during their senior year for graduate school placement and to further develop their skills.

Eligibility

Participants must meet grade point average standards; be U.S. citizens or permanent residents; and qualify as either a first generation college student with an income level established by the U.S. Department of Education, or a member of a group that is underrepresented in graduate education.

All students who wish to be involved submit an application to the program. A committee composed of faculty members and representatives from both the graduate dean’s office and the McNair Scholars Program selects participants and approves faculty mentors. Research internships are offered to those students who are juniors or seniors and are identified as having the greatest potential for pursuing doctoral studies.
Parenthood is a normative part of the adult experience in the United States. Most American men and women eventually become parents (United States Census Bureau, 2007). Because of the widespread experience of parenthood, parents are seldom asked why they chose to become parents in the first place. It is assumed that people parent out of a love of and concern for children. The same cannot be said for foster parents; the popular press, television and movies, and some academic writings speculate that foster parents engage in parenting for more instrumental reasons, especially financial gain. In this paper I explore the parenting motives that the general public attributes to foster parents.

Foster Care in the United States

The U.S. foster care system involves four main parties: the children, their biological parents, child protective services agencies, and foster parents. The U.S. Administration on Children and Families reports that over 500,000 children are in the foster care system. One-half of these children reside in non-kin placements or in what is known as traditional foster care. There are only 170,000 licensed foster homes nationwide (CWLA, 2006), and in most states, there is a shortage of licensed foster homes (Kirby, 1997).

A foster parent is a “resource that provides care for children in state custody” (ACF, 2006) and actively plays the role of a secondary parent with the state being the “ultimate parent” (Swartz, 2004). According to the National Foster Parent Association, foster parents feel that they are an “invisible” (Wozniak, 2002) part of the foster care system. They are made visible usually only when claims of abuse and neglect are made public in the press (Swartz, 2005). This portrayal of foster parents extends to television dramas and comedies that share the latent message that foster parents are corrupt. Storylines use abusive and uncaring foster parents as the catalyst for the actions of other characters. Foster parents are often presented as bad parents who take in children for monetary reasons only (Meyers, 2004; Swartz, 2005). Academic studies show that even though the foster parents get most of the blame for the inadequacies of the entire system, they are generally good, and state regulations provide an extra layer of protection for the children in care (Hendrix, 2003; Krebs, 2006; Meezan, 1985; Swartz, 2004; Wozniak, 2002).

Why would anyone want to be a foster parent? Studies have found that individuals become foster parents for several reasons. Explanations include, for example, infertility and a desire to parent, to increase family size, to continue the generational practices of fostering, to provide care to a child in need, and for financial gain (Baum, 2001; Cole, 2005; Orme et al, 2004; Hendrix, 2003; Andersson, 2001).

These motives are often characterized as selfish (e.g. infertility, money) or altruistic (provide care to a child). Swartz (2004) argues that this dichotomy between selfish and altruistic motives is too simplistic: multiple reasons underlie the decision to foster, and for many foster parents “changing a child’s life” and financial compensation are joint motives. Since the typical foster parent in the United States is also a member of the working class, often fiscal rewards often encourage these individuals to foster (Andersson, 2001; Cole, 2005; Swartz, 2004). Without the pay, Swartz (2005) argues, many foster parents simply could not take on the responsibility of fostering.
Gender, Parenting, and Paid Care Work

Parenting is heavily gendered and often refers to mothering more so than fathering. Motherhood is viewed as a natural part of womanhood and women are expected to be born knowing how to be a good mother whereas fatherhood is understood to be learned (Maill and March, 2003; Fox and Bruce, 2001). Foster care, as with other forms of care work, is assumed to be done by women following the traditional gendered roles of the male breadwinner and the female caretaker (Daly, 2001). Foster parents challenge the image of altruistic parenting because foster care is a sector of paid care work (Swartz, 2005). Since mothering is assumed to be natural, receiving pay for these services is frowned upon, and it is believed that women should foster out of the goodness of their hearts (Rhodes et al, 2003). However, it is assumed that men who foster father children are going out of their way to do something that they do not necessarily have to do and this is therefore, honorable.

Motherhood is central to women’s identity, and thus women may hold parents to a higher standard of proof of loving parenting than do men. Providing caring for pay is in direct contrast to the altruism that underlies the dominant care ethic in this country (Swartz, 2005). Thus, women may believe that anyone who receives payment in exchange for parenting is not practicing the virtue of caring (England, 2005). Therefore, it is expected that women will report more selfish motives to foster parenting than will men.

Methods

Data
Data for this study were provided by the Pew Commission on Children in Foster Care (hereafter PCCFC). The data were collected in 2003 from a nationally representative sample of 812 registered voters. The survey asked a range of questions addressing issues such as familiarity with the foster care system, accountability in the foster care system, reasons for children coming into foster care, and the motivations of foster parents. In addition, several demographic and background items were included.

Measures
Dependent Variable: This study focuses on the perceived motivations of foster parents. One question in the PCCFC data addresses the motives the respondent attributes to foster parents. The question used for the dependent variable is:

“Which of the following do you think BETTER describes why you think most people become foster parents? Do you think that most people become foster parents A) because of their concern for children, or B) because of the financial payments they receive?”

Respondents were read the first two responses, A and B, while response category C, both equally, was coded if the respondent offered this as an unsolicited response. A few respondents answered “not sure” (N=38) and were dropped from this study.

Independent Variables: Several independent variables are included in the analyses that follow. The primary relationship of interest is that between gender and perceived motives. For the gender variable women are coded as 1 and men as 0. Other variables of interest include age, race, education, income, parental status, and marital status. Age is measured as age in years and ranges from 18 to 75 and over. Race is categorized into two groups with whites coded 1 and people of color coded 0. Education is measured as highest year completed and ranges from a 1 indicating grade school to 9 for doctoral degrees. Household income is also treated continuously with 8 categories ranging from less than $10,000 to $100,000 and higher. Marital status is coded as 1 if the respondent is currently married and 0 if not currently married. Finally, parental status is coded 1 if the respondent has ever been the parent or legal guardian of a child and 0 if not. The sample characteristics are shown in Table 1. Individuals missing on any of these variables are excluded from the analyses.

<table>
<thead>
<tr>
<th>Table 1. Sample Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Age 18-29</td>
</tr>
<tr>
<td>Age 30-39</td>
</tr>
<tr>
<td>Age 40-49</td>
</tr>
<tr>
<td>Age 50-59</td>
</tr>
<tr>
<td>Age 60 and over</td>
</tr>
<tr>
<td>White</td>
</tr>
<tr>
<td>Black/ Hispanic/ Other</td>
</tr>
<tr>
<td>Less than high school</td>
</tr>
<tr>
<td>High school graduate</td>
</tr>
<tr>
<td>Some college</td>
</tr>
<tr>
<td>Bachelor’s</td>
</tr>
<tr>
<td>Advanced education</td>
</tr>
<tr>
<td>Married</td>
</tr>
<tr>
<td>Not Married</td>
</tr>
<tr>
<td>Parent</td>
</tr>
<tr>
<td>Not Parent</td>
</tr>
<tr>
<td>Household Income &lt;=$20,000</td>
</tr>
<tr>
<td>Household Income $21,000-$40,000</td>
</tr>
<tr>
<td>Household Income $41,000-$75,000</td>
</tr>
<tr>
<td>Household Income $76,000+</td>
</tr>
</tbody>
</table>

Source: Pew Commission on Children in Foster Care

Analyses
Two sets of results are presented. The first explores bivariate associations between the independent variables and the dependent variable. Chi-square tests of significance are reported. The second set presents the results of a multinomial logistic regression. In the regression analysis, the excluded category on the dependent variable is caring motive. This allows for comparisons of factors predicting the attribution of a caring versus financial motive and a caring versus both motives. The analyses for this study are adjusted with weights provided in the data. The data were analyzed using SPSS.
Findings

Bivariate Associations

Respondents differ in their attribution of motives to foster parents. Overall, 44% report that foster parents’ only motive is caring, 31% say the only motive is financial, and 25% say that both foster parents are equally motivated by both caring and financial reasons.

This study hypothesized that gender differences would exist in the attribution of motives to foster parents. However, given no other study has systematically examined this relationship, it is unclear whether men or women would show greater support for the caring versus financial motive. As shown in Table 2, there are significant differences by gender. Nearly 50% of men and 39% of women attribute a solely caring motive to foster parents. Conversely, women are more likely than men to report a financial only motive to foster parents (35% vs. 26%).

Table 2. Parenting Motives Attributed to Foster Parents

<table>
<thead>
<tr>
<th></th>
<th>Caring Motive</th>
<th>Financial Motive</th>
<th>Both</th>
<th>Chi-Square p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>49%</td>
<td>26%</td>
<td>25%</td>
<td>0.009</td>
</tr>
<tr>
<td>Female</td>
<td>39%</td>
<td>35%</td>
<td>25%</td>
<td></td>
</tr>
<tr>
<td>Age 18-29</td>
<td>56%</td>
<td>23%</td>
<td>21%</td>
<td>0.079</td>
</tr>
<tr>
<td>Age 30-39</td>
<td>49%</td>
<td>27%</td>
<td>24%</td>
<td></td>
</tr>
<tr>
<td>Age 40-49</td>
<td>37%</td>
<td>35%</td>
<td>28%</td>
<td></td>
</tr>
<tr>
<td>Age 50-59</td>
<td>42%</td>
<td>32%</td>
<td>26%</td>
<td></td>
</tr>
<tr>
<td>Age 60 and over</td>
<td>40%</td>
<td>35%</td>
<td>25%</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>45%</td>
<td>30%</td>
<td>25%</td>
<td>0.266</td>
</tr>
<tr>
<td>Black/ Hispanic/ Other</td>
<td>39%</td>
<td>34%</td>
<td>27%</td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>44%</td>
<td>30%</td>
<td>26%</td>
<td>0.044</td>
</tr>
<tr>
<td>High school graduate</td>
<td>45%</td>
<td>38%</td>
<td>17%</td>
<td></td>
</tr>
<tr>
<td>Some college</td>
<td>45%</td>
<td>25%</td>
<td>30%</td>
<td></td>
</tr>
<tr>
<td>Bachelor’s</td>
<td>45%</td>
<td>28%</td>
<td>28%</td>
<td></td>
</tr>
<tr>
<td>Advanced education</td>
<td>40%</td>
<td>30%</td>
<td>30%</td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>45%</td>
<td>30%</td>
<td>25%</td>
<td>0.840</td>
</tr>
<tr>
<td>Not Married</td>
<td>43%</td>
<td>31%</td>
<td>26%</td>
<td></td>
</tr>
<tr>
<td>Parent</td>
<td>43%</td>
<td>30%</td>
<td>27%</td>
<td>0.222</td>
</tr>
<tr>
<td>Not Parent</td>
<td>48%</td>
<td>31%</td>
<td>21%</td>
<td></td>
</tr>
<tr>
<td>Household Income &lt;=$20,000</td>
<td>39%</td>
<td>36%</td>
<td>25%</td>
<td>0.730</td>
</tr>
<tr>
<td>Household Income $21,000-$40,000</td>
<td>47%</td>
<td>26%</td>
<td>27%</td>
<td></td>
</tr>
<tr>
<td>Household Income $41,000-$75,000</td>
<td>44%</td>
<td>31%</td>
<td>24%</td>
<td></td>
</tr>
<tr>
<td>Household Income $76,000+</td>
<td>45%</td>
<td>32%</td>
<td>23%</td>
<td></td>
</tr>
</tbody>
</table>

Source: Pew Commission on Children in Foster Care

Discussion

In addition to gender, education is the only background characteristic significantly related to perceived motives of foster parents. In general, the more education someone has the more likely they are to attribute both caring and financial motives to foster parents. For example, 17% of high school graduates report both motives while 30% of advanced degree holders do.

There are several factors, though, that appear not to be systematically related to the attribution of motives. Age, race, marital status, parental status, and household income are all background factors that are not significantly related to the perceived motives of foster parents. For example, 21% of 18-29 year olds attribute both motives while 25% of individuals that are 60 and up do. 45% of whites attribute solely a financial motive while 39% of people in color attribute solely a financial motive to foster parents. Marital status is unrelated as well for example, 30% of married individuals attribute solely a financial motive while 31% of unmarried individuals do. Whether an individual is a parent is not related to the perceived motives.

Multinomial Regression Results

The results of a multinomial logistic regression are shown in Table 3. As shown, even after controls for several background characteristics, women are 1.8 times more likely than men to attribute a financial motive to foster parents than a caring motive. However, there are no gender differences in attributing both motives equally as compared to a caring motive only.

Although age was not significant in the bivariate analysis, age is a significant predictor of the motives attributed to foster parents in the regression analysis. After controls, it is shown that in general older people are more likely than younger people to believe foster parents are motivated by financial reasons more so than caring reasons (OR 1.088, p = 0.008). Although education was significant in the bivariate analysis, it is no longer a significant predictor of attributed motives once additional controls are added.

Table 3. Results from Multinomial Logistic Regression

<table>
<thead>
<tr>
<th></th>
<th>Financial Motive</th>
<th>Both Motives Equally</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-0.844</td>
<td>-1.165</td>
</tr>
<tr>
<td>Gender (1=female)</td>
<td>0.593</td>
<td>0.363</td>
</tr>
<tr>
<td>Age</td>
<td>0.085</td>
<td>1.055</td>
</tr>
<tr>
<td>Race (1=white)</td>
<td>-0.363</td>
<td>-0.700</td>
</tr>
<tr>
<td>Education</td>
<td>-0.062</td>
<td>0.116</td>
</tr>
<tr>
<td>Marital Status</td>
<td>-0.194</td>
<td>1.116</td>
</tr>
<tr>
<td>Parental Status</td>
<td>0.076</td>
<td>1.536</td>
</tr>
<tr>
<td>Household Income &lt;=$20,000</td>
<td>0.059</td>
<td>0.946</td>
</tr>
<tr>
<td>Household Income $21,000-$40,000</td>
<td>0.334</td>
<td>0.946</td>
</tr>
<tr>
<td>Household Income $41,000-$75,000</td>
<td>0.334</td>
<td>0.946</td>
</tr>
<tr>
<td>Household Income $76,000+</td>
<td>0.334</td>
<td>0.946</td>
</tr>
</tbody>
</table>

Source: Pew Commission on Children in Foster Care. Comparison response category is caring motive.

Despite the often negative images of foster parents shown by the press and media the majority of Americans believe that people that become foster parents do so because of altruistic motivations. Most people believe that individuals that become foster parents genuinely care for the child in need and are not
only motivated by the supplemental income they receive.

Nevertheless, women are more likely than men to see a selfish, especially monetary motive, at play for foster parents. An explanation of this is tied into the importance of motherhood on women’s identity and the virtue of caring (England, 2005). Receiving any type of supplemental income for care work, which foster parenting is a part of, is frowned upon and goes against the assumptions surrounding care. Yet, Swartz (2004) suggests that retaining naturalized and deeply gendered assumptions of care fails to recognize contemporary and future realities such as the foster care system.

It is also possible that women better understand the challenges of parenting and therefore, know that one would not take on foster parenting unless there was financial compensation involved. Women may not see foster parenting as any different from providing paid child care, and perhaps women are more likely to believe some care workers such as child care workers, are doing those jobs just for the compensation.

The belief that at least some foster parents foster simply for the money may discourage people from considering foster parenting in the United States. The number of licensed foster homes in the U.S. has declined over time; recruiting new foster parents is a significant challenge to foster care agencies (ACF, 2006; Meezan, 1985; NFCC, 2006). Only one in four adults in the United States would consider becoming a foster parent (ABC, 2006). Besides not having full autonomy over the children and often feeling “invisible” (Wozniak, 2002), the “for the money” motivation that is attributed to foster parents could have a negative impact on recruitment. It is likely some potential foster parents do not pursue fostering because they do not want to be stigmatized as someone who cares for kids only for the money.

There are several avenues for future research. First, as stated earlier, parenting is a gendered experience. However, little of the foster care literature systematically examines gender differences in the experiences of foster parents. Future research is needed to better understand how the experience of foster parenting differs for mothers and fathers. Second, research is needed to determine whether foster parents experience a stigma associated with being foster parents, especially the compensation stigma, and if so, how this stigma is manifested and how foster parents manage the stigma. Finally, much of the research on foster parent motivations has relied on samples of foster parents. Additional research is needed on representative samples of non-foster parents to explore which types of incentives (e.g. social, financial, moral) might influence them to become foster parents. This research could inform the recruitment process and lead to an increase in foster homes in the U.S.

References

Substance Use Among College Women
With Disordered Eating Attitudes and Behaviors

Disordered eating attitudes and behaviors, including anorexia nervosa, bulimia nervosa and binge eating disorders, represent a significant public health concern. Disordered eating (e.g., binge eating, purging, fasting) is associated with high rates of psychiatric comorbidity and serious medical complications including death (Herzog et al., 2006). Furthermore, rates of disordered eating have been rising in recent years among adolescents and young adults, leading many to call for greater research attention to this growing problem.

Past research has found substance-related disorders to be among the most common co-occurring problems for individuals with eating disorders (Milos, Spindler, & Schnyder, 2004) and for individuals with undiagnosed, but significant, eating pathology (Dunn, Larimer, & Neighbors, 2002; Fischer, Anderson, & Smith, 2004; Granner, Black, & Abood, 2002; Krahn, Kurth, Gomberg, & Drewnowski, 2005; Piran & Robinson, 2006; Ross & Ivis, 1999; Stewart, Angelopoulos, Baker, & Boland, 2000; von Ranson, Iacono, & McGue, 2002). However, it is unclear whether this increased risk for substance use and abuse is common to all types of disordered eating, or is specific to one particular pattern of disordered eating.

Types of Disordered Eating Attitudes and Behaviors

Eating disorders are characterized by severe disturbances in eating attitudes and behaviors (DE) such as bingeing, purging, dieting, fasting, and hard exercise. A binge is defined as eating an unusually large amount of food in a discrete period of time, usually accompanied with rapid consumption and feelings of loss of control. In an attempt to prevent weight gain after a binge episode, many individuals will compensate with other inappropriate behaviors. These compensatory behaviors include purging through self-induced vomiting, misuse of laxatives, diuretics and enemas and other methods such as fasting and excessive exercise.

Individuals with anorexia nervosa (AN) have an intense fear of weight gain and maintain a weight that is significantly below a healthy level for their age and height. Typically, these individuals achieve their low weight through reduction of total food intake, although some may binge, purge or use other compensatory behaviors. This diagnosis has two subtypes: restricting type (ANR) and binge/purge type (ANB). Individuals in the restricting type primarily achieve weight loss through fasting, dieting, or excessive exercise. ANR individuals do not regularly engage in bingeing or purging, whereas ANB individuals do engage in these behaviors.

Bulimia nervosa (BN) is characterized by frequent binge eating and subsequent compensatory behaviors to prevent weight gain. These individuals maintain a weight that is at or above normal. BN has two subtypes. BN purging type includes individuals who regularly engage in purging through self-induced vomiting, the use of laxatives, diuretics, or enemas. Individuals in the BN non-purging type use other compensatory methods such as excessive exercise, dieting or fasting.

Many individuals with disordered eating do not meet the strict criteria for BN and AN. These individuals may be diagnosed with an eating disorder not otherwise specified (EDNOS). This might include individuals who (a) despite significant weight loss
are still in the normal weight range or still have their menstrues, (b) use compensatory behaviors without binge eating, or (c) individuals who binge eat but do not use any compensatory behaviors. These latter individuals can also be classified as having Binge Eating Disorder (BED), a proposed new diagnosis currently being examined (American Psychological Association [APA], 1994).

The value and self-worth of individuals with disordered eating is often strongly influenced by weight and shape. They may also have an intense fear of gaining weight, intense desire to lose weight, and distorted body image. In addition, these individuals often experience feelings of inadequacy, insecurity, lack of confidence and inability to recognize and identify emotions and hunger sensations. Perfectionism, interpersonal distrust and maturity fears are also sometimes seen in individuals with eating disorders. (Garner, Olmstead, & Polivy, 1983)

Co-Occurrence of Substance Abuse and Disordered Eating

As noted above, the co-occurrence of substance use and abuse (SU) and disordered eating attitudes and behaviors (DE) has been well documented in clinical as well as non-clinical populations. Individuals with DE have shown higher rates of SU than individuals without DE (Krahn et al., 2005; Piran & Robinson, 2006; Ross & Ivis, 1999). Ross and Ivis (1999) found that individuals with DE were more likely than those without DE to have used tobacco, alcohol, marijuana, and a wide range of other illicit drugs including stimulants, cocaine, ecstasy, heroin, and hallucinogens. Similarly, Krahn et al. (2005) found that at-risk dieters were 50% more likely to report current drinking and more negative consequences from drinking than non-dieters. However, a few studies have failed to find these increased rates of SU and instead report similar rates of SU for those with and without DE (Dunn et al., 2002; Stock, Goldberg, Corbett, & Katzman, 2002).

Furthermore, research comparing rates of substance use among individuals with DE has some inconsistent findings. Some studies have found that there are no differences between individuals with AN versus BN and use of alcohol (Corte & Stein, 2000; Franko et al., 2005), marijuana (Corte & Stein, 2000; Herzog et al., 2006), amphetamines, cocaine, sedatives, hallucinogens, and solvents (Herzog et al., 2006). Others have found that individuals with BN are more likely than those with AN to have used alcohol and other substances (Wiederman & Pryor, 1996; Stock et al., 2002; Blinder et al., 2006). In a review of the literature of substance abuse and eating disorder comorbidities, Holderness et al. (1994) conclude that much of the inconsistent findings are the result of the overwhelming number of these studies that (a) do not include normative controls with no DE, or (b) fail to differentiate between different substances and different types of DE.

There is some indication that the high rate of comorbidity between DE and SU may be unique to those with binge and/or purging behaviors (e.g., BN, ANB, BED); the increased rates of comorbidity may not be apparent for those who show only restrictive dieting and exercising (e.g., ANR). Substance abuse is more frequently seen in bulimics and binge/purging anorexics rather than restricting anorexics (Blinder, Cumella, & Sanathara, 2006; Corcos et al., 2001; Stock et al., 2002; Wolfe & Maisto, 2000). For example, Blinder et al. (2006) found that individuals with ANR were half as likely to have any substance abuse problems compared to those with ANB or BN.

There are few studies that measure specific DE behaviors in relation to substance use. These studies found that several DE behaviors such as bingeing, purging, hard exercising, and caloric restraint predicted substance use. For example, Wiederman and Pryor (1996) found that (a) caloric restriction was predictive of amphetamine use, (b) purging was predictive of alcohol, cigarette, and cocaine use, and (c) binge eating was predictive of hallucinogen use. Similarly, Franko et al. (2005) found that vomiting is significantly related to alcohol use disorders, whereas binge eating was not.

Ross and Ivis (1999) examined how binge eating and compensatory behaviors were related to use of a variety of substances including tobacco, alcohol, stimulants, cocaine, and heroin. They found that females who binge ate and showed compensatory behaviors were considerably more likely to report use of all drugs measured in comparison to non-bingers, past-bingers and bingers who did not use compensating behaviors. These results emphasize the influence of compensatory behaviors (such as vomiting, laxatives, fasting, and excessive exercise) on substance use. Unfortunately, this study grouped all compensatory behaviors together, so it is impossible to discern which specific behaviors might be driving this relationship.

Of all the studies reviewed, only one (Piran & Robinson, 2006) measured dieting, bingeing, and purging behaviors, a broad range of different substances (including alcohol, tobacco, marijuana, cocaine, hallucinogens/heroin, and amphetamines) and included a control group (participants with no DE). They found that individuals who exhibited only bingeing behavior engaged in severe alcohol consumption more often than controls and more often than those who only dieted. The diet only individuals used hallucinogens and heroin at significantly higher rates than the controls and the binge only group. Presence of purging behavior was not included in this analysis. The rest of the analyses grouped these behaviors together in different combinations. For example, participants grouped in the “diet, purge, but no binge group” used both cocaine and stimulants/amphetamines at higher rates than controls and the “binge, diet and no purge group.” These results shed some light on the specific relationship between different eating disordered behaviors and substance use. Unfortunately, this study did not measure quantity or frequency of any of the substances besides alcohol.

Driving Mechanisms behind the Substance Abuse-Disordered Eating Association

The finding of a relationship between substance use and disordered eating, while informative, does not directly inform our understanding of the mechanism(s) driving this association (Wolfe & Maisto, 2000). An improved understanding of what is driving the relationship between SU and DE will guide the field toward better understanding of both SU and DE, and it may inform the development of more effective and efficient prevention and intervention programs. It may also further clarify some of the inconsistencies in the existing correlational literature (i.e., some studies fail to show the SU-DE association depending on the types of SU or DE measured).

There are several potential mechanisms that could be contributing to the SU-DE association. The SU-DE relationship
may be driven by a shared etiology. For example, substance use and disordered eating habits may each offer tension reduction to those experiencing heightened anxiety or distress (e.g., bingeing on food or alcohol may provide temporary relief; Wolfe & Maisto, 2000). Alternatively, disordered eating attitudes and weight concerns may lead to substance use, perhaps as yet another means of controlling appetite and weight. For example, Franko et al. (2005) found that the presence of an eating disorder has greater influence on alcohol use disorders than the reverse. Finally, substance abuse may lead to disordered eating habits (e.g., cigarettes, cocaine and other stimulants may decrease appetite). Most of the existing research indicates that the initial presence of an eating disorder leads to substance use problems (Wolfe & Maisto, 2000). These findings indicate that SU and DE may share a common cause or that DE may lead to SU, rather than SU leading to DE, but further research is needed in this area.

**SU and DE caused by shared psychological characteristics.** Several studies have looked at psychological characteristics that are common to SU and DE to see if these may be influencing the SU-DE relationship. Individuals may be engaging in SU and DE as a means of coping with anxiety, tension, or other problems. Milos et al. (2004) found that patients with a clinical eating disorder diagnosis and any co-morbid substance-related disorder had significantly elevated scores of Interoceptive Awareness (e.g., inability to recognize feelings and emotions) and Ineffectiveness (e.g., feelings of inadequacy, insecurity, and worthlessness). Interoceptive Awareness has also been weakly correlated with problematic alcohol use among university women (Kashubeck & Mintz, 1996).

Individuals may be using both SU and DE to reduce anxiety or tension (e.g. bingeing on food or alcohol may provide temporary relief; Wolfe & Maisto, 2000). Indeed, many individuals with DE have comorbid anxiety disorders. Killen et al. (1987) found that individuals with DE, specifically purging behavior, reacted with higher intensity to stressful situations and had more anxiety problems than those without DE. They found that individuals with DE were much more likely to use alcohol for stress management than those without DE. In addition, problematic eating behavior has been associated with the use of alcohol as an avoidant coping mechanism (Anderson, Simmons, Martens, Ferrier, & Sheehy, 2006).

**SU as a means of weight control.** Some individuals with DE may be using substances as a means to control their weight or shape. Of particular interest are substances with stimulant, diuretic, and appetite suppressant effects (such as cocaine, caffeine, amphetamines, tobacco, and other stimulants). Several studies have documented weight control as a reason for SU among individuals with DE (Cochrane, Malcolm, & Brewerton, 1998; Grauer & Black, 2001; Pomerleau et al., 1993; Welch & Fairburn, 1998). Individuals with DE put more emphasis on smoking cigarettes for weight control than individuals without DE (Welch & Fairburn, 1998). In addition, weight control is one of the most common reasons given by individuals with DE for using cocaine (Cochrane et al., 1998), alcohol and cigarettes (Grauer & Black, 2001; Stock et al., 2002).

**SU as a means of enhancing emotions.** In addition to decreasing negative emotions, research has shown that some individuals use substances to enhance positive emotions. These individuals may be using substances for fun, sensation seeking, or increased excitement. For example, Ross and Ivis (1999) found that bingers gave more importance to using marijuana for reasons such as “to feel good or get high” in comparison to nonbingers.

To date, there is little research that explores the various motives for SU among individuals with DE. In addition, these findings are very limited in that each involves a different population of individuals (e.g. clinical, community, university), none include reasons for a broad range of substances, and none compare different disordered eating behaviors and attitudes. No studies to date have made a comprehensive comparison of reasons for use of different substances for individuals with different disordered eating behaviors and attitudes.

In summary, previous research regarding the relationship between DE and SU has produced inconsistent findings. These inconsistencies may be the result of combining different DE behaviors into a single class and combining different substances. As noted above, differentiating between behaviors is essential because there is considerable overlap and variance in the type and frequency of DE behaviors among diagnostic groups. For example, Corte & Stein (2000) found that bingeing, vomiting, hard exercising, and food restriction occurred in all four of their diagnostic groups (sub-threshold and threshold AN and BN) and at varying intensities. Most studies do not include a full range of substances, behaviors, or even controls nor do they measure the quantity and frequency of SU and DE.

**Present Study**

In the present study, we will examine the relationship between disordered eating habits and weight concerns (DE) and substance use (SU). We will attempt to replicate the finding of a correlation between disordered eating and substance use. We will also address several gaps present in existing research. First, we will examine relationships between DE and SU within a non-clinical sample of young adult women. Second, we will distinguish between different types of substances (e.g., caffeine, nicotine, alcohol, illicit drugs) and between different types of DE (e.g., dieting, bingeing, purging, excessive exercise, weight concerns) in our analyses. Most of the existing research (outlined above) has focused on alcohol, marijuana, or tobacco use (Corte & Stein, 2000; Franko et al., 2005; Stock et al., 2002) or considered all substances in one lumped category (Milos et al., 2004; Stice, Burton, & Shaw, 2004), which can be problematic. Finally, we will examine hypothesized mechanisms driving the correlation between DE and SU.

The aims of the current study are to (a) determine the prevalence of DE and SU among a sample of college women, (b) compare rates of alcohol, tobacco, caffeine and illicit drug use (SU) for young women with disordered eating habits and weight concerns (DE) versus those without significant DE, (c) compare rates of SU for women with bingeing/purging behavior compared to those with only restricting behavior, and (d) examine possible attitudes and reasons for SU among women with DE. Based on previous findings and extent literature on the nature of disordered eating and substance use, we propose the following hypotheses:

**Hypothesis 1:** Substance use (SU) will be more prevalent among women with DE compared to those without DE. Specifically, alcohol, illicit drug use, and stimulant use will be more prevalent among women with DE compared to those without.
Hypothesis 2: Among women with DE, alcohol and non-stimulant drug use will be less prevalent for those with only dieting/food restricting behavior (pure-restrictors) compared to those with bingeing behavior.

Hypothesis 3: Among women with DE, stimulant use (tobacco, caffeine, cocaine and amphetamines) will be equally prevalent for the pure-restrictors and the bingers.

Hypothesis 4: Although existing research does not support a specific hypothesis, explanations and reasons for SU will be examined, including several DE psychological characteristics, weight control and the reduction of anxiety or tension. These exploratory analyses will shed light on potential mechanisms behind the association between DE and SU. However, due to the dearth of existing literature in this area, we have no formal hypotheses regarding these relationships.

Method
Participants
One hundred and twenty females enrolled in an introductory psychology course at a large Midwestern university participated in the study. Half of the sample (n=60) voluntarily signed up for the study, the other half (n=60) were recruited based on the presence of DE in a pre-test measure. The average age of participants was 18.89 years (SD = 1.002), average height was 65 inches (SD = 2.82, range 56-73 in.), and average weight was 137 pounds (SD = 23.26, range 85-224 lbs.). The sample was 84.2% Caucasian (n=101), 5% Asian/Pacific Islander (n=6), 5% African American (n=6), 2.5% Hispanic/Latino (n=3), 2.5% Native American (n=3), and 4% (n=5) listed their ethnicity as “other.”

Procedure
A description of the study was posted to the SONA online system accessible to all students enrolled in Psychology 1000, an introductory psychology course in which students are encouraged to participate in research studies as part of their learning process. All females enrolled in Psychology 1000 were allowed to participate. Males were excluded from the study due to the extremely low prevalence of disordered eating among this population.

In addition, all students enrolled in Psychology 1000 completed a pre-test for screening and recruitment purposes. This pre-test includes numerous items from several distinct studies. The study recruited individuals on the basis of their responses to the EAT-26 and three questions regarding specific disordered eating behaviors (described below). Female participants who endorsed significant eating pathology on this pre-test were sent an email asking them to consider participating in the study. Any participant who had a score of 20 or higher on the EAT-26 (the cut-off score for the measure, indicating abnormal, disturbed eating patterns), who answered “usually” or “always” to questions assessing fasting, vomiting, laxative/diuretic/enema use, and hard exercise to control their weight or shape were sent an email asking them to participate in the study. After two weeks, a second round of emails were sent out to those who did not respond to the first email and, because of the low rates of vomiting and laxative use in the sample, to participants who responded “often” to vomiting or laxative/diuretic/enema use and “always” or “usually” to binge eating.

Once participants signed up for the study, they were sent an email with an individual URL address that allowed them to confidentially complete both written consent and the complete study questionnaire online. After completion, each participant was provided with a written debriefing describing the purpose of the study and providing referrals for those interested in talking with a mental health professional about their eating habits or weight concerns. Each participant also received research participation points for their Psychology 1000 course.

Measures
Pre-Test Measure. The Eating Attitudes Test-26 (EAT-26; Garner, Olmstead, Bohr, & Garfinkel, 1982) is a self-report measure that consists of three subscales: Dieting, Bulimia and Food Preoccupation, and Oral Control. The EAT-26 is a widely used measure with established reliability (alpha = .90) and validity. This measure has also been used commonly in non-clinical samples to indicate the presence of disturbed eating patterns. Additionally, participants were asked three questions regarding specific disordered eating behaviors: a) I can eat an unusually large amount of food in a short period of time (e.g. several large bags of chips, two gallons of ice cream); b) I use diuretics (water pills), laxatives, or enemas to control my weight; and c) I exercise hard to control my weight. The response format for these questions was the six point likert scale used in the EAT-26 (ranging from never to always). The pre-test was used as an initial screening measure to select additional participants who demonstrated DE.

Eating Disorder Inventory. The Eating Disorder Inventory (EDI; Garner et al., 1983) is a widely used self-report measure of psychological and behavioral traits common to Anorexia Nervosa and Bulimia Nervosa. The inventory has eight subscales: Drive For Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, Interoceptive Awareness, and Maturity Fears. The EDI has demonstrated good criterion, discriminant, and convergent validity (Garner et al., 1983).

Disordered eating behaviors. The prevalence of disordered eating behaviors (binging, different types of purging, hard exercise, dieting and fasting) was assessed, as well as the age of onset of the particular behavior. All questions referred to prevalence in the past four weeks and had response choices ranging from never to twice in the past week.

Drinking Styles Questionnaire (DSQ; Smith, McCarthy, & Goldman, 1995). The Drinking Styles Questionnaire is a self-report measure of both quantity and frequency of alcohol use. This questionnaire shows good internal consistency and test-retest reliability.

Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). The FTND is a widely used measure of tobacco use and dependence. A FTND score of 6 or higher identifies subjects with high nicotine dependence.

Assessment of other substances. Additional questions were created to assess the participants’ current and past use of marijuana, cocaine/crack, amphetamines (e.g. crystal meth, uppers, speed, ecstasy, diet pills, Ritalin, Adderall),...
hallucinogens (e.g., PCP, LSD, mushrooms, peyote, mescaline) and opiates (e.g., heroin, morphine, opium, codeine, Vicodan). For each substance, participants were asked whether they had ever used the substance, their age when they first started using, lifetime use, use in the past 12 months, and use in the past 30 days. Each question has an ordinal response format ranging from not at all to more than 100 times. Caffeine consumption was assessed with the following two questions: 1) have you ever had a caffeinated drink? 2) and how many caffeinated drinks do you consume in a typical day?

Drinking Motives Measure (DMM; (Cooper, 1994). The Drinking Motives Measure is a 20 item self report measure that assesses motives for drinking with four subscales. For the purpose of this study, three subscales were used: Social, Coping, and Enhancement. Participants rate the frequency of drinking for each of the 20 reasons on a scale of 1 to 5, with 1 being almost never/never and 5 being almost always/always. Cooper (1994) demonstrated that these subscales are equally reliable and internally consistent and assess similar constructs across gender, race, and age groups. In addition, a weight-related subscale was added that contains five additional motives a) to avoid eating (as a substitute for food), b) to speed up your metabolism, c) to increase your energy and alertness, d) to help control your appetite, and e) to help control your weight. This measure has been adapted to be used as a motives measure for all substances in the present study by substituting each individual substance name for drinking (e.g. Because your friends pressure you to use [drug category here]).

Results

Before performing analyses, data was screened and distributions were examined. All data errors and outliers were omitted from the data. A series of bivariate correlation tables were created to examine possible, unanticipated relationships between variables.

Disordered Eating Prevalence

Prevalence of disordered eating was assessed for six different DE behaviors (bingeing, vomiting, laxative use, dieting, fasting, hard exercise). Figure 1 shows the prevalence of DE behavior one time a week or more in the past four weeks for the entire sample. Dieting and hard exercise were the most common behaviors (58% and 40%, respectively). Prevalence of binging and fasting was 12% and 14%, respectively. Purging, either by vomiting (4%) or use of laxatives/diuretics/enemas (3%) was less prevalent. Only 35% of the sample did not engage in any of these behaviors on a weekly basis. All of those with binging behavior compensated for their behavior with at least one of the other DE behaviors (e.g. dieting, hard exercise)

Substance Use Prevalence

Figure 2 shows the lifetime prevalence for alcohol, caffeine, tobacco, marijuana, cocaine, amphetamines, hallucinogens, and opiates. The most common drugs used were alcohol (89%), caffeine (98%), tobacco (49%), and marijuana (48%). Cocaine use (3%) and amphetamine use (8%) were combined in analyses as any illicit stimulant use (10%). In addition, 3% of the participants had used hallucinogens and 8% had used opiates in their lifetime.

Because of the high prevalence of caffeine use (98%) in the sample, a variable for excessive caffeine consumption was computed, for those who reported drinking three or more caffeine drinks daily (17%). Although lifetime tobacco use was fairly common (49%), only 6% reported currently smoking cigarettes.

In addition to lifetime prevalence of alcohol use, several questions assessed excessive drinking behavior. Specifically, of the 107 who reported any alcohol use, 22% (n=24) reporting drinking to drunkenness almost every time they drank; 54% (n=57) reported usually drinking one or more times a week; 38% (n=40) reported getting drunk at least once a week; and 45% (n=48) reporting binge drinking (typically drinking four or more drinks at one time).

Hypothesis 1: SU will be more prevalent among women with DE than among women without DE

Due to the nature of the SU variables, a series of binary logistic regressions were run to assess the relations between types of DE and SU. Logistic regression determines the percent of variance in the dependent variable explained by the independent variables (e.g., levels and types of disordered eating), ranks the relative importance of independent variables, and assesses interaction effects. Alcohol use and illicit drug use were more prevalent among women with any DE. Specifically, participants with DE, compared to those without, were more likely to have any lifetime
alcohol use (odds ratio = 3.4, p < .05); to binge drink (OR = 2.7, p < .05); to get drunk one or more times a week (OR = 3.3, p < .01); to get drunk almost every time they drank (OR = 6.9, p < .01); to have used any illicit substance (OR = 2.5, p < .05); and to have used marijuana (OR = 2.8, p < .05). No differences were found for stimulant use (excessive caffeine consumption, current and lifetime tobacco use, and illicit stimulant use) among those with DE compared to those without DE (all p > .05).

**Hypothesis 2:** Among women with DE, alcohol and non-stimulant drug use will be less prevalent for pure-restrictors than for bingers

The pure-restrictors were significantly less likely to have any lifetime alcohol use (OR = .852, p < .05); to binge drink (OR = .898, p < .05); to get drunk one or more times a week (OR = .890, p < .05); to get drunk almost every time they drank (OR = .850, p < .05); and to have used marijuana (OR = .913, p < .05).

**Hypothesis 3:** Stimulant use will be equally prevalent among women with DE

Among women with DE, stimulant use was equally prevalent for the pure-restrictors and the bingers. Specifically, no differences were found for lifetime tobacco use, current tobacco use, illicit stimulant use, and excess caffeine consumption (all p > .05).

**Hypothesis 4:** Attitudes leading to SU among women with DE

Scores on the EDI subscales were significantly higher for women with DE than without DE (see Table 1). To assess how certain psychological characteristics among women with DE may increase SU, a series of logistic regression analyses were run for the participants with DE. Tests were run only for substances that had significant effects in the above analyses with DE and SU; namely use of any illicit substance, marijuana, tobacco (both lifetime and current use), alcohol, illicit stimulants, excessive caffeine consumption, and excessive drinking. Five of the eight EDI subscales [Drive for Thinness (DT), Interoceptive Awareness (IA), Bulimia (BL), Ineffectiveness (IE), and Maturity Fears (MF)] had significant effects with SU. DT, IA, and BL increased the likelihood of using illicit stimulants (OR = 1.19, p < .05; OR = 1.14, p < .05; OR = 1.26, p < .001) and drinking excessive amounts of caffeine daily (OR = 1.15, p < .05; OR = 1.13, p < .05; OR = 1.28, p < .001) among women with DE. IE increased use of excessive caffeine (OR = 1.14, p < .05) and MF increased use of illicit stimulants (OR = 1.21, p < .01) among women with DE. Due to the nature of the data and the low base rates of SU, additional planned analyses regarding motives for substance use were not conducted.

<table>
<thead>
<tr>
<th>Mean Scores (and Standard Deviation) of EDI subscales</th>
<th>Participants with DE (n = 78)</th>
<th>Participants without DE (n = 42)</th>
<th>Difference Between Means (t-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drive for Thinness</td>
<td>9.7 (5.6)</td>
<td>2.2 (3.4)</td>
<td>p &lt; .0001</td>
</tr>
<tr>
<td>Bulimia</td>
<td>3.0 (4.9)</td>
<td>0.7 (1.5)</td>
<td>p &lt; .001</td>
</tr>
<tr>
<td>Body Dissatisfaction</td>
<td>13.3 (7.0)</td>
<td>6.6 (5.9)</td>
<td>p &lt; .0001</td>
</tr>
<tr>
<td>Ineffectiveness</td>
<td>4.2 (5.4)</td>
<td>0.7 (1.2)</td>
<td>p &lt; .0001</td>
</tr>
<tr>
<td>Perfectionism</td>
<td>7.1 (4.7)</td>
<td>6.8 (3.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Interpersonal Distrust</td>
<td>2.7 (3.6)</td>
<td>1.0 (1.3)</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>Interoceptive Awareness</td>
<td>4.8 (5.8)</td>
<td>1.1 (1.7)</td>
<td>p &lt; .0001</td>
</tr>
<tr>
<td>Maturity Fears</td>
<td>4.6 (4.2)</td>
<td>2.8 (3.4)</td>
<td>p &lt; .05</td>
</tr>
</tbody>
</table>

Note: NS = not significant

**Discussion**

This study used a behavior-specific approach to examine associations between disordered eating and substance use. We examined specific associations between various types of DE and various substances in a community sample with a range of DE and SU behaviors. Compared to a large national survey of substance use in the United States (US Dept Health and Human Services, 2005), marijuana, alcohol, and illicit substance use were much more common in the present sample. This was not unexpected, as increased use of substances is common among college students. Interestingly, cigarette use was much lower than national samples (only 5.8% identified themselves as current smokers), possibly suggesting a trend towards decreased tobacco use among younger generations. Prevalence of DE was higher in the present study, compared to other university samples.

As predicted, women with DE were more likely to use substances than women with no DE. Specifically, those with any DE were significantly more likely to have used any illicit substance, alcohol, marijuana, and to drink excessively, including binge drinking, getting drunk more often, and drinking to drunkenness almost every time they drank. However, contrary to expectations, no differences were found between participants with and without DE in use of stimulants (e.g. lifetime tobacco use, current tobacco use, caffeine consumption, and illicit stimulants such as cocaine and amphetamines).

As hypothesized, among women with DE, those who were pure-restrictors (that is did not engage in any binging or purging behavior) were significantly less likely to have used alcohol and other non-stimulant substances. Specifically, the pure-restrictors were less likely to have ever used alcohol or marijuana, to binge drink when they drank, to drink to drunkenness every time they drank, and to get drunk at least once a week. Also as predicted, no differences were found between the bingers and the pure-restrictors with regard to stimulant use.

These results are consistent with research suggesting that substance use is increased for those with disordered eating. Several studies have demonstrated an increased use of alcohol for those with DE (e.g., Krahn et al., 2005). Many other studies do not include controls for comparison, but do note a high prevalence of alcohol use and abuse among women with DE (e.g., Franko et al., 2005). In addition, Ross and Ivis (1999) found that individuals with DE were more likely to have used a wide range of substances including tobacco, alcohol, marijuana,
stimulants, cocaine, ecstasy, heroin, and hallucinogens. The present research did not find any significant effects for opiates or hallucinogens, possibly due to the low rates of use for these substances in the sample.

Additionally, although we found a significant relationship between DE and SU in general, this relationship was magnified for those with binging and compensatory behaviors. These results are consistent with the few other studies that also used a behavior-specific approach. Several other researchers have also found increased substance use among bingers/purgers compared to restrictors (Blinder et al., 2006; Piran & Robinson, 2006; Stock et al., 2002). Together, these findings indicate that the SU-DE association may be driven by the individuals with binge-purge behaviors.

A few studies have looked at EDI subscales and SU, none of which have found very conclusive results. For example, Kashubeck & Mintz (1996) concluded that the relationship between these subscales and SU was so small that it was unimportant. However, a significant and consistent relationship was presently found relating certain EDI subscales to stimulant use.

Exploratory analyses revealed several psychological traits among those with DE that are related to SU. Of the eight EDI subscales, five significantly increased the odds of certain SU among women with DE. Among those with DE, illicit stimulant use was more likely for those with higher scores on Drive for Thinness (DT, an intense need to be thinner and fear of gaining weight), Interoceptive Awareness (IA, confusion and lack of confidence in recognizing and identifying emotional states), Bulimia (BL, behaviors and attitudes related to bingeing and purging), and Maturity Fears (MF, wishes to return to preadolescent years due to overwhelming demands of adulthood). Similarly, excessive daily consumption of caffeine was more likely for those with higher scores on DT, IA, BL, and IE (Ineffectiveness: general feelings of inadequacy, insecurity, and worthlessness). These results suggest that for women with DE, higher levels of these five psychological traits increase their risk for stimulant use. As noted above, overall, those with DE were no more likely to use stimulants than those without DE. However, for women with DE who also had higher scores for Drive for Thinness, Interoceptive Awareness, Bulimia, Ineffectiveness, and Maturity Fears, the rates of stimulant use were significantly higher. This finding indicates that stimulant use is only increased for those with DE that also have these attitudes and suggests that these attitudes may represent a risk factor for stimulant use among women with DE. These individuals might be using substances for weight control, such as suppressed appetite or increased metabolism.

The present research was limited to a university sample of mostly Caucasian females. It will be important to see if these results replicate with non-university samples, clinical samples, and other ethnicities.

Although results here are consistent with some research regarding the DE-SU relationship, additional behavior-specific research is needed to replicate these findings and perhaps arrive at a consistent conclusion that binge-purge behaviors are associated with increased SU whereas pure restriction is not. Combined with studies of beliefs and motives for SU, these findings may shed light on who is at increased risk for substance use and what interventions may help ameliorate that risk. This study analyzed the psychological traits measured in the EDI, but this is by no means an exhaustive set of beliefs and motives that may drive the associations with SU. Additional reasons for SU such as weight control, coping with anxiety/tension, and emotion enhancement should be explored. This information can then be used to develop eating disorder interventions that incorporate a focus on these attitudes and beliefs about substances.

References


Introduction and Literature Review

Lyme disease is the most common arthropod-borne disease in the United States, affecting more than 20,000 people per year (1). Lyme disease is caused by infection with the spirochetal bacterium, *Borrelia burgdorferi*, and it may lead to the development of arthritis and carditis if individuals are not treated with antibiotics near the time of infection (2). More than 60% of humans develop arthritis and 8% develop carditis if untreated after a *B. burgdorferi* infection. Lyme carditis manifests through various lesions in the heart tissue, including the connective tissue, aorta, valves and blood vessels in a non-recurrent manner (2). In carditis the inflammatory infiltrate is composed primarily of macrophages, which are immune cells that are responsible for the killing and removal of microbial invaders. The presence of *B. burgdorferi* in tissues induces the recruitment of leukocytes to the area of infection and leads to the production of proinflammatory cytokines, which are thought to play a role in the development of pathology. The recruitment of immune cells and the role of various cytokines in disease pathogenesis are currently areas of interest to researchers.

The mechanisms responsible for the development of pathology in humans are currently unknown, therefore murine models are used to study Lyme disease pathogenesis. Using various murine models, such as susceptible C3H mice or resistant B6 mice, there is a consensus within the field that infection with *B. burgdorferi* causes “multisystem histopathologic lesions,” most frequently in the form of carditis and arthritis (3). Other mouse models such as BALB/c and NIH-3 mice have shown similar disease pathology (4,5). In 1990, researchers found that certain strains of mice infected with *B. burgdorferi* acquired severe pancarditis, with effects also in the endocardium, myocardium and epicardium (6). Lesions in the heart are located near blood vessels, in the connective tissue at the base of the heart, near the aorta and 60% of small arteries. Bradycardia, tachycardia, and atrioventricular blockage have been associated with *B. burgdorferi* infection in both mice and humans (7,8).

In *B. burgdorferi*-infected hearts, macrophages are present in larger numbers than any other immune cell, constituting 80-90% of the cells infiltrating into heart tissues (9). These mononuclear MAC-1+ cells serve as the first line of defense against the infection, infiltrating into areas of inflammation such as the aorta, blood vessels and connective tissue (6,10). The activation of macrophages, in addition to the infiltration, appear to be sufficient for disease pathogenesis. This was evident when SCID mice, who do not have T or B cells, still developed severe carditis following *B. burgdorferi* infection (6). Neutrophils and lymphocytes have also been found in *B. burgdorferi* infected hearts, but they do not appear to be as important for the development of carditis as macrophages (3). For example, CD4 and CD8 T. lymphocytes, although still functional, represent less than 5% of the infiltrated cells. Activated macrophages are known to be efficient killers of *B. burgdorferi* in vitro, and are thought to play an important role in clearance of the spirochetes from infected tissues (11).

The recruitment and activation of macrophages also results, directly or indirectly, in the production of numerous proinflammatory cytokines, and these are thought to drive the development of pathology. Previous studies on Lyme carditis have shown that IFN-γ, TNF-α, and IL-1β are consistently up
regulated in cardiac lesions (9,10,12,13). TNF-α, and IL-1β are up regulated during the early stages of the disease, while IFN-γ is up regulated at later time points. The increased production of these cytokines in Lyme disease susceptible mice are thought to be responsible for the development of severe carditis. The cytokine, IL-11 and its effect on Lyme carditis has also been studied. The expression of IL-11 assists in the regulation of macrophage production of pro-inflammatory cytokines (14). During B. burgdorferi infection, the expression of IL-11 was found to decrease arthritis development, but was not important in the pathology of Lyme carditis. The cytokines IL-4 and IL-6 were found to be present in low amounts in most studies on Lyme carditis. However, one study has shown that IL-4 may be effective in limiting the severity of cardiac inflammation after B. burgdorferi infection, but it has no effect on the resolution of the inflammation (15).

Inflammatory cells are originally attracted to the areas of infection by chemokines (chemotactic cytokines). Chemokines have been found in heart tissue during the development of Lyme carditis. Both macrophage chemoattractant protein-1 (MCP-1) and macrophage inhibitory protein (MIP)-2 (a neutrophil chemoattractant) were found to be expressed at high levels in B. burgdorferi infected hearts (16). MCP-1 is a powerful chemoattractant for the recruitment and activation of macrophages to sites of tissue injury or infection (17), contributing to the development of severe inflammation. In Lyme carditis, as well as other models of myocarditis, high levels of MCP-1 have been correlated with increased severity of cardiac pathology (18,19).

The overall goal of the current study was to examine the role and contribution of MCP-1 in the recruitment of macrophages to the heart and carditis severity. Surprisingly, we found that C3H MCP-1 KO mice developed the same severity of Lyme carditis as wild-type C3H mice following infection with B. burgdorferi. Levels of B. burgdorferi and proinflammatory cytokines were also not different between the two mouse strains. These results demonstrate that MCP-1 does not play a significant role in the pathogenesis of Lyme carditis.

Materials and Methods

Cultivation of Bacteria: A 0.5 ml stock quantity of B. burgdorferi strain N40 was grown in 7 ml BSK medium (Sigma Chemical Company, St. Louis, MO) containing 6% rabbit serum at 32°C for a period of 5 to 6 days. Live spirochetes were enumerated using dark field microscopy, diluted in BSK medium to 2 x 106/ml and used in experiments. Mice: C57BL/6j MCP-1 knockout mice (KO), a kind gift from Barrett J. Rollins (Harvard University) were backcrossed 10 generations onto the C3H/HeJ genetic background. Male and female C3H wild type (WT) and MCP-1 KO mice, 4 to 8 weeks old were used for experiments. The age and sex of the mice were kept constant among experimental groups. The mice were infected with approximately 1.0 x 105 B. burgdorferi strain N40 in each hind footpad. At 21 days post infection, the peak of Lyme disease pathogenesis, mice were sacrificed by CO2 asphyxiation. Both ankles, knees, as well as serum and the heart was harvested from each mouse. The harvested heart was cut in half longitudinally with a sterile blade. One-half was used for bacterial quantification and the other half was fixed in 10% buffered zinc-formalin (Anatech LTD, Battle Creek, MI) for histology.

B. burgdorferi burden: The heart samples were kept at -80°C until processed. DNA and RNA were isolated from the heart by TRizol extraction and resuspended in 200µl DEPC-H2O. B. burgdorferi flagellin and nidogen mouse genes were amplified by real time-PCR. The TAQ-man Real Time PCR kit was used according to the manufacturer’s instructions (Life Technologies, Gaithersburg, MD). The Applied Biosystems 7300 Real Time PCR system was used for amplification. Amplification started at 50°C for 2 min, followed by 10 min. at 95°C, 45 cycles of 95°C for 15 seconds each and completed with 1 min. of annealing at 60°C. The copy numbers for B. burgdorferi and mouse genomes were collected and analyzed.

Cytokine Quantification: The pro-inflammatory cytokines IL-1β, TNF-α, and IFN-γ levels were quantified using protein ELISA. Anti-inflammatory cytokines IL-4 and IL-10 were also quantified using ELISA. The total cytokine concentration was determined using the amount of cytokine per milliliter and data from a total protein assay (BCA kit, Pierce, Rockford, IL). Data are expressed as pg cytokine per mg total protein.

Histopathology: Hearts were cut in half, slicing longitudinally through the both atria and ventricles. The heart was fixed in 10% buffered zinc-formalin (Anatech LTD) until processed for histology. Through RADIL histology services at the University of Missouri-Columbia, the tissue was embedded in paraffin and H&E (hematoxylin-eosin) stained. After staining, the heart sections were analyzed for the amount of immune cell infiltration (neutrophil, macrophage, and lymphocytes) through microscopic examination. Those scoring were blinded to the study conditions until the results were given. The severity of inflammation was scored on a scale from 0 to 3 according to the number of inflammatory foci. A score of 0 showed no signs of inflammation; 1, mild inflammation with less than 2 foci of infiltration; 2, moderate inflammation, with 2 or more foci of infiltration and 3 severe inflammation, with wide spread immune cell infiltration.

Statistics: Statistical analysis was completed using SigmaStat software for Student’s t test. Critical values for statistical significance were set at α = 0.05.

Results

Development of Lyme carditis in MCP-1 KO mice: Previous studies have correlated the severity of Lyme carditis pathology with the expression of MCP-1 in cardiac tissue (18,20). Similarly, mice deficient in the receptor for MCP-1 (CCR2), also had altered development of Lyme carditis (16). CCR2 binds to more than one chemokine, therefore we wanted to determine if mice deficient in MCP-1 would also have altered Lyme carditis development. Wild type and MCP-1 KO C3H mice were infected with B. burgdorferi and sacrificed 21 days later. Hearts were removed and histological examination of heart tissue was performed in order to determine the number of immune cell infiltration into the heart tissue. Infiltration of neutrophils, macrophages and other leukocytes represented inflammation, and the high or low amounts of these cells signified increased or decreased carditis severity. A severity scale was used by two independent examiners, who scored the severity of carditis in the heart tissue samples in a blinded manner. The average severity score for MCP-1 KO heart sections was 1.8 ± 1.5, and the wild-type
heart sections were 1.3 ± 0.8. These results were not statistically different from one another (P < 0.45) and indicate that a deficiency in MCP-1 had no effect on the development of Lyme carditis severity. Representative histology sections are shown in Figure 1.

Figure 1. Histopathology of inflammatory cells in heart tissue 21 days post-infection.
Representative sections of heart tissue from A) MCP-1 KO and B) WT mice stained with H&E.

Borrelial loads in cardiac tissue: Macrophages are important for the clearance of *B. burgdorferi* from infected tissues (21). A defect in the recruitment of macrophages into the site of infection would likely lead to increased bacterial loads in the tissue. To determine the effect of MCP-1 deficiency on the number of *B. burgdorferi* found in the heart tissue after the initial infection, the number of *B. burgdorferi* genomes were measured by real-time PCR. The measurement was based on the number of *B. burgdorferi* flagellin gene copies per 1000 copies of mouse nidogen genes through RT-PCR (Figure 2). In the MCP-1 KO mice there were on average 315.83 ± 133.34 copies of the flagellin gene per 1000 copies of nidogen gene, while the wild-type mice had on average 265.75 ± 125.85 flagellin gene copies per 1000 nidogen gene copies. Again there were no significant differences between the number of *B. burgdorferi* genomes found in the MCP-1 KO mice and the wild-type mice (P < 0.67).

Figure 2. *Borrelia burgdorferi* genome quantification in heart tissue.
Wild-type and MCP-1 KO mice were infected with *B. burgdorferi* and were sacrificed at day 21 post-infection. Hearts were removed, cut in half, and DNA was extracted from one half. Quantitative real-time PCR was used to measure levels of *B. burgdorferi* flagellin and mouse nidogen genes in each sample. Results are expressed as copies of flagellin per 1000 copies of nidogen.

Cytokine levels in cardiac tissue: The production of pro-inflammatory cytokines was additionally investigated in order to better understand the role of other cytokines in the heart tissue during Lyme carditis. Higher levels of pro-inflammatory cytokines have been correlated with increased carditis severity and MCP-1 expression (22). In addition to MCP-1, pro-inflammatory cytokines could be responsible for varying carditis severity levels between MCP-1 KO mice and wild-type mice. The concentration of each cytokine was determined by ELISA and was normalized per milligram of protein determined through a total protein assay (Figure 3). Surprisingly, the results of this experiment were different from the original hypothesis, as there was no significant difference between the MCP-1 KO mice and the wild-type cytokine concentrations (P < 0.08). These results demonstrate that a deficiency in MCP-1 production had no effect on cytokine production in the hearts of *B. burgdorferi*-infected mice.

Figure 3. Cytokine concentrations in heart tissue.
Wild-type and MCP-1 KO mice were infected with *B. burgdorferi* and were sacrificed at day 21 post-infection. Hearts were removed, cut in half, and protein was extracted from one half. Levels of pro-inflammatory and anti-inflammatory cytokines were measured by ELISA and results are expressed on a per mg total protein basis.

Discussion
Murine genetics control the development of pathology during infections with *B. burgdorferi* with some mouse strains being resistant to the development of arthritis or carditis, and others being susceptible. While the presence of live spirochetes is required for the development of pathology, their absolute numbers within a tissue does not control the level of disease. Thus, resistant and susceptible mouse strains can harbor similar numbers of spirochetes within their tissues and yet retain their distinct disease phenotypes (23,24). This indicates that it is the host’s response to the infection that results in the development of disease. Susceptible mouse strains produce several chemokines in response to *B. burgdorferi* infection that recruit inflammatory cells to the site of infection (25). These inflammatory cells then amplify the response by the production of pro-inflammatory cytokines which recruit and activate even more inflammatory cells. While a certain level of immune response is required to clear the infection, an over-exuberant immune response is thought to cause disease.

In the current study, we examined the role of the chemokine MCP-1 in the development of Lyme carditis. In Lyme carditis the inflammatory infiltrate consists primarily of macrophages.
with lesser amounts of neutrophils (9). Macrophages from susceptible mouse strains are known to make higher levels of pro-inflammatory cytokines in response to in vitro stimulation with *B. burgdorferi* than those from resistant mouse strains (26). In addition, the severity of carditis following *B. burgdorferi* infection correlates with the production of MCP-1 within the heart tissue (20). MCP-1 is known to be a powerful stimulus for the recruitment of macrophages into sites of infection. Thus, we hypothesized that infection of MCP-1 KO mice on a genetically susceptible C3H background would prevent the development of Lyme carditis. We found that this was not true. C3H MCP-1 KO mice developed similar levels of Lyme carditis as wild-type C3H mice. The levels of *B. burgdorferi* within the heart tissues were also similar between the two strains of mice, and the production of pro- and anti-inflammatory cytokines were also unchanged. These results demonstrate conclusively that MCP-1 is not required for the development of Lyme carditis, nor for the activation of macrophages and the efficient removal of *B. burgdorferi* spirochetes.

These results are similar to a previous report from our laboratory examining the development of Lyme carditis in mice deficient in the receptor for MCP-1 (CCR2) (16). In that study, the authors found no difference in the severity of carditis in the CCR2 KO mice; however, the composition of the inflammatory infiltrate was altered so that neutrophils were now the predominant inflammatory cell type. In the current study, we have not yet examined the makeup of the inflammatory infiltrate to determine if this also occurs in the MCP-1 KO mice. It is possible that when there is a deficit in macrophage recruitment, neutrophils may compensate for this deficit. Further experiments are planned to examine the production of neutrophil chemokines in the heart tissue of infected MCP-1 KO mice. Similarly, other macrophage chemokines may also compensate for the lack of MCP-1 and act to recruit macrophages to the site of infection. Chemokines are known to be redundant and their receptors are capable of binding to more than one chemokine, so it is possible that other macrophage chemokines are acting in the place of MCP-1 in our experiments. Further studies are required to determine if this is happening.

**Reference List**


Introduction

Amphibian conservation research is an extremely pertinent subject to modern science. Although the world is seeing a serious loss of biodiversity in all species, amphibians are the most affected guild of animals, experiencing population declines and species extinctions on a worldwide scale (Alford and Richards 1999; Houlanah et al. 2000). According to the IUCN red list, the percentage of amphibian species facing extinction borders on 31% and includes a larger total number of endangered species than any other animal class (Groom et al. 2006). Semlitsch (2003) lists six major threats to amphibians identified by the Partners in Amphibian and Reptile Conservation, but it has been noted that most biologists agree that loss or decline in quality of local habitats is the major factor causing problems (Dodd and Smith 2003; Davidson et al. 2001). Amphibians are greatly impacted by slight microhabitat changes in soil moisture, temperature, and pH (Parmelee 1993), which allows them to be excellent indicator species for environmental health (Vitt et al. 1990).

Amphibian research has focused on factors that affect the success of larval amphibians because of the resulting repercussions on the juvenile and adult stages. Characteristics of larval amphibian environments can alter their physiology and morphology into adulthood, which has implications for future fitness (Relyea and Hoverman 2003). For example, larger sizes in earlier ontogenic stages have been correlated with greater survival in adult salamanders (Rothermel and Semlitsch 2006). Larger immature individuals have the advantage of experiencing less evaporative water loss from their skin due to a lower surface area to volume ratio (Spight 1968) and also a greater ability to resist predation (Berven 1990), compete for resources, and hunt for food. Increased tadpole size has also been correlated with larger size at reproductive maturity and earlier first reproduction (Semlitsch et al. 1988).

The loss of forest cover through habitat destruction has great potential to negatively impact the performance and distribution of amphibian populations by altering the light regime in the environment where the tadpoles live (Halverson et al. 2003). Increased levels of solar radiation hitting the surface of open canopy ponds has been found to produce water temperatures averaging 5° Celsius higher than in forested ponds (Skelly et al. 1990). Tadpoles in these ponds have been found to reach metamorphosis faster (Halverson et al. 2003), which is probably a precautionary measure to leave their natal environment before the pond dries from increased sunlight exposure (Semlitsch et al. 1988). With less time to develop, the metamorphs are likely to be smaller and less ecologically fit than those reared under closed canopy which, as discussed earlier, is due to their increased susceptibility to heat stress and desiccation (Parmelee 1993) and their inability to compete for food.

Other than light regime, the loss of forest cover also affects other important environmental factors that impact larval amphibians. One example of this is sight-selected predation in the pond community. Dragonfly larvae are predators that have the ability to significantly alter anuran larval populations. These odonates prefer to lay their eggs in open, sunlit ponds, and once hatched, the offspring (reaching body lengths of 60 mm) feed on tadpoles, utilizing a protrusible labium to capture and secure their prey (Skelly et al. 1990). Because these insect larvae use visual cues to hunt, tadpoles will be less likely to avoid predation.
in ponds where dragonfly density is high. Also, tadpoles attain refuge from predation by metamorphosing (Gunzburger et al. 2004), so this factor would also cause smaller, less fit individuals that will allocate their energy to quicker development rather than gathering food and resources. In fact, Skelly et al. (1990) found that tadpole activity decreased 41% in the presence of Anax species dragonfly larvae (which are sit-and-wait predators) compared to when these predators were absent. Therefore, open canopy ponds appear to offer less ideal larval treefrog habitat because of the presence of a major tadpole predator.

The purpose of this study is to determine the survival implications of clear-cut forests in Missouri on larval gray treefrogs and some future repercussions of these impacts. My hypothesis is that frogs reared in lower levels of shade and higher levels of predation (simulating the loss of trees through clear-cutting) will trade off the ability to easily gather food and resources in exchange for avoiding predators to better their chances of survival. As a result, they will metamorphose earlier and at smaller sizes. In contrast, individuals raised in conditions simulating closed canopy forest ponds with no predators will experience little pressure to develop and therefore will metamorphose later and attain larger sizes. I also predict that frogs reared in high shade will be more likely to survive in their juvenile stage and grow faster, because they will have greater energy stores, and will be less susceptible to desiccation.

Materials and Methods

The species of focus in my research is Hyla versicolor, the gray treefrog. Also called the common treefrog, it is an arboreal anuran that is present throughout the eastern United States except for the Florida panhandle (Leviton 2004). They begin breeding in late treefrog. Also called the common treefrog, it is an arboreal anuran throughout the rest of the experimental period, water levels were maintained with natural precipitation.

The tanks were then allowed to sit for a few days while the algae, bacteria, and zooplankton populations grew. This provided time for the collection of leaf litter from a nearby forested area. Once dried, 1 kg of deciduous leaf litter was added into each tank. Hyla versicolor frog larvae were hatched from eggs obtained from adult breeding pairs from Daniel Boone CA. Each cattle tank received a cohort of 100 tadpoles. Lastly, over-wintered insect larvae (dragonfly family Libellulidae) were also collected from ponds around Daniel Boone CA and transported back to Columbia where they were separated into the previously determined densities in 16 of the tanks (8 tanks received no dragonfly larvae), creating one of four predator density treatments: control (no larva), low (5 larvae), medium (10 larvae), and high (30 larvae). Eventually more of these dragonfly larvae needed to be collected to restock the tanks after some of the odonates emerged as fully grown dragonflies. All possible combinations of these two shade levels and four predator densities defined eight different tanks treatments, with three replicates of each.

The ponds were checked daily and observations were made on the tadpoles’ development and survival. Once the larvae had begun to metamorphose (defined by the emergence of their first forelimb), they were put into moist, covered containers and kept in the laboratory where they remained until achieving full tail-resorption and limb emergence (which normally took a day or two). They were then weighed, and this mass, along with their larval period (time from introduction into the tanks until metamorphosis), was recorded. They were then put back into containers until they were taken back out to Daniel Boone CA or were needed for another experiment (see next section).

Cage Study

This experiment began 27 June 2006 and used 20 wood frame cages, with hinging and locking lids and framed with fine mesh on the sides. The tops and bottoms were lined with wider, sturdier wire mesh, and the cages were dug in as flush against the ground as possible so that insects could crawl through and small plants could grow through. Once again, leaf litter was raked from the forest, and put into the cages to not only simulate the forest floor but to also inoculate the environment with small invertebrates on which the treefrogs could feed.

As mentioned in the previous section, we used recently metamorphosed frogs from the tank experiment to put in our cages. None from the low or medium predator treatment frogs were retained because these treatment types were not tested in this experiment. Therefore, there were four remaining treatment combinations with five replicates of each. All of the individuals from different tanks of the same treatment were put together. Each frog was then toe-clipped for identification and weighed to record initial mass. 300 frogs were used in all, 15 to each cage. A soaker hose watering system was stretched over the top of the cages to provide moisture twice a day.

Every 2-3 weeks, the frogs in the cages were surveyed. For two consecutive days during the survey period, all of the cages were searched at dusk when the individuals would be most active and easiest to collect. The frogs found were brought inside and their mark and mass were recorded to check for survival and growth. After the survey, they were immediately returned to their cages.
This was done a total of five times, and after the last survey the frogs were retained and released at the Daniel Boone CA.

**Statistical Analysis**

Several multivariate analysis of variance (MANOVA) tests were used to find the overall significance of data values for both experiments. For the pond experiment, tests were run to determine the statistical differences in larval period and average mass at metamorphosis as a response to larval pond environment for the parameters of shade level, predator density, and interaction effect of the two. A second set of MANOVAs was done to test the differences in the initial masses of the frogs when they were put into the cages and then in the survival and mass at the end of the experimental period due to the same previously stated natal parameters.

**Results**

As noted before, the parameters used to determine the impacts the different larval treatments had on the success of gray treefrog tadpoles were length of larval period and size at metamorphosis. Differences in the size at metamorphosis were determined by body weight once individuals had fully developed into froglets (Table 1). We found that frogs reared in low shade had statistically higher (P=0.0404) mass (0.3347 g) than those raised in high shade (.2960 g) (Fig. 1). However, there was no statistical difference (P>0.1) in weight between predator treatments and, again, there was no interaction effect.

Table 1  Summary of tank study results

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**Fig. 1** Average mass at metamorphosis by shade treatment. The correlation between shade level and body size is negative and significant (P=0.0404)

Differences in length of larval period between the treatments were found by comparing the average number of days that the tadpoles took to metamorphose (Table 1). In our shade treatments, we found that it took tadpoles in high shade tanks one day longer to reach metamorphosis than those in low shade tanks, but this difference was not significant. There were also no significant differences between the predator treatments. There was also no significant interaction effect between the shade and predator treatments.

In the terrestrial cage study, it was found that the sample of metamorphs added to the cages were representative of the entire cohort from the tank study because those that had been removed from low shade ponds (.2916 g) were statistically heavier (P= 0.0131) than those from high shade tanks (.2560 g) (Fig. 2). However, by the end of the experiment after they had grown for a few months, frogs from low (.4534 g) and high (.4952 g) shade tanks no longer possessed statistically different weights (Fig. 2). There was no statistical difference in either initial or final mass between predator treatments or as result of shade-predator interaction.

**Fig. 2** Average initial and final masses in cages due to shade level in natal ponds. Frogs reared in high shade were smaller at first were initially significantly smaller (P=0.0103) than those reared in low shade, but eventually there were no significant difference in weight between the treatments.
Finally, percent survival during the cage study was found to be statistically greater (P=0.0403) in low shade ponds (32.5%) than high shade ponds (12.5%) in treatments without predators, but this effect was suppressed in the presence of dragonfly larvae (Fig. 3). There was no significant difference in survival due to shade level alone or predator treatment alone.

Fig. 3 Average percent survival in cages due to natal pond shade and predator treatments. In treatments where predators were absent from the tanks, there is a significantly greater survival (P=0.0403) in frogs that were reared in low shade. This correlation is negative and insignificant when predators were absent.

**Discussion**

**Tank Study**

Our study confirmed that shade levels over the natal ponds impacts the development of gray treefrog tadpoles, particularly due to the light regime. We found that larger frogs emerged from low shade tanks, which indicates better conditions for their growth in ponds with less canopy cover. This finding is supported by Skelly et al. 2002 which found that another species of Missouri hydids, the spring peeper, selected ponds without canopy cover for breeding sites. However, because we found no significant difference in days to metamorphosis, we can disregard a longer larval period as the cause of this increased body size. One explanation for greater growth rates in the low shade ponds was increased periphyton levels due to increased solar radiation. Tadpoles utilize these algae for food and therefore had a larger resource base for their larval development. This accounts for the benefit of extra sunlight hitting the pond surface for the growing tadpoles, especially because there was no danger of desiccation and drying as the tank water levels were maintained throughout the experiment.

However, the presence of predators did not differentially impact the treefrog larvae size or length of development. Although Relyea and Hoverman (2003) found that tadpoles made body alterations in the mere presence of dragonfly larvae, morphology was not a parameter we tested and we found no difference in size at or time to metamorphosis in the different predator treatments. This could be due to different predator density levels triggering different conditions that the treefrogs dealt with in similar ways. Both pertain to limited feeding ability. Tadpoles raised in high predator treatments probably reduced their feeding activity in order to avoid predators, which caused similarly sized metamorphs as those raised with low predator density because increased survival rate created greater competition for food. Another explanation for this phenomenon was our use of over-wintered dragonfly larvae that had the ability to change into adults during our experimental period. In fact, many dragonflies emerged from our tanks throughout the study and more individuals had to be added frequently to maintain densities. Even though the larvae were replaced nearly as quickly as they emerged, this could not make up for the five days before emergence when it is known that the changing dragonflies do not feed. It was perhaps this lack of appreciable changes in predation pressure between different treatments that suppressed the effect the dragonflies would have had on the tadpoles’ development.

**Cage Study**

This study confirmed the existence of carry-over effects due to the differential development of the gray treefrogs in their larval environment, however, these impacts did not always persist under different population pressures. When placed in terrestrial cages, the frogs that had emerged from the low shade treatment ponds showed greater survival over time. This is probably due to their larger initial size, which made them more able to hunt a wider variety of prey but also allowing them to have greater energy store and less desiccation stress while carrying out this activity.

Relyea and Hoverman (2002) found a similar relationship between larger body sizes at metamorphosis and greater survival in the juvenile stage. However, unlike in this study, our experiment concluded that over time the tadpoles raised in different conditions eventually reached a size convergence in which there was no significant difference in body mass. One explanation for this occurrence is that the lower survival of metamorphs from the high shade tank treatment caused them to be less individuals competing for food within the cages. Because each cage was made to have a similar invertebrate immigration and emigration, each frog from high shade conditions had a greater share of resources to utilize for growth. This created comparatively better conditions in these cages and therefore suppressed some of the negative impacts from their larval environment. Therefore, we can conclude that while some characteristics of natal pond, such as shade level, can impact future success in gray treefrogs, it is only one of the many factors that will affect a cohort throughout their life cycle.

**Management Implications**

Though the result of this experiment implies that gray treefrog tadpoles benefit from the increased sunlight characteristic of open canopy ponds, forest loss should not be removed as a cause of amphibian declines. Many frogs have varying ecological requirements for their different life stages, so what may be true for tadpoles may differ greatly from the needs of mature frogs. The necessity of woody vegetation for treefrog adults can easily be ascertained. Once grown, these amphibians spend most their time resting and feeding on insects in the leafy refugia of trees, and call for mates from trees nearest to breeding ponds. Also, with 16 different amphibian species in the same forests in Missouri, it is likely that every one will have a different set of environmental requirements. Therefore, with amphibian populations declining on a global scale, the costs of habitat destruction must be studied extensively in the future to understand the ecological trade-offs made between human land-use and the conservation of biodiversity.
Acknowledgements
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Literature Cited


People turn to exercise for a myriad of reasons. Some engage in physical activity to improve overall health and build stronger bones, muscles, and joints, while others exercise to reduce or maintain body weight, or to reduce stress and the risk for health complications or illnesses. A great deal of research exists supporting the positive effects of exercise on both physical and mental health (Penedo & Dahn, 2005; Saxena, Van Ommeren, Tang, & Armstrong, 2005; Smith, 2006). However, as Lavallee, Kremer, Moran, and Williams (2004) note, it is important to distinguish between somatic and psychological effects of exercise. In terms of somatic effects, physical activity is associated with reduced risk of hypertension, Type 2 diabetes cardiovascular diseases, and osteoporosis (Cox, 2006; Morrato, Hill, Wyatt, Ghushchyan, & Sullivan, 2006; McGavock, Anderson, & Lewanczuk, 2006; Linden, Ahlborg, Besjakov, Gardsell, & Karlsson, 2006). In contrast, when considering the psychological effects of exercise, support for a similar healthy relationship is not as clear (Cockrell & Riddington, 1996). Lavallee et al. (2004) note that this is especially true when the motives for engaging in exercise are “less than healthy,” and exercise develops an addictive quality. Indeed, a number of terms have developed to characterize “unhealthy” exercise, including: “exercise addiction” (Hailey & Bailey, 1982), “obligatory exercise” (Pasman & Thompson, 1988), “compulsive exercise” (Brewerton, Stellefson, Hibbs, Hodges, & Cochrane, 1995), and “exercise dependence” (Cockrell & Riddington, 1996).

Thus, exercise as a positive behavior is not necessarily true for all types of people. According to Thome and Espelage (2004), whereas exercise is associated with positive psychological benefits in men, it is associated with both positive and negative psychological health in women. Results from their study indicate that the relationship between exercise and psychological health may be influenced by gender and the presence of eating pathology. In fact, exercise has been linked to the development and maintenance of eating pathology (Davis et al., 1997). In the current study, we compare several dimensions of exercise (motivation, guilt, obsessive cognitions, and typical weekly exercise) across individuals at different stages of an eating disorder (i.e., active eating disorder vs. recovered from an eating disorder).

Exercise as a means to control weight is common among individuals with eating disorders, predominantly those with anorexia nervosa (Davis et al., 1997). For those with bulimia nervosa, the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) lists “excessive exercise” as a defining feature of the non-purging subtype of the disorder. The current criteria for excessive exercise in the DSM-IV include exercise that interferes with important activities, takes place at inappropriate times of day or settings, and persists despite injury or illness. Nowhere in this definition are reasons for exercise or guilt considered, although an obsessive quality is implied with the persistence despite injury/illness criterion.

Most of the past research on excessive exercise has been focused on frequency and intensity of exercise as the sole criteria to identify those with unhealthy exercise patterns (Steffen & Brehm, 1999). For example, Brewerton et al. (1995) divided their sample of eating disorder patients into two groups,
compulsive exercisers and non-compulsive exercisers, based only on frequency and duration of exercise. Those who reported exercising to control weight for at least 60 minutes per day were defined as compulsive exercisers and had a higher degree of body dissatisfaction and distress after weight gain. When Long, Smith, Midgley, and Cassidy (1993) compared exercise in a group of individuals with anorexia and a healthy control group, they found that those in the clinical sample participated in significantly more different exercise activities each week, were more likely to engage in daily exercise, and were more likely to be secretive about their exercise. Sixty percent of the control group exercised for 15-20 minutes per day whereas 63% of the anorexies exercised in excess of 30 minutes.

Researchers now acknowledge that excessive exercise is an activity that is multidimensional and, more recently, both psychological and behavioral characteristics have been included in the assessment of excessive exercise. Mond, Hay, Rodgers, and Owen (2006) suggest that both motivation for exercise and experience of guilt after exercise postponement should be part of the definition of excessive exercise. In assessing motivations for exercise using the Reasons for Exercise Inventory (REI; Silberstein, Striegel-Moore, Timko, & Rodin, 1988), they found that exercising for weight control, tone, and to improve appearance, in addition to feeling guilt after postponement of exercise, was linked to higher levels of eating disorder psychopathology and lower quality of life (Mond, Hay, Rodgers, Owen, & Beumont, 2004; Mond, Hay, Rodgers, and Owen, 2006). The finding related to guilt is consistent with that of Ackard, Brehm, and Steffen (2002), who reported that negative emotions after missing an exercise session were highly correlated with multiple dimensions of eating pathology as reflected by the subscales of the Eating Disorder Inventory (EDI; Garner, Olmsted, & Polivy, 1983). Stephen and Brehm (1999) found that it is not the amount of exercise, but rather the negative emotionality linked to a particular exercise activity that may connect exercise and eating disorders. In their study, they used factor analysis to reduce the original 20-item Obligatory Exercise Questionnaire (OEQ) to a 10-item measure with three factors: (1) Emotional Element of Exercise, which refers to the degree of emotional stress resulting from missed exercise sessions; (2) Exercise Frequency and Intensity, which refers to the amount and intensity at which an individual engages in exercise; and (3) Exercise Preoccupation, which refers to obsessive thoughts about exercise. This last factor, exercise preoccupation, has also been examined by Ackard et al. (2002) who combined it with the exercise emotionality factor from Steffen and Brehm’s study (1999) to create the exercise fixation factor. They found exercise fixation to be most highly correlated with measures of psychological maladjustment, regardless of exercise frequency, and suggested that cognitions related to excessive exercise could be critical to understanding how it contributes to eating disorder psychopathology.

Despite the harmful associations between exercise and eating disorders, there is also evidence that exercise, with appropriate guidance, may be an important element in the treatment of eating disorders. For example, Sundgot-Borgen, Rosenvinge, Bahr, and Schneider (2002) found that physical exercise was more effective than cognitive behavioral therapy in reducing bulimic symptoms in female patients with bulimia nervosa. Similarly, a more recent study by Calogero and Pedrotty (2004) illustrated that a guided exercise program designed to reduce exercise abuse can in fact be successful in the treatment of eating disorders. In just under four weeks, women with eating disorders who participated in the exercise program reported less disordered thoughts, feelings, and behaviors about exercise than those who had not participated in the exercise program. Just as guided experiences with food are used to change destructive thinking about food in the treatment of eating disorders, so too may guided experiences with exercise be helpful in changing unhealthy behaviors and thoughts about exercise.

The aim of the current study was to examine the connection between exercise and eating disorders by comparing two groups (active eating disorder and recovered eating disorder) on four dimensions of exercise: reasons for exercise, guilt associated with postponement of exercise, obsessive thoughts related to exercise, and typical weekly exercise patterns. While other studies of exercise have compared clinical samples to normal samples (Long et al. 1993), examining how a recovered eating disorder group looks on these factors as compared to their currently eating disordered peers is novel. It will be valuable to know whether those recovered from eating disorders exhibit psychological and behavioral characteristics of exercise similar to those with active eating disorders, which would argue for the need to better address exercise in the treatment of eating disorders.

It was hypothesized that 1) those in the active eating disorder group would score higher on exercise motives related to appearance and weight and lower on the motives related to enjoyment and mood improvement than the recovered group; 2) those in the active group would report greater guilt after postponement of exercise and greater obsessive thoughts about exercise than those in the recovered group; and 3) those in the active group would engage in more exercise on a weekly basis than those in the recovered group.

**Method**

**Participants**

The present study is part of a larger study investigating a wide array of psychosocial outcomes to help better define recovery from eating disorders. A sample of female participants from the University of Missouri Adolescent Clinic (MUAC) was used for this study. The MUAC is a general health clinic; however, two of the attending physicians are well-known for treating eating disorders in the mid-Missouri area and thus have seen a significant number of eating disorder patients. All current and former female eating disorder patients of MUAC were contacted for participation, and the current project reports on the preliminary data from 19 study participants. Eating disorder status was determined based on the Semi-structured Clinical Interview for DSM-IV Axis I disorders (SCID; First, Spitzer, Gibbon & Williams, 1995) resulting in eleven women with a current eating disorder (active cases) and eight women who no longer met criteria for an eating disorder (recovered cases). Participants who were in the active group ranged in age from 17 to 28; 27.3% had a current diagnosis of anorexia nervosa and 72.7% had a current diagnosis of Eating Disorder Not Otherwise Specified (EDNOS). Participants who were in the recovered group ranged in age from 19 to 29; 87.5% had a lifetime diagnosis of anorexia nervosa, 12.5% had a lifetime diagnosis of bulimia.
nervosa, and 12.5% had a lifetime diagnosis of EDNOS. See Table 1 for descriptive statistics of the sample.

Procedure
The paper charts of all former and current eating disorder patients were first scanned for contact information, demographic information about the patient (e.g., age), and information concerning the course of the eating disorder (i.e., eating disorder diagnosis or symptoms). The information from the paper charts was then cross-referenced with patient information contained in the hospital chart database system (Power Chart) in order to retrieve the most updated contact information possible.

Once the contact information for all potential participants was collected, a letter introducing the general study and study consent form was sent to all current and former eating disorder patients. A follow-up phone call was then made by the principal investigator to address any questions and to request participation in the study. In order to participate, all participants had to provide written consent, and those who were minors (under age 18) required parental written consent.

Those who agreed to participate first completed a questionnaire session. The questionnaires covered a wide range of topics, including, for the purposes of the current study, motivations for exercise, guilt after the postponement of exercise, obsessive thoughts related to exercise, and current typical exercise patterns. At some date after the questionnaires, the diagnostic interview (SCID; First et al. 1995) was used to determine DSM-IV Axis I diagnoses (eating disorders, mood disorders, anxiety disorders, substance-related disorders). Interviewers were an advanced undergraduate psychology student and McNair scholar, a doctoral clinical psychology student, and the principal investigator. All interviewers received extensive training before the interview process began to achieve good inter-rater reliability.

Measures
Questionnaires covered an array of variables considered important in relation to eating disorder development, maintenance, and recovery. For the purposes of this study, the following questionnaires related to exercise were administered: 1) the Reasons for Exercise Inventory (REI; Silberstein et al. 1989), a 24-item questionnaire that represents seven areas of exercise motivation: exercising for weight control, for fitness, for improvement of body tone, for health, for improving overall physical attractiveness, for enjoyment, and for improving mood; 2) a question assessing guilt after the postponement of exercise found to be critical in past research (Mond et al. 2006): “Do you feel ‘guilty’ that you have somehow ‘let yourself down’ when you miss an exercise session?” (response format: 1 = not at all guilty to 5 = extremely guilty); 3) a measure of cognitions related to exercise via two questions: “Typically, what amount of your waking hours do you spend thinking about exercise (e.g., planning when you will exercise next or which exercises you’ll do, thinking about exercise you did, thinking about opportunities to exercise that you missed)? (This would include both positive and negative thinking about exercise.)” (response format: 1 = no time or almost no time to 5 = almost all the time or all the time) and “How easy is it for you to stop thinking about exercise when you find yourself thinking about it (e.g., change your thoughts and think about something else)?” (response format: 1 = extremely easy to 5 = extremely difficult); and 4) a measure of current typical exercise patterns, including type of exercise and exercise frequency (i.e., typical number of days per week spent exercising and typical number of minutes per exercise session).

Results
Data Analytic Strategy
T-tests were used to determine whether or not statistically significant differences existed between the active and recovered eating disorder groups on motivation, guilt, cognitions related to exercise, and typical weekly exercise patterns.

Motivation
The results of the t-tests revealed a significant difference between groups on the weight control motive, $t(15.97) = -2.48$, $p < .05$. As hypothesized, participants in the active eating disorder group scored higher on this motive than did participants in the recovered eating disorder group (active: $M = 15.64$, $SD = 3.91$; recovered: $M = 12.00$, $SD = 2.31$). It is noteworthy that although differences in the means on the other subscales were not statistically significant, the means were in the hypothesized direction for appearance. That is, the mean scores on motives related to appearance (i.e., attractiveness and tone) were higher (although not significant) in the active group than in the recovered group. Refer to Table 2 and Figure 1 for more information related to group differences on this measure.

Guilt
The results of the t-tests revealed a marginally significant difference between groups in terms of how much guilt they reported, $t(17) = -1.63$, $p = .122$. Participants in the active eating disorder group scored marginally higher on experiencing guilt after exercise postponement than did participants in the recovered eating disorder group (active: $M = 4.09$, $SD = 1.22$; recovered: $M = 3.13$, $SD = 1.36$) (see Table 2).

Cognitions
The results of the t-tests revealed a significant difference between groups on how difficult it was to stop thinking about exercise, $t(15.92) = -2.52$, $p < .05$. As hypothesized, participants in the active eating disorder group found it more difficult to stop thinking about exercise than did participants in the recovered eating disorder group (active: $M = 1.88$, $SD = .83$). Although differences in the other obsessive cognition related to exercise (amount of time spent thinking about exercise) were not significant, the mean scores fell in the hypothesized direction (see Table 2).

Typical Weekly Exercise
Exercise activities were divided into either a cardiovascular exercise category or a strength training category. Activities such as running, biking, and swimming were included in the cardiovascular exercise category, and activities such as weightlifting and crunches were included in the strength training category. The results of the t-tests revealed a significant difference between groups on the amount of weekly cardiovascular exercise, $t(16.68) = -2.22$, $p < .05$. As hypothesized, participants in the active eating disorder group spent more
minutes engaging in cardiovascular exercise each week than did participants in the recovered eating disorder group (active: \(M = 343.18, SD = 174.65\); recovered: \(M = 181.25, SD = 142.95\)). While there were no significant differences between groups in terms of amount of time spent doing strength training exercises, the mean scores were higher in the active group than in the recovered group, illustrating a possible trend (see Table 2).

**Discussion**

Few studies have taken such an in-depth look at psychological aspects of exercise in a clinical sample of eating disorder patients. The results of this preliminary study support and extend findings suggested by other studies, namely, that those with eating disorders are more likely to engage in exercise in order to control their weight (Davis et al., 1997), find it more difficult to stop thinking about exercise (Ackard, Brehm, & Steffen, 2002; Steffen & Brehm, 1999), and spend more time doing cardiovascular exercise on a weekly basis. It is important to note that although significant differences were not found on other measures, including the motives related to attractiveness, tone, mood improvement and, as well as guilt, amount of time spent thinking about exercise, and weekly strength training exercise, all but the exercise motive related to mood improvement showed a possible trend in the hypothesized direction. While these findings are preliminary, they suggest that there is a clear difference in both exercise attitudes and behaviors between those who have recovered from an eating disorder and those who currently have an eating disorder.

The current study has several strengths: 1) comparing an active eating disorder group to a recovered eating disorder group, 2) including several psychological variables in one study, and 3) using a well-established semi-structured interview to determine eating disorder diagnosis. In terms of limitations, there is no healthy control comparison group, meaning that it is unclear how the recovered group would compare to a non-eating disorder sample on the exercise factors of motivation, guilt, cognitions, and typical weekly patterns. Another limitation is the small sample size. However, both of these limitations will be addressed with the ongoing data collection of more current and former eating disorder patients and a healthy control group. The current p-values suggest that an increase in the sample size will probably yield significant differences between groups on more measures.

Much more research needs to be conducted examining exercise participation and its link to the development and maintenance of eating disorders. Future research should look at what factors contribute to change in these exercise factors from active to recovered stages in eating disorders. Similarly, future research should examine these issues separately for anorexia nervosa and for bulimia nervosa as there may be differences between these two eating disorder diagnoses in terms of how exercise attitudes and behaviors may change with recovery from the eating disorder. Just as Sundgot et al. (2002) noted, these findings also suggest that exercise, with appropriate guidance, may be an important element in the treatment of eating disorders. A successful exercise component may emphasize motives for exercise related to mood improvement and enjoyment as well as illustrate safe and healthy exercise techniques and behaviors. Lastly, exercise attitudes and behaviors may be a key factor in more fully defining recovery from an eating disorder and thus should be included in assessments of outcome.

**Table 1**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Recovered ED</th>
<th>Active ED</th>
<th>t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>23.63 (2.97)</td>
<td>22.00 (3.55)</td>
<td>-2.48</td>
<td>.025</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.9 (3.29)</td>
<td>20.66 (3.77)</td>
<td>-2.22</td>
<td>.041</td>
</tr>
<tr>
<td>SES (years of highest parent education)</td>
<td>17.25 (2.96)</td>
<td>17.36 (2.20)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>87.5% Caucasian</td>
<td>100% Caucasian</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marital Status</td>
<td>37.5% Married</td>
<td>9.1% Married</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifetime ED Diagnosis</td>
<td>72.7% EDNOS</td>
<td>27.3% AN</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current ED Diagnosis</td>
<td>Not Applicable</td>
<td>72.7% EDNOS</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note.** ED = eating disorder; BMI = body mass index; SES = socio-economic status; AN = anorexia nervosa; BN = bulimia nervosa; EDNOS = eating disorder not otherwise specified. Means (standard deviations) are reported for age, BMI, and SES. The percentiles for lifetime ED diagnosis sum to greater than 100% since the individuals could have had more than 1 ED diagnosis.

**Table 2**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Recovered ED</th>
<th>Active ED</th>
<th>t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight Control</td>
<td>12.00 (2.31)</td>
<td>15.64 (3.91)</td>
<td>-2.48</td>
<td>.025</td>
</tr>
<tr>
<td>Attractiveness</td>
<td>12.38 (4.34)</td>
<td>14.73 (4.82)</td>
<td>-1.09</td>
<td>.289</td>
</tr>
<tr>
<td>Tone</td>
<td>14.38 (4.91)</td>
<td>16.45 (3.86)</td>
<td>-1.90</td>
<td>.089</td>
</tr>
<tr>
<td>Fitness</td>
<td>20.88 (5.38)</td>
<td>21.91 (3.36)</td>
<td>-0.52</td>
<td>.62</td>
</tr>
<tr>
<td>Health</td>
<td>22.38 (4.34)</td>
<td>20.73 (4.10)</td>
<td>0.94</td>
<td>.41</td>
</tr>
<tr>
<td>Mood</td>
<td>19.86 (4.60)</td>
<td>22.64 (4.37)</td>
<td>-1.29</td>
<td>.215</td>
</tr>
<tr>
<td>Enjoyment</td>
<td>12.13 (3.40)</td>
<td>10.64 (4.65)</td>
<td>0.77</td>
<td>.45</td>
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</table>

**GUILT**

<table>
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<th>Active ED</th>
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<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>Guilt</td>
<td>3.13 (1.36)</td>
<td>4.09 (1.22)</td>
<td>-1.62</td>
<td>.122</td>
</tr>
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</table>

**COGNITIONS RELATED TO EXERCISE**

<table>
<thead>
<tr>
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<th>Active ED</th>
<th>t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount of time</td>
<td>2.25 (1.04)</td>
<td>3.00 (1.26)</td>
<td>-1.37</td>
<td>.18</td>
</tr>
<tr>
<td>Difficult to stop</td>
<td>1.88 (8.3)</td>
<td>3.27 (1.56)</td>
<td>-2.52</td>
<td>.013</td>
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**TYPICAL EXERCISE PATTERNS**

<table>
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<tr>
<th>Variable</th>
<th>Recovered ED</th>
<th>Active ED</th>
<th>t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular Minutes/Week</td>
<td>181.25 (142.95)</td>
<td>343.18 (174.65)</td>
<td>-2.22</td>
<td>.041</td>
</tr>
<tr>
<td>Strength Training Minutes/Week</td>
<td>56.13 (70.04)</td>
<td>92.05 (87.08)</td>
<td>-0.96</td>
<td>.35</td>
</tr>
</tbody>
</table>

**Note.** ED = eating disorder. Means and (standard deviations) are presented in the second and third columns.
**References**


Introduction

Cell survival depends on homeostasis, the delicate equilibrium between anabolic and catabolic processes. Autophagy, literally ‘self-eating’, is a highly regulated catabolic process essential in the maintenance of eukaryotic cells. It is the process in which defective and unneeded organelles along with cytosol are taken into an autophagic vesicle, known as an autophagosome, and transported to the vacuole or lysosome to be degraded. The resulting monomers are recycled back into the cytosol and used to build new macromolecules (Klionsky and Emr, 2000; Nair and Klionsky, 2005). In humans both excessive and insufficient autophagy are linked to many diseases including cancer, pathogen infections, Alzheimer’s-, Huntington’s-, and Parkinson’s disease as well as ageing. The precise role of autophagy in disease is unknown, but it has been proposed that autophagy may help to prevent the accumulation of denatured proteins and aggregates of denatured proteins that may be toxic to the cells and cause cell death (Hara et al., 2006; Komatsu et al., 2006). Autophagy is also essential for the survival of higher organisms. New born mice deficient for Atg5, which is essential for autophagosome formation, die within 24 hours, despite appearing almost normal at birth (Kuma et al., 2004; Mizushima et al., 2001). A cause for certain cancers may be the inhibition of autophagy; however, autophagy may also be important to the survival of the nutrient poor cells at the interior of a tumor or the survival of a tumor during the stress of cancer treatments. Therefore, the manipulation of autophagy could potentially aid in tumor suppression (Shintani and Klionsky, 2004). Autophagy also works as a defense against pathogens that invade mammalian cells.

Autophagy takes place in all eukaryotic cells. However, it is the study of autophagy in yeast that has led to the discovery of most of the proteins required for autophagy (Klionsky, 2003; Thumm et al., 1994; Tsukada and Ohsumi, 1993). However, the specific functions of many autophagy proteins are still unknown. In yeast, the simplest eukaryote, autophagy is primarily a response to nutritional stress. *Saccharomyces cerevisiae*, commonly known as Baker’s yeast, is a model organism for studying autophagosome formation. *S. cerevisiae* contains a specialized pathway for the delivery of certain proteins to the vacuole by selective autophagy known as the Cytoplasm to vacuole targeting (Cvt) pathway. The Cvt pathway is highly specific, transporting only a few selected proteins to the vacuole, whereas autophagy is generally believed to be non-selective. Also, the Cvt pathway is biosynthetic and occurs in nutrient rich conditions, unlike non-selective autophagy, which is induced by starvation.

The two known cargo proteins of the Cvt pathway are the precursor of aminopeptidase1 (prApe1) and α-mannosidase 1 (Ams1) (Oda et al., 1996; Thumm et al., 1994; Tsukada and Ohsumi, 1993). The pathway begins in the cytosol when prApe1 oligomerizes into dodecamers, which then aggregate to form the Ape1 complex (Stromhaug and Klionsky, 2003). This is followed by Atg19 (autophagy-related protein 19), which acts as a receptor and binds to the Ape1 complex (Figure 1). Atg19 also binds and recruits Ams1 to the Ape1 complex; the resulting structure is often referred to as the Cvt complex. Atg19 next interacts with Atg11, Atg8 and Atg1 (Kim et al., 2001; Stromhaug and Klionsky, 2003). The function of these interactions is believed
to be to recruit autophagic membrane and the vacuole forming machinery. In nutrient rich conditions, Atg1, a serine/threonine protein kinase, which is essential in all forms of autophagy, has low activity. In starvation conditions, Atg1 binds to and is activated by dephosphorylated Atg13; and this, results in the activation of Atg1. Atg13 dephosphorylation is regulated by Tor, by an unknown mechanism (Schmelzle and Hall, 2000). It appears that low activity of Atg1 leads to the formation of small autophagosomes or Cvt vesicles and selective autophagy of the Cvt complex, whereas activated Atg1 induces the creation of large autophagosomes and non-selective autophagy (Kim et al., 2001; Kamada et al., 2000; Scott et al., 2000).

Atg8, a marker for autophagic membrane, is involved in the association of the Ape1 complex with autophagic membrane. Atg8 is an ubiquitin-like protein, which is first activated by the ubiquitin-activating-like enzyme, Atg7, and next conjugated to the lipid phosphatidylethanolamine by the ubiquitin-ligase-like enzyme, Atg3. Atg7 also activates the ubiquitin-like protein, Atg12, and conjugates it to Atg5 with the help of Atg10 (Komatsu et al., 2001; Shintani et al., 1999). Following this conjugation, Atg16 dimerizes and joins a pair of Atg12-Atg5 conjugates resulting in a 350 kDa protein complex, which is required for efficient lipidation of Atg8 (Ichimura et al., 2000). These proteins along with several others induces the formation of the Cvt vesicle. In this process, all of Atg11 and some of Atg8 are sorted outside the vesicle. Upon completion of the Cvt vesicle and transport to the vacuole, the Cvt vesicle’s outer membrane fuses with the vacuolar membrane allowing the now single membrane bound Cvt body to enter the vacuolar lumen. In the lumen, the membrane is degraded, the pro-peptide of prApe1 is cleaved off by proteinase B, and the complex dissociates into active Ape1 dodecamers (Stromhaug and Klionsky, 2003).

Until recently S. cerevisiae was the only organism known to have a Cvt pathway. However, it is now known that the methylotrophic yeast, Pichia pastoris, also has a Cvt pathway (Farré et al., 2007). P. pastoris is also capable of non-selective autophagy and selective autophagy of peroxisomes. P. pastoris and S. cerevisiae are related yeasts, in which most of the autophagy genes are highly conserved between the two species (Kim et al. 2001). However, P. pastoris has no detectable ortholog to S. cerevisiae Atg19 (Farré et al., 2007). The proteins PpAtg26 and PpAtg28 are involved in the Cvt pathway in P. pastoris, but not in the Cvt pathway in S. cerevisiae. Moreover, the pro-peptide of P. pastoris Ape1 (PpApe1) is similar to the pro-peptide of S. cerevisiae Ape1 (ScApe1) in secondary structure, but not in primary structure (Farré et al., 2007). In S. cerevisiae, the pro-peptide of Ape1 interacts with Atg19. Atg19 is a receptor protein that interacts with Atg11, Atg8, Atg1 and Ams1 and is necessary for the formation of the Cvt complex in S. cerevisiae (Stromhaug and Klionsky, 2003). We have introduced prApe1 from S. cerevisiae into P. pastoris in order to identify the factors required for the uptake of a foreign protein aggregate to the vacuole. We believe this could help in understanding how autophagic membrane recognizes and is recruited to a protein aggregate, and how autophagosomes form.

**Strains and Media**
Yeast strains used in this study are listed in Table 1. The P. pastoris strain, JC308 (ade1, arg4, his4, ura3), is from James Cregg (Keck Graduate Institute of Applied Life Science, Claremont, CA). The S. cerevisiae strain, By4742 (MATa his3, leu2, lys2, ura3), is from EUROSCARF. Strains were grown at 30°C in an orbital shaker at 250 rpm (New Brunswick Innova 4300) generally to mid-log phase in liquid SMD medium (0.67% yeast nitrogen base without amino acids, 2% glucose, and vitamins and auxotrophic amino acids as required) or in YPD medium (1% yeast extract, 2% bacto-peptone, 2% dextrose), or on plates supported with 2% agar.
Table 1: Yeast strains used in this study

<table>
<thead>
<tr>
<th>Strain</th>
<th>Genotype</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>P. pastoris strains</td>
<td></td>
<td></td>
</tr>
<tr>
<td>JC308</td>
<td>ade1 arg4 his4 ura3</td>
<td>Cregg et al.</td>
</tr>
<tr>
<td>RR3</td>
<td>JC308 his4::PpAtg1-AP4:His3</td>
<td>This study</td>
</tr>
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Plasmid Construction

pR3 expressing GFP-tagged S. cerevisiae prApe1 from the pGAP promoter was constructed by amplifying GFP-prApe1 by PCR (IDProof TM DNA Polymerase, IDLabs, Ontario), from pPS130 (Stromhaug, MBC 2004) and inserted into the SacI and SpeI sites of pIB2 (Sears, Yeast 1998). The plasmid was linearized with Sall and integrated into his4 of JC308 resulting in the strain RRY3. pR2 was made by amplifying GFP-Atg19 (Shintani, Dev. Cell 2002) by PCR and inserted into SacI and SphI sites of pBLURA (Sears, Yeast 1998), and next pGAP, amplified by PCR, was inserted into the SacI site. The plasmid was linearized with XbaI and inserted into RRY3 cells to make the strain KSY2. pKSY1 was made by replacing the pGAP promoter of pR2 with pYPT1. pGAP was removed from pR2 by cutting with SacI and pYPT1 was amplified from pHK (Sears, Yeast 1998) by PCR and inserted into the SacI site of pBLURA. The strain KSY3 is made by inserting pKSY1 linearized with XbaI into RRY3 after replacing the GFP tag of Atg19 with an HA tag.

Yeast Transformations

Electro-competent P. pastoris cells were made by incubations with LiAc and DTT. Competent cells are resuspended in sorbitol with 20% glycerol and frozen at -80°C. Transformations were carried out by electroporation in which a mix of 80 µl of competent yeast cells and 9 µl of linearized plasmid are incubated on ice for 10 minutes and then pulsed with 2000V for 5ms in a 2mm gap width cuvette, and immediately suspended in 1mL 1M sorbitol. Transformants were plated on SMD plates with selection and grown at 30°C and cloned by streaking single colonies on an SMD plate with selection.

Two-Hybrid Assay

The two-hybrid assay was done according to the Matchmaker Two-Hybrid System (Clontech, CA). Genes were cloned into the plasmids pGBDU-C2 and pGAD-C2. These two plasmids were co-transformed into PJ69-4A and colonies patched on histidine selection plates.

Protein Electrophoresis and Western Blotting

All protein samples were made by TCA precipitation, resolved by SDS-PAGE, transferred onto Immobilon-P membrane (Millipore) by SemiDry (BioRad) transfer and blocked in 5% nonfat milk in TBST. The blots were probed with primary rabbit antiserum to Ap (diluted 1:20,000 in TBST), primary rabbit antiserum to Atg19 (diluted 1:5,000), mouse antibody against hemagglutinin (HA) (diluted 1:4,000), or GFP (diluted 1:2,000). Blots were washed and probed with secondary goat anti-rabbit or rabbit anti-mouse IgG conjugated to horseradish peroxidase both diluted 1:10,000 in TBST. Blots were then developed by luminol chemiluminescence (West Pico, Pierce).

Fluorescence Microscopy

Cell strains expressing fluorescent fusion proteins were taken from plate or liquid cultures (concentrated by centrifugation at 600g for 30 seconds and resuspended in SMD media). Cell strains were analyzed by fluorescence microscopy in a Leica DM5000 Microscope equipped with a digital camera and pictures were captured by Leica FW4000 software.

Results

S. cerevisiae prApe1 aggregates into an Ape1 complex in P. pastoris. The pathology of a number of human diseases involves the formation of protein aggregates that perturb normal cell function. Intriguingly, in yeast, the vacuolar protease, precursor aminopeptidase I (prApe1), is synthesized in the cytosol and aggregates before delivery to the vacuole by the Cvt pathway. The study of the delivery of this aggregate can shed light on how protein aggregates are removed by autophagy.

The delivery of prApe1 to the vacuole can be monitored using red fluorescent protein tagged prApe1 (RFP-prApe1). In normal S. cerevisiae cells, the majority of RFP-prApe1 can be seen in the vacuole (Figure 2). However, if the vacuolar delivery is blocked by the removal of the prApe1 receptor, Atg19, all RFP-prApe1 is found in a single, cytosolic aggregate referred to as an Ape1 complex. How this aggregate forms is currently unknown. It is also possible that additional components are present in the prApe1 complex. When the same RFP-prApe1 construct was expressed in P. pastoris cells, a single red aggregate was seen in the cytosol (Figure 2). This suggests that prApe1 aggregation is an intrinsic feature of prApe1 and that additional components are not required. Identical results were obtained using GFP-tagged prApe1, additionally when only RFP or GFP was expressed, no fluorescent aggregate was seen (data not shown). These results suggest that the aggregation was not caused by the fluorescent protein itself. Furthermore, the RFP protein used is a monomeric form of RFP (mRFP), which does not have a tendency to form tetramers (Campbell et al., 2002).
prApe1 aggregation is an intrinsic feature of *S. cerevisiae* prApe1.

The fusion protein RFP-ScprApe1 is expressed in a *S. cerevisiae* strain lacking Atg19. In this strain, RFP-prApe1 aggregates, and can be seen using a fluorescent microscope. The same fusion protein is also expressed in the *P. pastoris* strain JC308. In the *P. pastoris* strain, ScprApe1 also aggregates spontaneously, indicating that it is an intrinsic feature of ScprApe1.

ScAtg19 binds to the ScprApe1 aggregate in *P. pastoris*. In rich media conditions, both aggregated mRFP-ScprApe1 and GFP-ScprApe1 in *P. pastoris* remained in the cytosol and there did not appear to be any fluorescence in the vacuole. Only upon starvation did a weak fluorescent signal appear in the vacuole, indicating some uptake by non-selective autophagy (data not shown). This suggests that there is not a receptor, like ScAtg19, in *P. pastoris* or that PpAtg19 does not bind to ScprApe1. We next expressed GFP-tagged ScAtg19 in *P. pastoris* and found the protein to be evenly distributed in the cytosol with no apparent localization to any cell structures or to the vacuole (Figure 3). However, when GFP-ScAtg19 was co-expressed with RFP-prApe1, the two proteins co-localized showing that GFP-Atg19 can bind to the Ape1 complex in *P. pastoris* and forms a Cvt complex (Figure 3). We could still detect very little RFP-prApe1 in the vacuole in cells grown in rich media, suggesting that *P. pastoris* autophagy proteins are not able to bind properly to Atg19 and recruit autophagic membrane.

**Figure 2:** prApe1 aggregation is an intrinsic feature of *S. cerevisiae* prApe1.

The fusion protein RFP-ScprApe1 is expressed in a *S. cerevisiae* strain lacking Atg19. In this strain, RFP-prApe1 aggregates, and can be seen using a fluorescent microscope. The same fusion protein is also expressed in the *P. pastoris* strain JC308. In the *P. pastoris* strain, ScprApe1 also aggregates spontaneously, indicating that it is an intrinsic feature of ScprApe1.

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**Figure 3:** Atg19 binds to *S. cerevisiae* prApe1 aggregate in *P. pastoris*.

In a *P. pastoris* strain expressing RFP-ScprApe1 and GFP-Atg19, GFP-Atg19 co-localizes with the prApe1 aggregate, as can be seen using fluorescent microscopy. This indicates that these two proteins interact.

**Figure 4:** Two-hybrid assay confirms that *P. pastoris* Atg11 does not bind to Atg19.

Atg19, Atg11, and Atg8 from *S. cerevisiae* and Atg11 and Atg8 from *P. pastoris* were fused to the binding domain (BD) or activating domain (AD). Pairs of BD- and AD-fusion proteins were co-expressed in *S. cerevisiae*. Growth indicates interaction. This two-hybrid assay show, that PpAtg11 does not interact with ScAtg11 or with PpAtg8 and that PpAtg8, ScAtg8, and ScAtg11, all can bind to ScAtg19.

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Figure 5: Atg11/11 fusion protein.
This Atg11/11 fusion protein contains the C-terminal domain of ScAtg11, which binds to Atg19, and the N-terminal domain of PpAtg11, which directs the protein to the vacuolar membrane. In this diagram, GFP is fused to the N-terminal domain of the Atg11/11 fusion protein.

The C-terminus of ScAtg11 has been shown to bind to ScAtg11 by the two-hybrid assay and this domain fused to GFP as well as the GFP-Atg11/11 fusion protein co-localize with RFP-prApe1 in *S. cerevisiae* in a similar fashion as GFP-ScAtg11 (data not shown). When the GFP-Atg11/11 fusion protein was expressed in *P. pastoris*, it localized to the rim of the vacuole, confirming that the N-terminus of Atg11 contains the domain that directs the protein to the vacuolar membrane (Figure 6). Co-localization with RFP-prApe1 was only found in cells also expressing HA-tagged ScAtg19. In these cells, the Cvt complex was locked to the vacuolar membrane and not moving randomly about in the cytosol. However, little RFP-prApe1 could be detected in the vacuole in cells grown in rich media even though the Cvt complex appeared to be tethered to the vacuole.

Figure 6: Atg11/11 fusion protein tethers the *S. cerevisiae* Ape1 complex to the vacuole.
In the *P. pastoris* cell line expressing RFP-ScprApe1, high copy GFP-Atg19, and high copy of ScprApe1, a green ring can be visualized around the outside of the prApe1 complex using fluorescent microscopy.

However, when high levels of GFP-Atg19 were expressed, strong vacuolar staining and smaller or no cytosolic RFP-prApe1 aggregates were observed (Figure 8). Surprisingly, this uptake occurred in rich medium and did not have to be induced by starvation or by induction of peroxisome autophagy, and the uptake occurred even though the aggregate did not seem to be tethered to the vacuole. This suggests that the uptake was not by microautophagy and also that Atg11 may not be involved in the vacuolar uptake of the aggregate. In fact, the Atg11/11 fusion protein was also expressed in these cells and the prApe1 aggregate was tethered to the vacuolar membrane. However, little fluorescence was seen in the vacuole (data not shown). Western blotting confirmed that little mature Ape1 was formed in these cells (Figure 9). Based on these results we propose that the binding of Atg8 to Atg19 may be sufficient for recruitment of autophagic membrane to the prApe1 aggregate followed by autophagic sequestration.

Atg19-induced uptake of prApe1 to the vacuole. We next wanted to see if we could induce microautophagy of the prApe1 aggregate. In order to better see the vacuolar arms engulfing the aggregate, we expressed high amounts of untagged prApe1 in addition to RFP-tagged prApe1. GFP-Atg19 expressed from a weak promoter was seen forming a ring around the prApe1 aggregate, indicating that GFP-Atg19 binds to the surface of the prApe1 complex (Figure 7). In the cells expressing RFP-ScprApe1, occasional weak red staining of the vacuole was observed, suggesting that there was some vacuolar uptake of the prApe1 aggregate in rich media conditions.

Figure 7: Atg19 binds to the surface of *S. cerevisiae* Ape1 complex.
In a *P. pastoris* cell line expressing RFP-ScprApe1, GFP-Atg19, and high copy of ScprApe1, a green ring can be visualized around the outside of the prApe1 complex using fluorescent microscopy.

Figure 8: High expression of *S. cerevisiae* prApe1 and Atg19 allow transport to the vacuole in *P. pastoris*.
In the *P. pastoris* cell line expressing RFP-ScprApe1, high copy ScprApe1, high copy GFP-Atg19, a red diffused signal can be seen in the vacuole, indicating that RFP-ScprApe1 is transported to the vacuole. The vacuolar staining is not seen in a similar strain line expressing a lower copy of GFP-Atg19.
required for the specific pathway. Removal of common and pathway specific autophagy genes could therefore tell us by which mechanism ScprApe1 is delivered to the vacuole. We are currently in the process of knocking out Atg7, which is necessary for all forms of autophagy, in order to verify that the uptake into the vacuole we see is indeed by autophagy. Our data suggests that Atg11, which is required for selective autophagy of both prApe1 and peroxisomes in S. cerevisiae and P. pastoris, is not required for delivery of ScprApe1 to the vacuole when expressed in P. pastoris, but this has to be verified by removing Atg11. Intriguingly, an orthologue of Atg11 has not yet been identified in higher organisms, and it is not yet clear how protein aggregates are recognized and sequestered in animals.

It is possible that the ScprApe1 aggregate that forms in P. pastoris could actually be an aggregate of PpprApe1 and ScprApe1. If the aggregate is a mix of PpprApe1 and ScprApe1, then there should be some uptake to the vacuole via the P. pastoris Cvt pathway. However, we see little uptake of RFP-prApe1 into the vacuole when ScAtg19 is not expressed. Perhaps this lack of uptake can be explained by there being a less efficient Cvt pathway in P. pastoris than in S. cerevisiae, and that by expressing high amounts of ScprApe1 the Cvt pathway is saturated. It is also possible that ScprApe1 could be binding to a yet to be discovered P. pastoris homologue of ScAte19, or to another autophagy protein that functions as a prApe1 receptor. However, our fluorescence microscopy data suggests that ScAtg19 is not binding to PpprApe1. In control cells expressing only GFP-Atg19 and not ScprApe1, there is only diffuse cytosolic staining and no visible punctate structure. Furthermore, the amino acid sequence of the pro-peptide of prApe1 from S. cerevisiae and P. pastoris is not conserved.

The interaction of PpAtg8 with ScAtg19 could be sufficient for the recruitment of phagophore membrane to the ScApe1 complex. This interaction along with the machinery from the recently discovered Cvt pathway in P. pastoris could be causing uptake of the Ape1 complex to the vacuole. Atg8, which recruits autophagic membrane, could be used as a marker to confirm that PpAtg8 is interacting with ScAtg19 in vivo. A study involving GFP-Atg8 expressed in P. pastoris could show if Atg8 is indeed interacting with Atg19 and with P. pastoris autophagy proteins. Much work still needs to be done in order to determine which factors are actually causing vacuolar uptake of ScApe1 in P. pastoris.

**References**


Leyland Young, PhD
Mechanical and Aerospace Engineering
NASA Balloon Program Office, Wallops Flight Facility

I believe graduate school success is predetermined by the correct selections of school, advisor, and research topic. For me, the Ronald E. McNair Post-baccalaureate Achievement Program aided me in these selections. In part, I am a living testament of the McNair Scholar’s motto, “Before you can make a dream come true, you must first have one.” The truth is, I had a post-baccalaureate dream; the problem was: I didn’t know how to make that dream come true. In 1996, my sophomore year, I began thinking about graduate school; however, as a first generation college student, my family and I were clueless about graduate school and its requirements. Ten years later, I can honestly say that I was very fortunate to have been accepted into the MU McNair Scholars Program.

As a McNair’s Scholar, I had an advantage over other students from the time I began the graduate application process to the time I completed graduate school. I attribute this advantage to the MU McNair Program Director, Dr. Vicki Curby and the program’s staff who helped me through the graduate school application process as well as the support for the GRE test; moreover, they provided me with the necessary skills needed as a graduate professional. I believe the most difficult part of graduate school is presenting your research. However, my confidence level was high stemming from the opportunities of attending and presenting my McNair research at national conferences. There is no doubt that I owe my success today and during my graduate studies to what I’ve learned as a Ronald E. McNair Scholar. The program knowingly and unknowingly provides the motivation for one to succeed.

My McNair faculty mentor, Dr. P. Frank Pai, became my M.S. Thesis and Ph.D. Dissertation advisor. My M.S. Thesis was a continuation of my McNair research; thus, I was able to complete my M.S. Thesis in just two semesters. Even though I received solicitations from other graduate schools, I chose to stay at MU and work with Dr. Pai because of his experience and the structural dynamics research I conducted as a McNair’s Scholar. Just the same, I believe Dr. Pai accepted me as one of his graduate students because he knew of the goals set by the McNair Scholar program.

In May 2003, I received a Ph.D. in Mechanical and Aerospace Engineering. The motivation I had as a McNair intern, the commitment and effort of the MU McNair program’s staff, and the steady guidance of my McNair mentor and advisor, allowed me to achieve a personal goal of finishing both my M.S. and Ph.D. degrees with a 4.0 gpa. I am currently employed with New Mexico State University Physical Science Laboratory at the NASA Balloon Program Office, Wallops Flight Facility. Because Ronald E. McNair was a NASA astronaut, I feel blessed to be working at NASA. There is no hesitation in saying, I am very proud to have been a McNair Scholar, and my graduate education and professionalism today are reinforced because of my experiences in the University of Missouri’s Ronald E. McNair Post-baccalaureate Achievement Program.

Lisa Molix, PhD
School of Science and Engineering
Assistant Professor, Psychology

In 1998, I was selected to participate in the Ronald E. McNair Post-baccalaureate Achievement Program. As a participant in this remarkable program, I gained valuable research experience, guidance with the graduate school application process, and information about how to succeed in graduate school. During my time as a McNair Scholar I also had opportunities to attend workshops and conferences that enabled me to foster life-long relationships with faculty and students from all over the U.S.

The most valuable part of my McNair experience was working with my faculty mentor, Dr. B. Ann Bettencourt. Although I had worked as research assistant prior to being in the McNair program, this was my first opportunity to be involved in all aspects of the research process (e.g., design, data collection, data analysis, manuscript preparation, formal presentation). Because Dr. Bettencourt interacted with me as if I was a graduate student, my McNair experience was essentially like taking a graduate school test drive.

The relationship I fostered with Dr. Bettencourt as a McNair scholar convinced me that she would also be an ideal graduate school mentor, and influenced my decision to go on to work with her as a graduate student. In May of 2007, I received a Ph.D. in Social and Personality Psychology with a minor in Quantitative Methodology from the University of Missouri-Columbia. I am currently an Assistant Professor at Tulane University, teaching courses that I love and conducting research that aims to answer some of the questions I initially proposed in my McNair project on improving intergroup relations. I am grateful to have been afforded the opportunity to participate in the McNair program, as I have truly benefited from the guidance I received.
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2006-2007 McNair Scholars

Back row: Jeremy Bloss (Student Services Advisor), Darlene Dixon (Program Assistant), NaTashua Davis (Assistant Director), Vicki Curby (Director)

Fifth row: Robert Jinkerson, Enjoli Sims, Mary Stilwell, Samuel Grinter

Fourth row: Njabulo Ngwenyama, Katherine Speichinger, Ashley Acevedo, Amber Bell

Third row: Michelle Carpentier, Bridgette Adams, Sara Mijares

Second row: Marcus Adair, Tracy Johnson, Anthony Minarovic (alternate scholar), Stanley Ikpe

First row: Allison Barlows, Kari Rott, Lauren Lewis

(Not pictured): Valeska Araujo, David Aguayo, Tamela Smith