

## Original Article

### Life History Strategy and Disordered Eating Behavior

Catherine Salmon, Department of Psychology, University of Redlands, Redlands, CA, USA. Email: [Catherine\\_Salmon@redlands.edu](mailto:Catherine_Salmon@redlands.edu).

Aurelio José Figueredo, Department of Psychology, University of Arizona, Tucson, AZ, USA. Email: [ajf@u.arizona.edu](mailto:ajf@u.arizona.edu) (Corresponding author).

Lindsey Woodburn, Department of Psychology, University of Arizona, Tucson, AZ, USA. Email: [woodburn@email.arizona.edu](mailto:woodburn@email.arizona.edu).

**Abstract:** A sample of female undergraduates completed a packet of questionnaires consisting of the Arizona Life History Battery, a modified version of the Eating Disorders Inventory, the Behavioral Regulation scales from the Behavior Rating Inventory of Executive Function, and two measures of Female Intrasexual Competitiveness that distinguished between competition for mates and competition for status. As predicted, Executive Functions completely mediated the relation between Slow Life History Strategy and Disordered Eating Behavior. Surprisingly, however, the relation between Female Intrasexual Competitiveness (competition for mates and competition for status) and Disordered Eating Behavior was completely spurious, with executive functions serving as a common cause underlying the inhibition of both Disordered Eating Behavior and Female Intrasexual Competitiveness. The protective function of Slow Life History Strategy with respect to Disordered Eating Behavior apparently resides in a higher degree of Behavioral Regulation, a type of Executive Function. The enhanced Behavioral Regulation or self-control, of individuals with a Slow Life History Strategy is also protective against hazardously escalated levels of Female Intrasexual Competitiveness.

**Keywords:** eating disorders, anorexia, bulimia, life history strategy, executive functions, female intrasexual competition

---

## Introduction

A compelling puzzle of our modern world is the disturbing obsession of some women with body image and dieting. Why do so many women in North America place such an emphasis on being thin? Why do these desires lead to eating disorders in only some women? It is commonly assumed that the desire for a thin female physique and its

pathological expression in eating disorders result from a social pressure for thinness. In recent years, anorexia nervosa and bulimia have become the most attention grabbing eating disorders with a multitude of studies being published from a variety of perspectives. With all this attention, one would think that we would have a concrete understanding of the causes of eating disorders and yet there have been a plethora of theories that have been proposed in the literature. We will briefly review the predominant mainstream theories before discussing evolutionary approaches to the study of dieting behavior and our own research findings. We would also like to note that we use the term “disordered eating behavior” to refer to a range of behaviors including various degrees of dieting from mild restriction to anorexia as we are not considering only those who have a clinical diagnosis but those women who are engaging in dieting that temporarily impairs their reproductive function.

### *Psychoanalytic and Sociocultural Approaches*

Early psychoanalytic approaches to anorexia focused on refusing to eat being part of a struggle for psychological control and autonomy and emphasized the role overly controlling parents might play in its development (Bruch, 1988). This also led to a family systems approach and the belief that anorexics were trying to regress to a prepubertal state (Crisp, 1980). The non-reproductive aspect of such a prepubertal state was not an early emphasis but it returned to the spotlight in later evolutionary theorizing about the reproductive impact of such excessive dieting.

Sociocultural theories have held the greatest influence in terms of public opinion on eating disorders (and have also been the focus on many researchers and clinicians) in recent years, attributing major risk to exposure to western ideals of female beauty that focus on a thin form (Botta, 2003; Garner and Garfinkel, 1997; Groesz, Levine, and Murnen, 2002). Excellent coverage of the sociocultural perspective can be found in reviews by Polivy and Herman (2002) and Striegel-Moore and Bulik (2007). At the heart of the cultural models is the idea that eating disorders develop from the internalization of a thin ideal of beauty. Individuals are exposed to such ideals, which they then internalize. When they experience an inevitable discrepancy between their self and the ideal, the result is body dissatisfaction and extreme dieting behavior. Other factors such as social pressure, class, and perfectionism are also used to explain why this cultural ideal affects some young women but not all. However, most of these models focus too much on western media images and do not take into account the role played by interactions between female reproductive roles and the social environment. There is an assumption that the western media has created a thin standard of beauty. However, cross-cultural explorations of ideal body type suggest that the role of women in a society (valued in traditional reproductive roles vs. modern workplace roles) is an excellent predictor of whether a society has a thin or heavier standard of female beauty (Anderson, Crawford, Nadeau, and Lindberg, 1992). The social dominance of women outside the home is a better predictor of a thin standard of beauty than exposure to media images of thinness.

### *Biological approaches*

There has also been some attention paid to the possible influence of genetics, particularly in terms of susceptibility to such cultural forces. Twin studies have suggested some genetic contribution is likely to play a role, though the specifics remain very unclear

(Klump, Miller, Keel, McGue, and Iacono, 2001, Striegel-Moore and Bulik, 2007). Studies of treated twin samples and twin samples from the general population suggest a heritability of close to 70% and also implicate certain alleles as risk factors for anorexia nervosa specifically (Gorwood, Kipman, and Foulon, 2003).

### *Evolutionary approaches*

There have been a number of evolutionary approaches to eating disorders, most focusing on reproductive suppression or female competition for mates or status (for a recent overview of evolutionary approaches to eating disorders, see Kardum, Gracanin, and Hudek-Knezevic, 2008). Much of the work in this area has focused on the reproductive suppression hypothesis (RSH) which suggests that natural selection shaped a mechanism for adjusting female reproduction to sociological conditions by altering the amount of body fat (Anderson and Crawford, 1992; Surbey, 1987; Wasser 1990; Wasser and Barash, 1983). Since female body fat stores significant amounts of estrogen and converts androgens to estrogen, adjusting body fat in response to environmental stress is an effective mechanism for adjusting women's reproduction (Frisch, 1990, 1996). While the threshold varies from woman to woman, and others factors also contribute, most women require adipose tissue to make up about 22% of their body weight to maintain ovulation. In female athletes, for example, who are close to this threshold, menstruation can be turned off or on by the loss or gain of only a few pounds. Thus, alterations in the rate of adolescent weight gain or weight control in adult lean women (Ellison, 2008; Rippon, Nash, Myburgh, and Noakes, 1988) could have been an efficient mechanism for adjusting ancestral reproductive effort in response to environmental conditions.

In contemporary western culture, social and ecological cues (such as high levels of social competition among women and stressful sexual attention from undesirable males), which would have signaled the need for temporary postponement of reproduction in ancestral environments, may now be experienced to an unprecedented intensity and duration, leading to fears related to fatness and body image (Salmon, Crawford, Dane, and Zuberbier, 2008). Such a mechanism could also serve to delay puberty in response to conditions indicative of a currently poor environment for reproduction (Surbey, 1987). Several studies (Juda, Campbell, and Crawford, 2004; Salmon et al., 2008) have suggested that high levels of female competition, as well as perceived low levels of social support, may increase dieting behavior, as both may be indications that the conditions for reproduction are not as good as they might be in future. Mealey (2000) took the RSH in a slightly different direction, arguing that modern anorexia is the consequence of intrasexual competition, a manipulative strategy in which dominant females suppress the reproduction of subordinate females (hijacking the existing mechanism for reproductive control). The majority, over 95%, of eating disorders are found in women, which should not be surprising from an evolutionary perspective as reproduction is more closely linked to body fat in women than in men.

Other evolutionary minded approaches have included the AFFH, or adapted to flee famine hypothesis (Guisinger, 2003), which suggests that anorexia's characteristic restriction of food, denial of weight loss, and hyperactivity are evolved mechanisms that would have aided nomadic foragers in leaving depleted environments. In this model, genetic susceptibility is also raised as the reason some people are more likely to develop anorexia. Another recent study (Li, Smith, Griskevicius, Cason, and Bryan, 2009; cited

with permission) looked at the relationship between intrasexual competition (in the form of exposure to high status and competitive same sex individuals) and unhealthy eating in heterosexual and homosexual individuals. They found exposure to cues of intrasexual competition resulted in unhealthy attitudes and body image in women but not men and that for homosexuals, only the men were negatively influenced in terms of attitudes and body image by such exposure. The lesbian participants were not impacted. This study emphasizes the role intrasexual competition may be playing in the development of eating disorders, in particular cues of high status competitors.

Faer, Hendriks, Abed, and Figueredo (2005) studied the relationship between eating disorders and female intrasexual competitiveness, predicting that female intrasexual competitiveness for mates would be the strongest predictor of bulimia (BN), and that, in contrast, female intrasexual competitiveness for status would be the strongest predictor of anorexia (AN). The resulting structural equations model demonstrated that intrasexual competitiveness for mates was the driving factor that ultimately contributed to female competitiveness for status, general competitiveness, perfectionism, body dissatisfaction, drive for thinness, and both bulimia and anorexia. Contrary to initial expectations, the results indicated a mostly spurious causal relationship between female competitiveness for status and anorexia, with the only indirect causal effect being through the influence of Perfectionism, which was uniquely on anorexia and not on bulimia. The role of perceived personal and ideal partner mate value was also explored. Although strongly positively related to each other, perceived personal and ideal partner mate value had nearly equal and opposite effects on body dissatisfaction.

Abed, Mehta, Figueredo, Aldridge, Balson, Meyer, and Palmer (2009) recently performed a constructive replication in the United Kingdom, which included several measures of reproductive life history strategy. These measures permitted a close examination of possible evolutionary sources of female intrasexual competitiveness. This hypothesis was derived from Life History Theory, which is a mid-level theory from evolutionary biology that describes the allocation of limited bioenergetic and material resources to the twin goals of survival and reproduction. According to this theory, resources are first partitioned into *somatic* effort, invested in the survival of the individual organism, and *reproductive* effort, invested in the production of offspring. Reproductive effort is further partitioned into *mating* effort, invested in the acquisition and retention of sexual partner, and *parental* effort, invested in the survival of existing offspring. A *slow* life history strategy (K-selected) is one that emphasizes somatic and parental effort, whereas a *fast* life history strategy (r-selected) is one that instead emphasizes reproductive and mating effort (Bogaert and Rushton, 1989; Figueredo et al, 2005; Stearns, 1992). Thus, a slower life history strategy would be *inconsistent* (and a faster life history strategy would be *consistent*) with the increased expenditure of mating effort entailed in heightened levels of female intrasexual competitiveness (Geary, 2005).

As in the original (Faer et al., 2005) study, the respondents completed the Female Competitiveness for Mates Scale, the Female Competitiveness for Status Scale, the General Competitiveness Scale, and the Eating Disorders Inventory. In addition, respondents also completed the Eating Disorders Examination. All of the measures of life history converged upon a single common Slow Life History factor, all of the measures of competitiveness converged upon a single common Intrasexual Competitiveness factor, and both of the outcome measures converged upon a single Disordered Eating Behavior Factor. As

predicted by Life History Theory, the Slow Life History factor had a negative (inhibitory) effect upon the Intrasexual Competitiveness factor. The Intrasexual Competitiveness factor partially mediated the effect of the Slow Life History factor upon Disordered Eating Behavior, but Slow Life History had a negative direct effect on Disordered Eating Behavior, indicating that Slow Life History inhibits Disordered Eating Behavior by an unmeasured mechanism as well as by the suppression of intrasexually competitive behavior for mates or for status.

The purpose of the present study is to identify one possible neuropsychological mechanism for this inhibitory effect. Executive functions include the abilities entailed in planning for the future, inhibiting or delaying responding, initiating behavior, and shifting between activities flexibly. The ability to set goals, plan, sequence, prioritize, organize, initiate, inhibit, pace, shift, monitor, control, and complete actions all involve executive functions (Lezak, Howieson, and Loring, 2004).

We therefore hypothesized that enhanced Executive Functions, and more specifically Behavioral Regulation (Self-Control), would mediate the effects of Slow Life History Strategy on female intrasexually competitive behavior and on disordered eating behavior. However, we were uncertain as to whether the enhanced Executive Functions possessed by slower life history strategists would serve to reduce disordered eating behavior *indirectly* by inhibiting the heightened intrasexually competitive behavior for mates and for status that has been associated with it in previous research, or whether enhanced Executive Functions would *directly* inhibit disordered eating behaviors as well as intrasexually competitive behavior for mates and for status. Thus, based on the results of the Abed, Mehta, Figueredo, Aldridge, Balson, Meyer, and Palmer (2009) study, we also tested the possibility that enhanced Executive Functions would also have a direct effect upon disordered eating behavior that was not mediated by inhibited intrasexually competitive behavior either for mates or for status.

## **Materials and Methods**

### *Participants*

Participants included one hundred female undergraduate students who received course credit in return for completing the questionnaires. Their mean age was 18.8 years (range 18-47). Ninety-five percent were heterosexual, while 92% were single, 6% living together, and 2% were married. Eighty-six percent came from middle or upper middle class backgrounds, while 6% came from high income families and 9% came from lower income family backgrounds. In addition, the mean age at which they reached menarche was 12.6 years (range 5-16).

### *Procedures*

Respondents completed a packet of questionnaires consisting of the Arizona Life History Battery (ALHB), a modified version of the Eating Disorders Inventory (EDI-2), the Behavioral Regulation scales from the Behavior Rating Inventory of Executive Function (BRIEF-A), and two measures of Female Intrasexual Competitiveness: the Female Intrasexual Competitiveness for Status Scale and the Female Intrasexual Competitiveness for Mates Scale.

*Measures*

*The Arizona Life History Battery (ALHB;* Figueredo, 2007; Gladden, Figueredo, and Jacobs, 2009) is a battery of cognitive and behavioral indicators of life history strategy compiled and adapted from various original sources. These self-report psychometric indicators measure graded individual differences along various complementary facets of a coherent and coordinated life history strategy, as specified by Life History Theory, and converge upon a single multivariate latent construct. They are scored directionally to indicate a “slow” (K-selected) life history strategy on the “fast-slow” (r-K) continuum. The Cronbach’s alphas for these subscales were .63 for the Mini-K Short Form, .83 for Insight, Planning, and Control, .76 for Mother/Father Relationship Quality, .88 for Family Social Contact and Support, .85 for Friends Social Contact and Support, .90 for Secure Romantic Partner Attachment, .87 for General Altruism, and .96 for Religiosity.

*The Eating Disorders Inventory (EDI-2;* Garner, 1991) includes 11 subscales measuring several different aspects of disordered eating behavior, and was expanded to include a twelfth subscale with seven items specific to Anorexia (restriction of eating behavior) taken from the Oral Control subscale of the Eating Attitudes Test (EAT) developed by Garner and Garfinkel (1979), as had been done in a previous study (Faer et al., 2005). The Cronbach’s alphas for these subscales were .92 for Drive for Thinness, .75 for Bulimia, .74 for Body Dissatisfaction, .91 for Ineffectiveness, .79 for Perfectionism, .81 for Interpersonal Distrust, .80 for interoceptive Awareness, .82 for Maturity Fears, .61 for Asceticism, .77 for Impulse Regulation, .75 for Social Insecurity, and .60 for Anorexia.

*The Female Intrasexual Competitiveness Scales* (Faer et al., 2005) distinguished between intrasexual competitiveness for mates and intrasexual competitiveness for status. These two scales each contained several third-person vignettes, which consisted of questions in which participants read a situation and rated the behavior of the fictional character named Mary. Both measures contained statements requiring participants to rate their level of endorsement on 7-point Likert scales of the behavior of the hypothetical protagonist (“Mary”) on how likely, how appropriate, and how understandable her behavior was for each of the given scenarios. In the previous (Faer et al., 2005; Abed et al., 2009) studies, only a single rating was taken on a 6-point Likert scale from “Completely Inappropriate” to “Completely Appropriate”, but the present study expanded upon that procedure to enhance the density of measurement. The Cronbach’s alphas for these scales were .70 for Female Intrasexual Competitiveness for Mates and .69 for Female Intrasexual Competitiveness for Status.

*The Behavioral Regulation Scales of the Behavior Rating Inventory of Executive Function - Adult version (BRIEF-A;* Gioia, Isquith, Retzlaff, and Espy, 2002) were used to measure Executive Functions. This portion of the *BRIEF-A* is a 30-item self-report instrument of adult executive functions or self-regulation in everyday environments that assess Inhibition (e.g., “I tap my fingers or bounce my legs”), Set Shifting (e.g., “I have trouble changing from one activity or task to another”) and Emotional control (e.g., “I overreact emotionally”). The Cronbach’s alpha for this scale was .94.

*Balanced Inventory of Desirable Responding (BIDR-6;* Paulhus, 1991) is a 40-item measure that includes two subscales commonly used to statistically control for socially desirable responding: Self-deceptive Enhancement (e.g., “I am fully in control of my own fate”) and Impression Management (e.g., “I never swear”). The Cronbach’s alphas for the two scales were 0.54 for Self-deceptive Enhancement and 0.75 for Impression

Management.

### Statistical Analyses

All univariate and multivariate analyses were performed using SAS 9.1. Because it was not possible to analyze all of the individual subscales within a single multivariate model simultaneously due to the limitations of our sample size, a hierarchical analytical strategy was employed. Unit-weighted common factor scores (Gorsuch, 1983) were estimated, using SAS PROC STANDARD and DATA, as the means of the standardized scores for all non-missing subscales on each factor (Figueredo, McKnight, McKnight, and Sidani, 2000).

Also computed were both the Cronbach's alphas and the covariance matrices of the subscales using SAS PROC CORR. The loadings (scale-factor correlations) of the unit-weighted factors on the subscales are presented in Tables 1 and 2.

**Table 1.** The Arizona Life History Battery.

Subscales	Slow LH Factor
Mini-K	.80*
Insight, Planning, Control	.51*
Parent Relationship Quality	.57*
Family Contact/Support	.67*
Friends Contact/Support	.48*
Partner Attachment	.25*
General Altruism	.60*
Religiosity	.55*

\* $p < 0.05$

All the unit-weighted factor scales were entered as manifest variables for multivariate causal analysis within a single structural equation model. Structural equation modeling was performed by SAS PROC CALIS. Structural equation modeling between these constructs then provided a multivariate causal analysis of the structural relations between them. Structural equations models were evaluated by use of chi-square, the Bentler-Bonett Normed Fit Index (*NFI*), the Bentler-Bonett Comparative Fit Index (*CFI*), and the Root Mean Squared Error of Approximation (*RMSEA*). Index values of the *NFI* and *CFI* greater than 0.90 are considered satisfactory levels of practical goodness-of-fit (Bentler and Bonett, 1980; Bentler, 1995), whereas *RMSEA* values of 0.05 or less are considered indications of good fit, values between 0.08 and 0.10 are considered indications of a mediocre fit, and values greater than 0.10 are considered indications of a poor fit (Steiger and Lind, 1980; Browne and Cudeck, 1993). The *CFI* was selected because it is adjusted for model parsimony and performs well with moderate to small sample sizes ( $N < 250$ ), especially with Maximum Likelihood estimation (Bentler, 1990; Hu and Bentler, 1995). Alternative fit indices, such as the Bentler-Bonett Non-Normed Fit Index (*NNFI*), provide

poor estimates of model fit with smaller samples (Hu and Bentler, 1995).

Prior to these structural analyses, all the variables were residualized on both Self-Deceptive Enhancement and Impression Management to statistically control for any socially desirable responding.

**Table 2.** The Eating Disorders Inventory.

Subscales	Disordered Eating Behaviors
Drive for Thinness	.72*
Bulimia	.63*
Body Dissatisfaction	.65*
Ineffectiveness	.87*
Perfectionism	.48*
Interpersonal Distrust	.60*
Interoceptive Awareness	.77*
Maturity Fears	.53*
Asceticism	.73*
Impulse Regulation	.64*
Social Insecurity	.74*
Anorexia	.59*

\* $p < 0.05$

#### *Statistical Power*

A sample size of  $N = 100$  is considered a “small” sample for the purposes of structural equations modeling. However, the absolute size of the sample must also be considered in terms of the relative complexity or parsimony of the model. Bentler (1995) has recommended a ratio of at least five cases for every structural parameter freely estimated in confirmatory models. A sample size of  $N = 100$  could therefore in principle support  $k = 20$  parameter estimates according to this ratio. Both alternative models tested with the present data, however, only contained 4 structural path coefficients to be estimated.

Because a structural equation model is ultimately no more than a system of cross-linked multiple regressions, another approach to estimating the statistical power available for a given sample size is to estimate the power available to test the significance of the model parameters that were omitted in the restricted structural models. In the case of the present data, the most complex regression one could specify within a fully saturated model would contain four predictors of disordered eating behaviors: Slow Life History, Executive Functions, Competition for Mates, and Competition for Status. Using G\*Power (Erdfelder, Faul, and Buchner, 1996), we estimated the *post hoc* statistical power to detect a “moderate” increment in the squared multiple correlation ( $f^2 = 0.15$ ) of a prediction equation using 100 cases, 4 predictors, and  $\alpha = 0.05$ . The results were a non-centrality parameter ( $\lambda$ ) of 15.00, a critical F-ratio of 3.94, and a power ( $1 - \beta$ ) of 0.97. We therefore



concluded that we indeed had sufficient statistical power to detect any additional effects of “moderate” magnitude ( $f^2 = 0.15$ ) that had not been specified in either of the alternative restricted structural equation models. Thus, rather than estimate the power available to reject an entire alternative model, which we clearly had because we in fact rejected one of them, we applied and we satisfied the more stringent criterion of whether we would have had sufficient statistical power to detect any of the effects that were omitted in either or both of our restricted structural models.

## **Results**

### *Descriptive Statistics*

Garner, Olmstead, and Polivy (1983) claim that the presence or absence of bulimia differentiates subtypes of anorexia. These two subtypes are termed “restricters” and “binge-eating/purging” by DSM-IV (1994). In the EDI-2 Professional Manual (Garner, 1991), means are reported for each EDI subscale for “anorexic” females versus “comparison” group females (university students without a diagnosed ED). In comparing EDI subscale means from the EDI manual with EDI means from the present sample, the approximate location of the present sample on the eating disorder continuum can be examined. The present sample scored lower than the clinical anorexic group on all subscales, except for bulimia, which was higher in our sample than for the clinical anorexic restricting sample and the college non-anorexic comparison group (*AN diagnosed*:  $M = 1.8$ ,  $SD = 3.5$ , *comparison*:  $M = 1.2$ ,  $SD = 1.9$ , *present sample*:  $M = 2.2$ ,  $SD = 0.7$ ). The present sample had lower mean scores than the clinical sample and higher mean scores than the “comparison non-anorexic” group on Ineffectiveness (*AN diagnosed*:  $M = 11.4$ ,  $SD = 8.4$ , *comparison*:  $M = 2.3$ ,  $SD = 3.6$ , *present sample*:  $M = 2.5$ ,  $SD = 0.8$ ), Interpersonal Distrust (*AN diagnosed*:  $M = 6.9$ ,  $SD = 5.3$ , *comparison*:  $M = 2.0$ ,  $SD = 3.1$ , *present sample*:  $M = 2.6$ ,  $SD = 0.7$ ), and Maturity Fears (*AN diagnosed*:  $M = 4.8$ ,  $SD = 5.1$ , *comparison*:  $M = 2.7$ ,  $SD = 2.9$ , *present sample*:  $M = 3.0$ ,  $SD = 0.8$ ).

A frequency analysis was also conducted to see how many women in the present sample exceeded the cut off for clinical screening purposes suggested by Garner. For the present sample, no women exceeded a score of 14 on Drive for Thinness, or a score of 11 on Bulimia, or a score of 14 on Body Dissatisfaction.

From a combination of these descriptive analyses, we may conclude the following. First, although some of our sample means were higher than the “comparison” group means, overall they were closer to the “comparison” group means than to the means of women who had been specifically diagnosed as “anorexic.” This is reasonable because our sample was composed of university students who had not been screened for either presence or absence of anorexia. On the other hand, our sample did contain a small proportion of women who had scores on subscales that were greater than the means of the women diagnosed with “anorexia”, whether they were restricters or bulimics, in these previous studies.

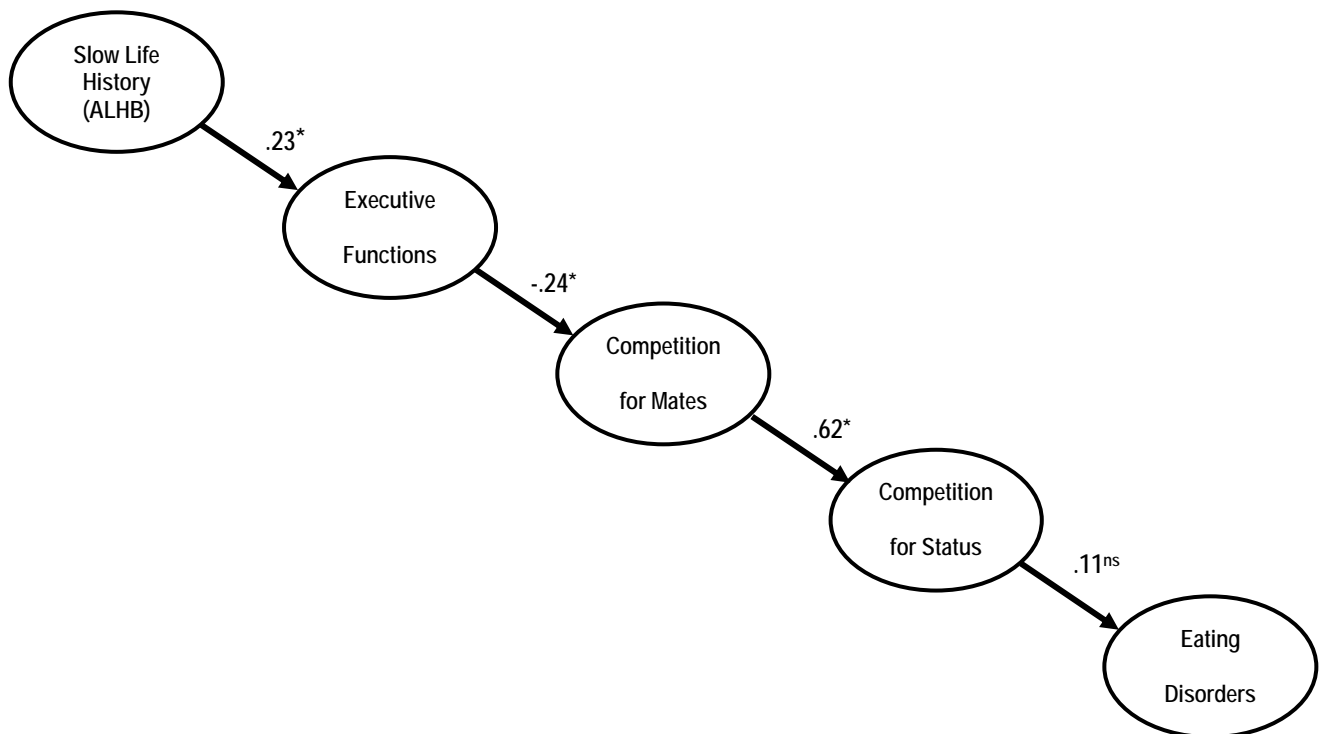
### *Structural Equation Models*

In both alternative structural models, we hypothesized that enhanced Executive Functions, and more specifically Behavioral Regulation (Self-Control), would mediate the effects of Slow Life History Strategy on female intrasexually competitive behavior and on

disordered eating behavior. However, in Alternative Model 1, the enhanced Executive Functions possessed by slower life history strategists were hypothesized to reduce disordered eating behavior *indirectly* by inhibiting the heightened intrasexually competitive behavior for mates and for status. In Alternative Model 2, the enhanced Executive Functions possessed by slower life history strategists were hypothesized to *directly* inhibit disordered eating behaviors as well as to directly inhibit intrasexually competitive behavior for mates and for status.

Alternative Model 1 was rejectable by all statistical and practical indicators of goodness-of-fit ( $X^2(6) = 15.795$ ,  $p = 0.0149$ ,  $CFI = 0.853$ ,  $RMSEA = 0.1291$ ). These results are displayed graphically in Figure 1.

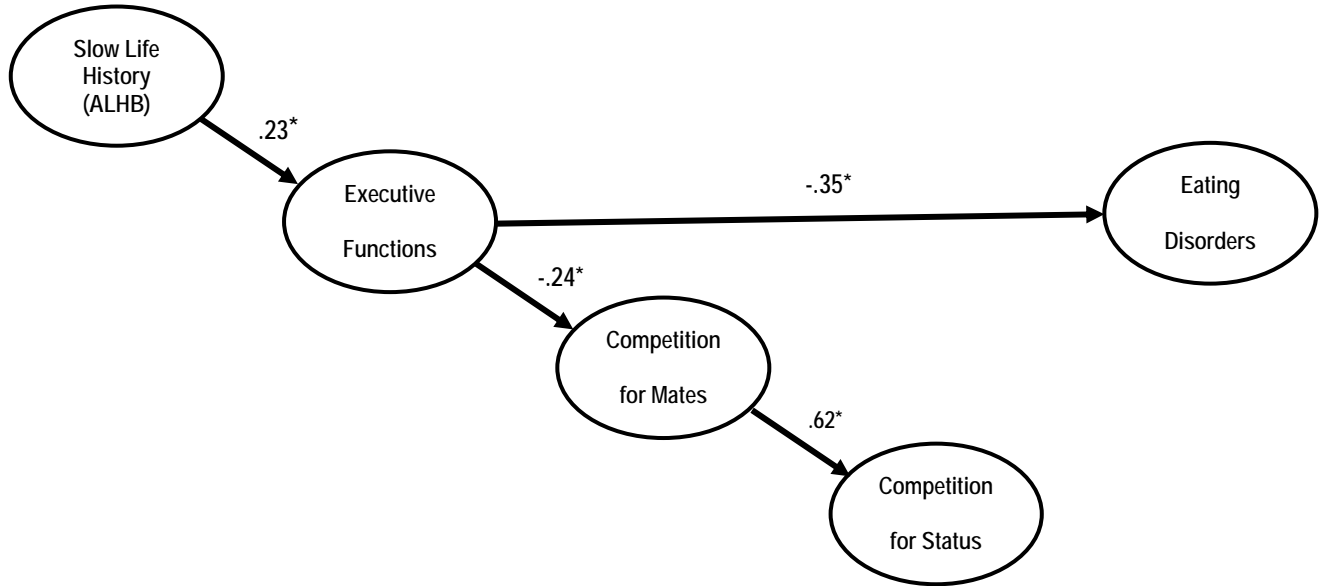
Figure 1. Alternative Structural Equations Model 1 (see text for details).



The standardized path coefficients for Alternative Model 1 were as follows: (1) Slow Life History Strategy predicted higher Executive Functions ( $\beta = 0.23$ ,  $t = 2.38$ ,  $p < 0.05$ ); (2) Higher Executive Functions predicted lower Intrasexual Competitiveness for Mates ( $\beta = -.24$ ,  $t = -2.46$ ,  $p < 0.05$ ); (3) Higher Intrasexual Competitiveness for Mates predicted higher Intrasexual Competitiveness for Status ( $\beta = 0.62$ ,  $t = 7.89$ ,  $p < 0.05$ ); and (4) Higher Intrasexual Competitiveness for Status failed to significantly predict lower Disordered Eating Behaviors ( $\beta = 0.11$ ,  $t = 1.14$ ,  $p > 0.05$ ).

In contrast, Alternative Model 2 was acceptable by all statistical and practical indicators of goodness-of-fit ( $X^2(6) = 4.205$ ,  $p = 0.6490$ ,  $CFI = 1.000$ ,  $RMSEA = 0.0000$ ). These results are displayed graphically in Figure 2.

Figure 2. Alternative Structural Equations Model 2 (see text for details).



The standardized path coefficients for Alternative Model 2 were as follows: (1) Slow Life History Strategy predicted higher Executive Functions ( $\beta = 0.23$ ,  $t = 2.38$ ,  $p < 0.05$ ); (2) Higher Executive Functions predicted lower Intraseual Competitiveness for Mates ( $\beta = -0.24$ ,  $t = -2.46$ ,  $p < 0.05$ ); (3) Higher Intraseual Competitiveness for Mates predicted higher Intraseual Competitiveness for Status ( $\beta = 0.62$ ,  $t = 7.89$ ,  $p < 0.05$ ); and (4) Higher Executive Functions predicted lower Disordered Eating Behaviors ( $\beta = -0.35$ ,  $t = -3.71$ ,  $p < 0.05$ ).

We therefore accepted Alternative Model 2 as superior to Alternative Model 1 by all criteria applied.

## Discussion

As predicted, Executive Functions completely mediated the relation between Slow Life History and Disordered Eating Behaviors. The relation between Intraseual Competitiveness (competitiveness for mates and competitiveness for status) and the Disordered Eating Behaviors, however, was completely spurious. This means that although Executive Functions indeed served to inhibit both Intraseual Competitiveness and Disordered Eating Behaviors, the inhibition of Disordered Eating Behaviors was not even partially mediated by Intraseual Competitiveness, as it had been represented to be in previous studies. The data were most consistent with Executive Functions serving as a common cause underlying the inhibition of both Disordered Eating Behaviors and Intraseual Competitiveness.

These data patterns suggest that the function of a Slow Life History strategy in protecting against Disordered Eating Behaviors resides in the higher degree of Behavioral Regulation exhibited by individuals with a Slow Life History Strategy. Enhanced

Behavioral Regulation, or self-control, is also protective against escalated levels of intrasexual competitiveness. Previous work, looking at both intrasexual competitiveness for mates and intrasexual competitiveness for status, has suggested that some women may be more vulnerable to the pressures of intrasexual competition than others and indicated that this increased vulnerability might pose a health hazard (Salmon et al., 2008; Li et al., 2009; cited with permission), whether or not it is directly causal to disordered eating behavior. For example, subjects who rated low in susceptibility were less affected by the stressful scenarios in this study than subjects who rated high in susceptibility (Salmon et al., 2008). Because some women were more vulnerable to cues of intrasexual competition, a clearer understanding of why they are more susceptible may point the way toward developing more effective prevention and intervention methods for those at risk. Salmon et al. suggested that the factors that might play a role in the development of susceptibility, deserving of further examination, included rearing environment, sexual experience, and social support. It may be that, in addition to these, slow life history strategy, or more specifically enhanced executive functioning, is another major factor influencing such susceptibility to intrasexually competitive pressures.

In the past, there has been a great deal of emphasis in non-evolutionary explanations of eating disorders on the role of rearing environment in the development of disordered eating behavior. In particular, the focus has been on the impact of mother-daughter relationships on the development of eating disorders in young women (Ogden and Steward, 2000). This relationship might facilitate the development of anorexic behavior if the mother's perceptions of female competition or other stressors is exacerbated (through the media or other sources) and then communicated, perhaps through overprotection, to a daughter. In the absence of high executive function, this might result in a greater likelihood of developing eating disordered behavior. More research needs to be done in this area to clarify exactly how this relationship can contribute (in a positive or negative way) to the susceptibility of young women to eating disorders.

A majority of the sociocultural approaches to eating disorders have focused on the internalization of a thin standard of beauty and the role the media plays in fostering this. And while body dissatisfaction is surely increased by heavy exposure to individuals with ideal bodies such approaches fail to explain why eating disorders are not significantly more prevalent clinically than they appear to be. Current prevalence estimates of anorexia and bulimia among young females are 0.3% and 1% respectively with little change over the past twenty years (Hoek, 2006). Our theory, which suggests that executive function (specifically behavioral regulation) mediates the effects of slow life history strategy on disordered eating behavior, accounts for such a pattern. High levels of executive functioning serve to buffer young women against the impact of intrasexual competition while at the same time slow life history tends to suppress the intrasexually competitive behavior itself. The women who will be most at risk of developing an eating disorder will be those who have lower executive function and who experience environments with more abundant cues of intrasexual competition.

Thus, our results are somewhat counterintuitive in the context of the common conception that eating disorders are pathologies of *excessive* self-control rather than the result of *deficient* behavioral regulation. It might be that disordered eating behaviors represent extreme attempts to control tendencies towards overeating when the mechanisms of moderation used by normal individuals are perceived to be failing. This might be

analogous to the plight of alcoholics. Most non-alcoholics are able to drink moderately and avoid the pathological consequences associated with drinking to excess. In fact, many studies have revealed that moderate intake of alcohol may instead have some salubrious effects. Alcoholics, on the other hand, are apparently unable to drink moderately and must instead abstain altogether or risk losing control and drinking to excess.

By analogy, individuals with disordered eating behaviors might be unable to eat in moderation due to insufficient competencies in behavioral regulation, as in the cyclical bingeing of bulimics, who rely on purging afterwards to control their weight. Anorexics instead restrict their food intake to unhealthy and even life-threatening levels, possibly as a paradoxical overreaction to tendencies towards overeating. These adverse health consequences may be seen as unintended side-effects of these alternative weight-control strategies in girls who perceive themselves to be under high degrees of competitive pressure from other girls. Although the reproductive suppression hypothesis considers amenorrhea and infertility as the ultimate function of anorexic behavior, the present results indicate that they might also represent unintended and fitness-reducing consequences of disordered eating behavior. The present results indirectly associate disordered eating behaviors with *faster* life history strategies, instead of the *slower* ones that might be expected to correlate with adaptive reproductive suppression. The present results are therefore inconsistent with the reproductive suppression hypothesis, because sacrificing present reproduction in favor of potential future reproduction is a tactic more aligned with slow life history strategy (Ellis, Figueredo, Brumbach, and Schlomer, 2009).

#### *Limitations of the Study*

We recognize that the major limitation of this study is the reliance on data collected from a non-clinical population with an average age of 18.8. The lack of clinically diagnosed bulimics and anorexics probably accounted for the relatively small effect sizes found in this study. Presumably, the effect sizes would be much larger with more severe cases than with a non-clinical population. In addition, childhood and adult onset eating disorders cannot be examined in this study due to the restricted participant age range. We only had the power to detect effects of moderate magnitude. Our findings, therefore, await replication in future studies using clinical populations of anorexics and bulimics with a more diverse age range.

#### *Conclusion*

Our results indicate that a slow life-history strategy is indicative of greater executive function, which indicates enhanced self control and manifests as increased behavioral regulation, serving as a protective factor against both escalated female intrasexual competitiveness and the development of disordered eating behaviors.

**Received 27 July 2009; Revision submitted 13 October 2009; Accepted 13 November 2009**

#### **References**

Abed, R.T., Mehta, S., Figueredo, A.J., Aldridge, S., Balson, H., Meyer, C., and Palmer, R. (2009). Eating disorders and intrasexual competition: Testing an evolutionary

- hypothesis among young women. Manuscript submitted for publication.
- Anderson, J.L., and Crawford, C.B. (1992). Modeling costs and benefits of adolescent weight control as a mechanism for reproductive suppression. *Human Nature*, 3, 299-334.
- Anderson, J.L., Crawford, C.B., Nadeau, J., and Lindberg, T. (1992). Was the Duchess of Windsor right? A cross-cultural review of the socioecology of ideals of female body shape. *Ethology and Sociobiology*, 13, 197-227.
- Bentler, P.M. (1990). Fit indices, Lagrange Multipliers, constraint changes, and incomplete data in structural models. *Multivariate Behavioral Research*, 25, 163-172.
- Bentler, P.M. (1995). *EQS: Structural equations program manual*. Los Angeles: Multivariate Software.
- Bentler, P.M., and Bonett, D.G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*, 88, 588-606.
- Bogaert, A.F., and Rushton, J.P. (1989). Sexuality, delinquency, and r/K reproductive strategies: data from a Canadian university sample. *Personality and Individual Differences*, 10, 1072-1077.
- Botta, R.A. (2003). For your health? The relationship between magazine reading and adolescents' body image and eating disturbances. *Sex Roles*, 48, 389-399.
- Browne, M.W., and Cudeck, R. (1993). Alternative ways of assessing model fit. *Sociological Methods and Research*, 21, 230-258.
- Bruch, H. (1988). *Conversations with anorexics*. Northvale, N.J.: Aronson.
- Crisp, A.H. (1980). *Anorexia nervosa: let me be*. London: Plenum Press.
- Ellis, B.J., Figueredo, A.J., Brumbach, B.H., and Schlomer, G.L. (2009). Mechanisms of environmental risk: The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. *Human Nature*, 20, 204-268.
- Ellison, P. (2008). Energetics, reproductive ecology, and human evolution. *PaleoAnthropolgy*, 2008, 172-200.
- Erdfelder, E., Faul, F., and Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research Methods, Instruments, and Computers*, 28, 1-11.
- Faer, L.M., Hendriks, A., Abed, R., and Figueredo, A.J. (2005). The evolutionary psychology of eating disorders: Female competition for mates or for status? *Psychology and Psychotherapy: Theory, Research and Practice*, 78, 397-417.
- Figueredo, A.J. (2007). *The Arizona Life History Battery* [Electronic Version]. <http://www.u.arizona.edu/~ajf/allhb.html>
- Figueredo, A.J., McKnight, P.E., McKnight, K.M., and Sidani, S., (2000). Multivariate modeling of missing data within and across assessment waves. *Addiction*, 95 (Supplement 3), pp. S361-S380.
- Figueredo, A.J., Vasquez, G., Brumbach, B.H., Sefcek, J.A., Kirsner, B.R., and Jacobs, W.J. (2005). The K-factor: Individual differences in life history strategy. *Personality and Individual Differences*, 39, 1349-1360.
- Frisch, R.E. (1990). Body fat, menarche, fitness, and fertility. In R.E. Frisch (Ed.) *Adipose tissue and reproduction* (pp. 1-26). Basel: Karger.
- Frisch, R.E. (1996). The right weight: Body fat, menarche, and fertility. *Nutrition*, 12, 452-453.
- Garner, D.M. (1991). *Eating Disorder Inventory-2: Professional Manual*. Psychological Assessment Resources.

- Garner, D.M., and Garfinkel, P.E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine*, 9, 273-279.
- Garner, D.M., and Garfinkel, P.E. (1997). *Handbook of psychotherapy for anorexia and bulimia* (2<sup>nd</sup> ed.). New York: Guilford Press.
- Garner, D.M., Olmstead, M.P., and Polivy, J. (1983). The eating disorders inventory: A measure of cognitive behavioral dimensions of anorexia nervosa and bulimia. In D.L. Darby, P.E. Garfinkel, D.E. Gardner, and D.V. Coscina (Eds.) *Anorexia nervosa: Recent developments in research* (pp. 173-184). New York: Alan R. Liss.
- Geary, D.C. (2005). *The origin of mind: Evolution of brain, cognition, and general intelligence*. Washington, DC: American Psychological Association.
- Gioia, G.G., Isquith, P.K., Retzlaff, P., and Espy, K.A. (2002). Confirmatory Factor Analysis of the BRIEF in a Clinical Sample. *Child Neuropsychology*, 8, 249-257.
- Gladden, P.R., Figueredo, A.J., and Jacobs, W.J. (2008). Life history strategy, psychopathic attitudes, personality, and general intelligence. *Personality and Individual Differences*, 46, 270-275.
- Gorsuch, R. L. (1983). *Factor Analysis*. Hillsdale, N.J.: Erlbaum.
- Gorwood, P., Kipman, A., and Foulon, C. (2003). The human genetics of anorexia nervosa. *European Journal of Pharmacology*, 480, 163-170.
- Groesz, L.M., Levine, M.P., and Murnen, S.K. (2002). The effect of experimental presentation of thin media images on body satisfaction: A meta-analytic review. *International Journal of Eating Disorders*, 31, 1-16.
- Guisinger, S. (2003). Adapted to flee famine: Adding an evolutionary perspective on anorexia nervosa. *Psychological Review*, 110, 745-761.
- Hoek, H.W. (2006). Incidence, prevalence, and mortality of anorexia nervosa and other eating disorders. *Current Opinion in Psychiatry*, 19, 389-394.
- Hu, L.T., and Bentler, P.M. (1995). Evaluating model fit. In R. H. Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 76-99). Thousand Oaks, CA: Sage
- Juda, M.N., Campbell, L., and Crawford, C. (2004). Dieting symptomatology in women and perceptions of social support: An evolutionary approach. *Evolution and Human Behavior*, 25, 200-208.
- Kardum, I., Gracanin, A., and Hudek-Knezevic, J. (2008). Evolutionary explanations of eating disorders. *Psychological Topics*, 17, 247-263.
- Klump, K.L., Miller, K.B., Keel, P.K., McGue, M., and Iacono, W.G. (2001). Genetic and environmental influences on anorexia nervosa syndromes in a population-based twin sample. *Psychological Medicine*, 31, 737-740.
- Lezak, M.D., Howieson, D. B., Loring, D. W. (2004). *Neuropsychological Assessment* (4<sup>th</sup> Edition). New York: Oxford University Press
- Li, N.P., Smith, A.R., Griskevicius, V., Cason, M.J., and Bryan, A. (2009). Intrasexual competition and unhealthy eating in heterosexual and homosexual individuals. Unpublished manuscript.
- Mealey, L. (2000). Anorexia: A “losing” strategy? *Human Nature*, 11, 105-116.
- Ogden, J. and Steward, J. (2000). The role of the mother-daughter relationship in explaining weight concern. *International Journal of Eating Disorders*, 28, 78-83.
- Paulhus, D.L. (1991). Measurement and control of response bias. In J.P. Robinson, P.R. Shaver, and L.S. Wrightsman (Eds.), *Measures of personality and social*

- psychological attitudes* (pp. 17-59). New York: Academic Press.
- Polivy, J. and Herman, C.P. (2002). Causes of eating disorders. *Annual Review of Psychology*, 53, 187-213.
- Rippon, C., Nash, J., Myburgh, K.H., and Noakes, T.D. (1988). Abnormal eating attitude test scores predict menstrual dysfunction in lean females. *International Journal of Eating Disorders*, 7, 617-624.
- Salmon, C., Crawford, C., Dane, L., and Zuberbier, O. (2008). Ancestral mechanisms in modern environments: impact of competition and stressors on body image and dieting behavior. *Human Nature*, 19, 103-117.
- Stearns, S.G. (1992). *The evolution of life histories*. Oxford, UK: Oxford University Press.
- Steiger, J.H., and Lind, J.C. (1980). Statistically-based tests for the number of common factors. Paper presented at the annual Spring Meeting of the Psychometric Society in Iowa City. May 30, 1980.
- Striegel-Moore, R.H. and Bulik, C.M. (2007). Risk factors for eating disorders. *American Psychologist*, 62, 181-198.
- Surbey, M. K. (1987). Anorexia nervosa, amenorrhea and adaptation. *Ethology and Sociobiology*, 8, 47-61.
- Wasser, S.K. (1990). Infertility, abortion, and biotechnology: When it's not nice to fool Mother Nature. *Human Nature*, 1, 3-24.
- Wasser, S.K. and Barash, D.P. (1983). Reproductive suppression among female mammals: Implications for biomedicine and sexual selection theory. *Quarterly Review of Biology*, 58, 513-538.