



## 'Slow' reproductive strategy: A negative predictor of depressive symptomatology

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### Abstract

The present study examined the associations between a high-K (slow) life history strategy and depressive symptomatology. The participants were a sample of 494 male utility workers who underwent psychological evaluations. It was hypothesised that high-K will correlate negatively with, and will be a negative predictor of, depressive symptomatology. The results confirmed the predictions, showing that high-K accounts for 15% of the variance in depressive symptomatology after controlling for risk factors for depression such as demographics, prior traumatic experiences, past depression, and recent negative life events. Implications of the results are discussed.

**Key words:** depression, evolutionary psychopathology, fitness, high-K strategy, Life History Theory

The high and persistent incidence of mental disorder in the general population has triggered attempts to explain it from an evolutionary perspective. Such attempts centred on the very taxonomy or definition of mental disorder (Wakefield, 1997; Wakefield & First, 2003), or on the possible functions of distinct mental conditions (Crow, 1991, 1995, 1997; Fiske & Haslam, 1997; Mealey, 2000).

Generally, the approach in explaining mental illness from an evolutionary perspective is to identify the possible utility of the symptoms. For instance, modern excessive fears have been explained as adaptive mechanisms designed to yield false positives gone awry. Indeed, optimal systems protecting the organism from harm should be designed to yield false positives, since one single false negative (e.g., to mistake a poisonous snake for a non-dangerous one) could have resulted in significant fitness costs in the environment of evolutionary adaptedness.

Depression, because of its high (5–10%) prevalence in the USA (Kessler et al., 2003), cross-cultural universality (Nesse & Williams, 2004), upward course in symptom severity (National Institute of Mental Health, 1994), and puzzling potential consequences to fitness, including suicide, (American Psychiatric Association, 2000; Fisher &

Thompson, 2007) has been tackled in the evolutionary psychology literature. While no single specific cause for depression has been unequivocally identified, factors such as demographics (Lewinsohn, Hoberman, & Rosenbaum, 1988), prior major depression (Lewinsohn et al., 1988), early traumatic experiences such as physical or sexual abuse (Heim, Owens, Plotsky, & Nemeroff, 1997; Maercker, Michael, Fehm, Becker, & Margraf, 2004), or negative life experiences, such as job loss or loss of a loved one (Shrout et al., 1989), are believed to be involved.

Evolutionary psychologists attempt to explain depression through the functions it may serve. For instance, Leith and Baumeister (1996) showed that low mood is associated with less likelihood of engaging in risk-taking behaviours, and other authors argued that low levels of depression seem to be 'normal' or functional states (Hagen, 2003; McGuire, Troisi, & Raleigh, 1997; Nesse, 1998).

Some authors see depression as a mechanism that allocates effort proportional to propitiousness (Nesse, 2000). Low mood, in this view, blocks unfavourable investments which motivates changes in behavioural strategies and goal pursuit (Watson & Andrews, 2002). Others see depression as a result of unsuccessful attempts to reach a critical goal, and clinicians often note that depression sometimes subsides when the individual gives up the pursuit of an unattainable goal (Nesse, 2000; Price, Sloman, Gardner, Gilbert, & Rohde, 1994). Yet, other authors see depression as an 'involuntary yielding' adaptation that serves the purpose of inhibiting aggressive behaviours towards others of superior rank (Evans, 1999; Nesse, 2000). Thus, the adaptive role of

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depression may be a switching function: When one's activities result in low status (and decreased fitness as a consequence), the loss of interest in that activity may lead to trying out other alternatives that might increase fitness.

There are multiple ways by which an organism achieves fitness (i.e., reproductive success). According to the Life History theory (Bogaert & Rushton, 1989; Figueredo et al., 2006; MacArthur & Wilson, 1967; Pianka, 1970), an organism's available resources are finite, which translates into trade-offs in their allocation for solving various fitness-relevant tasks. There are two extremes of these reproductive strategies: 'r', representing maximum egg output with no investment, and 'K', representing the opposite. Humans appear to be highly K selected; therefore in the human species, these strategies are termed 'differential K'. At the extremes of the differential K, there are two cohesive fitness strategies: 'high-K', where the investment is predominantly in somatic and parental effort, and 'low-K', where the investment is predominantly in mating efforts. Traits associated with a high-K fitness strategy are parental investment, deep social networks, engagement in long-term relationships, significant investment in children, careful planning, cooperation, and altruism (Figueredo et al., 2006; Figueredo, Vasquez, Brumbach, & Schneider, 2005; Giosan, 2006). Figueredo et al. (2005) showed that slow life history strategy is associated with Big Five personality traits that are commonly associated with social success, and Wenner, Figueredo, and Jacobs (2005) have shown that high-K is negatively correlated with a cluster of socially undesirable behaviours and positively correlated with impulse control.

Directly related to the object of the present article are the studies of Kirsner, Figueredo, and Jacobs (2003), who showed that depressive symptoms distort mating behaviour by lowering the perceived mate value of the individual and thus distorting the normal mechanisms of assortative mating. Fast life history strategies (i.e., low-K) have been explicitly linked to these effects (Kirsner, Figueredo, & Jacobs, 2009). Elsewhere, it has been shown that low perceived mate quality is associated with faster life history strategies (Gladden, Figueredo, & Snyder, 2010). A preliminary study found negative associations between depressive symptomatology and high-K (Giosan, 2007), while other authors linked high-K to a co-vitality factor that included a measure of negative affect (Figueredo et al., 2005, 2006).

The present article extends the above-mentioned studies by testing whether the contribution of high-K to current depressive symptomatology goes beyond that of the factors that are commonly identified in literature to be predictors of depression. The study also adds to the prior research on the topic by examining associations between high-K and a clinical diagnosis of past depression.

Specifically, three hypotheses were tested: (1) High-K strategy is negatively associated with clinical diagnosis of

past depression (Hypothesis 1); (2) High-K strategy is negatively associated with current depressive symptomatology (Hypothesis 2); and (3) This relationship will hold even after controlling for risk factors for depression (Hypothesis 3).

Two Life History measures have been developed in the literature: the Mini-K (Figueredo et al., 2006) and the High-K Strategy Scale (HKSS) (Giosan, 2006). Recent research has found that HKSS and Mini-K are significantly correlated and also have the strongest correlation with a general personality factor, representing better measures of Life History Strategy than proxy variables (Dunkel & Decker, 2010). The items on the Mini-K are generally oriented towards personality traits/behaviours (e.g., 'I often make plans in advance'), while the items on the HKSS are oriented towards the successful resolution (or lack thereof) of indicators that make up a high-K strategy (e.g., 'I am able to provide a decent quality of life for myself and my family'). Elsewhere, it was shown that both measures load on a K-factor (.66 for HKSS and .59 for the mini-K) (Gladden, Welch, Figueredo, & Jacobs, 2009). The HKSS has been used in the present study to test the associations with depression, because prior research has shown that this construct is broadly associated with psychopathology (Giosan & Wyka, 2009).

## METHODS

### Participants and procedure

Participants in this study were a sample of 494 males from a population of utility workers deployed at the World Trade Center site in New York City during or after the September 11th 2001 attack. The participants underwent a medical and psychological evaluation conducted under the auspices of the company's Occupational Health Department. The psychological evaluations, which were performed by doctoral-level clinical psychologists, were piggybacked onto annual fitness-for-duty evaluations for all utility workers who were deployed to work at the World Trade Center in the aftermath of 9/11. This sample comes from the same population that Giosan and Wyka (2009) used to test associations between HKSS and general psychopathology.

### Measures

**Life History Strategy.** Life History Strategy was measured with *High-K Strategy Scale* (HKSS) (Giosan, 2006). HKSS is a 26-item scale tapping into domains that are theorised to make up the high-K independent criterion of fitness, such as resource access, upward mobility, health, and attractiveness, mate value, social capital, risks consideration. The scale enjoys high-internal consistency (Cronbach's alpha = .92)

(Giosan, 2006). Items are coded on a Likert scale and are summed up to yield a total HKSS score. The total possible HKSS score is 130.

**Depressive symptomatology.** The Beck Depression Inventory—Second Edition (BDI-II) (Beck, Steer, Ball, & Ranieri, 1996; Beck, Steer, & Brown, 1996), a 21-item version of the original BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), was used to assess depressive symptomatology. BDI-II is one of the most widely used self-report measures of depression. BDI-II items consist of four statements each, scored 0–3, reflecting increasing severity of specific depression symptoms. Items are summed to yield a total depression score, as well as scores for cognitive and somatic symptoms. The psychometric properties of the original BDI are well-established, and the BDI-II also appears to be psychometrically strong (Beck, Steer, Ball et al., 1996).

**Clinical diagnosis of depression.** To determine the past depression diagnostic status of the participants, the Structured Clinical Interview for the DSM-IV (SCID) was used (First, Spitzer, Williams, & Gibbon, 1997). The SCID is a semi-structured clinical interview designed to determine DSM-IV diagnoses. SCID psychometric properties have been well-established. The SCID with the past depression module for the DSM-IV was used for the purposes of the present study.

**Trauma History.** Trauma history was assessed with Trauma History Questionnaire (THQ) (Green, 1996), a 13-item measure developed to document lifetime trauma history. The THQ inquires if the participant has ever witnessed or experienced traumatic events such as natural disasters, accident/injury, sudden life-threatening illness, military combat, death of a friend/family member in an accident or by murder, sudden unexpected death of close family member, assault, childhood abuse, and coerced sexual contact. For the purposes of the present study, a dichotomous variable (history of trauma 'yes/no') was used in the statistical analyses.

**The Life Experiences Survey (LES).** The Life Experiences Survey (Sarason, Johnson, & Siegel, 1978) is an instrument used to assess positive and negative life experiences, as well as individualised ratings of the impact of events. It consists of 57 items scored on a Likert scale, which can take values from –3 to 3 (negative for events whose valence was perceived negatively and positive for events whose valence was perceived positively). The instrument has two sections, one for the general public and one for the students. Because of the demographics of the sample, the module for the general public was used in the present study, and the participants were asked whether the event happened in the past five years and in the past three months. The LES yields a total

score, calculated by summing up the absolute values of the items and two dimensions: a negative events score, yielded by the addition of all the negative values, and a positive events score, obtained through the addition of all the positive values. Sarason et al. (1978) showed that the negative life events score of the LES was associated with depressive symptomatology as measured by the BDI. This module (the negative life events score) was used in the analyses.

## RESULTS

### Demographic and clinical characteristics of the sample

Table 1 depicts the demographic characteristics of the sample ( $N = 494$ ). The participants were males, predominantly white and married or cohabitating. Almost all were high school graduates, and many completed at least some college. In the statistical analyses, race, education, and marital status were dichotomised as follows: race (whites vs other races), education (high school or less vs college or more), and marital status (married or cohabitating vs separated, divorced, or single).

The SCID yielded a prevalence of 12.6% for past major depression, and 50.8% of the sample reported having experienced prior trauma as assessed by the traumatic events interview. About 50% of the sample (51.4%) reported having experienced at least one negative life event. The mean BDI total score of this sample was 3.43 ( $SD = 5.74$ ), and the mean HKSS score was 96.84 ( $SD = 16.74$ ). In accord with published prevalence of depression, which puts it at 5–10% in the general population (Kessler et al., 2003), 7.3% in our sample reported at least mild depressive symptomatology, indicated by a BDI-II score greater than 13. A small

**Table 1** Socio-demographic characteristics of the sample ( $N = 494$ )

	Variable of interest	Per cent (%)
Age, $M$ ( $SD$ )	45.68 (8.80)	
Race		
	Caucasian	68.9
	African American	15.7
	Hispanic	13.5
	Asian	.4
	Other	1.4
Education		
	Some or no high-school	2.0
	High school graduate	47.7
	Some college or training	34.9
	College graduate	11.2
	More than college	4.3
Marital status		
	Married	73.6
	Separated or divorced	11.0
	Single	13.6
	Cohabitating	1.8

Note.  $M$  = mean;  $SD$  = standard deviation.

**Table 2** Bivariate correlation matrix between high-K, demographics, prior trauma, past depression, negative life events, and depressive symptomatology

	High-K	Race	Education	Age	Marital status	Prior trauma	Past depression	Negative life events	Depressive symptoms
High-K	1	-.17***	.08	-.03	-.28***	-.03	-.10*	.13**	-.45***
Race		1	.04	.05	-.12**	.04	.06	.10*	-.00
Education			1	.01	-.01	.03	.05	.02	-.07
Age				1	-.22***	.09	-.02	.02	-.03
Marital status					1	-.00	.14**	-.04	.09*
Trauma						1	.19***	.03	.03
Past depression							1	-.00	.21***
Negative life events								1	-.10*
Depressive symptoms									1

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

Note. High-K was measured with HKSS (Giosan, 2006); Trauma was measured with the THQ (Green, 1996); Negative life events were captured by the LES (Sarason et al., 1978); Past depression was assessed with the SCID (First et al., 1997); Depressive symptomatology was assessed with the BDI-II (Beck, Steer, Ball, et al., 1996).

number of people ( $N = 12$ ) reported moderate or severe depressive symptomatology, indicated by BDI-II scores greater than 19 or 28, respectively.

### Associations between HKSS and depressive symptomatology

*T*-tests between HKSS and past depression were first performed. People who were diagnosed with past depression had significantly lower HKSS scores compared to those who were not diagnosed with past depression ( $t = 2.72$ ,  $p = .01$ ), confirming Hypothesis 1.

Furthermore, HKSS correlated significantly with the BDI-II ( $r = -.45$ ,  $p = .000$ ), confirming Hypothesis 2.

In accord with prior findings (Lewinsohn et al., 1988; Sarason et al., 1978; Shrout et al., 1989), past depression ( $t = -4.47$ ,  $p = .001$ ), demographics (marital status only;  $t = -2.01$ ,  $p = .05$ ), and negative life events ( $r = -.01$ ,  $p = .05$ ) were also significantly associated with the BDI-II. The full correlation matrix is presented in Table 2.

The following strategy was employed to deal with the skewness of the outcome variable BDI-II (skewness = 2.60,  $SE = .11$ ; Kurtosis = 8.43,  $SE = .22$ ). First, the data were transformed by taking the square root of the outcome variable BDI-II (sqrtBDI-II), which yielded a skewness of .99 ( $SE = .11$ ) and Kurtosis of .25 ( $SE = .22$ ). A linear regression on sqrtBDI-II on the predictor variables was then run. The results showed that HKSS accounted for significant variance in the sqrtBDI-II (21%) after controlling for demographics, prior trauma, past depression, and negative life events (Table 3).

Second, all subjects with a total BDI-II score of 0 ( $N = 221$ ) or mild or moderate BDI-II scores ( $\geq 20$ , approximately three standard deviations above the mean,  $N = 12$ ) were removed from the analyses. This reduced the sample size from  $N = 494$  to  $N = 261$  while maintaining the same sample composition (93% of the participants reported BDI-II scores

**Table 3** Hierarchical regression of the square root of BDI-II on high-K

		Cumulative			
		$R^2$	$F$	Beta	$t$
Step 1	Demographics	.02	1.86		
	Age			.01	.25
	Race			.04	.73
	Marital status			.14	2.43*
	Education			-.03	-.54
Step 2	Prior trauma	.03	1.81	.07	1.27
Step 3	Past depression	.05	2.87**	.16	2.83**
Step 4	Negative life events	.06	2.72**	-.07	-1.33
Step 6	High-K	.27	14.96***	-.50	-9.75***

Note. High-K was measured with HKSS (Giosan, 2006); Prior trauma was measured with the THQ (Green, 1996); Past depression was assessed with the SCID (First et al., 1997); Negative life events were captured by the LES (Sarason et al., 1978); Depressive symptomatology was assessed with the BDI-II (Beck, Steer, Ball, et al., 1996).

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

lower than 14 in the full sample, and 91% reported the same in the reduced sample). The mean BDI-II in this subsample was 5.28 ( $SD = 4.80$ ).

Regression analyses of the BDI-II on the HKSS were run on this subsample (Table 4). The results showed that HKSS was a significant predictor of the BDI-II scores, accounting for 15% of the variance, after controlling for risk factors for depression (demographics, prior trauma, past depression, and recent negative life events). Residual analyses performed on this reduced sample showed that none of the regression analyses assumptions were violated.

## DISCUSSION

Several aspects emerge from the above findings. First, we have shown that in this sample, people who report higher scores on the HKSS also report significantly lower scores on

**Table 4** Hierarchical regression of depressive symptomatology (BDI-II) on high-K

		Cumulative		Beta	<i>t</i>
		<i>R</i> <sup>2</sup>	<i>F</i>		
Step 1	Demographics	.02	.69		
	Age			.01	.17
	Race			-.05	-.73
	Education			.11	1.42
	Marital status			.05	.67
Step 2	Prior trauma	.02	.63	-.05	-.63
Step 3	Past depression	.03	.96	.12	1.61
Step 4	Negative life events	.03	.82	.00	.01
Step 6	High-K	.18	5.01***	-.41	-5.77***

Note. High-K was measured with HKSS (Giosan, 2006); Prior trauma was measured with the THQ (Green, 1996); Past depression was assessed with the SCID (First et al., 1997); Negative life events were captured by the LES (Sarason et al., 1978); Depressive symptomatology was assessed with the BDI-II (Beck, Steer, Ball, et al., 1996).

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

the BDI-II. Thus, it appears that successful resolution of problems associated with a high-K reproductive strategy (e.g., social capital, upward mobility, resourcefulness, etc.) is associated with lower current depressive symptomatology. We have also shown that HKSS is associated with lower incidence of retrospectively diagnosed past depression assessed with clinician-administered measures.

Moreover, we have shown that significant variance in the BDI-II is accounted for by the HKSS. Indeed, the combined controlled factors (demographics, prior trauma, past depression, and significant negative life events) accounted for 3% of the variance in the BDI-II, while HKSS accounted for an additional 15%. Past depression was significantly associated with current depressive symptomatology in the correlational analyses ( $r = .21$ ,  $p < .001$ ) reconfirming prior findings, such as Lewinsohn et al.'s (1988), who found the same magnitude of the correlations ( $r = .23$ ,  $p < .001$ ) between self-rated symptoms of depression and a categorical measure of depression. When past depression was entered in the regression model, it no longer retained significance. This is likely due to the fact that HKSS, as predicted, is associated with both past depression and BDI-II scores. Also worth noting is that, with the exception of marital status, the demographic variables were not significantly correlated with the BDI-II, further adding to the inconsistency of the results in this respect, as detailed in recent meta-analyses (Hölzel, Härter, Reese, & Kriston, 2011).

The results from the present study, showing links between depressive symptomatology and Life History, further cement similar associations described in prior research (Figueredo et al., 2006; Kirsner et al., 2003, 2009), this time on a sample with unique characteristics, evaluated both through self-reports and clinician-administered instruments.

Despite these findings, however, several caveats are in order. First and foremost, as in any correlational study, the direction of causality cannot be established with certainty. Indeed, one could argue that people score highly on HKSS precisely because they enjoy better mental health in the first place, therefore they can channel their resources towards this fitness strategy. This is a legitimate claim and testing the direction of causality is not a small feat. Further longitudinal studies are required to test whether changes in high-K strategy are associated with changes in depressive symptomatology, and whether active interventions on the indicators of high-K will reflect in reductions in depressive symptomatology. Second, the generalisability of the results to other categories of people may be in question, as the study was conducted on a unique population, which, while not clinical, was characterised by exposure to stressors and traumatic events. Also, the generalisability of the results to populations with severe depression may be called into question, since the present sample was characterised by mild to moderate BDI-II scores. Moreover, the data collection constraints led to a male-only sample, therefore the generalisability of these findings to women is also in question. However, the significant associations found in this study suggest that high-K may serve as a buffer of depressive symptomatology at least in male populations exposed to traumas. Further studies are needed to test these associations on samples that are more comparable to the general population. Finally, the outcome variable in the present study was a self-report (BDI-II). Future research should be conducted with clinician-administered diagnostic measures for current depression to test if these associations hold. Related to this, collecting data about other risk factors for depression and controlling for them would be useful in increasing the explanatory power of this model. The design constraints of this study did not allow us to collect information about the duration of the past depressive episode, the number of past depressive episodes, family history of mood disorders, or negative social interactions, all of which are thought to be risk factors for chronic depression (Hölzel et al., 2011).

Despite these limitations, however, the present study documents a clear relationship between high-K and depressive symptomatology. This relation can have far-reaching clinical implications. Indeed, since many of the indicators presumed to make up the high-K independent criterion of fitness as operationalised by HKSS are partially under the individual's control (e.g., physical appearance, social capital, safety), a legitimate important practical and clinical question arises: If one succeeds in improving on some of these indicators, will this translate into decreased depressive symptomatology? A positive answer to this empirical question would likely aid therapists in their quests for finding more effective interventions for depression.

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## REFERENCES

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders*. (4th, text rev.). Washington, DC: American Psychiatric Association.
- Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. (1996). Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of personality assessment*, 67(3), 588–597. doi:10.1207/s15327752jpa6703\_13
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory*. San Antonio, TX: The Psychological Corporation.
- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.
- Bogaert, A. F., & Rushton, P. J. (1989). Sexuality, delinquency and r/K reproductive strategies: Data from a Canadian university sample. *Personality and Individual Differences*, 10(10), 1071–1077. doi:10.1016/0191-8869(89)90259-6
- Crow, T. J. (1991). The origins of psychosis and 'The Descent of Man'. *The British Journal of Psychiatry. Supplement*, 14, 76–82.
- Crow, T. J. (1995). A Darwinian approach to the origins of psychosis. *The British Journal of Psychiatry: The Journal of Mental Science*, 167(1), 12–25.
- Crow, T. J. (1997). Is schizophrenia the price that Homo sapiens pays for language? *Schizophrenia Research*, 28(2-3), 127–141.
- Dunkel, C. S., & Decker, M. (2010). Convergent validity of measures of life-history strategy. *Personality and Individual Differences*, 48(5), 681–684. doi:10.1016/j.paid.2009.12.014
- Evans, D. (1999). The social competition hypothesis of depression. *ASCAP Newsletter*, 12(3), 12–15.
- Figueredo, A. J., Vasquez, G., Brumbach, B., & Schneider, S. M. R. (2005). The K-factor, covitality, and personality: A psychometric test of Life History Theory. *Human Nature*, 18, 47–73.
- Figueredo, A. J., Vasquez, G., Brumbach, B. H., Schneider, S. M. R., Sefcek, J. A., Tal, I. R., . . . Jacobs, W. J. (2006). Consilience and life history theory: From genes to brain to reproductive strategy. *Developmental Review*, 26(2), 243–275.
- First, M. B., Spitzer, R. L., Williams, J. B. W., & Gibbon, M. (1997). *Structured clinical interview for DSM-IV SCID*. Washington, D.C.: American Psychological Association.
- Fisher, H. E., & Thompson, J. A. (2007). Lust, romance, attachment: Do the side effects of serotonin-enhancing antidepressants jeopardize romantic love, marriage and fertility? In S. M. Platek, J. P. Keenan & T. K. Shakeford (Eds.), *Evolutionary cognitive neuroscience* (pp. 248–283). Cambridge, MA: MIT Press.
- Fiske, A. P., & Haslam, N. (1997). Is obsessive-compulsive disorder a pathology of the human disposition to perform socially meaningful rituals? Evidence of similar content. *The Journal of Nervous and Mental Disease*, 185(4), 211–222.
- Giosan, C. (2006). High-K Strategy Scale: A measure of the High-K independent criterion of fitness. *Evolutionary Psychology*, 4, 394–405.
- Giosan, C. (2007). *High-K reproductive strategy as a negative predictor of depressive symptomatology*. Presented at the Northeast Evolutionary Psychology Conference, SUNY at New Paltz.
- Giosan, C., & Wyka, K. E. (2009). Is a successful High-K fitness strategy associated with better mental health? *Evolutionary Psychology*, 7(1), 28–39.
- Gladden, P. R., Figueredo, A. J., & Snyder, B. (2010). Life history strategy and evaluative self-assessment. *Personality and Individual Differences*, 48(6), 731–735.
- Gladden, P. R., Welch, J., Figueredo, A. J., & Jacobs, W. J. (2009). Moral intuitions and religiosity as spuriously correlated life history traits. *Journal of Evolutionary Psychology*, 7, 167–184.
- Green, B. L. (1996). Trauma History Questionnaire (Self-report). In B. H. Stamm (Ed.), *Measurement of stress, trauma, and adaptation* (pp. 366–368). Lutherville, MD: Sidran.
- Hagen, E. H. (2003). The bargaining model of depression. In P. Hammerstein (Ed.), *Genetic and cultural evolution of cooperation* (pp. 95–123). Cambridge: MIT Press.
- Heim, C., Owens, M. J., Plotsky, P. M., & Nemeroff, C. B. (1997). The role of early adverse life events in the etiology of depression and posttraumatic stress disorder. Focus on corticotropin-releasing factor. *Annals of the New York Academy of Sciences*, 821, 194–207.
- Hölzel, L., Härter, M., Reese, C., & Kriston, L. (2011). Risk factors for chronic depression—A systematic review. *Journal of Affective Disorders*, 129, 1–13.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., . . . Wang, P. S. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *JAMA: The Journal of the American Medical Association*, 289(23), 3095–3105. doi:10.1001/jama.289.23.3095
- Kirsner, B. R., Figueredo, A. J., & Jacobs, W. J. (2003). Self, friends, and lovers: Structural relations among Beck Depression Inventory scores and perceived mate values. *Journal of Affective Disorders*, 75(2), 131–148.
- Kirsner, B. R., Figueredo, A. J., & Jacobs, W. J. (2009). Structural relations among negative affect, mate value, and mating effort. *Evolutionary Psychology*, 7(3), 374–397.
- Leith, K. P., & Baumeister, R. F. (1996). Why do bad moods increase self-defeating behavior? Emotion, risk taking, and self-regulation. *Journal of Personality and Social Psychology*, 71(6), 1250–1267.
- Lewinsohn, P. M., Hoberman, H. M., & Rosenbaum, M. (1988). A prospective study of risk factors for unipolar depression. *Journal of Abnormal Psychology*, 97(3), 251–264.
- Mac Arthur, R. H., & Wilson, E. O. (1967). *The theory of island biogeography*. Princeton, NJ: Princeton University Press.
- Maercker, A., Michael, T., Fehm, L., Becker, E. S., & Margraf, J. (2004). Age of traumatisation as a predictor of post-traumatic stress disorder or major depression in young women. *The British Journal of Psychiatry: The Journal of Mental Science*, 184, 482–487.
- McGuire, M. T., Troisi, A., & Raleigh, M. M. (1997). Depression in evolutionary context. In S. Baron-Cohen (Ed.), *The maladapted mind: Classic readings in evolutionary psychopathology* (pp. 255–282). London: Erlbaum/Taylor & Francis.
- Mealey, L. (2000). Anorexia: A 'losing' strategy? *Human Nature*, 11, 105–116.
- National Institute of Mental Health (1994). Depression fact sheet (No. 00-4501). National Institutes of Health.
- Nesse, R. M. (1998). Emotional disorders in evolutionary perspective. *British Journal of Medical Psychology*, 71, 397–415.
- Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry*, 57(1), 14–20.

- Nesse, R. M., & Williams, C. G. (2004). *Why do we get sick? The new science of Darwinian medicine*. New York: Times Books.
- Pianka, E. R. (1970). On r- and K-selection. *American Naturalist*, 104, 592–596.
- Price, J., Sloman, L., Gardner, R., Jr, Gilbert, P., & Rohde, P. (1994). The social competition hypothesis of depression. *The British Journal of Psychiatry: The Journal of Mental Science*, 164(3), 309–315.
- Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. *Journal of Consulting and Clinical Psychology*, 46(5), 932–946.
- Shrout, P. E., Link, B. G., Dohrenwend, B. P., Skodol, A. E., Stueve, A., & Mirotznik, J. (1989). Characterizing life events as risk factors for depression: the role of fateful loss events. *Journal of Abnormal Psychology*, 98(4), 460–467.
- Wakefield, J. C. (1997). Diagnosing DSM-IV—Part I: DSM-IV and the concept of disorder. *Behaviour Research and Therapy*, 35(7), 633–649.
- Wakefield, J. C., & First, M. (2003). Clarifying the distinction between disorder and non-disorder: Confronting the overdiagnosis ('false positives') problem in DSM-IV. In K. A. Phillips, M. B. First, & H. A. Pincus (Eds.), *Advancing DSM: Dilemmas in psychiatric diagnosis* (pp. 23–56). Washington, DC: American Psychiatric Press.
- Watson, P. J., & Andrews, P. W. (2002). Toward a revised evolutionary adaptationist analysis of depression: the social navigation hypothesis. *Journal of Affective Disorders*, 72(1), 0165–0327.
- Wenner, C. J., Figueredo, A. J., & Jacobs, W. J. (2005). Validation of the 'Mini-K' using socially problematic behaviors. *Annual meeting of the Human Behavior and Evolution Society*, Austin, TX.