Research Report

You've Gotta Know When to Fold 'Em

Goal Disengagement and Systemic Inflammation in Adolescence

Gregory E. Miller[1] and Carsten Wrosch[2]

[1]University of British Columbia, Vancouver, British Columbia, Canada, and [2]Concordia University, Montreal, Quebec, Canada

ABSTRACT—*The notion that persistence is essential for success and happiness is deeply embedded in popular and scientific writings. However, when people are faced with situations in which they cannot realize a key life goal, the most adaptive response for mental and physical health may be to disengage from that goal. This project followed 90 adolescents over the course of 1 year. Capacities for managing unattainable goals were assessed at baseline, and concentrations of the inflammatory molecule C-reactive protein (CRP) were quantified at that time, as well as 6 and 12 months later. To the extent that subjects had difficulties disengaging from unattainable goals, they displayed increasing concentrations of CRP over the follow-up. This association was independent of potential confounds, including adiposity, smoking, and depression. Because excessive inflammation contributes to a variety of adverse medical outcomes, these findings suggest that in some contexts, persistence may actually undermine well-being and good health.*

The notion that persistence is essential for success is deeply embedded within American culture. America's founding mythology suggests that by doggedly pursuing their goals, people can overcome significant obstacles and go on to lead lives of privilege and happiness. This sentiment was captured by Calvin Coolidge, the 30th president of the United States:

> Nothing in this world can take the place of persistence. Talent will not; nothing is more common than unsuccessful people with talent. Genius will not; unrewarded genius is almost a proverb. Education will not; the world is full of educated derelicts. Persistence and determination alone are omnipotent. The slogan 'press on' has solved and always will solve the problems of the human race. (cited in Knowles, 1999, p. 537)

Persistence is accorded similar virtues in theories of motivation and adaptation (Bandura, 1997; Taylor & Brown, 1988). These models posit that "pressing on" is adaptive; it maximizes the probability that life goals will be realized and thereby fosters well-being and good health. By contrast, giving up has often been equated with helplessness and depression (Seligman, 1975). There is mounting evidence to support these views. To the extent that people persist at life goals, they report higher subjective well-being, and fare better under stress (Bandura, 1997; Carver & Scheier, 1998; Seligman, 1975).

Despite the popular and scientific enthusiasm for persistence, there are contexts in which it is likely to be maladaptive (Heckhausen & Schulz, 1995; Klinger, 1975; Nesse, 2000; Wrosch, Scheier, Carver, & Schulz, 2003). Specifically, when people find themselves in situations in which they are unlikely to realize a goal, the most adaptive response may be to disengage from it. By withdrawing from a goal that is unattainable, a person can avoid repeated failure experiences and their consequences for mind and body. Accordingly, we have shown that people who can disengage from unattainable goals enjoy better well-being, have more normative patterns of cortisol secretion, and experience fewer symptoms of everyday illness than do people who have difficulty disengaging from unattainable goals (Wrosch, Miller, Scheier, & Brun De Pontet, 2007; Wrosch, Scheier, Miller, Schulz, & Carver, 2003). Withdrawal also may enable people to recoup personal resources that can be used to reengage in new goals. The capacity to adaptively reengage has also been related to markers of well-being and health. It seems to be

Address correspondence to Gregory Miller, Department of Psychology, University of British Columbia, 2136 West Mall, Vancouver, BC, Canada V6T 1Z4, e-mail: gemiller@psych.ubc.ca.

especially beneficial in people who cannot easily withdraw from goals that have become unrealizable (Wrosch et al., 2007; Wrosch, Scheier, Miller, et al., 2003).

This pattern of findings suggests that when people encounter serious obstacles to achieving life goals, their self-regulatory strategies can have important downstream implications for mental and physical health. However, this hypothesis has yet to be tested rigorously, because extant research has been cross-sectional or has relied on self-reports of everyday symptoms, which are poor indicators of underlying disease (Feldman, Cohen, Doyle, Skoner, & Gwaltney, 1999; Watson & Pennebaker, 1989). In the study reported here, we sought to provide a more rigorous test by following a cohort of adolescents over the span of 1 year. Adolescents are a theoretically interesting population for such research because they are actively engaged in forming identities (Markus & Nurius, 1986). This often entails pursuing goals that later prove to be unrealizable. To evaluate the impact of goal regulation on health, we tracked changes in levels of C-reactive protein (CRP), a marker of systemic inflammation. When the immune system detects an infection or injury, it launches an inflammatory response, with the goal of eliminating pathogens and repairing tissue damage. Although this response is critical for survival, its magnitude and duration must be carefully regulated, because excessive inflammation contributes to numerous medical conditions. Thus, CRP functions as a useful prognostic indicator; to the extent that people exhibit higher levels of it, they have greater long-term risk for diabetes, heart disease, and other medical conditions (Willerson & Ridker, 2004).

We predicted that capacities for managing pursuits that had become unattainable would have downstream consequences for inflammation. Specifically, we expected that to the extent subjects experienced difficulties disengaging from unattainable goals, they would show increasing concentrations of CRP over time. Because depression often contributes to difficulties in managing goals (Nesse, 2000), and is typically accompanied by inflammation (Miller & Blackwell, 2006), we also sought to rule out the possibility that any observed association between ability to disengage from unattainable goals and CRP change over time was due to this mood state.

METHOD

Subjects

Data were collected as part of a larger project on depression and atherosclerosis among young women at high risk for affective disorders. Adolescent females were recruited from Vancouver, British Columbia, through advertisements in local media. Potential recruits were eligible for the larger study if they had (a) no history of psychiatric disorders and were (b) 15 to 19 years old, (c) fluent in English, (d) free of acute and chronic medical conditions, and (e) at high risk for developing an initial episode of depression. High risk for depression was defined as having a first-degree relative with a history of depression or as scoring in the top quartile of the population distribution on either of two indices of cognitive vulnerability, the Dysfunctional Attitudes Scale (Alloy et al., 2006) or the Adolescent Cognitive Style Questionnaire (Hankin & Abramson, 2002).

This article focuses on 90 subjects who completed three waves of data collection, timed to occur at entry into the study (Wave 1) and approximately 6 and 12 months later (Waves 2 and 3). The sample had a mean age of 17.23 years ($SD = 1.37$). The majority identified themselves as East Asian (49%) or as Caucasian (41%); the others identified themselves as East Indian, Aboriginal, or "other." Participants' parents averaged 15.12 years of education. The project was approved by the University of British Columbia's Research Ethics Board, and written consent was obtained from all participants. For those who were younger than 18, a parent or guardian also provided consent.

Measures

To measure tendencies for managing unattainable goals, we administered a previously validated questionnaire when subjects entered the study (Wrosch et al., 2007; Wrosch, Scheier, Miller, et al., 2003). This questionnaire included 10 items measuring how subjects usually reacted when they had to stop pursuing an important goal. Four items measured tendencies to disengage (e.g., "It's easy for me to reduce my effort towards the goal"), and 6 measured tendencies to reengage with new goals (e.g., "I seek other meaningful goals"). Response options ranged from 1, *almost never true*, to 5, *almost always true*. Responses were summed to obtain scores for disengagement and reengagement tendencies. The sample's average scores on the disengagement and reengagement scales were 9.72 ($SD = 2.82$) and 22.54 ($SD = 3.41$), respectively. The scales were internally consistent (αs = .67 and .81) and modestly correlated with each other, $r = .21$, $p = .02$.

To measure systemic inflammation, we collected blood through antecubital venipuncture following a 12-hr fast. It was centrifuged at 1,000 × g for 25 min; the serum was then aspirated and frozen at −20 °C. CRP was analyzed in batches using a high-sensitivity, chemiluminescent technique on an IMMULITE 2000 (Diagnostic Products Corporation, Los Angeles, CA). This assay has an interassay coefficient of variation of 2.2% and a detection threshold of 0.20 mg/L. The mean CRP values at Waves 1, 2, and 3 were 0.61 ($SD = 0.89$), 0.68 ($SD = 1.17$), and 0.92 mg/L ($SD = 1.67$), respectively. CRP levels were moderately stable over the year, Spearman $r = .53$, $p = .001$.

We also collected information regarding a number of potential confounds. These included demographic (age, ethnicity) and behavioral (smoking, adiposity) characteristics that have been linked with systemic inflammation (Irwin, 2001; Miller, Stetler, Carney, Freedland, & Banks, 2002). Smoking was indexed as whether or not cigarettes were used daily, and adiposity as body mass index (BMI). These variables were modeled as covariates, except in the case of smoking, which was reported by only 3 women. Depressive symptoms were assessed with the Beck

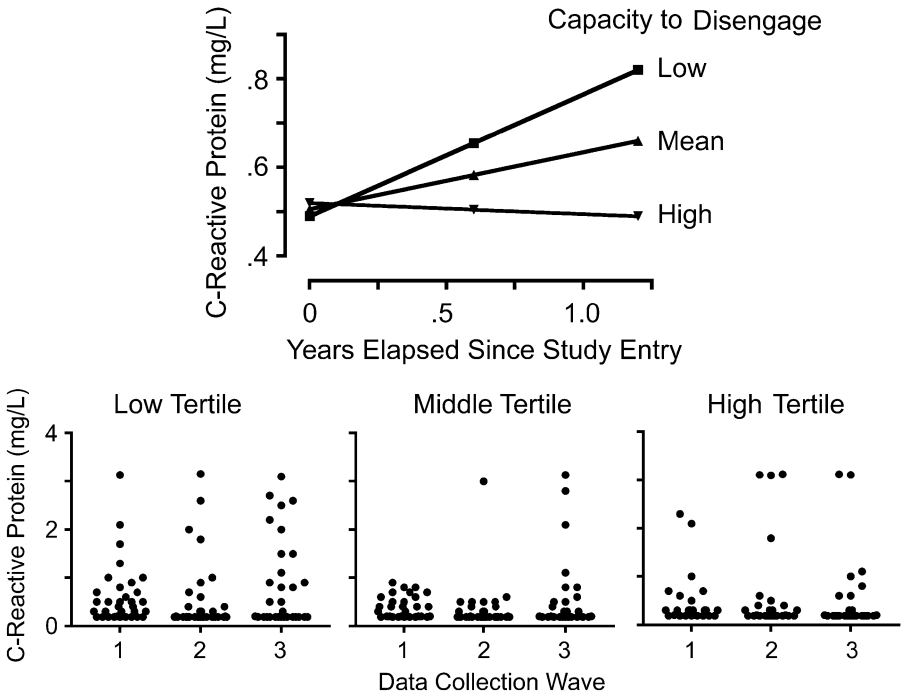

Fig. 1. Systemic inflammation over the course of 1 year. The top graph displays changes in the level of C-reactive protein as a function of time and capacity to disengage from unattainable goals. Estimated trajectories are plotted for low, medium, and high levels of disengagement, corresponding to 1 *SD* below the mean, the sample average, and 1 *SD* above the mean. The three graphs at the bottom display raw data for C-reactive protein levels at each collection wave, for subjects in each tertile of capacity to disengage.

Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at the time of entry into the study.

RESULTS

To determine whether goal-regulation strategies presage changes in systemic inflammation, we estimated a series of growth-curve models utilizing HLM 6.03 (Raudenbush, Bryk, & Congdon, 2006). In the within-person (or Level 1) model, we estimated CRP as a function of time since study entry, BMI, and a residual term. This model yielded a series of person-specific intercepts reflecting CRP at study entry (β_{0i}) and person-specific trajectories reflecting the rate of CRP change over the year (β_{1i}). In the between-person (or Level 2) model, we estimated β_{0i} and β_{1i} values for each subject as a function of age, ethnicity, goal-disengagement and goal-reengagement tendencies, and a product term reflecting the interaction of these two tendencies. These models also included random variables specifying the amounts by which each subject deviated from the sample's average β_{0i} and β_{1i}. The critical parameters were the coefficients γ_{13}, γ_{14}, and γ_{15}, which reflect associations between goal-regulation variables and CRP trajectories, after the influence of confounds had been eliminated.

We began by estimating a simple Level 1 model to describe patterns of change. Over the 1-year follow-up, the sample exhibited a marginally significant increase in CRP, which amounted to 0.13 mg/L per year after adiposity was controlled, $\beta_{1i} = .13$, $SE = .07$, $p_{rep} = .85$. However, there was significant variability around this average, $p_{rep} = .93$, indicating the presence of reliable between-person differences. The Level 1 model further indicated that BMI and CRP covaried over time, $\beta_{2i} = .05$, $SE = .02$, $p_{rep} = .97$. As a woman's adiposity increased, so did her level of inflammation. Next, demographics were added to the Level 2 model, but they did not predict CRP trajectories, p_{rep}s $< .60$.

Finally, goal disengagement, goal reengagement, and their interaction were added to the model. Disengagement was a significant predictor of CRP trajectories, $\gamma_{13} = -0.15$, $SE = 0.07$, $p_{rep} = .89$, with each 1-*SD* increment in persistence associated with a 0.15 gain in inflammation (Fig. 1). Thus, for subjects with average disengagement tendencies, CRP increased at a rate of 0.13 mg/L annually. For those with poor disengagement (i.e., 1 *SD* below average), CRP increased at twice this rate, slope = 0.28. And for subjects who could easily disengage (i.e., 1 *SD* above average), inflammation declined slightly over the year, slope = −0.02. These effects were fairly large in magnitude. The between-person variance in CRP dropped from 0.09 to 0.08, or by 11%, in the model with disengagement, relative to a model in which demographics only were included. Though disengagement was a robust predictor of CRP, neither reengagement nor the interaction of disengagement and reengagement was associated with CRP, p_{rep}s $< .66$.

The final analyses evaluated the contribution of depressive symptoms. When scores on the Beck Depression Inventory were added to the Level 2 model, disengagement remained a significant predictor of CRP trajectories, $\gamma_{13} = -0.14$, $SE = 0.07$, $p_{rep} = .89$, suggesting that our findings do not reflect a spurious association fostered by affective problems.

DISCUSSION

Although the virtues of persistence have been extolled in the popular and scientific literatures, a tendency to "press on" can be maladaptive when there are serious obstacles to realizing goals (Wrosch, Scheier, Carver, & Schulz, 2003). We have shown that the inability to disengage from goals in such situations has downstream biological consequences in the form of systemic inflammation. This effect was fairly large. The rate of CRP increase was twice as rapid among women with poor disengagement tendencies as it was among women at the sample average. And CRP actually declined slightly over the year among women who easily disengaged. Because the sample comprised healthy teenagers whose CRP levels were within the normal range, the escalating inflammation they exhibited would be unlikely to have immediate medical consequences. However, to the extent that such trends persist over time, they would heighten risk for a variety of diseases, including diabetes, osteoporosis, and atherosclerosis (Papanicoloaou, Wilder, Manolagas, & Chrousos, 1998; Willerson & Ridker, 2004). Though none of these conditions is typically diagnosed until middle adulthood, the pathology underlying them begins in childhood (Berenson & Srnivasan, 2005). Thus, inflammation is probably contributing to the early stages of disease in our study's "persisters." Because inflammation is recognized as contributing to the formation of depressive symptoms (Miller & Blackwell, 2006), our findings may also shed light on why the inability to disengage from unrealized goals often results in affective difficulties (Nesse, 2000).

These findings have widespread theoretical implications. They suggest that psychological models of adaptation would benefit from a more balanced perspective on persistence. When opportunities for goal realization are favorable, persistence is likely to result in beneficial outcomes. But as our findings illustrate, giving up can be adaptive, particularly when goals are unattainable. The findings also suggest a need to broaden the scope of models in health psychology, which have focused on severe chronic stressors and intense negative emotions as the central psychosocial determinants of health (Kiecolt-Glaser, Mcguire, Robles, & Glaser, 2002; Miller & Blackwell, 2006; Schneiderman, Ironson, & Siegel, 2005). Our data suggest that even normative differences in self-regulation may have consequences for health.

Future research will need to identify mechanisms through which goal-regulation tendencies "get inside the body." Our data indicate that depression, smoking, and adiposity are not the culprits. We suspect that the distal psychological mediators are mood states involving frustration, ambivalence, and exhaustion, which in turn modify activities of proximal biological mediators such as the autonomic nervous system and the hypothalamic-pituitary-adrenocortical axis (Pavlov & Tracey, 2005; Webster, Tonelli, & Sternberg, 2002). It is also possible that persistent individuals have difficulties sleeping because they are ruminating about failures to progress toward goals. Sleep is an important regulator of immune functions, and when it is disturbed, there are robust increases in systemic inflammation (Irwin, Wang, Campomayor, Collado-Hidalgo, & Cole, 2006). In addition to evaluating these mediational hypotheses, future research will need to clarify the influence of goal reengagement. Unlike previous studies (Wrosch et al., 2007; Wrosch, Scheier, Miller, et al., 2003), the present study did not find a buffering effect of reengagement. This may have been a result of the life stage of the women in our sample, who were not yet at a point where they were pursuing major long-term goals (e.g., getting married, having children, building a career).

This project had several limitations. Because the sample was chosen to be at high risk for depression, it is not representative of the general population. Though this constrains the generalizability of the findings, it does not seriously complicate interpretation of them. Also, we relied on self-reports of goal-management capacities; future research must substantiate our findings with behavioral indicators. It must also examine specific goal contexts. Our previous research indicates that people's goal-regulation tendencies are consistent across different pursuits (Wrosch, Scheier, Miller, et al., 2003), so we would not be surprised if the biological consequences of persistence were most pronounced among people experiencing difficulties with multiple self-relevant goal domains.

Despite its limitations, this study had a number of strengths, including its longitudinal design and repeated assessments of a clinically important biomarker. Its findings provide fresh insights into the benefits and liabilities of persistence, a construct long viewed as adaptive in popular and scientific circles. But like most interesting phenomena in social psychology, the maladaptive effects of persistence were anticipated long ago by a country-western singer. In his 1978 hit "The Gambler," Kenny Rogers issued what turns out to be a wise prescription to goal seekers: "If you're gonna play the game, boy, you gotta learn to play it right. You've got to know when to hold 'em; know when to fold 'em. Know when to walk away, and know when to run" (Rogers, 1978).

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REFERENCES

Alloy, L.B., Abramson, L.Y., Whitehouse, W.G., Hogan, M.E., Panzarella, C., & Rose, D.T. (2006). Prospective incidence of first onsets and recurrences of depression in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 115*, 145–156.

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, *5*, 462–467.

Berenson, G.S., & Srnivasan, S.R. (2005). Cardiovascular risk factors in youth with implications for aging: The Bogalusa Heart Study. *Neurobiology of Aging*, *26*, 303–307.

Carver, C.S., & Scheier, M.F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Feldman, P.J., Cohen, S., Doyle, W., Skoner, D., & Gwaltney, J.M., Jr. (1999). The impact of personality on the reporting of unfounded symptoms and illness. *Journal of Personality and Social Psychology*, *77*, 370–378.

Hankin, B.L., & Abramson, L.Y. (2002). Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. *Journal of Clinical Child and Adolescent Psychiatry*, *31*, 491–504.

Heckhausen, J., & Schulz, R. (1995). A life-span theory of control. *Psychological Review*, *102*, 284–304.

Irwin, M.R. (2001). Depression and immunity. In R. Ader, D. Felten, & N. Cohen (Eds.), *Psychoneuroimmunology* (3rd ed., pp. 383–398). New York: Academic Press.

Irwin, M.R., Wang, M., Campomayor, C., Collado-Hidalgo, A., & Cole, S. (2006). Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. *Archives of Internal Medicine*, *166*, 1756–1762.

Kiecolt-Glaser, J.K., Mcguire, L., Robles, T.F., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology*, *53*, 83–107.

Klinger, E. (1975). Consequences of commitment to and disengagement from incentives. *Psychological Review*, *82*, 1–25.

Knowles, E. (1999). *The Oxford dictionary of quotations*. New York: Oxford University Press.

Markus, H., & Nurius, P. (1986). Possible selves. *American Psychologist*, *41*, 954–969.

Miller, G.E., & Blackwell, E. (2006). Turning up the heat: Inflammation as a mechanism linking chronic stress, depression, and heart disease. *Current Directions in Psychological Science*, *15*, 269–272.

Miller, G.E., Stetler, C.A., Carney, R.M., Freedland, K.E., & Banks, W.A. (2002). Clinical depression and inflammatory risk markers for coronary heart disease. *The American Journal of Cardiology*, *90*, 1279–1283.

Nesse, R.M. (2000). Is depression an adaptation? *Archives of General Psychiatry*, *57*, 14–20.

Papanicoloaou, D.A., Wilder, R.L., Manolagas, S.C., & Chrousos, G.P. (1998). The pathophysiologic roles of interleukin-6 in human disease. *Annals of Internal Medicine*, *128*, 127–137.

Pavlov, V., & Tracey, K. (2005). The cholinergic anti-inflammatory pathway. *Brain, Behavior, and Immunity*, *19*, 493–499.

Raudenbush, S.W., Bryk, A.S., & Congdon, R.T. (2006). *HLM Version 6.03*. Chicago: Scientific Software International.

Rogers, K. (1978). The gambler. On *The gambler* [Record]. Hollywood, CA: United Artists.

Schneiderman, N., Ironson, G., & Siegel, S.D. (2005). Stress and health: Psychological, behavioral, and biological determinants. *Annual Review of Psychology*, *1*, 607–628.

Seligman, M.E.P. (1975). *Helplessness*. San Francisco: W.H. Freeman.

Taylor, S.E., & Brown, J.D. (1988). Illusion and well-being: A social psychological perspective on mental health. *Psychological Bulletin*, *103*, 193–210.

Watson, D., & Pennebaker, J.W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*, *96*, 234–254.

Webster, J.I., Tonelli, L., & Sternberg, E.M. (2002). Neuroendocrine regulation of immunity. *Annual Review of Immunology*, *20*, 125–163.

Willerson, J.T., & Ridker, P.M. (2004). Inflammation as a cardiovascular risk factor. *Circulation*, *109*, 2–10.

Wrosch, C., Miller, G.E., Scheier, M.F., & Brun De Pontet, S. (2007). Giving up on unattainable goals: Benefits for health? *Personality and Social Psychology Bulletin*, *33*, 251–265.

Wrosch, C., Scheier, M.F., Carver, C.S., & Schulz, R. (2003). The importance of goal disengagement in adaptive self-regulation: When giving up is beneficial. *Self and Identity*, *2*, 1–20.

Wrosch, C., Scheier, M.F., Miller, G.E., Schulz, R., & Carver, C.S. (2003). Adaptive self-regulation of unattainable goals: Goal disengagement, goal reengagement, and subjective well-being. *Personality and Social Psychology Bulletin*, *29*, 1494–1508.

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