

Short Communication

Stress response and binge eating disorder

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Abstract

In clinical practice, obese patients report stress as a primary trigger for binge eating. However, the biological mechanism underlying this relationship is poorly understood. This paper presents, a theoretical overview of how cortisol secretion, a major component of the stress response, could play a role in binge eating, given that exogenous glucocorticoids can lead to obesity by increasing food intake. I will discuss findings from recent studies demonstrating links between laboratory stress, cortisol, food intake and abdominal fat in humans. Cortisol is elevated following laboratory stressors in women with anorexia nervosa (AN), bulimia nervosa (BN), and obesity, but has not been widely studied in women with binge eating disorder (BED). Additionally, I will review recent findings demonstrating a greater cortisol response to stress in obese women with BED compared to non-BED.

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Background

Obesity, defined as a body mass index [BMI, in kg/m^2] > 30 , has become a medical epidemic in the US (Caballero, 2001). There is a subset of obese individuals who have binge eating disorder (BED) (Mitchell & Mussell, 1995). These individuals exhibit greater psychopathology, often do less well in weight loss treatment and relapse more quickly than non-BED (Yanovski, Nelson, Dubbert, & Spitzer, 1993a). BED is characterized by the consumption of objectively large amounts of food and a sense of loss of control overeating (Wilfley, Agras, Telch, Rossiter, Schneider and Cole, 1993). BED affects about 2% in the general community (Spitzer, Yanovski, Wadden, Wing, Marcus and Stunkard, 1993) and a much larger proportion (18–46%) of obese individuals presenting for weight loss treatment (de Zwaan, Mitchell, Raymond, & Spitzer, 1994).

The lifetime prevalence of psychiatric diagnoses in BED ranges from 60–72% as compared to only 28–49% in controls (Marcus, Wing, Ewing, & Kern, 1990; Specker, de Zwaan, Raymond, & Mitchell, 1994). There is evidence for

moderate heritability of .50 in BED (Bulik, Sullivan, & Kendler, 2003) and for a genetic association between BED and melanocortin 4 receptor (MC4R) mutation (Branson, Potoczna, Kral, Lentjes, Hoehe and Horber, 2003). Further support for a biological basis for BED includes physiological abnormalities in the actions of peripheral hormones such as insulin (Hellstrom, Geliebter, Naslund, Schmidt, Yahav and Hashim, 2004), leptin (Adami, Campostano, Cella, & Scopinaro, 2002), and ghrelin (Geliebter, Yahav, Gluck, & Hashim, 2004).

Cortisol and appetitive behavior

Cortisol is involved in appetite regulation (Wolkowitz, Epel, & Rues, 2001) and energy balance by increasing available energy through gluconeogenesis and lipolysis (Epel, Lapidus, McEwen, & Brownell, 2001). In animals, glucocorticoid administration (Bray, 1985) and corticosterone replacement (Castonguay, 1991) led to hyperphagia and weight gain. Corticosterone also increased fat intake in a dose-dependent fashion in rats (Castonguay, 1991). Dallman and colleagues recently described a 'chronic stress-response network' in rats, based on greater consumption of sucrose following administration of glucocorticoids, leading to increased abdominal fat depots. They suggest that

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overeating of ‘comfort foods’ in humans may be stimulated by cortisol in response to stress, which can result in abdominal obesity (Dallman, Pecoraro, Akana, La Fleur, Gomez and Houshyar, 2003).

The relationship between cortisol and appetitive behavior has been studied less frequently in humans than in rats (Epel et al., 2001; Wolkowitz et al., 2001). In healthy males, exogenous glucocorticoid administration increased daily food intake compared to placebo (Tataranni, Larson, Snitker, Young, Flatt and Ravussin, 1996). Cushing’s patients, with elevated cortisol levels selected high-fat foods twice as often as normal weight subjects and three times as often as overweight controls (Castonguay, 1991). In the night eating syndrome, the nighttime elevation of cortisol might contribute to inducing awakenings to eat (Birketvedt, Florholmen, Sundsfjord, Osterud, Dinges and Bilker, 1999). Only one study has examined both cortisol changes and food intake following a laboratory stressor and found that among healthy women, high cortisol reactors ate significantly more food following a cognitive stress task compared to low cortisol reactors (Epel et al., 2001). No work to date has examined cortisol and food intake in obese individuals with BED.

Cortisol stress response in eating disorders

The key hormonal pathway that governs the endocrine response to stress is the hypothalamic-pituitary-adrenal (HPA) axis. The dexamethasone suppression test (DST) is widely used in clinical settings to test for hypercortisolemia and diminished sensitivity to HPA negative feedback (Walsh, Gladis, & Roose, 1987a). Oral dexamethasone characteristically suppresses the morning rise in cortisol. DST non-suppression and hypercortisolemia have been reported in anorexia nervosa (AN) (Brambilla, Ferrari, Panerai, Manfredi, Petraglia and Catalano, 1993; Walsh et al., 1987b) and BN (Fricther, Pirke, Pollinger, Wolfram, & Brunner, 1990), but not in BED (Gluck, Geliebter, Hung, & Yahav, 2004a; Yanovski, Yanovski, Gwirtsman, Bernat, Gold & Chrousos 1993b). Compared to controls, women with AN (Putignano, Dubini, Toja, Invitti, Bonfanti and Redaelli, 2001), BN (Pirke, Platte, Laessle, Seidl, & Fichter, 1992), BED (Gluck et al., 2004a) and the night eating syndrome (Birketvedt et al., 1999) had higher basal cortisol compared to controls.

Exaggerated cortisol responses to stress have been observed in women with AN (Abell, Malagelada, Lucas, Brown, Camilleri and Go, 1987), BN, (Koo-Loeb, Costello, Light, & Girdler, 2000) and obesity (Marin, Darin, Amemiya, Andersson, Jern and Bjorntorp, 1992). Few studies have examined cortisol responsivity to stress over the long-term, although higher 24-h urinary cortisol in women with BN were observed on the day following an interpersonal speech task compared to a control group (Koo-Loeb et al., 2000). Only one study failed to observe

raised cortisol levels following a mental challenge stress test in BN, despite higher baseline levels compared to healthy controls (Pirke et al., 1992). Our recent study was the first to examine cortisol following stress in BED and found a trend towards a greater AUC for cortisol in BED than non-BED following a CPT, unrelated to depression scores.

Chronic hypercortisolemia and stress responsivity

Although acute elevation of cortisol plays a protective role during stress, persistently elevated levels promote insulin resistance and abdominal obesity (Bjorntorp & Rosmond, 2000; Jayo, Shively, Kaplan, & Manuck, 1993). Most studies have observed that chronic stress over-activates the hypothalamic-pituitary-adrenal (HPA) axis and fuels insulin release, in turn activating abdominal fat storage (Dallman, Akana, Strack, Hanson, & Sebastian, 1995). However, the literature is mixed, in that some studies show a sluggish HPA axis response to waking in men with greater abdominal fat (Rosmond, Holm, & Bjorntorp, 2000).

Abdominal fat distribution is also related to vulnerability to stress. For example, in response to laboratory stressors, women with a high WHR reported feeling more threatened (Epel, McEwen, Seeman, Matthews, Castellazzo and Brownell, 2000), and had higher cortisol reactivity than women with a low WHR (Moyer, Rodin, Grilo, Cummings, Larson and Rebuffe-Scrive, 1994). Another study of overweight women found a positive correlation between cortisol levels following a cold pressor test (CPT) and sagittal diameter of the abdomen (Marin et al., 1992). We observed a relationship between WHR and cortisol levels following a CPT, only in women with BED (Gluck et al., 2004). This relationship persisted, even after a 6 week treatment consisting of a liquid diet and cognitive behavioral (Gluck, Geliebter, & Lorence, 2004b).

Emotional stress responses and food intake

The majority of studies have measured food intake following laboratory stressors without measuring biological correlates. Surprisingly, many have failed to observe differences in overall levels of consumption, but rather differences in macronutrient content. For example, Levine and Marcus found no differences between women with bulimic symptoms versus control women following an interpersonal speech task (Levine & Marcus, 1997). However, both bulimic and control women increased their consumption of carbohydrates following the stress task compared to the no-stress control condition. Lattimore and colleagues observed increased ice cream consumption following the Stroop task and watching a fearful film compared to a control day, but consumption did not differ between normal weight binge eaters and non-binge eaters (Lattimore, 2001). Only one laboratory study has examined

stress and food intake in BED and found no differences on calories ingested during a multi-item buffet compared to controls following a negative mood versus neutral mood induction (Telch & Agras, 1996).

There is strong evidence that restrained and emotional eaters overeat in response to stress (Wallis & Hetherington, 2004). Restrained eaters consumed more than unrestrained following a reaction time task, while the opposite was observed following a relaxation condition (Lattimore & Caswell, 2004). Following an interpersonal stressor, restrained eaters ate more than did non-restrained eaters (Stroud, Tanofsky-Kraff, Wilfley, & Salovey, 2000). Moreover, the greater the restraint, the more participants ate (Tanofsky-Kraff, Wilfley, & Spurrell, 2000). Stressed emotional eaters ate more sweet, high-fat foods and a more energy-dense meal than unstressed and non-emotional eaters following a speech preparation task (Oliver, Wardle, & Gibson, 2000).

Subjective ratings are often used to assess hunger and desire to eat in studies where food intake is not measured. Cattanach and colleagues reported greater desires to binge eat following four different laboratory stress tests in women with eating disordered symptomatology compared to a control group (Cattanach, Malley, & Rodin, 1988). Bulimic patients reported increases in hunger and desires to binge eat compared to restrained eaters and controls following an interpersonal imagery task (Tuschen-Caffier & Voegelé, 1999). Obese BED subjects reported greater levels of hunger and desire to binge eat following a CPT compared to obese non-BED subjects (Gluck et al., 2004b).

Thus, it appears that while macronutrient content appears to be more relevant to the stress response in all subjects, subjective ratings of hunger and binge eating differ between individuals with and without eating pathology. Moreover, some of the relationships reported on in this paper could be due to restrained eating and dieting, rather than to differences in eating pathology. Future studies should control for diet history and current levels of restrained eating to better differentiate these effects.

Research limitations and future directions

There are several limitations to the stress and eating studies to date. Researchers use a variety of methods by which to induce stress in the laboratory and it is therefore difficult to compare results from study to study. None of the approaches used to date are naturalistic and therefore they reveal nothing about factors that might actually lead to binge eating in real life. Moreover, these laboratory stressors last for various durations, ranging in length from 2 to 45 min. The psychological factors of disordered eating have been more widely studied in humans than the biological factors. The most striking limitation remaining is that very few studies integrate both behavioral and physiological models of stress-induced eating. No studies to

date have manipulated cortisol in BED to examine effects on food intake, a study that our lab plans to undertake.

Emotional eating has been associated with both increased and decreased food intake (Greeno & Wing, 1994), and little is known about the mechanisms that underlie the direction of change. Typically, responses to stress result in anorexia and, if the stress is sufficiently persistent, weight loss. The longstanding view is that stress produces sympathetic arousal that results in reductions, rather than increases in eating. For example, in rats, both a single social defeat stressor (Berton, Durand, Aguerre, Mormede, & Chaouloff, 1999) and a 2-h immobilization stressor (Shimizu, Oomura, & Kai, 1989) resulted in a significant reduction of food intake and body weight. More recently, however, incongruent paradigms of stress physiology have emerged (Sapolsky, Romero, & Munck, 2000) and it is unclear why some animals and humans show opposite eating reactions to stressors. As previously mentioned, the direction of change in intake could be predicted by restrained eating. Overeating has been observed in rats following a stress, following a period of caloric restriction (Hagan, Wauford, Chandler, Jarrett, Rybak and Blackburn, 2002) only in those given highly palatable food (Hagan, Chandler, Wauford, Rybak, & Oswald, 2003). Likewise, in humans, dieters are more likely to report stress hyperphagia compared to non-dieters who are more likely to report stress hypophagia (Oliver & Wardle, 1999). To address all of the other possible mechanisms would be outside the scope of this review. However, it is important to note that this paper addresses only this anomalous group of subjects who show increased, rather than decreased, food intake and weight gain when stressed.

Appetitive behavior is complex and multifaceted. Stress reactivity, both physiological and psychological, may distinguish overeaters from under eaters (Cattanach et al., 1988). A better, more sophisticated understanding of these mechanisms that have to date not been widely studied, would go a long way toward understanding the stress eating relationship. Integration of these factors is paramount for understanding obesity and binge eating from a biopsychosocial perspective, which could help lead to better treatment options for this group of co-morbid obese patients who experience marked psychological distress and impairment.

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References

- Abell, T. L., Malagelada, J. R., Lucas, A. R., Brown, M. L., Camilleri, M., Go, V. L., et al. (1987). Gastric electromechanical and neurohormonal function in anorexia nervosa. *Gastroenterology*, *93*, 958–965.
- Adami, G. F., Campostano, A., Cella, F., & Scopinaro, N. (2002). Serum leptin concentration in obese patients with binge eating disorder. *International Journal of Obesity Related Metabolic Disorders*, *26*, 1125–1128.
- Berton, O., Durand, M., Aguerre, S., Mormede, P., & Chaouloff, F. (1999). Behavioral, neuroendocrine and serotonergic consequences of single social defeat and repeated fluoxetine pretreatment in the Lewis rat strain. *Neuroscience*, *92*, 327–341.
- Birketvedt, G. S., Florholmen, J., Sundsfjord, J., Osterud, B., Dinges, D., Bilker, W., et al. (1999). Behavioral and neuroendocrine characteristics of the night-eating syndrome. *The Journal of the American Medical Association*, *282*, 657–663.
- Bjorntorp, P., & Rosmond, R. (2000). Neuroendocrine abnormalities in visceral obesity. *International Journal of Obesity and Related Metabolic Disorders*, *24*(Suppl. 2), S80–S85.
- Brambilla, F., Ferrari, E., Panerai, A., Manfredi, B., Petraglia, F., Catalano, M., et al. (1993). Psychoimmunoendocrine investigation in anorexia nervosa. *Neuropsychobiology*, *27*, 9–16.
- Branson, R., Potoczna, N., Kral, N., Lentz, K. U., Hoehe, M. R., & Horber, F.F. (2003). Binge eating as a major phenotype of melanocortin 4 receptor gene mutations. *The New England Journal of Medicine*, *348*, 1096–1103.
- Bray, G. A. (1985). Autonomic and endocrine factors in the regulation of food intake. *Brain Research Bulletin*, *14*, 505–510.
- Bulik, C. M., Sullivan, P. F., & Kendler, K. S. (2003). Genetic and environmental contributions to obesity and binge eating. *The International Journal of Eating Disorders*, *33*, 293–298.
- Caballero, B. (2001). Introduction. Symposium: Obesity in developing countries: Biological and ecological factors. *The Journal of Nutrition*, *131*, 866S–870S.
- Castonguay, T. W. (1991). Glucocorticoids as modulators in the control of feeding. *Brain Research Bulletin*, *27*, 423–428.
- Cattanach, L., Malley, R., & Rodin, J. (1988). Psychologic and physiologic reactivity to stressors in eating disordered individuals. *Psychosomatic Medicine*, *50*, 591–599.
- Dallman, M. F., Akana, S. F., Strack, A. M., Hanson, E. S., & Sebastian, R. J. (1995). The neural network that regulates energy balance is responsive to glucocorticoids and insulin and also regulates HPA axis responsivity at a site proximal to CRF neurons. *Annals of the New York Academy of Sciences*, *771*, 730–742.
- Dallman, M. F., Pecoraro, N., Akana, S. F., La Fleur, S. E., Gomez, F., Houshyar, H., et al. (2003). Chronic stress and obesity: a new view of 'comfort food'. *Proceedings of the National Academy of Sciences of the United States of America*, *100*, 11696–11701.
- de Zwaan, M., Mitchell, J. E., Raymond, N. C., & Spitzer, R. L. (1994). Binge eating disorder: Clinical features and treatment of a new diagnosis. *Harvard Review of Psychiatry*, *1*, 310–325.
- Epel, E. S., Lapidus, R., McEwen, B., & Brownell, K. (2001). Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. *Psychoneuroendocrinology*, *26*, 34–49.
- Epel, E. S., McEwen, B., Seeman, T., Matthews, K., Castellazzo, G., Brownell, K. D., et al. (2000). Stress and body shape: stress-induced cortisol secretion is consistently greater among women with central fat. *Psychosomatic Medicine*, *62*, 623–632.
- Fricther, M. M., Pirke, K. M., Pollinger, J., Wolfram, G., & Brunner, E. (1990). Disturbances in the hypothalamic–pituitary–adrenal and other neuroendocrine axes in bulimia. *Biological Psychiatry*, *27*, 1021–1037.
- Geliebter, A., Yahav, E. K., Gluck, M. E., & Hashim, S. A. (2004). Gastric capacity, test meal intake, and appetitive hormones in binge eating disorder. *Physiology and Behavior*, *81*, 735–740.
- Gluck, M. E., Geliebter, A., Hung, J., & Yahav, E. (2004a). Cortisol, hunger, and desire to binge eat following a cold stress test in obese women with binge eating disorder (BED). *Psychosomatic Medicine*, *66*, 876–881.
- Gluck, M. E., Geliebter, A., & Lorence, M. (2004b). Cortisol stress response is positively correlated with central obesity in obese women with binge eating disorder (BED) before and after cognitive-behavioral treatment. *Annals of the New York Academy of Sciences*, *1032*, 202–207.
- Greeno, C. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin*, *115*, 444–464.
- Hagan, M., Wauford, P., Chandler, P., Jarrett, L., Rybak, R., & Blackburn, K. (2002). A new animal model of binge eating. Key synergistic role of past caloric restriction and stress. *Physiology and Behavior*, *77*, 45.
- Hagan, M. M., Chandler, P. C., Wauford, P. K., Rybak, R. J., & Oswald, K. D. (2003). The role of palatable food and hunger as trigger factors in an animal model of stress induced binge eating. *The International Journal of Eating Disorders*, *34*, 183–197.
- Hellstrom, P. M., Geliebter, A., Naslund, E., Schmidt, P. T., Yahav, E. K., Hashim, S. A., et al. (2004). Peripheral and central signals in the control of eating in normal, obese and binge-eating human subjects. *The British Journal of Nutrition*, *92*(Suppl. 1), S47–S57.
- Jayo, J. M., Shively, C. A., Kaplan, J. R., & Manuck, S. B. (1993). Effects of exercise and stress on body fat distribution in male cynomolgus monkeys. *International Journal of Obesity and Related Metabolic Disorders*, *17*, 597–604.
- Koo-Loeb, J. H., Costello, N., Light, K. C., & Girdler, S. S. (2000). Women with eating disorder tendencies display altered cardiovascular, neuroendocrine, and psychosocial profiles. *Psychosomatic Medicine*, *62*, 539–548.
- Lattimore, P., & Caswell, N. (2004). Differential effects of active and passive stress on food intake in restrained and unrestrained eaters. *Appetite*, *42*, 167–173.
- Lattimore, P. J. (2001). Stress-induced eating: an alternative method for inducing ego-threatening stress. *Appetite*, *36*, 187–188.
- Levine, M., & Marcus, M. (1997). Eating behavior following stress in women with and without bulimic symptoms. *Annals of Behavior Medicine*, *19*, 132–138.
- Marcus, M. D., Wing, R. R., Ewing, L., & Kern, E. (1990). Psychiatric disorders among obese binge eaters. *The International Journal of Eating Disorders*, *9*, 69–77.
- Marin, P., Darin, N., Amemiya, T., Andersson, B., Jern, S., & Bjorntorp, P. (1992). Cortisol secretion in relation to body fat distribution in obese premenopausal women. *Metabolism*, *41*, 882–886.
- Mitchell, J. E., & Mussell, M. P. (1995). Comorbidity and binge eating disorder. *Addictive Behaviors*, *20*, 725–732.
- Moyer, A., Rodin, J., Grilo, C., Cummings, N., Larson, L., & Rebuffe-Scrive, M. (1994). Stress-induced cortisol response and fat distribution in women. *Obesity Research*, *2*, 255–262.
- Oliver, G., Wardle, J., & Gibson, E. L. (2000). Stress and food choice: a laboratory study. *Psychosomatic Medicine*, *62*, 853–865.
- Pirke, K. M., Platte, P., Laessle, R., Seidl, M., & Fichter, M. M. (1992). The effect of a mental challenge test of plasma norepinephrine and cortisol in bulimia nervosa and in controls. *Biological Psychiatry*, *32*, 202–206.
- Putignano, P., Dubini, A., Toja, P., Invitti, C., Bonfanti, S., Redaelli, G., et al. (2001). Salivary cortisol measurement in normal-weight, obese and anorexic women: Comparison with plasma cortisol. *European Journal of Endocrinology*, *145*, 165–171.

- Rosmond, R., Holm, G., & Bjorntorp, P. (2000). Food-induced cortisol secretion in relation to anthropometric, metabolic and haemodynamic variables in men. *International Journal of Obesity and Related Metabolic Disorders*, 24, 416–422.
- Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, 21, 55–89.
- Specker, S., de Zwaan, M., Raymond, N., & Mitchell, J. (1994). Psychopathology in subgroups of obese women with and without binge eating disorder. *Comprehensive Psychiatry*, 35, 185–190.
- Spitzer, R. L., Yanovski, S., Wadden, T., Wing, R., Marcus, M. D., Stunkard, A., et al. (1993). Binge eating disorder: Its further validation in a multisite study. *The International Journal of Eating Disorders*, 13, 137–153.
- Stroud, L. R., Tanofsky-Kraff, M., Wilfley, D. E., & Salovey, P. (2000). The Yale interpersonal stressor (YIPS): Affective, physiological, and behavioral responses to a novel interpersonal rejection paradigm. *Annals of Behavioral Medicine*, 22, 204–213.
- Tanofsky-Kraff, M., Wilfley, D. E., & Spurrell, E. (2000). Impact of interpersonal and ego-related stress on restrained eaters. *The International Journal of Eating Disorders*, 27, 411–418.
- Tataranni, P. A., Larson, D. E., Snitker, S., Young, J. B., Flatt, J. P., & Ravussin, E. (1996). Effects of glucocorticoids on energy metabolism and food intake in humans. *American Journal of Physiology*, 271, E317–E325.
- Telch, C. F., & Agras, W. S. (1996). Do emotional states influence binge eating in the obese? *The International Journal of Eating Disorders*, 20, 271–279.
- Tuschen-Caffier, B., & Vogele, C. (1999). Psychological and physiological reactivity to stress: An experimental study on bulimic patients, restrained eaters, and controls. *Psychotherapy and Psychosomatics*, 68, 333–340.
- Wallis, D. J., & Hetherington, M. M. (2004). Stress and eating: The effects of ego-threat and cognitive demand on food intake in restrained and emotional eaters. *Appetite*, 43, 39–46.
- Walsh, B. T., Roose, S. P., Katz, J. L., Dyrenfurth, I., Wright, L., Vande, W. R., et al. (1987b). Hypothalamic–pituitary–adrenal–cortical activity in anorexia nervosa and bulimia. *Psychoneuroendocrinology*, 12, 131–140.
- Wilfley, D. E., Agras, W. S., Telch, C. F., Rossiter, E. M., Schneider, J. A., Cole, A. G., et al. (1993). Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: a controlled comparison. *Journal of Consulting and Clinical Psychology*, 61, 296–305.
- Wolkowitz, O. M., Epel, E. S., & Rues, V. (2001). Stress hormone-related psychopathology: pathophysiology and treatment implications. *The World Journal of Biological Psychiatry*, 2, 115–143.
- Yanovski, S. Z., Nelson, J. E., Dubbert, B. K., & Spitzer, R. L. (1993a). Association of binge eating disorder and psychiatric comorbidity in obese subjects. *The American Journal of Psychiatry*, 150, 1472–1479.
- Yanovski, S. Z., Yanovski, J. A., Gwirtsman, H. E., Bernat, A., Gold, P. W., & Chrousos, G. P. (1993b). Normal dexamethasone suppression in obese binge and nonbinge eaters with rapid weight loss. *The Journal of Clinical Endocrinology and Metabolism*, 76, 675–679.