and external environments that tax resources available to the individual. Thus, all organisms cope in some way and understanding this potentially adaptive process and how to increase the effectiveness of such efforts is of fundamental importance to all people. Future investigations that provide new perspectives on successful coping and are amenable to empirical validation are needed. The degree to which such models and investigations can provide specific information about which strategies work, in what contexts, and for whom, will determine the degree to which such research can be applied to increase positive adaptations. Several recent investigations have begun to demonstrate preliminary support for fostering successful coping; however, further investigations in the area of intervention research are warranted and could potentially have enormous impact on coping.

SEE ALSO: ▶ Emotional approach coping ▶ Global well-being ▶ Mature defense mechanisms

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Cortisol

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Cortisol is a hormone that is released from the adrenal gland. A common misconception is that the hormone cortisol is "bad for you" (i.e., always exacerbates health problems and/or causes feelings of distress). While it is true that chronically elevated cortisol levels can have deleterious effects on health, mild elevations of cortisol are beneficial in many ways. Acute cortisol elevation is adaptive insofar as it mobilizes and directs bodily energy consumption, modulates psychological processes, and restrains (or "turns off") stress-responsive systems.

Downstream Regulation of Cortisol

Several upstream hormonal regulators control the release of cortisol. Corticotropin-releasing hormone (CRH; which is sometimes called corticotrophin-releasing factor, or CRF) and arginine vasopressin (AVP) are released from the hypothalamus and control the release of adrenocorticotropin hormone (ACTH) from the pituitary gland. ACTH is released into the blood stream and controls the production and secretion of cortisol from the adrenal gland. Because the hypothalamus and the pituitary gland are the structures that most proximally control cortisol release from the adrenal, the system that regulates cortisol is called the "hypothalamic pituitary adrenal axis" (or, "HPA axis"). Recently, investigators have highlighted the important role of brain circuitry associated with emotion in the regulation of the HPA axis by coining the term "limbic hypothalamic pituitary adrenal axis" (or "LHPA axis").

Cortisol is a hormone in primates that is very similar in structure and function to the hormone corticosterone in rodents. Both cortisol and corticosterone are also called "glucocorticoids." Changes in glucocorticoid levels occur as a function of many factors, including circadian variation (i.e., in humans glucocorticoids are high in the morning and low in the evening), and in response to food intake, physical activity, injury, and psychological stress (e.g., psychosocial threat). Active tasks (such as exercise or public speaking) typically cause greater glucocorticoid elevations than passive tasks (such as watching an emotionally arousing movie). The types of psychological stressors that most potently cause glucocorticoid elevations are those that involve unpredictability or uncontrollability of a perceived threat.

Humans are particularly sensitive to social stressors. In the journal *Psychological Bulletin* in 2004, Dickerson and Kemeny published a meta-analysis of studies using laboratory based stressors in humans. They found that the types of stressors that cause the largest glucocorticoid increases are those that involve both uncontrollability and social-evaluative threat (i.e., the possibility of being negatively evaluated by others). Thus, when people are actively engaged in a task (e.g., presenting information to others), and they feel that they are not in control of others' negative evaluations of themselves, cortisol elevations will often occur. In contrast, several investigators have shown that greater perceived social support is associated with better regulation of cortisol levels.

Upstream Effects of Cortisol on the Brain and Psychological Processes

Endogenous glucocorticoids modulate activity in target tissues throughout the body and brain primarily via the two types of corticosteroid receptors, mineralocorticoid receptors (MR) and glucocorticoid receptors (GR). As mentioned above, one

function of glucocorticoids in target tissues is restraint of stress-related physiological processes, including restraint of sympathetic nervous system activation, inflammation, and their own further production and release (by suppressing release of CRH and ACTH) via negative feedback at the level of the pituitary, hypothalamus, and other brain regions (most importantly, dorsal regions of the medial prefrontal cortex [mPFC] and the hippocampus). However, not all glucocorticoid effects on the brain dampen HPA activity. In addition to negative feedback pathways, positive feedback loops exist through the amygdala and other brain regions, in which glucocorticoid elevations serve to increase brain CRH and HPA activation. Thus, positive and negative feedback circuitry operate in parallel.

The effects of glucocorticoids on many target tissues and behavioral processes follow an inverted U-shaped function in which moderate elevation of glucocorticoids enhances functioning, while extreme or prolonged glucocorticoid elevation impairs functioning. Research has associated cortisol with stress-related pathology. For instance, chronic overproduction of glucocorticoids in animals contributes to alterations (e.g., cell atrophy, cell death) in brain structures essential for regulation of the HPA axis, such as the hippocampus. However, short-term mild elevations of glucocorticoids enhance hippocampal neuronal activity. Partially related to their effects on the hippocampus, glucocorticoids affect memory. The relation between cortisol and memory follows the same inverted U-shaped function: mild glucocorticoid elevations often benefit memory consolidation, but extreme or prolonged elevations or deficiency in glucocorticoid levels impair memory formation.

With regard to effects of glucocorticoids on memory, there appear to be some very important interactive effects between emotional arousal and glucocorticoid levels. Both animal and human data show that variation in cortisol facilitates memory formation in individuals experiencing emotional arousal, but not in individuals who are not emotionally aroused at the time of learning. Research suggests that emotion-related brain circuitry (in particular, noradrenergic processes in the basolateral nucleus of the amygdala) must be activated in order for cortisol to affect memory. Thus, the hormone cortisol affects memory differentially depending on the pattern of regional neural activity and emotional arousal at the time of cortisol elevations. These data suggest that glucocorticoid elevations that occur during "non-stress" or "non-emotional" situations (such as with exercise or eating) do not facilitate the formation of memories. In a stress response with negative emotional arousal, however, cortisol *does* help memory centers of the brain (such as the amygdala and hippocampus) in laying down new memories.

The direct effects of cortisol on emotion vary. Contrary to common conceptions, many studies show that cortisol does not cause distress. Often, pharmacological manipulation of cortisol in the physiological range has no effect on self-reported emotional state. Even though physiological levels of cortisol do not have reliable effects on global measures of subjective emotional state, cortisol subtly modulates activity in brain structures related to emotion and arousal. Cortisol also has been

found to affect emotional behaviors and affective ratings of emotional and neutral stimuli. For instance, mild systemic elevations of glucocorticoids in humans and nonhumans cause reductions in physiological and behavioral indices of fear. However, corticosterone infused directly into the amygdala in animals causes anxiogenesis. Cortisol administration has also been found to heighten arousal ratings of objectively neutral stimuli. Cortisol's effects on neural circuitry related to emotion and brain excitability may cause only slight variations in affect or arousal, which may not be reliably reflected in subjective experience or detected on global assessments of affect, but may be apparent on indices that detect subtle changes in affective experience.

Dexamethasone and Prednisone

It should be noted that many of the studies of the effects of glucocorticoids on psychological processes use synthetic glucocorticoids, such as prednisone or dexamethasone. Unlike cortisol or hydrocortisone (or cortisone, which converts to cortisol), synthetic glucocorticoids do not readily cross the blood brain barrier. Furthermore, dexamethasone exhibits different binding affinities for the two types of corticosteroid receptors than cortisol. Thus, studies of the effects of prednisone and dexamethasone on psychological processes must be interpreted with extreme caution when making inferences about the psychological effects of endogenous variation in cortisol.

Cortisol and Psychopathology

The relation between cortisol elevations and psychopathology has long been studied. Cortisol hyperactivity has most reliably been found in severe, melancholic, and/or psychotic depression. In fact, preliminary evidence suggests that short-term blockade of the glucocorticoid receptor in psychotic depression may have salubrious effects. Chronic cortisol elevations have also been found in other forms of psychopathology, but less reliably than in severe depression. Cortisol hyperactivity in depression has been characterized alternatively as a trigger of depressive symptoms (i.e., HPA disturbance causes depression) or as a neuroendocrine response to psychological suffering (i.e., depression causes HPA disturbance). However, these polarized causal views are oversimplified. More likely, mechanisms associated with HPA dysregulation are intimately intertwined with the mechanisms that underlie psychological processes involved in depression.

Research has also shown associations between glucocorticoid alterations and fearful temperament, such that basal glucocorticoid levels have been found to be elevated in nonhuman primates and humans with inhibited behavior and anxiety. In addition, many studies have shown cortisol alterations in posttraumatic stress disorder (PTSD), with some studies showing chronic HPA hyperactivity and other

studies showing HPA hypoactivity with overactive HPA negative feedback in PTSD. This disparity in the PTSD literature may be partially accounted for by differences in subgroups of individuals with PTSD (e.g., combat veterans vs. interpersonal assault victims, or children vs. adults). Interesting preliminary data suggests that moderately elevated cortisol levels following a traumatic incident are protective against the development of PTSD. Several researchers have investigated using glucocorticoids as a preventative treatment for PTSD, and have found decreased incidence of PTSD after administering small doses of glucocorticoids following or during traumatic experiences.

In summary, feelings associated with social and physical threat are associated with heightened cortisol. However, acute cortisol elevation typically does not *cause* feelings of distress. Rather, stress-related cortisol elevations *result* from neural processing of perceived and actual threat, which results in stimulation of the HPA axis. Although chronically elevated cortisol levels are associated with psychopathology and poor health, mild elevations in cortisol facilitate many physiological, psychological, and behavioral processes.

SEE ALSO: ▶ Health psychology ▶ Neurobiology ▶ Social support

Counseling Psychology

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Counseling psychology is one of 11 specialties recognized by the American Psychological Associations' Commission for the Recognition of Specialties and Proficiencies in Professional Psychology (CRSPP). As described in CRSPP's archival description of counseling psychology, counseling psychology is a general practice and health service provider specialty in professional psychology. Practitioners of counseling psychology provide assessment, diagnosis, and treatment of psychopathology, while focusing on personal and interpersonal functioning across the lifespan and on emotional, social, vocational, educational, health-related, developmental and organizational concerns. While attentive to disturbances in functioning, practitioners generally focus on healthy aspects and strengths of their clients and the environmental/situational factors that affect their development and functioning.

Counseling psychology focuses both on typical or normal developmental issues and on atypical, dysfunctional, or disordered development as it applies to human experience from individual, family, group, systems, and organizational perspectives. Counseling psychology strives to help people with physical, emotional, and mental disorders to improve their well-being, alleviate distress and maladjustment, resolve crises, and increase their ability to live more highly functioning and satisfying lives.