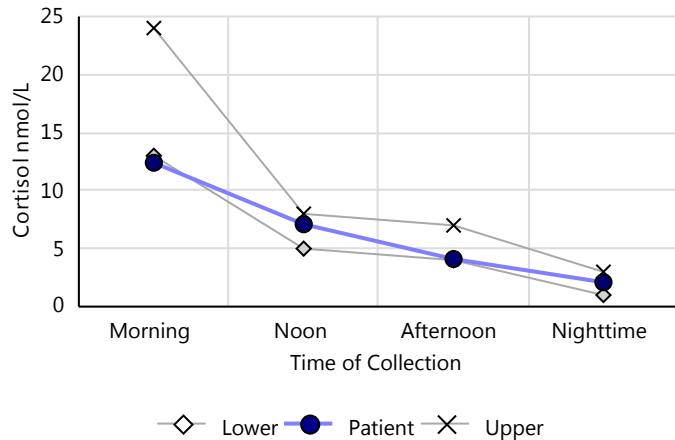


Authorizing Clinician	Patient	Gender:	Collected	Received	Reported
BioHealth Laboratory 23900 Hawthorne Blvd, Suite 150 Torrance, CA 90505	Rebecca Stein	Female	01/08/2017	01/09/2017	01/09/2017
		DOB: 11/26/1968			

HPA Stress Profile (#201A)

Cortisol Diurnal Rhythm



Cortisol and DHEA-S Results

Parameter	Result	Reference Range	Units
Cortisol - Morning	12.4	13.0 - 24.0	nmol/L
Cortisol - Noon	7.1	5.0 - 8.0	nmol/L
Cortisol - Afternoon	4.1	4.0 - 7.0	nmol/L
Cortisol - Nighttime	2.1	1.0 - 3.0	nmol/L
Cortisol - Sum	25.7	23.0 - 42.0	nmol/L
DHEA-S Morning	2.4	1.0 - 10.0	nmol/L
Cortisol:DHEA-S Ratio	5.2 : 1	**	Ratio

** A new Cortisol:DHEA-S ratio reference range is currently being determined by analysis of patient data from the newly improved test conditions.

Cortisol has one of the most distinct circadian rhythms in human physiology. This is regulated by the central clock located in the suprachiasmatic nucleus of the hypothalamus. Cortisol acts as a secondary messenger between central and peripheral clocks, hence its importance in the synchronization of body circadian rhythms. Optimal regulation of the hypothalamic-pituitary-adrenal (HPA) axis is critical for a successful response to any stressor as well as in non-stressful situations. Dysregulation of the HPA axis in basal conditions or in response to acute or chronic (including psychosocial) stress is closely related to the onset and/or progression of many diseases. The anabolic steroid, dehydroepiandrosterone sulfate (DHEA-S), is secreted from the adrenal cortex. It plays a significant role in the body as a precursor to sex steroids as well as a role in HPA axis response to stress.

The Cortisol to DHEA-S ratio provides a snapshot of the waking values of these hormones and is reported specific to the age and gender of the patient. In general, an elevated cortisol:DHEA-S ratio is indicative of progressive HPA axis dysfunction in which acute and/or chronic stressors have taken their toll on homeostasis. The cortisol:DHEA-S ratio is generally considered to be a measure of catabolic vs. anabolic activities, but it may be better described as the overall burden of glucocorticoid signaling on tissues, since DHEA acts not only as an anabolic hormone, but appears to function to down-regulate the cellular effects of cortisol. Therefore, the signaling burden of cortisol is not just a function of available free cortisol, but of the DHEA-S available as an opposing signal.