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HORMONE METABOLITES SCAN

Patient Name Sample Demo

Signal	Lvl	Current Value	6/22/26	
Hormone Metabolites Assessment -> Estrogens -> 2-Hydroxy Estradiol *2-Hydroxyestradiol is generated from estradiol by a Cytochrome P450. 2-Hydroxyestradiol binds, with a low affinity, to estrogen receptors. It inhbits catechol-O-methyltransferase (COMT) activity. Inactivity of COMT blocks inactivation of catechol hormones and catecholamine neutransmitters. 2-Hydroxyestradiol is also reported to inhibit angiongensis and tumor cell growth (PMID: 9472688)*	10		99	
Hormone Metabolites Assessment -> Enzymes -> Catechol-O-Methyl-Transferase (COMT) *The COMT gene provides instructions for making an enzyme called catechol-O-methyltransferase. Two versions of this enzyme are made from the gene. The longer form, called membrane-bound catechol-O-methyltransferase (MB-COMT), is chiefly produced by nerve cells in the brain. Other tissues, including the liver, kidneys, and blood, produce a shorter form of the enzyme called soluble catechol-O-methyltransferase (S-COMT). This form of the enzyme helps control the levels of certain hormones.* *In the brain, catechol-O-methyltransferase helps break down certain chemical messengers called neurotransmitters. These chemicals conduct signals from one nerve cell to another. Catechol-O-methyltransferase is particularly important in an area at the front of the brain called the prefrontal cortex, which organizes and coordinates information from other parts of the brain. This region is involved with personality, planning, inhibition of behaviors, abstract thinking, emotion, and working (short-term) memory. To function efficiently, the prefrontal cortex requires signaling by neurotransmitters such as dopamine and norepinephrine. Catechol-O-methyltransferase helps maintain appropriate levels of these neurotransmitters in this part of the brain.* *Catechol-O-methyltransferase is involved in the inactivation of the catecholamine neurotransmitters (dopamine, epinephrine, and norepinephrine). The enzyme introduces a methyl group to the catecholamine, which is donated by S-adenosyl*	8		99	

methionine (SAM). Any compound having a catechol structure, like catecholestrogens and catechol-containing flavonoids, are substrates of COMT. Estrogen is broken down by the COMT gene – this reaction produces a calming, anti-cancer type of estrogen called 2-OH methoxy estrogen. 2-OH methoxy estrogen is very important for health and it helps to prevent other symptoms of estrogen dominance such as PMS, heavy bleeding, fibroids, endometriosis, etc. *Even though the COMT gene breaks down estrogen, it is also epigenetically slowed down by estrogen. Meaning the more estrogen a woman or man has, the slower the COMT system will be working as cells will produce fewer copies of the enzyme. A research article from 2003 describes how hormonal therapy can be effective for dopamine-related diseases like Parkinson's Disease. Because estrogen slows COMT pathways it may be of therapeutic value in Parkinson's patients who need a more steady supply of dopamine in their brain.* *A recent review article published in 2015 shows how through environmental and epigenetic mechanisms, estrogen inhibits COMT pathways by about 30%. The researchers suggest this is why many women have a predisposition towards a phenotype of high anxiety and a lower tolerance for stress.4 So even if you aren't born with a SNP in your COMT pathway, just being female or having too much estrogen can make it feel like you do have a +/- or +/+ SNP.* *In addition to the epigenetic effects that estrogen has on COMT pathways, it also greatly impacts the MAO system. Because estrogen impacts both COMT and MAO pathways, it is capable of having a major impact on the levels of frontal-lobe dopamine and stress hormones throughout the body. Research has clarified that a woman's mood is largely a reflection of how fast or slow her MAO-A system is working.*				
Hormone Metabolites Assessment -> Estrogens -> 4-Methoxy Estradiol *COMT dependent metabolite of 4-Hydroxy Estrone.*	12		99	
Hormone Metabolites Assessment -> Enzymes -> Aromatase *Aromatase, also called estrogen synthetase or estrogen synthase, is an enzyme responsible for a key step in the biosynthesis of estrogens. It is CYP19A1, a member of the cytochrome P450 superfamily (EC 1.14.14.1), which are monooxygenases that catalyze many reactions involved in steroidogenesis. In particular, aromatase is responsible for the aromatization of androgens into estrogens. The aromatase enzyme can be found in many tissues including gonads, brain, adipose tissue, placenta, blood vessels, skin, and bone, as well as in tissue of endometriosis, uterine fibroids, breast cancer, and endometrial cancer. It is an important factor in sexual development.*	6		98	
Hormone Metabolites Assessment -> Androgens -> 5a-Dihydrotestosterone *Dihydrotestosterone (commonly abbreviated to DHT), or 5a-dihydrotestosterone (5a-DHT), also known as*	6		95	

5a-androstan-17ß-ol-3-one, is a sex steroid and androgen hormone. The enzyme 5a-reductase synthesizes DHT from testosterone in the prostate, testes, hair follicles, and adrenal glands. This enzyme reduces the 4,5 double-bond of the testosterone. Relative to testosterone, DHT is much more potent as an agonist of the androgen receptor. DHT is also known as androstanolone (INN) and stanolone (BAN), and under brand names including Anabolex, Anaprotin, Andractim, Androlone, Gelovit, Neoprol, Pesomax, and Stanaprol, is used clinically as an androgen and anabolic steroid. Unlike testosterone and some anabolic steroids, DHT cannot be aromatized, and hence, has no risk of producing estrogenic effects such as gynecomastia. *An excess of 5a-DHT contributes to hair loss, acne, and hirsutism in men and women and increased risk for BPH and prostate cancer in men (but only when associated with high estrogens). While this highly androgenic metabolite is of great interest and importance, it is largely created intracellularly and quickly metabolized to 3a-androstanediol.*				
Hormone Metabolites Assessment -> Glucocorticoids -> Tetrahydrocortisol *Metabolite of cortisol dependent on 5B-Reductase and 3a-HSD. Tetrahydrocortisol is the most powerful natural angiostatic steroid. It is involved in C21-Steroid hormone metabolism pathway (KEGG).* *Cortisol is irreversibly metabolized for disposal in the liver to dihydrocortisol and then tetrahydrocortisol (THF); there is a corresponding pathway for disposal of cortisone to form tetrahydrocortisone (THE). Liver metabolism to THF and THE is catalyzed by 5a and 5ß reductases, therefore elevations in the activity of these enzymes will increase cortisol metabolism and levels of THF and THE. Increased cortisol metabolism in turn drives increased cortisol production from the adrenal glands via the HPA axis, which stimulates ACTH (adrenocorticotrophic hormone) secretion by the pituitary gland. This has the side effect of also increasing adrenal androgen production, as seen for example in PCOS when insulin resistance results in increased 5a-reductase activity. The ratio of THF to THE reflects the tissue cortisol/cortisone ratio. A high THF/ THE ratio is seen in essential hypertension, while a low ratio is seen in insulin resistance and obesity. Cortisol metabolite levels are also affected by the increased and decreased metabolism of cortisol seen in hyper- and hypothyroidism.*	3		95	
Hormone Metabolites Assessment -> Thyroid -> Reverse Triiodothyronine rT3 *Reverse triiodothyronine (3,3’,5’-triiodothyronine, reverse T3, or rT3) is an isomer of triiodothyronine (3,5,3’ triiodothyronine, T3). It is an inactive form, and increased levels can compete with active T3 molecules for receptor binding sites and trigger hypothyroid symptoms. Reverse T3 is the third-most common iodothyronine the thyroid gland releases into the bloodstream, of which 0.9% is rT3; tetraiodothyronine (levothyroxine, T4) constitutes 90% and T3 is 9%.*	12		94	

However, 95% of rT3 in human blood is made elsewhere in the body, as enzymes remove a particular iodine atom from T4. *The production of hormone by the thyroid gland is controlled by the hypothalamus and pituitary gland. The physiological activity of thyroid hormone is regulated by a system of enzymes that activate, inactivate or simply discard the prohormone T4 and in turn functionally modify T3 and rT3. These enzymes operate under complex direction of systems including neurotransmitters, hormones, markers of metabolism and immunological signals.* *The levels of rT3 increase in conditions such as euthyroid sick syndrome, stress, diabetes, liver imbalances, chronic inflammation, etc., because its clearance decreases while its production stays the same. The decreased clearance is possibly from lower 5'-deiodinase activity in the peripheral tissue or decreased liver uptake of rT3.*				
Hormone Metabolites Assessment -> Estrogens -> Estriol *Estriol is a metabolite of estrone metabolized via 16alpha-hydroxyestrone through the enzyme 16alpha-hydroxysteroid dehydrogenase (EC 1.1.1.147) or to 2- or 4-hydroxyestrone (catechol estrogens) by the action of catecho-O-methyltransferase (EC 2.1.1.6). The latter metabolites can be formed in the brain and may compete with receptors for catecholamines. Metabolites are conjugated with sulfate or glucuronide before excretion by the kidney. During pregnancy, estriol constitutes 60-70% of the total estrogens, increasing to 300-500-fold in relation to non-pregnant women. The late term human fetus produces relatively large amounts of 16 alphahydroxy DHEA, which serves the mother as a precursor of estriol. It has been shown that 90% of the precursors for the formation of estriol are of fetal origin. If abnormal maternal serum screening results, specifically low levels of unconjugated estriol in the second trimester are detected, a diagnosis of Smith-Lemli-Opitz syndrome (SLOS),or RSH is suspected. SLOS is an autosomal recessive disorder caused by mutations of the gene encoding 7-dehydrocholesterol reductase (EC 1.3.1.21, DHCR7). (PMID: 16202579 , 16112271 , 16097001).*	2		94	
Hormone Metabolites Assessment -> Androgens -> DHEA *Dehydroepiandrosterone (DHEA) is a natural steroid hormone produced from cholesterol by the adrenal glands. DHEA is also produced in the gonads, adipose tissue and the brain. DHEA is structurally similar to, and is a precursor of, androstenedione, testosterone, estradiol, estrone and estrogen. It is the most abundant hormone in the human body. Most of DHEA is sulfated (dehydroepiandrosterone sulfate- DEHAS) before secretion. DHEAS is the sulfated version of DHEA; - this conversion is reversibly catalyzed by sulfotransferase (SULT2A1) primarily in the adrenals, the liver, and small intestines. In blood, most DHEA is found as DHEAS with levels that are about 300 times higher than free DHEA.*	9		93	
Hormone Metabolites Assessment -> Glucocorticoids -> Tetrahydrocortisone	3		93	

Metabolite of cortisone dependent on 5B-Reductase and 3a-HSD. *Cortisol is irreversibly metabolized for disposal in the liver to dihydrocortisol and then tetrahydrocortisol (THF); there is a corresponding pathway for disposal of cortisone to form tetrahydrocortisone (THE). Liver metabolism to THF and THE is catalyzed by 5a and 5ß reductases, therefore elevations in the activity of these enzymes will increase cortisol metabolism and levels of THF and THE. Increased cortisol metabolism in turn drives increased cortisol production from the adrenal glands via the HPA axis, which stimulates ACTH (adrenocorticotrophic hormone) secretion by the pituitary gland. This has the side effect of also increasing adrenal androgen production, as seen for example in PCOS when insulin resistance results in increased 5a-reductase activity. The ratio of THF to THE reflects the tissue cortisol/cortisone ratio. A high THF/ THE ratio is seen in essential hypertension, while a low ratio is seen in insulin resistance and obesity. Cortisol metabolite levels are also affected by the increased and decreased metabolism of cortisol seen in hyper- and hypothyroidism.*				
Hormone Metabolites Assessment -> Enzymes -> 17a-Hydroxylase (17a-OH) *Cytochrome P450 17A1, also called steroid 17-alpha-monooxygenase, 17a-hydroxylase, 17,20 lyase, or 17,20 desmolase, is an enzyme of the hydroxylase type that in humans is encoded by the CYP17A1 gene. It is found in the zona reticularis of the adrenal cortex and zona fasciculata as well as gonadal tissues. This gene encodes a member of the cytochrome P450 superfamily of enzymes. The cytochrome P450 proteins are generally regarded as monooxygenases that catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids, and other lipids, including the remarkable carbon-carbon bond scission catalyzed by this enzyme. This protein localizes to the endoplasmic reticulum. It has both 17alpha-hydroxylase and 17,20-lyase activities, and is a key enzyme in the steroidogenic pathway that produces progestins, mineralocorticoids, glucocorticoids, androgens, and estrogens.* *More specifically, CYP17A1 acts upon pregnenolone and progesterone to add a hydroxyl (-OH) group at carbon 17 of the steroid D ring (the hydroxylase activity), or acts upon 17-hydroxyprogesterone and 17-hydroxypregnenolone to split the side-chain off the steroid nucleus (the lyase activity).*	6		88	
Hormone Metabolites Assessment -> Glucocorticoids -> Free Cortisol *Cortisol is a glucocorticoid secreted by the adrenal cortex after adrenocortico-tropic hormone (ACTH) stimulation.* *More than 90% of circulating cortisol is bound to cortisol-binding globulin (CBG or transcortin). CBG levels are increased in pregnancy, estrogen therapy, and oral contraceptives, and*	1		85	

decreased in nephrotic syndrome, starvation, and chronic liver disease. Thus, total cortisol levels can be affected by these conditions. Free cortisol, however, is the physiologically active form. Free cortisol levels change independently and accurately reflect the clinical status; thus free cortisol is the most reliable test for glucocorticoid status. Cortisol is reversibly metabolized to cortisone in tissues. Relative amounts of cortisol and cortisone are determined by the relative activity of 11ß-hydroxysteroid dehydrogenases (11ßHSD): 11ßHSD type 1 converts inactive cortisone to cortisol, while 11ßHSD type 2 converts cortisol to cortisone, effectively suppressing cortisol activity. 11ßHSD1 predominates in liver, adipose tissue, gonads, brain, vascular smooth muscle, and skeletal muscle; 11ßHSD2 predominates in the kidney, colon, GI tract, and salivary glands. Cortisol is irreversibly metabolized for disposal in the liver to dihydrocortisol and then tetrahydrocortisol (THF); there is a corresponding pathway for disposal of cortisone to form tetrahydrocortisone (THE). Liver metabolism to THF and THE is catalyzed by 5a and 5ß reductases, therefore elevations in the activity of these enzymes will increase cortisol metabolism and levels of THF and THE. Increased cortisol metabolism in turn drives increased cortisol production from the adrenal glands via the HPA axis, which stimulates ACTH (adrenocorticotrophic hormone) secretion by the pituitary gland. This has the side effect of also increasing adrenal androgen production, as seen for example in PCOS when insulin resistance results in increased 5a-reductase activity. The ratio of THF to THE reflects the tissue cortisol/cortisone ratio. A high THF/ THE ratio is seen in essential hypertension, while a low ratio is seen in insulin resistance and obesity. Cortisol metabolite levels are also affected by the increased and decreased metabolism of cortisol seen in hyper- and hypothyroidism.				
Hormone Metabolites Assessment -> Enzymes -> 5B-Reductase *The human enzyme efficiently acts on progesterone, androstenedione, 17alpha-hydroxyprogesterone and testosterone to 5beta-reduced metabolites. It can also act on aldosterone, corticosterone and cortisol, to some extent.*	6		82	
Hormone Metabolites Assessment -> Estrogens -> 4-Hydroxy Estrone *4-Hydroxyestrone is metabolite originating from 17beta-estradiol (testosterone metabolite via aromatase or estrone metabolite via 17B-HSD pathway), dependent CYP1B1 activity.*	5		77	
Hormone Metabolites Assessment -> Enzymes -> Membrane Bound Catechol-O-Methyl-Transferase (MB-COMT)	3		75	

Hormone Metabolites Assessment -> Progestogens -> 20a-Dihydroprogesterone *20a-Dihydroprogesterone, or 20a-hydroxyprogesterone, is a naturally-occurring, endogenous progestogen. It is a metabolite of progesterone, converted by the 20a-hydroxysteroid dehydrogenases AKR1C1 and AKR1C3, and although still active as a progestogen, is much less potent in comparison.*	12		72	
Hormone Metabolites Assessment -> Glucocorticoids -> Total Cortisol *Cortisol is a corticosteroid hormone produced by the adrenal cortex that is involved in the response to stress; The main glucocorticoid secreted by the adrenal cortex.* *Cortisol is synthesized from pregnenolone. The amount of cortisol present in the serum undergoes diurnal variation, with the highest levels present in the early morning, and lower levels in the evening, several hours after the onset of sleep. Cortisol is a corticosteroid hormone produced by the adrenal cortex that is involved in the response to stress; it increases blood pressure, blood sugar levels, may cause infertility in women, and suppresses the immune system. Synthetic cortisol, also known as hydrocortisone, is used as a drug mainly to fight allergies and inflammation; it increases blood pressure, blood sugar levels, may cause infertility in women, and suppresses the immune system. Synthetic cortisol, also known as hydrocortisone, is used as a drug mainly to fight allergies and inflammation.*	12		70	
Hormone Metabolites Assessment -> Estrogens -> Estradiol *Estradiol is the most potent form of mammalian estrogenic steroids. Estradiol is produced in the ovaries. The ovary requires both luteinizing hormone (LH) and follicle-stimulating hormone (FSH) to produce sex steroids. LH stimulates the cells surrounding the follicle to produce progesterone and androgens. The androgens diffuse across the basement membrane to the granulosa cell layer, where, under the action of FSH, they are aromatized to estrogens, mainly estradiol. The ovary shows cyclical activity, unlike the testis that is maintained in a more or less constant state of activity. Hormone secretions vary according to the phase of the menstrual cycle. In the developing follicle LH receptors (LH-R) are only located on the thecal cells and FSH receptors (FSHR) on the granulosa cells. The dominant pre-ovulatory follicle develops LH-Rs on the granulosa cells prior to the LH surge. Thecal cells of the preovulatory follicle also develop the capacity to synthesize estradiol and this persists when the thecal cells become incorporated into the corpus luteum. After ovulation, the empty follicle is remodelled and plays an important role in the second half or luteal phase of the menstrual cycle. This phase is dominated by progesterone and, to a lesser extent, estradiol secretion by the corpus luteum. estradiol is also synthesized locally from cholesterol through testosterone in the hippocampus*	3		68	

and acts rapidly to modulate neuronal synaptic plasticity. Localization of estrogen receptor alpha (ERalpha) in spines in addition to nuclei of principal neurons implies that synaptic ERalpha is responsible for rapid modulation of synaptic plasticity by endogenous estradiol. estradiol is a potent endogenous antioxidant which suppresses hepatic fibrosis in animal models, and attenuates induction of redox sensitive transcription factors, hepatocyte apoptosis and hepatic stellate cells activation by inhibiting a generation of reactive oxygen species in primary cultures. This suggests that the greater progression of hepatic fibrosis and hepatocellular carcinoma in men and postmenopausal women may be due, at least in part, to lower production of estradiol and a reduced response to the action of estradiol. estradiol has been reported to induce the production of interferon (INF)-gamma in lymphocytes, and augments an antigen-specific primary antibody response in human peripheral blood mononuclear cells. IFN-gamma is a potent cytokine with immunomodulatory and antiproliferative properties. Therefore, female subjects, particularly before menopause, may produce antibodies against hepatitis B virus e antigen and hepatitis B virus surface antigen at a higher frequency than males with chronic hepatitis B virus infection. The estradiol-Dihydrotestosterone model of prostate cancer (PC) proposes that the first step in the development of most PC and breast cancer (BC) occurs when aromatase converts testosterone to estradiol. (PMID: 17708600 , 17678531 , 17644764).				
Hormone Metabolites Assessment -> Androgens -> Androstenedione *DHEA metabolite dependent on 3B-HSD. A weak androgen and an intermediate in the biosynthesis of testosterone from DHEA.* *Androstenedione is metabolized to estrone and testosterone is metabolized to estradiol via the enzyme aromatase. In men, high aromatase activity can contribute to gynecomastia and testosterone deficiency symptoms. In women, aromatase has been implicated in the etiology of breast cancer.*	8		64	
Hormone Metabolites Assessment -> Estrogens -> Estrone *Estrone is a major mammalian estrogen. The conversion of the natural C19 steroids, testosterone and androstenedione into estrone is dependent on a complex key reaction catalyzed by the cytochrome P450 aromatase (EC 1.14.14.1, unspecific monooxygenase), which is expressed in many tissues of the adult human (e.g. ovary, fat tissue), but not in the liver. The ovaries after menopause continue to produce androstenedione and testosterone in significant amounts and these androgens are converted in fat, muscle, and skin into estrone. 17beta-hydroxysteroid dehydrogenases (EC 1.1.1.62, 17-HSDs) are enzymes involved in the formation of active sex steroids. estrone is interconverted by two enzymes 17-HSD types. Type 1 converts estrone to estradiol and Type 2 catalyzes the reverse reaction.*	6		63	

Hormone Metabolites Assessment -> Enzymes -> 20a-Hydroxysteroid dehydrogenase (20a-HSD) *The 3 substrates of this enzyme are 17alpha,20alpha-dihydroxypregn-4-en-3-one, NAD+, and NADP+, whereas its 4 products are 17-alpha-hydroxyprogesterone, NADH, NADPH, and H+.* *This enzyme belongs to the family of oxidoreductases, specifically those acting on the CH-OH group of donor with NAD+ or NADP+ as acceptor. The systematic name of this enzyme class is 20alpha-hydroxysteroid:NAD(P)+ 20-oxidoreductase. Other names in common use include 20alpha-hydroxy steroid dehydrogenase, 20alpha-hydroxy steroid dehydrogenase, 20alpha-HSD, and 20alpha-HSDH. This enzyme participates in c21-steroid hormone metabolism.*	3		62	
Hormone Metabolites Assessment -> Androgens -> Androstenendiol *DHEA metabolite dependent on 17B-Hydroxysteroid Dehydrogenase (17B-HSD). A weak androgen, an estrogen, and an intermediate in the biosynthesis of testosterone from DHEA.*	8		60	
Hormone Metabolites Assessment -> Progestogens -> Progesterone *In women the primary source of progesterone is ovarian. During the luteal phase of the menstrual cycle, progesterone is synthesized in the ovaries in large amounts and released into the bloodstream where it reaches target tissues throughout the body. Progesterone has a relatively short half-life in the bloodstream and is metabolized in the liver by various phase I and II enzymes. These metabolites are excreted by the kidneys into urine. Very little progesterone itself enters urine, whereas progesterone metabolites are readily excreted in urine. One of the primary progesterone metabolites is a glucuronide conjugate of pregnanediol. Levels of this progesterone metabolite are very similar to active levels of progesterone in the bloodstream, making pregnanediol a convenient surrogate marker of progesterone synthesis. About half of progesterone is metabolized in the liver (hepatic metabolism) and the other half in other organs or tissues (extrahepatically). Hepatic metabolism is usually about 50% by 5a-reductase and 50% by 5ß-reductase. In total 60-70% follows the 5a pathway and only 25% the 5ß pathway. The 5a pathway creates allopregnanolone, which enters the brain and binds to GABA receptors and has a calming, sleep-inducing action6. Progesterone used as a supplement raises both circulating and urine levels of progesterone and its metabolites. Topical progesterone has little effect on urinary levels of progesterone metabolites since little of it enters the liver or kidneys. Oral progesterone, on the other hand, passes through the liver where it is extensively converted to metabolites that are excreted by the kidneys into urine.*	10		60	
Hormone Metabolites Assessment -> Thyroid -> Triiodothyronine T3 *T3 is the true hormone. Its effects on target tissues are roughly four times more potent than*	8		59	

those of T4. Of the thyroid hormone that is produced, just about 20% is T3, whereas 80% is produced as T4. Roughly 85% of the circulating T3 is later formed in the liver and pituitary by removal of the iodine atom from the carbon atom number five of the outer ring of T4. In any case, the concentration of T3 in the human blood plasma is about one-fortieth that of T4. This is observed in fact because of the short half-life of T3, which is only 2.5 days. This compares with the half-life of T4, which is about 6.5 days. *The thyroid hormones, triiodothyronine (T3) and its prohormone, thyroxine (T4), are tyrosine-based hormones produced by the thyroid gland that are primarily responsible for regulation of metabolism. T3 and T4 are partially composed of iodine (see molecular model). A deficiency of iodine leads to decreased production of T3 and T4, enlarges the thyroid tissue and will cause the disease known as simple goitre. The major form of thyroid hormone in the blood is thyroxine (T4), which has a longer half-life than T3. In humans, the ratio of T4 to T3 released into the blood is between 14:1 and 20:1. T4 is converted to the active T3 (three to four times more potent than T4) within cells by deiodinases (5'-iodinase). These are further processed by decarboxylation and deiodination to produce iodothyronamine (T1a) and thyronamine (T0a). All three isoforms of the deiodinases are selenium-containing enzymes, thus dietary selenium is essential for T3 production.* *Thyroid hormones (T4 and T3) are produced by the follicular cells of the thyroid gland and are regulated by TSH made by the thyrotropes of the anterior pituitary gland. The effects of T4 in vivo are mediated via T3 (T4 is converted to T3 in target tissues). T3 is 3- to 5- fold more active than T4.*				
Hormone Metabolites Assessment -> Enzymes -> 21-Hydroxylase (21-OH) *Steroid 21-hydroxylase is a cytochrome P450 enzyme that is involved with the biosynthesis of the steroid hormones aldosterone and cortisol.* *In humans, 21-Hydroxylase is encoded by the gene CYP21A2.*	1		58	
Hormone Metabolites Assessment -> Estrogens -> 4-Methoxy Estrone *COMT dependent metabolite of 4-Hydroxy Estrone.*	12		54	
Hormone Metabolites Assessment -> Estrogens -> 2-Hydroxy Estrone *Estrone (also oestrone) is an estrogenic hormone secreted by the ovary. Its molecular formula is C18H22O2. Estrone is one of the three estrogens, which also include estriol and estradiol. Estrone is the least prevalent of the three hormones, estradiol being prevalent almost always in a female body, estriol being prevalent primarily during pregnancy. Estrone sulfate is relevant to health and disease due to its conversion to estrone sulfate, a long-lived derivative of estrone. Estrone sulfate acts as a pool of estrone which can be converted as needed to the more active estradiol.*	9		53	

Hormone Metabolites Assessment -> Androgens -> Testosterone *Testosterone is a steroid hormone from the androgen group and is found in humans and other vertebrates. In humans and other mammals, testosterone is secreted primarily by the testicles of males and, to a lesser extent, the ovaries of females. Small amounts are also secreted by the adrenal glands. It is the principal male sex hormone and an anabolic steroid.* *In men, testosterone plays a key role in the development of male reproductive tissues such as the testis and prostate, as well as promoting secondary sexual characteristics such as increased muscle and bone mass, and the growth of body hair. In addition, testosterone is essential for health and well-being, and for the prevention of osteoporosis.* *Testosterone is the most important androgen in potency and quantity. Testosterone is synthesized and released by the Leydig cells that lie between the tubules and comprise less than 5% of the total testicular volume. testosterone diffuses into the seminiferous tubules where it is essential for maintaining spermatogenesis. Some binds to an androgen-binding protein (ABP) that is produced by the Sertoli cells and is homologous to the sex-hormone binding globulin that transports testosterone in the general circulation. The ABP carries testosterone in the testicular fluid where it maintains the activity of the accessory sex glands and may also help to retain testosterone within the tubule and bind excess free hormone. Some testosterone is converted to estradiol by Sertoli cell-derived aromatase enzyme. Leydig cell steroidogenesis is controlled primarily by luteinizing hormone with negative feedback of testosterone on the hypothalamic-pituitary axis. The requirement of spermatogenesis for high local concentrations of testosterone means that loss of androgen production is likely to be accompanied by loss of spermatogenesis. Indeed, if testicular androgen production is inhibited by the administration of exogenous androgens then spermatogenesis ceases. This is the basis of using exogenous testosterone as a male contraceptive. testosterone is converted to dihydrotestosterone by 5a-reductase type 2 (EC 1.3.1.22, SRD5A2), the androgen with the highest affinity for the androgen receptor.*	6		53	
Hormone Metabolites Assessment -> Progestogens -> Corticosterone *In many species, including amphibians, reptiles, rodents and birds, corticosterone is a main glucocorticoid, involved in regulation of energy, immune reactions, and stress responses.* *However, in humans, cortisol is the primary glucocorticoid that is produced primarily in the zona fasciculata of the adrenal cortex.* *Corticosterone has only weak glucocorticoid and mineralocorticoid potencies in humans and is important mainly as an intermediate in the steroidogenic pathway from pregnenolone to aldosterone. Corticosterone is converted to*	2		52	

aldosterone by aldosterone synthase, found only in the mitochondria of glomerulosa cells. Glomerulosa cells are found in the Zona glomerulosa, which is the most superficial region of endocrine cells in the adrenal cortex. *Corticosterone is the precursor molecule to the mineralocorticoid aldosterone, one of the major homeostatic modulators of sodium and potassium levels in vivo.*				
Hormone Metabolites Assessment -> Glucocorticoids -> Total Cortisone *Metabolite of the active cortisol dependent on 11B-HSD. Cortisone is a naturally occurring glucocorticoid. It has been used in replacement therapy for adrenal insufficiency and as an anti-inflammatory agent. Cortisone itself is inactive. It is converted in the liver to the active metabolite cortisol/hydrocortisone.*	4		44	
Hormone Metabolites Assessment -> Androgens -> 5-Androstenediol *5-Androstenediol is a direct metabolite of the most abundant steroid produced by the human adrenal cortex, dehydroepiandrosterone (DHEA). 5-Androstenediol is less androgenic than 4-androstenediol, and stimulates the immune system. When administered to rats in vivo, 5-androstenediol has approximately 1/70 the androgenicity of DHEA, 1/185 the androgenicity of androstenedione, and 1/475 the androgenicity of testosterone (Wikipedia). An intermediate in testosterone biosynthesis, found in the testis or the adrenal glands. 5-Androstenediol, derived from dehydroepiandrosterone by the reduction of the 17-keto group (17-hydroxysteroid dehydrogenases), is converted to testosterone by the oxidation of the 3-beta hydroxyl group to a 3-keto group (3-fydroxysteroid dehydrogenase).*	8		42	
Hormone Metabolites Assessment -> Estrogens -> 16a-Hydroxy Estrone *Estrone metabolite.*	7		42	
Hormone Metabolites Assessment -> Androgens -> 5a, 3a-Androstanediol *Metabolite of 5a-DHT, dependent on 3a-Hydroxysteroid dehydrogenase.* *3a-Androstanediol (often abbreviated as 3a-diol), also known as 5a-androstane-3a,17ß-diol, is an endogenous inhibitory androstane neurosteroid and weak androgen, and a major metabolite of dihydrotestosterone (DHT).As a neurosteroid, it acts as a potent positive allosteric modulator of the GABAA receptor, and has been found to have rewarding, anxiolytic,pro-sexual, and anticonvulsant effects. As androgens such as testosterone and DHT are known to have many of the same effects as 3a-diol and are converted into it in vivo, it is thought that this compound may in part be responsible for said effects.*	11		41	
Hormone Metabolites Assessment -> Enzymes -> 5a-Reductase *5a-reductases, also known as 3-oxo-5a-steroid 4-dehydrogenases, are enzymes involved in steroid metabolism. They participate in 3 metabolic pathways: bile acid biosynthesis, androgen and*	7		40	

estrogen metabolism, and prostate cancer. There are three isoenzymes of 5-alpha reductase, which vary in different tissues with age. *pecific substrates include testosterone, progesterone, androstenedione, epi-testosterone, cortisol, aldosterone, and deoxycorticosterone. Outside of dihydrotestosterone, much of the physiological role of 5a-reduced steroids is unknown. Beyond reducing testosterone to dihydrotestosterone, 5alpha-reductase enzyme isoforms I and II reduce progesterone to dihydroprogesterone (DHP) and deoxycorticosterone to dihydrodeoxycorticosterone (DHDOC).*				
Hormone Metabolites Assessment -> Progestogens -> Pregnanediol *Pregnanediol is an inactive metabolic product of progesterone. It can be detected in adults and newborns urine in variable concentrations. Pregnanediol is abnormally elevated in patients with cytochrome P450 (P450C17, steroid 17alpha-monooxygenase, EC 1.14.99.9) oxidoreductase deficiency (Antley-Bixler syndrome, PORD, OMIM 201750).* *Pregnanediol is one of the most important markers of pregnenolone administration, which can potentially be abused by athletes to maintain an equilibration of the steroidal environment after sex steroids administrations. Patients with recurrent vulvovaginal candidiasis have significantly lower levels of urinary pregnanediol.*	9		39	
Hormone Metabolites Assessment -> Glucocorticoids -> Free Cortisone *A naturally occurring corticosteroid, C21H28O5, that is converted in the body to cortisol. It is used in synthetic form as a drug, especially to treat adrenal insufficiency, certain allergies, and inflammation, as from rheumatoid arthritis.*	4		38	
Hormone Metabolites Assessment -> Thyroid -> Thyroglobulin *Thyroglobulin (Tg) is a 660 kDa, dimeric protein produced by the follicular cells of the thyroid and used entirely within the thyroid gland. Thyroglobulin protein accounts for approximately half of the protein content of the thyroid gland. The protein is a precursor of the thyroid hormones; these are produced when thyroglobulin's tyrosine residues are combined with iodine and the protein is subsequently cleaved. Each thyroglobulin molecule contains approximately 100-120 tyrosine residues, but only a small number (20) of these are subject to iodination by thyroperoxidase in the follicular colloid. Therefore, each Tg molecule forms only approximately 10 thyroid hormone molecules.*	12		37	
Hormone Metabolites Assessment -> Androgens -> DHEAS *Dehydroepiandrosterone (DHEA) is a natural steroid hormone produced from cholesterol by the adrenal glands. DHEA is also produced in the gonads, adipose tissue and the brain. DHEA is structurally similar to, and is a precursor of, androstenedione, testosterone, estradiol, estrone and estrogen. It*	1		37	

is the most abundant hormone in the human body. Most of DHEA is sulfated (dehydroepiandrosterone sulfate- DEHAS) before secretion. DHEAS is the sulfated version of DHEA; - this conversion is reversibly catalyzed by sulfotransferase (SULT2A1) primarily in the adrenals, the liver, and small intestines. In blood, most DHEA is found as DHEAS with levels that are about 300 times higher than free DHEA.				
Hormone Metabolites Assessment -> Androgens -> Epitestosterone *Epitestosterone is an endogenous antiandrogen steroid, an epimer of the hormone testosterone. It is a weak competitive antagonist of the androgen receptor (AR) and a potent 5a-reductase inhibitor. Structurally, it differs from testosterone only in the configuration at the OH-bearing carbon, C17. The 17-alpha isomer of testosterone, derived from pregnenolone via the delta5-steroid pathway, and via 5-androstene-3-beta,17-alpha-diol. Epitestosterone acts as an antiandrogen in various target tissues. The ratio between testosterone/epitestosterone is used to monitor anabolic drug abuse.* *Epi-T and testosterone are produced in near equal amounts from DHEA/ androstenedione; however, Epi-T is metabolically inert. Epi-T is well known in the context of sports doping. While typically found in a 1:1 ratio with testosterone, individuals taking exogenous testosterone have a high T/Epi-T ratio, which usually rises above 6. The T/Epi-T ratio can also be helpful in interpreting cases with testosterone supplementation.*	3		36	
Hormone Metabolites Assessment -> Progestogens -> Deoxycorticosterone *Deoxycorticosterone is a steroid or mineralocorticoid secreted by the zona fasiculata of the adrenal cortex. Deoxycorticsterone acts as a precursor to aldosterone. Deoxycorticosterone is not a major secretory hormone. It is produced from progesterone by 21beta-hydroxylase and is converted to corticosterone by 11beta-hydroxylase. Corticosterone is then converted to aldosterone by aldosterone synthase. Deoxycorticosterone stimulates the collecting tubules in the kidney to continue to excrete potassium in much the same way that aldosterone does. Deoxycorticosterone has about 1/20 of the sodium retaining power of aldosterone and about 1/5 the potassium excreting power of aldosterone (Wikipedia).* *Deoxycorticosterone can be used to treat adrenal insufficiency. In particular, desoxycorticosterone acetate (DOCA) is used as replacement therapy in Addison's disease.*	12		35	
Hormone Metabolites Assessment -> Thyroid -> Thyroxine T4 *Thyroxine (3,5,3',5'-tetraiodothyronine) is produced by follicular cells of the thyroid gland. It is produced as the precursor thyroglobulin (this is not the same as TBG), which is cleaved by enzymes to produce active T4.* *The thyroid hormones, triiodothyronine (T3) and its prohormone, thyroxine (T4), are tyrosine-based hormones produced by the thyroid gland that are*	11		35	

primarily responsible for regulation of metabolism. T3 and T4 are partially composed of iodine (see molecular model). A deficiency of iodine leads to decreased production of T3 and T4, enlarges the thyroid tissue and will cause the disease known as simple goitre. The major form of thyroid hormone in the blood is thyroxine (T4), which has a longer half-life than T3. In humans, the ratio of T4 to T3 released into the blood is between 14:1 and 20:1. T4 is converted to the active T3 (three to four times more potent than T4) within cells by deiodinases (5'-iodinase). These are further processed by decarboxylation and deiodination to produce iodothyronamine (T1a) and thyronamine (T0a). All three isoforms of the deiodinases are selenium-containing enzymes, thus dietary selenium is essential for T3 production. *Thyroid hormones (T4 and T3) are produced by the follicular cells of the thyroid gland and are regulated by TSH made by the thyrotropes of the anterior pituitary gland. The effects of T4 in vivo are mediated via T3 (T4 is converted to T3 in target tissues). T3 is 3- to 5- fold more active than T4.*				
Hormone Metabolites Assessment -> Glucocorticoids -> Free Cortisol 12pm *The level of cortisol normally rises and falls in a 'diurnal variation' pattern. It peaks early in the morning, then declines throughout the day, reaching its lowest level about midnight. This pattern can change when a person works irregular shifts (such as the night shift) and sleeps at different times of the day, and it can become disrupted when an imbalance or condition either limits or stimulates cortisol production.*	12		32	
Hormone Metabolites Assessment -> Enzymes -> 3a-Hydroxysteroid dehydrogenase (3a-HSD) *3a-hydroxysteroid dehydrogenase (3a-HSD), also known as aldo-keto reductase family 1 member C4, is an enzyme that in humans is encoded by the AKR1C4 gene. It is known to be necessary for the synthesis of the endogenous neurosteroids allopregnanolone, THDOC, and 3a-androstanediol. It is also known to catalyze the reversible conversion of 3a-androstanediol (5a-androstane-3a,17ß-diol) to dihydrotestosterone (DHT) (5a-androstan-17ß-ol-3-one) and vice versa.*	9		27	
Hormone Metabolites Assessment -> Androgens -> 4-Androstenediol *4-Androstenediol is an androstenediol that is converted to testosterone. The conversion rate is about 15.76%, almost triple that of 4-androstenedione, due to utilization of a different enzymatic pathway. There is also some conversion into estrogen, since testosterone is the metabolic precursor of the estrogens.* *4-Androstenediol is a metabolite of testosterone. Conversely, the conversion of 4-Androstenediol to testosterone has been demonstrated to occur in homogenates of hyperplastic human female adrenal glands. 4-Androstenediol is an anabolic agent that has been found in increased concentration in athletes suspected of doping. 4-Androstenediol has also been found in aqueous and solid nutritional*	10		25	

supplements that are commercially available. Studies showing that non-hormonal supplements such as vitamins, minerals and amino acids can contain anabolic androgenic steroids not declared on the labels of the products have been published. These undeclared substances (often prohormones of testosterone) can cause health risks to consumers and might lead to positive results in sports doping control. It has been demonstrated that 4-Androstenediol taken by month is capable of producing in vivo increases in testosterone concentration in apparently healthy young men an women. (PMID: 15808000 , 15103700 , 10638382 , 15370836).				
Hormone Metabolites Assessment -> Glucocorticoids -> Free Cortisol 6pm	11		23	
Hormone Metabolites Assessment -> Estrogens -> 2-Methoxy Estradiol *Natural metabolite of estradiol. 2-Methoxyestradiol (2ME2) is a drug that has been developed (Panzem) that prevents the formation of new blood vessels that tumors need in order to grow (angiogenesis). It is derived from estradiol, although it binds poorly to known estrogen receptors, and belongs to the family of drugs called angiogenesis inhibitors. It has undergone Phase 1 clinical trials against breast cancers (induces apoptosis). Preclinical models also suggest that 2ME2 could also be effective against inflammatory diseases such as rheumatoid arthritis. The CAS name for 2ME2 is (17 beta)-2-methoxyestra-1,3,5(10)-triene-3,17-diol. It also acts as a vasodilator.*	7		22	
Hormone Metabolites Assessment -> Enzymes -> 17a-Hydroxysteroid dehydrogenase (17a-HSD) *Involved in conversion of androstenedione to epitestosterone.*	6		22	
Hormone Metabolites Assessment -> Progestogens -> Allopregnanediol *Metabolite of Allopregnanolone, dependent on 20a-Hydroxysteroid Dehydrogenase (20a-HSD).*	1		21	
Hormone Metabolites Assessment -> Enzymes -> 17B-Hydroxysteroid dehydrogenase (17B-HSD) *17ß-Hydroxysteroid dehydrogenases are a group of alcohol oxidoreductases which catalyse the dehydrogenation of 17-hydroxysteroids in steroidogenesis. This includes interconversion of DHEA and androstenediol, androstenedione and testosterone, and estrone and estradiol, respectively.*	10		20	
Hormone Metabolites Assessment -> Glucocorticoids -> Free Cortisol 12am *The level of cortisol normally rises and falls in a 'diurnal variation' pattern. It peaks early in the morning, then declines throughout the day, reaching its lowest level about midnight. This pattern can change when a person works irregular shifts (such as the night shift) and sleeps at different times of the day, and it can become disrupted when an imbalance or condition either limits or stimulates cortisol production.*	8		19	
Hormone Metabolites Assessment -> Glucocorticoids -> Free Cortisol 6am	8		18	

The level of cortisol normally rises and falls in a 'diurnal variation' pattern. It peaks early in the morning, then declines throughout the day, reaching its lowest level about midnight. This pattern can change when a person works irregular shifts (such as the night shift) and sleeps at different times of the day, and it can become disrupted when an imbalance or condition either limits or stimulates cortisol production.				
Hormone Metabolites Assessment -> Enzymes -> 11B-Hydroxylase (11B-OH) *Steroid 11ß-hydroxylase is a steroid hydroxylase found in the zona glomerulosa and zona fasciculata. Named officially the cytochrome P450 11B1, mitochondrial, it is a protein that in humans is encoded by the CYP11B1 gene.* *This gene encodes a member of the cytochrome P450 superfamily of enzymes. The cytochrome P450 proteins are monooxygenases that catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids and other lipids. This protein localizes to the mitochondrial inner membrane and is involved in the conversion of 11-deoxycortisol to cortisol in the adrenal cortex. Transcript variants encoding different isoforms have been noted for this gene.*	6		16	
Hormone Metabolites Assessment -> Androgens -> Etiocholanolone *Etiocholanolone is the 5-beta-reduced isomer of androsterone. Etiocholanolone is a major metabolite of testosterone and androstenedione in many mammalian species including humans. It is excreted in the urine and is androgenically inactive. Classified a ketosteroid, it causes fever (it is a pyrogen), immunostimulation and leukocytosis. The pyrogenic effect of Etiocholanolone has been shown to be due to the release of interleukin-1 (IL-1) from the leukocytes that are mobilized in response to its production or injection. Etiocholanolone has anticonvulsant activity and may be an endogenous modulator of seizure susceptibility. Significantly increased values of etiocholanolone (along with testoterone and androsterone) an be detected in the urine of men with androgenic alopecia (male pattern baldness).* *Etiocholanolone, 3a-hydroxy-5ß-androstan-17-one, is an endogenous 17-ketosteroid that is produced from the metabolism of testosterone. It causes fever, immunostimulation, and leukocytosis, and is used to evaluate adrenal cortex function, bone marrow performance, and in neoplastic disease to stimulate the immune system. Etiocholanolone is also known to be an inhibitory androstane neurosteroid, acting as a positive allosteric modulator of the GABAA receptor, and possesses anticonvulsant effects. The unnatural enantiomer of etiocholanolone is more potent as a positive allosteric modulator of GABAA receptors and as an anticonvulsant than the natural form.*	12		15	
Hormone Metabolites Assessment -> Enzymes -> 3B-Hydroxysteroid dehydrogenase (3B-HSD) *3ß-Hydroxysteroid dehydrogenase/?5-4 isomerase (3ß-HSD) (EC 1.1.1.145) is an enzyme that catalyzes the*	4		13	

biosynthesis of progesterone from pregnenolone, 17a-hydroxyprogesterone from 17a-hydroxypregnenolone, and androstenedione from dehydroepiandrosterone (DHEA) in the adrenal gland. It is the only enzyme in the adrenal pathway of corticosteroid synthesis that is not a member of the cytochrome P450 family. In humans, there are two 3ß-HSD isozymes encoded by the HSD3B1 and HSD3B2 genes. *3ß-HSD is also known as delta 5-4-isomerase, which catalyzes the oxidative conversion of 5-3-ß-hydroxysteroids to the 4-3-keto configuration and is, therefore, essential for the biosynthesis of all classes of hormonal steroids, namely progesterone, glucocorticoids, mineralocorticoids, androgens, and estrogens.* *The 3ß-HSD complex is responsible for the conversion of:* *Pregnenolone to progesterone* *17a-Hydroxypregnenolone to 17a-hydroxyprogesterone* *DHEA to androstenedione* *Androstenediol to testosterone* *Androstadienol to androstadienone*				
Hormone Metabolites Assessment -> Enzymes -> Soluble Catechol-O-Methyl-Transferase (S-COMT)	1		8	
Hormone Metabolites Assessment -> Progestogens -> Allopregnanolone *Allopregnanolone (ALLO), also known as 3a-hydroxy-5a-pregnan-20-one or 3a,5a-tetrahydroprogesterone (3a,5a-THP). Progesterone metabolite dependent on 5a-reductase and 3a-Hydroxysteroid reductase.* *It is an endogenous inhibitory pregnane neurosteroid. It is synthesized from progesterone, and is a potent positive allosteric modulator of the action of gamma-amininobutyric acid (GABA) at GABAA receptor. Allopregnanolone has effects similar to those of other positive allosteric modulators of the GABA action at GABAA receptor such as the benzodiazepines, including anxiolytic, sedative, and anticonvulsant activity.* *Allopregnanolone is a neuroactive metabolite of progesterone and a barbiturate-like modulator of central gamma-aminobutyric acid receptors that modify a range of behaviors, including the stress response. is a steroid created in the body when progesterone, the female sex hormone, is metabolized. Typically, THP (allopregnanolone) is released in the brain in response to stress, and quiets the neural system within 30 minutes of escalation. This steroid hormone has recently been found to be responsible for the extreme mood swings found in teenagers. In adults and pre-pubescent chlidren THP normally helps soothe the activity of brain cells by binding to GABA receptors that inhibit accelerating electrical activity. However, in pubescent teenagers THP actually becomes a GABA receptor antagonist. GABA (gamma-aminobutyric acid) is the primary inhibitory neurotransmitter in the brain with most sedatives (tranquilizers, anesthetics and alcohol) acting on the GABA receptor.*	10		6	

Hormone Metabolites Assessment -> Estrogens -> 2-Methoxy Estrone *2-methoxyestrone is a steroid derivative that is a byproduct of estrone and 2-hydroxyestrone metabolism and is dependent on CYP1A1 activity. It is part of the androgen and estrogen metabolic pathway. The acid ionization constant (pKa) of 2-methoxyestrone is 10.81 (PMID: 516114). 2-Methoxyestrone can be metabolized to a sulfated derivative (2-Methoxyestrone 3-sulfate) via steroid sulfotransferase (EC 2.8.2.15). It can also be glucuronidated to 2-Methoxyestrone 3-glucuronide by UDP glucuronosyltransferase (EC 2.4.1.17).*	1		0	
Hormone Metabolites Assessment -> Thyroid -> Thyroid Peroxidase *Thyroid peroxidase or thyroperoxidase (TPO) is an enzyme expressed mainly in the thyroid where it is secreted into colloid. Thyroid peroxidase oxidizes iodide ions to form iodine atoms for addition onto tyrosine residues on thyroglobulin for the production of thyroxine (T4) or triiodothyronine (T3), the thyroid hormones. In humans, thyroperoxidase is encoded by the TPO gene. Thyroid peroxidase is a frequent epitope of autoantibodies in autoimmune thyroid disease, with such antibodies being called anti-thyroid peroxidase antibodies (anti-TPO antibodies). This is most commonly associated with Hashimoto's thyroiditis. Thus, an antibody titer can be used to assess disease activity in patients that have developed such antibodies.*	10		0	
Hormone Metabolites Assessment -> Estrogens -> 4-Hydroxy Estradiol *4-Hydroxyestradiol is an oncogenic catechol estrogen produced by metabolism of estrogen.*	9		0	
Hormone Metabolites Assessment -> Estrogens -> Bisphenol A *Bisphenol A, commonly abbreviated as BPA, is an organic compound with two phenol functional groups. It is a difunctional building block of several important plastics and plastic additives. With an annual production of 2-3 million metric tonnes, it is an important monomer in the production of polycarbonate. It is a potential food contaminant arising from its use in reusable polycarbonate food containers such as water carboys, baby bottles and kitchen utensils. Suspected of being hazardous to humans since the 1930s, concerns about the use of bisphenol A in consumer products were regularly reported in the news media in 2008 after several governments issued reports questioning its safety, and some retailers removed baby bottles and other children's products made from it from their shelves.* *BPA has been found to bind to both of the nuclear estrogen receptors (ERs), ERa and ERß. It is 1000- to 2000-fold less potent than estradiol. BPA can both mimic the action of estrogen and antagonize estrogen, indicating that it is a selective estrogen receptor modulator (SERM) or partial*	5		0	

agonist of the ER. At high concentrations, BPA also binds to and acts as an antagonist of the androgen receptor (AR). In addition to receptor binding, the compound has been found to affect Leydig cell steroidogenesis, including affecting 17a-hydroxylase/17,20 lyase and aromatase expression and interfering with LH receptor-ligand binding.				
Hormone Metabolites Assessment -> Progestogens -> 3a-Dihydroprogesterone *3a-Dihydroprogesterone (3a-DHP), also known as 3a-hydroxy-4-pregnen-20-one, is an endogenous neurosteroid. It is biosynthesized by 3a-hydroxysteroid dehydrogenase from progesterone. 3a-DHP has been found to act as a positive allosteric modulator of the GABA receptor and is described as being as active as allopregnanolone in regard to this action. In accordance, it has anxiolytic effects in animals. 3a-DHP has also been found to inhibit the secretion of follicle-stimulating hormone (FSH) from the rat pituitary gland, demonstrating possible antigonadotropic properties. Unlike the case of most other inhibitory neurosteroids, 3a-DHP production is not blocked by 5a-reductase inhibitors like finasteride.*	4		0	
Hormone Metabolites Assessment -> Enzymes -> Aldosterone synthase *Aldosterone synthase is a steroid hydroxylase cytochrome P450 enzyme involved in the biosynthesis of the mineralocorticoid aldosterone. It is a protein which is only expressed in the zona glomerulosa of the adrenal cortex and is primarily regulated by the renin-angiotensin system. It is the sole enzyme capable of synthesizing aldosterone in humans and plays an important role in electrolyte balance and blood pressure.* *Involved in conversion of corticosterone to aldosterone.*	3		0	