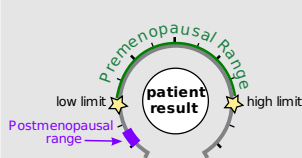


TEST NAME: DUTCH Complete Female Sample Report

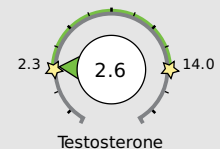
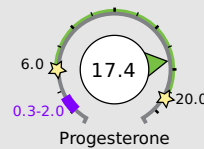
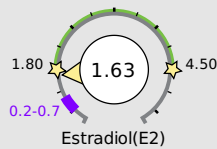
Hormone Testing Summary

Key (how to read the results):



Sex Hormones

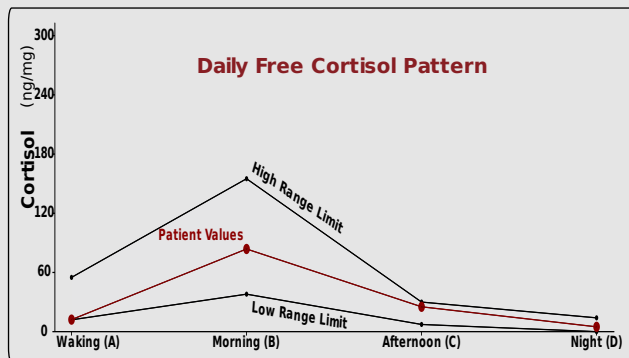
See Pages 2 and 3 for a thorough breakdown of sex hormone metabolites



(Serum Equivalent, ng/mL)
 Progesterone Serum Equivalent is a calculated value based on urine pregnanediol.

Adrenal Hormones

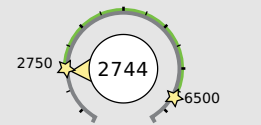
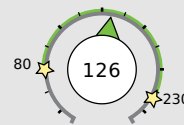
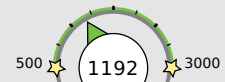
See pages 4 and 5 for a more complete breakdown of adrenal hormones



Free cortisol best reflects tissue levels. Metabolized cortisol best reflects total cortisol production.

Total DHEA Production

Age	Range
20-39	1300-3000
40-60	750-2000
>60	500-1200



cortisol
metabolism

The following videos (which can also be found on the website under the listed names along with others) may aid your understanding:
[DUTCH Complete Overview](#) [Estrogen Tutorial](#) [Female Androgen Tutorial](#) [Cortisol Tutorial](#)

PLEASE BE SURE TO READ BELOW FOR ANY SPECIFIC LAB COMMENTS. More detailed comments can be found on page 8.

- The patient shows significantly higher free cortisol compared to metabolized cortisol. It may be advisable to check thyroid hormones if you have not. See comments in the notes for more details.



Nordic Laboratories
dutch
Direct Urine Test for Cortisol and Cortisone

PATIENT: **Sample Report**

TEST REF: **TST-##-####**

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 35
 DATE OF BIRTH: dd-mm-yyyy

COLLECTED: dd/mm/yyyy
 RECEIVED: dd/mm/yyyy
 TESTED: dd/mm/yyyy

PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

TEST NAME: DUTCH Complete Female Sample Report

Category	Test	Result	Units	Normal Range
Creatinine (Urine)				
	Creatinine A (Waking)	Within range	0.82 mg/ml	0.2 - 2
	Creatinine B (Morning)	Within range	1.14 mg/ml	0.2 - 2
	Creatinine C (Afternoon)	Within range	0.35 mg/ml	0.2 - 2
	Creatinine D (Night)	Within range	0.46 mg/ml	0.2 - 2
Daily Free Cortisol and Cortisone (Urine)				
	Cortisol A (Waking)	Low end of range	12.2 ng/mg	12 - 55
	Cortisol B (Morning)	Within range	83.8 ng/mg	38 - 155
	Cortisol C (Afternoon)	Within range	25.2 ng/mg	7.3 - 30
	Cortisol D (Night)	Within range	4.9 ng/mg	0 - 14
	Cortisone A (Waking)	Within range	56.0 ng/mg	40 - 120
	Cortisone B (Morning)	Above range	254.6 ng/mg	90 - 230
	Cortisone C (Afternoon)	Above range	116.8 ng/mg	32 - 95
	Cortisone D (Night)	Within range	30.0 ng/mg	0 - 55
	24hr Free Cortisol	Within range	126.1 ng/mg	80 - 230
	24hr Free Cortisone	Above range	457.5 ng/mg	220 - 450
Cortisol Metabolites and DHEA-S (Urine)				
	a-Tetrahydrocortisol (a-THF)	Within range	139.0 ng/mg	75 - 370
	b-Tetrahydrocortisol (b-THF)	Below range	1035.0 ng/mg	1050 - 2500
	b-Tetrahydrocortisone (b-THE)	Low end of range	1570.0 ng/mg	1550 - 3800
	Metabolized Cortisol (THF+THE)	Below range	2744.0 ng/mg	2750 - 6500
	DHEA-S	Low end of range	55.0 ng/mg	20 - 750



Nordic Laboratories
dutch
Diced Urine Test for Competitive Hormones

PATIENT: Sample Report

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 35
 DATE OF BIRTH: dd-mm-yyyy

COLLECTED: dd/mm/yyyy
 RECEIVED: dd/mm/yyyy
 TESTED: dd/mm/yyyy

TEST REF: **TST-##-#####**

PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

TEST NAME: DUTCH Complete Female Sample Report

Category	Test	Result	Units	Normal Range
Nutritional Organic Acids				
Vitamin B12 Marker (may be deficient if high) - (Urine)				
	Methylmalonate (MMA)	Within range	1.0	ug/mg 0 - 2.2
Vitamin B6 Markers (may be deficient if high) - (Urine)				
	Xanthurenate	Within range	0.5	ug/mg 0 - 1.4
	Kynurenate	Within range	2.6	ug/mg 0 - 7.3
Glutathione Marker (may be deficient if low or high) - (Urine)				
	Pyroglutamate	Within range	52.4	ug/mg 32 - 60
Neurotransmitter Metabolites				
Dopamine Metabolite - (Urine)				
	Homovanillate (HVA)	Within range	6.6	ug/mg 4 - 13
Norepinephrine/Epinephrine Metabolite - (Urine)				
	Vanilmandelate (VMA)	Within range	5.1	ug/mg 2.4 - 6.4
Melatonin (*measured as 6-OH-Melatonin-Sulfate) - (Urine)				
	Melatonin* (Waking)	Within range	31.3	ng/mg 10 - 85
Oxidative Stress / DNA Damage, measured as 8-Hydroxy-2-deoxyguanosine (8-OHdG) - (Urine)				
	8-OHdG (Waking)	Within range	2.6	ng/mg 0 - 5.2

Nordic Laboratories Aps

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11 Old Factory Buildings • Stonegate • E. Sussex TN5 7DU • UK
 Tel: +44 (0)1580 201 687

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 35
 DATE OF BIRTH: dd-mm-yyyy

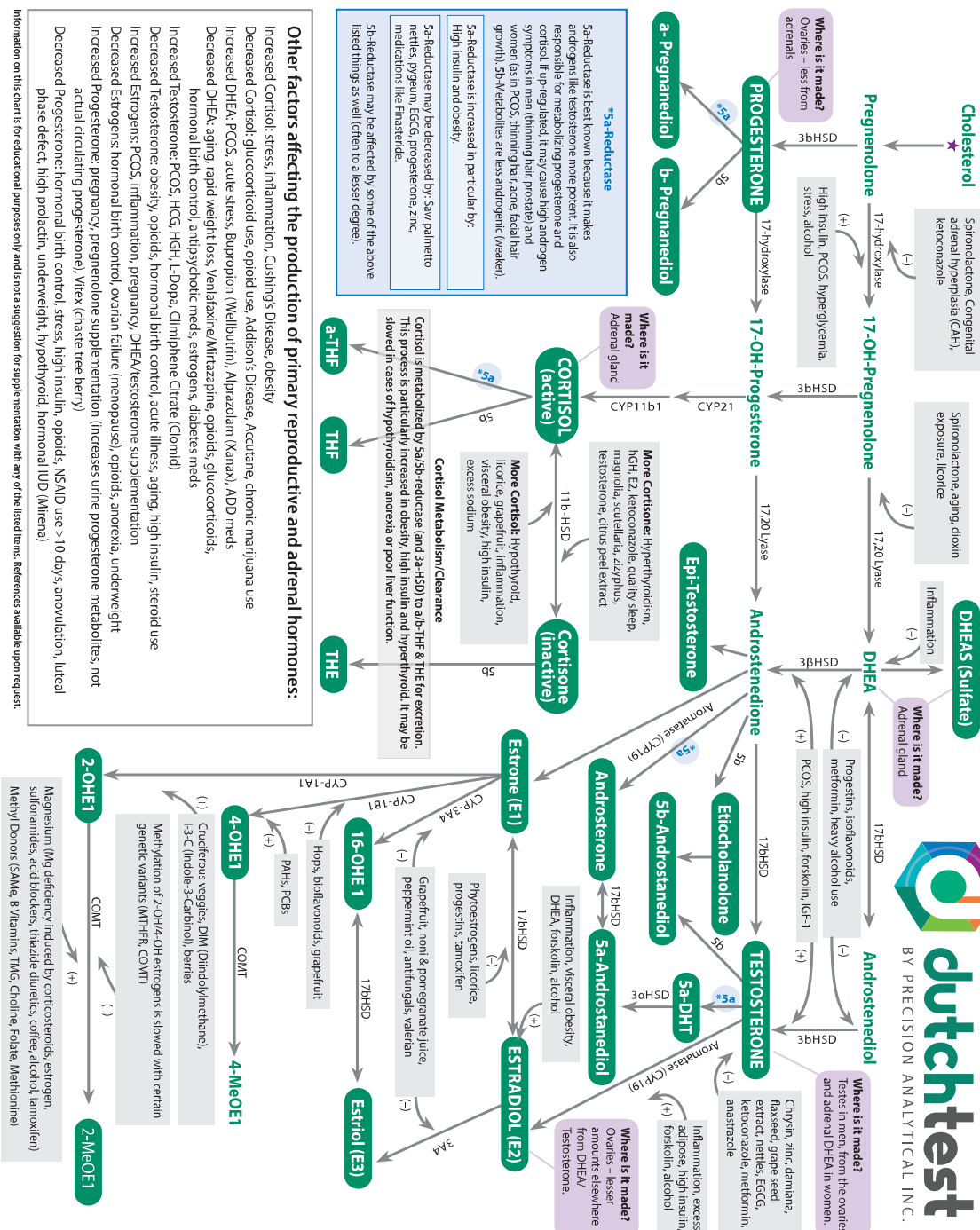
COLLECTED: dd/mm/yyyy
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PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

TEST NAME: DUTCH Complete Female Sample Report

Steroid Pathways Find these **Hormones** on the **DUTCH Complete**

Primary hormones (in CAPS) are made by organs by taking up cholesterol * and converting it locally, for example, progesterone. Much less is made from circulating precursors like pregnenolone. For example, taking DHEA can create testosterone and estrogen, but far less than is made by the testes or ovaries, respectively.



Other factors affecting the production of primary reproductive and adrenal hormones:

- Increased Cortisol: stress, inflammation, Cushing's Disease, obesity
- Decreased Cortisol: glucocorticoid use, opioid use, Addison's Disease, chronic marijuana use
- Increased DHEA: PCOS, acute stress, Bupropion (Wellbutrin), Alprazolam (Xanax), ADD meds
- Decreased DHEA: aging, rapid weight loss, Venlafaxine/Milnacipran, opioids, glucocorticoids, hormonal birth control, antipsychotic meds, estrogen, diabetes meds
- Increased Testosterone: PCOS, HCG, HGH, L-Dopa, Climbiphen Citrate (Clomid)
- Decreased Testosterone: obesity, opioids, hormonal birth control, acute illness, aging, high insulin, steroid use
- Increased Estrogens: hormonal birth control, ovarian failure (menopausal), opioids, anorexia, underweight
- Increased Progesterone: pregnancy, pregnenolone supplementation (increases urine progesterone metabolites, not actual circulating progesterone), Vitex (chaste tree berry)
- Decreased Progesterone: hormonal birth control, stress, high insulin, opioids, NSAID use > 10 days, anovulation, luteal phase defect, high prolactin, underweight, hypothyroid, hormonal IUD (Mirena)

Information on this chart is for educational purposes only and is not a suggestion for supplementation with any of the listed items. References available upon request.

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 35
 DATE OF BIRTH: dd-mm-yyyy

COLLECTED: dd/mm/yyyy
 RECEIVED: dd/mm/yyyy
 TESTED: dd/mm/yyyy

PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

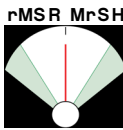
TEST NAME: DUTCH Complete Female Sample Report

UoDop

polDpNSoop

SR SR :: R HMR:RSSMM:SSS M: M:rS SR
 ::RM:SR RRMRRS R R Sr SNrR r S S::rR
 :NrRr Sr SN S S:S:: M::rSr S
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R r R:M RRSrN:S S R : M:rS SN:S S M :rS
 MRRS RSrRRFMMrR



rMSR MrSHRrSFS rSRRRSHRrSR NrM
 N TTN NRrS rRrS r RR:rRRSHRrSR R
 NrMN MR SRr:MR SR NRr S S:SSrRR RRRRSR
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SRRSR NRr Srr:

UppolCp

RR R:MR SRrSRMR SSMM RRS RRsr SSMR Srr
 NR: SSM RR R SM RSR R SR SMRS SMM RRMS
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NlppoopoAHClopoHIC C:

UoApompIC

RrRRS RS RNrM S RMSRS RRS R NRr S S::
 M RS RrrSrrNMSr rRS RSR:HSRNF:RrS
 SR NMS S RS N: NRr HN rSR S MRSR:F:Rr
 R R NRr SSR :RRS RrrS rR R MMR:
 RS RrrSHS RSR RRMS RRMM
 E: Rr HRrRS RS R r R
 NRr SRRS RNR:RMRr S RrR RS RS R RR: Sr
 MRr RS S RS RrS RSrr r NRr SM S Sr
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S U

UoApooHCploolAol CAIpNCHpICNIDlpAHCpCC Hood
 Bl opNICCCpICHpNICpN oApoCClo
 ICC CIC ploloppNpHo:

ICI pNpoA

poAmplC

rES RrrSH SR RSSSS RrrR
 • NplpHCdCoNANpoAoDhp:
 ArSR RR:M HS :Rr S RS S RHSrrS rS RSMRS::
 RRR RHMS: R RSrRRS RSr

• UNl mplC:
 S RS NRr:rN r:R S S CFDSMRS:: SS



PATIENT: Sample Report		TEST REF: TST-##-####
TEST NUMBER: #####	COLLECTED: dd/mm/yyyy	PRACTITIONER: Nordic Laboratories ADDRESS:
PATIENT NUMBER: #####	RECEIVED: dd/mm/yyyy	
GENDER: Female	TESTED: dd/mm/yyyy	
AGE: 35		
DATE OF BIRTH: dd-mm-yyyy		

TEST NAME: DUTCH Complete Female Sample Report

because of the anti-cancer properties of 2-OH metabolites. Conversely, the 4-OH pathway is considered the most genotoxic as its metabolites can create reactive products that damage DNA. The third pathway, 16-OH creates the most estrogenic of the metabolites (although still considerably less estrogenic than estradiol) - 16-OH-E1. If overall estrogen levels are high, production of 16-OH-E1 may exacerbate high estrogen symptoms. Similarly, a woman with very low levels of estrogens, may have less low estrogen symptoms if 16-OH metabolism is preferred. For example Armamento-Villareal showed that a higher 2-OH-E1/16-OH-E1 ratio correlated to bone loss (a low estrogen symptom). Estriol is thought of as a safer (weaker) estrogen metabolite, but it is important to remember that estriol is actually 16-OH-E2, so generally patients that make a lot of the potentially protective/weak estriol may also make a lot of the estrogenic 16-OH-E1.

When evaluating phase I metabolism, it may be important to look at the ratios of the three metabolites to see which pathways are preferred relative to one another. It may also be important to compare these metabolites to the levels of the parent hormones (E1, E2). If the ratios of the three metabolites are favorable but overall levels of metabolites are much lower than E1 and E2, this may imply sluggish phase I clearance of estrogens, which can contribute to high levels of E1 and E2. Similarly, patients with excessive phase I metabolism may have low E1 and E2 levels because of high rates of clearance (as opposed to simply not making a lot of estrogen).

The pie chart will assist you in comparing the three pathway options of phase I metabolism compared to what is "normal." 2-OH metabolism can be increased by using products containing D.I.M. or I-3-C. These compounds are found (or created from) in cruciferous vegetables and are known for promoting this pathway.

Patients typically metabolize a much higher percentage of their estrogens down the more protective 2-OH pathway in phase I detoxification. Diindolylmethane (DIM) or Indole-3-Carbinol containing products can help move estrogens more efficiently down this pathway. Be aware that this typically lowers most of the other estrogens, including E1 and E2 as well. If the patients are taking or considering hormone replacement therapy, these products may be considered but a higher dose of estrogen may be needed for the same clinical effect if taken at the same time.

• Methylation (part of phase II metabolism) of estrogens:

After phase I metabolism, both 4-OH and 2-OH (not 16-OH) estrogens can be deactivated and eliminated by methylation. The methylation-activity index shows the patient's ratio of 2-Methoxy-E1 / 2-OH-E1 compared to what is expected. Low methylation can be caused by low levels of nutrients needed for methylation and/or genetic abnormalities (COMT, MTHFR). The COMT enzyme responsible for methylation requires magnesium and methyl donors. Deficiencies in folate or vitamin B6 or B12 can cause low levels of methyl donors. MTHFR genetic defects can make it more difficult for patients to make sufficient methyl donors. Genetic defects in COMT can make methylation poor even in the presence of adequate methyl donors.

Androgen Metabolism

When evaluating androgen levels, it is important to assess the following:

• The status (low, normal or high?) of DHEA:

DHEA and androstenedione are made almost exclusively by the adrenal gland (although a smaller amount is made in the ovaries). These hormones appear in urine as DHEA-S (DHEA-Sulfate), androsterone and etiocholanolone. The best way to assess the total production of DHEA is to add up these three metabolites. This total can be seen on the first page of the DUTCH Complete (and DUTCH Plus). DHEA production decreases quite significantly with age. Age-dependent ranges can be seen on the graphical page of results.

The Total DHEA Production (page 1) was about 1,192ng/mg which is within the overall range but is below the range for the patient's age-dependent range. This implies that the adrenal glands are not producing appropriate DHEA levels for the patient's age. Low DHEA is associated with depression, diabetes, heart disease, inflammation and immune disorders. It can be caused by hypothyroidism. It can cause fatigue, low mood and low libido. Supplementing DHEA in women often raises both testosterone and estrogen, which may or may not be desirable here. DHEA may increase with adaptogens such as maca and rhodiola, which improve overall adrenal output.

• The status (low, normal or high?) of testosterone:

Females make most of their DHEA in the adrenal gland and a fraction of that DHEA trickles down metabolically to testosterone. For premenopausal women, some testosterone is also made by the ovaries. Levels of testosterone do drop somewhat with age, but not to the degree that DHEA decreases.

Testosterone levels for this patient were approximately 2.60ng/mg, which is within range, but is below "normal" levels for a young, healthy woman. You may want to also carefully evaluate 5a-metabolism (see below) and testosterone's downstream metabolites, 5a-androstenediol and 5b-androstenediol on page 2 of the DUTCH Plus or DUTCH Complete. They are on page 1 of a DUTCH Sex Hormone. These two metabolites generally parallel testosterone production, although they can also be generated from DHEA without going through testosterone. If all markers are on the lower end of the range and she reports low androgen symptoms (fatigue, loss of libido, bone loss, etc.), you may want to consider testosterone HRT or symptomatic support with Zinc, Maca, Tribulus or Shatavari.

• The metabolic preference for the 5a (5-alpha) or 5b (5-beta) pathway:

5a-reductase converts testosterone into 5a-DHT (DHT), which is even more potent (~3x) than testosterone. High levels of DHT can lead to symptoms associated with too much testosterone. Metabolites created down the 5b-pathway are significantly less androgenic than their 5a counterparts. In the examples below, the example on the left shows a patient with 5b-metabolism preference. A patient with a pattern like the example on the right may have high androgen symptoms even though the hormones are in the normal range because of the likely preference for turning a lot of her testosterone into DHT. The fan-style gauge below the hormones shows the 5a or 5b preference based on etiocholanolone (5b) and androsterone

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 35
 DATE OF BIRTH: dd-mm-yyyy

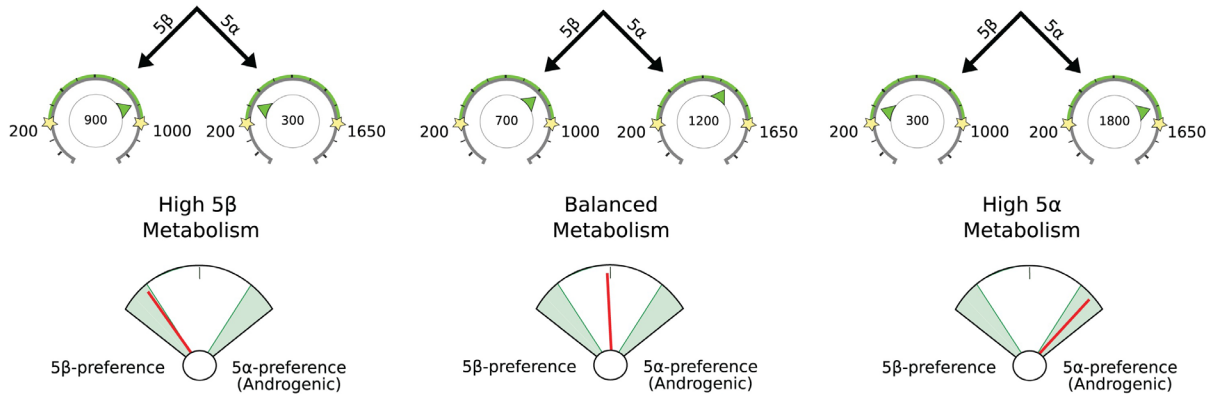
COLLECTED: dd/mm/yyyy
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PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

TEST NAME: DUTCH Complete Female Sample Report

(5a) results Progesterone metabolites are also metabolized by 5a and 5b enzymes and the balance between these two metabolites can be useful to confirm a 5a or 5b preference

Example of how to read fan-style gauge for 5a-reductase activity:



Neither testosterone or overall levels of DHEA are elevated, and 5a-metabolism is not elevated. This is consistent with the patient's lack of reporting androgen excess symptoms.

It is important to consider DHEA and testosterone production, 5a metabolite patterns as well as the patient symptoms. For example, a woman with higher levels of DHEA and testosterone who often has high androgen symptoms (facial hair, thinning scalp hair, etc.) exacerbated by 5a metabolism, on the other hand, she prefers 5b metabolism she may not express high androgen symptoms in spite of higher levels of testosterone because 5b is the essential androgenic pathway. Testosterone levels may be better understood by also considering its downstream metabolites (5α androstenedione, 5β androstenedione). Technically, these metabolites can also be formed from DHEA metabolites without going through the testosterone pathway, but they generally tend to correlate with testosterone production. You will also see levels of epitestosterone, which is not androgenic like testosterone, that happens to be produced in about the same concentrations as testosterone (this is an approximate relationship). This can be helpful to assess testosterone therapy and rare cases where testosterone may have other complexities.

DUTCH Adrenal

The HPA Axis refers to the communication and interaction between the hypothalamus (H) and pituitary (P) in the brain down to the adrenal glands (A) that sit on top of your kidneys. When a physical or psychological stressor occurs, the hypothalamus tells the pituitary to make ACTH, a hormone that stimulates the adrenal glands to make the stress hormone, cortisol and to a lesser extent DHEA and DHEAS. Normally, the HPA axis production follows a daily pattern in which cortisol rises rather rapidly in the first 10-30 minutes after waking in order to help with energy, then gradually decreases throughout the day so that it is low at night for sleep. The cycle starts over the next morning. Abnormal high activity occurs in Cushing's Disease where the HPA axis is hyperstimulated causing cortisol to be elevated all day. The opposite is known as Addison's Disease, where cortisol is abnormally low because it is not made appropriately in response to ACTH's stimulation. These two conditions are somewhat rare. Examples of more common conditions related to excess severe abnormal cortisol levels include fatigue, depression, insomnia, fibromyalgia, anxiety, inflammation and more.

Only a fraction of cortisol is "free" and bioactive. This fraction of cortisol is very important, but levels of metabolized cortisol best represent overall production of cortisol therefore both should be taken into account to correctly assess adrenal function.

When evaluating cortisol levels, it is important to assess the following:

- **The overall up-and-down pattern of free cortisol throughout the day, looking for low and high levels:** Abnormal results should be considered along with related symptoms. Remember that with urine results, the "waking" sample reflects the night's total for free cortisol. The sample collected two hours after waking captures the cortisol awakening response, which is typically the time with the most cortisol secretion.
- **The sum of the free cortisol as an expression of the overall tissue cortisol exposure:** This total of four free cortisol measurements is the best way to assess the total of free cortisol throughout the day, and this result correlates reasonably well to a true 24-hour urine free cortisol. Do be aware that this measurement does not take into account transient shifts in cortisol in the late morning or early afternoon.
- **The total level of cortisol metabolites:** We calculate this as a "Metabolized Cortisol" which is the sum of a THF, b THF and b THE (the most abundant cortisol metabolites). While free cortisol is the best assessment for tissue levels of cortisol, it only represents 1-3% of the total produced. The majority of cortisol results in a urine metabolite and the total of these metabolites best represents the total glandular output of cortisol for the day. When overall production is much higher than free cortisol levels, cortisol clearance may be increased (as seen in hyperthyroidism, obesity, etc.). The most common reason for sluggish cortisol clearance (assumed when free cortisol levels are much higher than metabolized cortisol) is slow thyroid.

PATIENT: Sample Report		TEST REF: TST-##-####
TEST NUMBER: #####	COLLECTED: dd/mm/yyyy	PRACTITIONER: Nordic Laboratories ADDRESS:
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GENDER: Female	TESTED: dd/mm/yyyy	
AGE: 35		
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TEST NAME: DUTCH Complete Female Sample Report

Overall free cortisol levels are within range, but metabolized cortisol (the best marker for overall cortisol production) is low. This implies that overall HPA-Axis A is low. Cortisol clearance may be a bit sluggish, which keeps free cortisol levels within range in spite of low overall production. Hypothyroidism and other conditions may lead to low cortisol metabolism. If treating the patient for potential thyroid issues be sure to take into account the interplay between the thyroid and adrenals.

• **A potential preference for cortisol or cortisone (the inactive form):**

Looking at the comparison between the total free cortisol and free cortisone is NOT the best indication of a person's preference for cortisol or cortisone. The kidney converts cortisol to cortisone in the local tissue. This localized conversion can be seen by comparing cortisol (free) and cortisone levels. To see the patient's preference system clearly, it's best to look at which metabolite predominates (THF or THE). This preference can be seen in the fan style gauge. This is known as the 11b HSD index. The enzyme 11b HSD converts cortisol to cortisone in the kidneys, saliva and and colon. 11b HSD is more active in the liver, fat cells and the periphery and responds better for reacting cortisol to cortisol. Both are then metabolized by 5a reductase to become tetrahydrocortisol (THF) and tetrahydrocortisone (THE) respectively.

The patient's THF/THE and cortisol to cortisone ratios imply a preference for cortisone (relative to cortisol). Because cortisol levels are not low, this may have some beneficial effect in keeping cortisol levels from being even higher than reported here.

Nutritional Organic Acids

The following three organic acids are functional markers for vitamin deficiency. These compounds essentially back up in human biochemistry when a key nutrient is missing. These three metabolites have fairly straightforward interpretations. When the markers are elevated, it's likely that the patient's cellular levels of the related nutrient may be insufficient.

Methylmalonate (MMA)

Methylmalonate (also known as methylmalonic acid or MMA) is a functional marker of vitamin B12 (also known as cobalamin) deficiency. When cellular levels of B12 are low either from deficiency or due to a B12 transporter gene mutation, levels of MMA increase. This marker is considered superior to measuring serum B12 levels directly. A 2012 publication by Miller showed that 20% of those tested had a genetic defect in the protein that transports B12 to cells. These patients may have a functional B12 deficiency even if serum levels of B12 are normal. Levels of MMA are elevated, it may be advisable to increase B12 consumption. Common foods high in B12 include beef liver, sardines, lamb, wild caught salmon, grass fed beef, nutrient yeast and eggs. Vitamin B12 levels can also be increased through supplementation of B12 (taken as cobalamin, methylcobalamin, hydroxycobalamin, or adenosylcobalamin). Symptoms of a vitamin B12 deficiency include: fatigue, brain fog, memory problems, muscle weakness, unsteady gait, numbness, tingling, depression, migraines/headaches and low blood pressure.

Xanthurenate

Xanthurenate (also known as xanthurenic acid) is a functional marker of vitamin B6 (also known as pyridoxine). Vitamin B6 is a critical cofactor to over 100 important reactions that occur in the human body and is stored in the highest concentrations in muscle tissue. Tryptophan is readily converted to NAD by the liver. One of the steps in this pathway requires B6. When there is insufficient B6, xanthurenate is made instead. Not only is xanthurenate an indicator of a lack of B6, it is also harmful to the human body. It complexes with insulin and decreases insulin sensitivity. In fact, rats fed xanthurenate eventually developed diabetes because of the effects on insulin. Xanthurenate levels are elevated, B6 supplementation may be considered. Food high in B6 include turkey breast, grass fed beef, pinto beans, avocado, potatoes, chicken, sesame and sunflower seeds. While there is a way some tryptophan goes down the kynurenine pathway towards NAD (and possibly xanthurenate), this process is upregulated by inflammation, estrogen and cortisol. Levels of estrogen or cortisol are high, it may exacerbate xanthurenate elevations and increase the need for B6. Xanthurenate can also bind to iron and create a complex that increases DNA oxidative damage resulting in higher 8-OHdG levels. If both markers are elevated, there is likely an antioxidant insufficiency.

Pyroglutamate

Pyroglutamate (also known as pyroglutamic acid) is a functional marker of glutathione deficiency. Pyroglutamate is a step in the production/recycling of glutathione. If the body cannot convert pyroglutamate forward, it will show up elevated in the urine. High pyroglutamate is an established marker for glutathione deficiency. Glutathione is one of the most potent antioxidants in the human body. It is especially important in getting rid of toxins, including the reactive quinone species formed by 4-OH-E1 and 4-OH-E2. These reactive species can damage DNA if not detoxified by either methylation or glutathione. Some have reported that low pyroglutamate may also be indicative of a need for glutathione; however, this is not established in the scientific literature.

Neurotransmitter Metabolites

The neurotransmitters dopamine, norepinephrine and serotonin are important for human health. Measuring neurotransmitters directly (direct testing of serotonin, for example) is difficult because of the instability and the urinary measurements are controversial with respect to how well they reflect the body's levels of these neurohormones. Each of these three neurotransmitters can be assessed indirectly by measuring the urinary metabolites. While these metabolites are not a perfect reflection of what's going on in the brain, the scientific literature does affirm the use for a good representation of overall levels of these neurotransmitters.

Homovanillate (HVA)

PATIENT: Sample Report		TEST REF: TST-##-####
TEST NUMBER: #####	COLLECTED: dd/mm/yyyy	PRACTITIONER: Nordic Laboratories ADDRESS:
PATIENT NUMBER: #####	RECEIVED: dd/mm/yyyy	
GENDER: Female	TESTED: dd/mm/yyyy	
AGE: 35		
DATE OF BIRTH: dd-mm-yyyy		

TEST NAME: DUTCH Complete Female Sample Report

Homovanillic acid (also known as HVA) is the primary metabolite of dopamine, a brain and adrenal neurotransmitter that comes from tyrosine (with BH4 and iron as cofactors) and goes on to create norepinephrine (noradrenaline) and epinephrine (adrenaline).

Low levels of HVA can be due to low levels of dopamine or poor conversion of dopamine to HVA. The latter may be due to insufficient levels of SAM, Magnesium, FAD and NAD which are needed to metabolize dopamine. Low circulating dopamine may be due to insufficient BH4, iron or tyrosine. It may also be seen when adrenal function is generally low. Low dopamine levels may be associated with addictions, cravings and pleasure seeking (to boost levels) in addition to sleepiness, impulsivity, tremors, stress motivation, fatigue and low mood.

Elevated HVA may be caused by generally increased adrenal hormone output or because of a copper or vitamin C deficiency (which are needed for dopamine conversion to norepinephrine). Elevations may also be caused by a number of medications or supplements including: MAO inhibitors, quercetin, tyrosine, DL-phenylethylamine (DLPEA), L-dopa, macuna, dopamine medication (Levodopa, Sinemet, Methyl-dopa), SNR medication (Wellbutrin), tricyclic antidepressants, amphetamines, appetite suppressants, and caffeine. Bananas also contain dopamine. Elevated dopamine may be associated with loss of memory, insomnia, agitation, hyperactivity, mania, hyperfocus, high stress and anxiety as well as addictions, cravings and pleasure seeking (to maintain high levels).

When HVA is very high, consider if the previously discussed foods, supplements or medications may be the cause. Rarely, tumors associated with increased HVA may be present. In these cases, further testing is necessary for diagnosis. High HVA alone is not diagnostic of a tumor.

Vanilmandelic acid (VMA)

Vanilmandelic acid (also known as VMA) is the primary metabolite of norepinephrine and epinephrine (adrenaline). The adrenal gland makes cortisol and DHEA as well as norepinephrine and epinephrine. When adrenal hormone output is generally low, VMA levels may be low. If HVA levels are significantly higher than VMA, there may be a conversion problem from dopamine to norepinephrine. This case can be caused by a copper or vitamin C deficiency. The enzymes COMT (methylation) and MAO are needed to make VMA from norepinephrine. If these enzymes are not working properly, VMA may be low when circulating norepinephrine and/or epinephrine are not low. Low levels of norepinephrine and epinephrine may be associated with addictions, cravings, fatigue, low blood pressure, low muscle tone, intolerance to exercise, depression, loss of alertness. When the body is under physical or psychological stress, VMA levels may increase. Because dopamine gets converted to norepinephrine and ultimately to VMA, the use of medications and supplements that increase HVA may also increase VMA. Elevated levels may be associated with feeling stressed, aggression, violence, impatience, anxiety, panic, worry, insomnia, paranoia, increased tingling/burning, loss of memory, paresthesia, high blood pressure and heart palpitations. If VMA and HVA are both extremely high, it may be necessary to rule out a neuroblastoma tumor.

Melatonin (measured as 6-OHMS)

Melatonin is not technically an adrenal or sex hormone however it is highly involved in the entire endocrine system. It is made in small amounts in the pineal gland in response to darkness and stimulated by Melanocyte Stimulating Hormone (MSH). A low MSH is associated with insomnia, an increased perception of pain, and mood exposure. Pineal melatonin (melatonin is also made in significant quantities in the gut) is associated with the circadian rhythm of all hormones (including female hormone release). It is also made in small amounts in the bone marrow, lymphocytes, epithelial cells and mast cells. Studies have shown that a urine sample collected upon waking has levels of 6-Hydroxymelatonin sulfate (6-OHMS) that correlate well to the total levels of melatonin in blood samples taken continuously throughout the night. The DUTCH test uses the waking sample only to test levels of melatonin production.

Low melatonin levels may be associated with insomnia, poor immune response, constipation, weight gain or increased appetite. Elevated melatonin is usually caused by ingestion of melatonin through melatonin supplementation or eating melatonin containing foods. Elevated melatonin production that is problematic is rare, but levels can be higher in patients with Chronic Fatigue Syndrome and may be phase shifted (peaking later) in some forms of depression.

8-OHdG (8-Hydroxy-2-deoxyguanosine)

8-OHdG (8-hydroxy-2-deoxyguanosine) results can be seen on page 6 of the DUTCH Complete (or DUTCH Plus) report. It is a marker for estimated DNA damage due to oxidative stress (ROS creation). 8-OHdG is considered pro-mutagenic as it is a biomarker for various cancer and degenerative diseases. In addition and promotion, it can be increased by chronic inflammation, increased cell turnover, chronic stress, hypertension, hyperglycemia/pre-diabetes/diabetes, kidney disease, BD, chronic skin conditions (psoriasis/eczema), depression, atherosclerosis, chronic liver disease, Parkinson's (increasing levels with worsening stages), Diabetic neuropathy, COPD, bladder cancer, or insomnia. Studies have shown higher levels in patients with breast and prostate cancers. When levels are elevated, it may be prudent to eliminate or reduce any causes and increase the consumption of antioxidant containing foods and/or supplements.

The reference range for 8-OHdG is a more aggressive range for Functional Medicine that puts the range limit at the 80th percentile for each gender. A classic range (average plus two standard deviations) would result in a range of 0.6ng/mg for women and 0.10ng/mg for men. Seeking out the cause of oxidative stress may be more crucial if results exceed these limits.

Urine Hormone Testing - General Information

What is actually measured in urine? In blood, most hormones are bound to binding proteins. A small fraction of the total hormone levels are "free" and unbound such that they are active hormones. These free hormones are not found readily in urine except for cortisol and cortisone (because they are much more water soluble than, for example, testosterone). As such, free cortisol and cortisone can be measured in urine and this is the measurement that nearly all urinary cortisol research is based upon. In the DUTCH Adrenal Profile the diurnal patterns of free cortisol and cortisone are measured by LC-MS/MS.

Other hormones measured (cortisol metabolites, DHEA, and androgen hormones) are excreted in urine predominantly after



Nordic Laboratories
dutch
Direct Urine Test for Competitive Hormones

PATIENT: **Sample Report**

TEST REF: **TST-##-####**

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 35
 DATE OF BIRTH: dd-mm-yyyy

COLLECTED: dd/mm/yyyy
 RECEIVED: dd/mm/yyyy
 TESTED: dd/mm/yyyy

PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

TEST NAME: DUTCH Complete Female Sample Report

the addition of a glucuronide or sulfate group (to increase water solubility for excretion). As an example, Taic (Natura Sciences, 1968 publication) found that of the testosterone found in urine, 57.80% was testosterone glucuronide, 14.42% was testosterone sulfate, and negligible amounts (<1% for most) was free testosterone. The most likely source of free sex hormones in urine is from contamination from hormonal supplements. To eliminate this potential, we remove free hormones from conjugates (our testing can be used even if vaginal hormones have been given). The glucuronides and sulfates are then broken off of the parent hormones, and the measurements made. These measurements reflect the bioavailable amount of hormone in most cases as it is only the free, nonprotein bound fraction in blood/tissue that is available for phase metabolism (glucuronidation and sulfation) and subsequent urine excretion.
 Disclaimer: the filter paper used for sample collection is designed for blood collection, so this technology considered "research only" for urine collection. Its proper use for urine collection has been thoroughly validated.

