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ABOUT US

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What Is Methylation?

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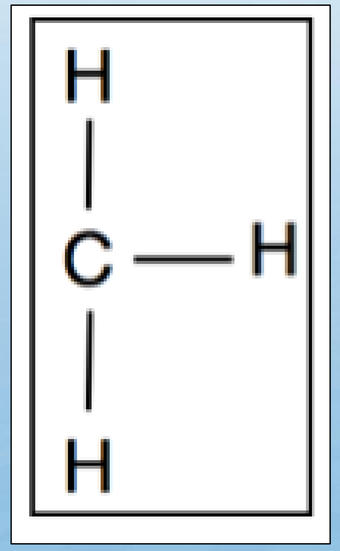
What Is Methylation?

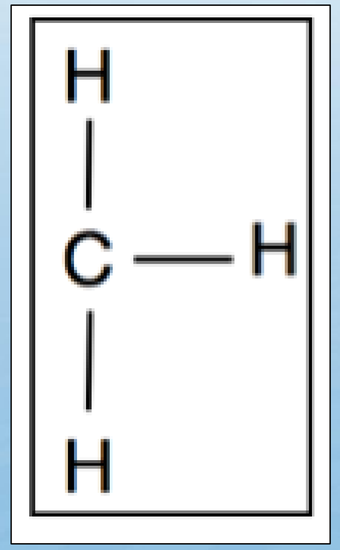
- A biochemical process taking place in every cell of the body, continuously
- The addition of a methyl group (CH3) to a substrate



Methylation is Essential For

- Genetic expression
- DNA & RNA synthesis and repair
- Biosynthesis of active form of B-12 (methylcobalamin)
- Biosynthesis of active form of Folate (5-methyltetrahydrofolate)
- Neurotransmitter synthesis
- Detoxification/biotransformation: Glutathione synthesis, SOD synthesis, processing of heavy metal & chemical toxins, estrogen methylation
- Myelination of nerves, lipid methylation
- Cardiovascular & endothelial health: synthesis & removal of homocysteine, nitric oxide synthesis/urea cycle function
- Immune cell synthesis: NK cells, T cells
- Blood clotting
- Healthy pregnancy

Common Issues Which May Involve Methylation



- Birth defects
- Estrogen dominance
- Autoimmune conditions and immune disorders
- Heart disease or genetic heart disease history
- Anxiety, autism, depression, bipolar, schizophrenia, OCD, ADD/ADHD, etc
- Environmental chemical toxicity and/or intolerance
- History of toxic metal poisoning
- Cancer
- Viral infections
- Diabetes
- Neurological inflammatory conditions: lyme, MS, ALS, Parkinson’s, Alzheimer’s, Huntington’s, Wilson’s

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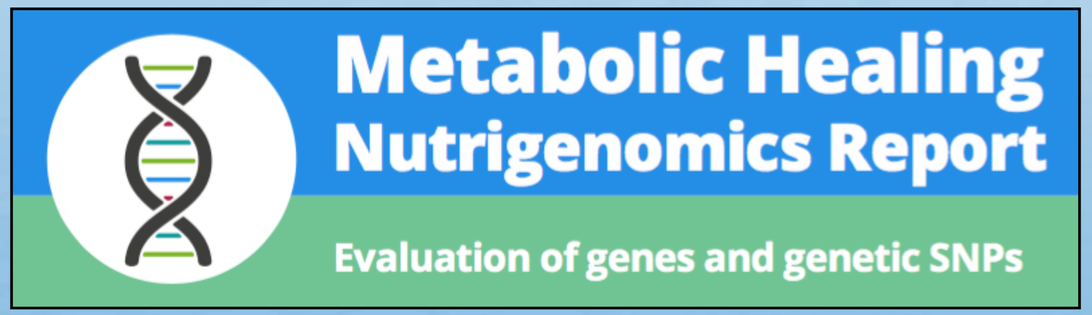
Methylation Genetics:
Assessing The Genes That Regulate Genes



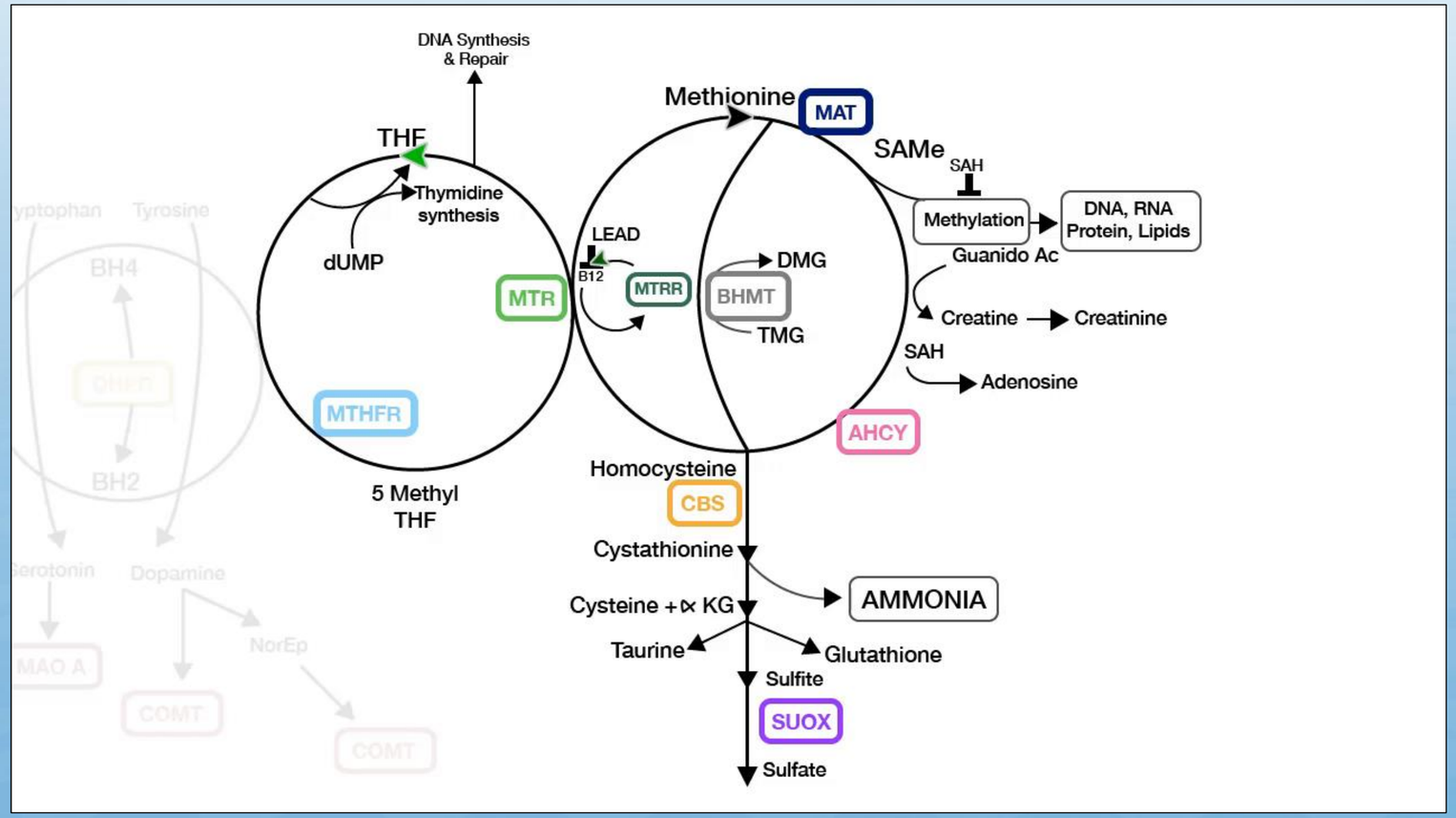
Methylation — MTHFR, MTR & MTRR

RS#	Call	Risk Allele	Gene	Variation	Result
rs1801131	TT	G	MTHFR	A1298C	-/-
rs1801133	GG	A	MTHFR	C677T	-/-
rs17367504	AA	G	MTHFR	A1572G	-/-
rs2066470	GG	A	MTHFR 03	P39P	-/-
rs2274976	CC	T	MTHFR	G1793A (R594Q)	-/-
rs1805087	AG	G	MTR	A2756G	+/-
rs1801394	GG	G	MTRR	A66G	+/+
rs162036	AA	G	MTRR	K350A	-/-
rs1802059	GG	A	MTRR-11	A664A	-/-
rs1532268	CC	T	MTRR		-/-
rs3776467	AA	G	MTRR		-/-
rs9332	GG	A	MTRR		-/-

RS#	Call	Risk Allele	Gene	Variation	Result
rs6269	AA	G	COMT		-/-
rs769224	GG	A	COMT	-61 P199P	-/-
rs4633	TT	T	COMT	H62H	+/+
rs4680	AA	A	COMT	V158M	+/+

RS#	Call	Risk Allele	Gene	Variation	Result
rs2071010	GG	A	FOLR1		-/-
rs651933	GG	A	FOLR2		-/-
rs7925545	AA	G	FOLR3		-/-
rs1643649	CT	C	DHFR		+/-
rs1076991	CT	C	MTHFD1	C105T	+/-
rs2236225	AG	A	MTHFD1	G1958A	+/-
rs11754661	GG	A	MTHFD1L		-/-
rs17349743	CT	C	MTHFD1L		+/-
rs6922269	GG	A	MTHFD1L		-/-
rs803422	GG	A	MTHFD1L		-/-

Methylation Made Simple



Deficiencies of cofactors/nutrients prevent proper methylation

- Methionine/SAMe
- Serine
- Glycine
- Zinc
- Magnesium
- B-vitamins: B-6, B-2, B-12, TMG, folate, choline
- Redox cofactors: NADH, FADH

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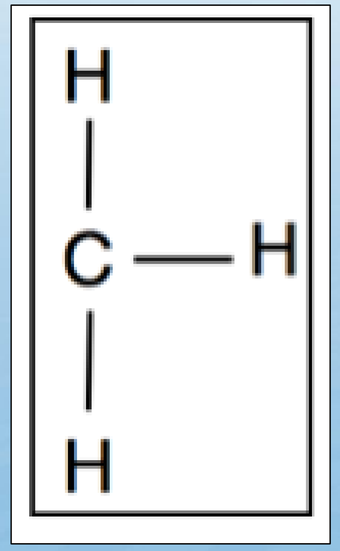
MTHFR



Methylene Tetra Hydro Folate Reductase (MTHFR)

- Gene/enzyme central to methylation
- Necessary to make the active form of folate (5-methylfolate)
- More than 5,000 published studies on MTHFR gene
- Associated with a number of health risks (cardiovascular disease, cancer, birth defects, stroke)
- 10% of US population is +/+ for MTHFR C677T

Unmetabolized Folic Acid Controversy

Am J Clin Nutr. 1997 Jun;65(6):1790-5.

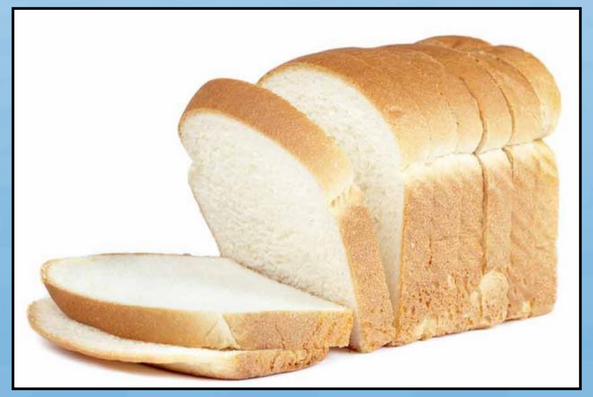
Unmetabolized folic acid in serum: acute studies in subjects consuming fortified food and supplements.

Kelly P[1], McPartlin J, Goggins M, Weir DG, Scott JM.

Author information

Abstract

Periconceptual consumption of folic acid has been shown to decrease the incidence of neural tube defects. The strategy of universal fortification of staple foodstuffs with folic acid presents the possibility of life-long exposure to unmetabolized folic acid. Chief among the risks of exposure to folic acid in the circulation is that of masking the diagnosis of cobalamin deficiency in pernicious anemia and the progression of neurologic disease. Other effects are unknown. For instance, the effect of in vivo chronic exposure of adult and fetal cells to the synthetic form of the vitamin has never been investigated at the population level. This study examined the acute appearance of unmetabolized folic acid in serum in response to the consumption of some fortified foodstuffs by young and elderly volunteers. Subjects on a 5-d regimen of fortified ready-to-eat-cereal and bread in addition to their normal diet had a threshold intake of 266 micrograms folic acid per meal at which unaltered folic acid appeared in the serum. Subjects given folic acid in either isotonic saline, milk, or white bread also had a threshold > 200 micrograms. From patterns of food consumption in the United States, the implementation of flour fortification at 1.4 mg/kg is unlikely to lead to folic acid appearance in serum, assuming that consumption is spread throughout the day. Increasing this level of fortification, however, as has been advocated by some agencies, may result in the repeated appearance of folic acid in serum over many years, particularly in consumers in nontargeted populations of large amounts of fortified foods. The "safe level of intake" of 1 mg folate/d set by the US Food and Drug Administration may cause a serum folic acid effect. Furthermore, a repeated serum folic acid response is likely to be found in many women complying with the advice to take 400 micrograms folic acid/d to prevent the occurrence of neural tube defects.



BMC Public Health. 2007; 7: 41. PMCID: PMC1839088

Published online 2007 Mar 22. doi: 10.1186/1471-2458-7-41

Folic acid fortification and public health: Report on threshold doses above which unmetabolised folic acid appear in serum

Mary Rose Sweeney,[1,3] Joseph McPartlin,[1] and John Scott[2]

Author information ▶ Article notes ▶ Copyright and License information ▶

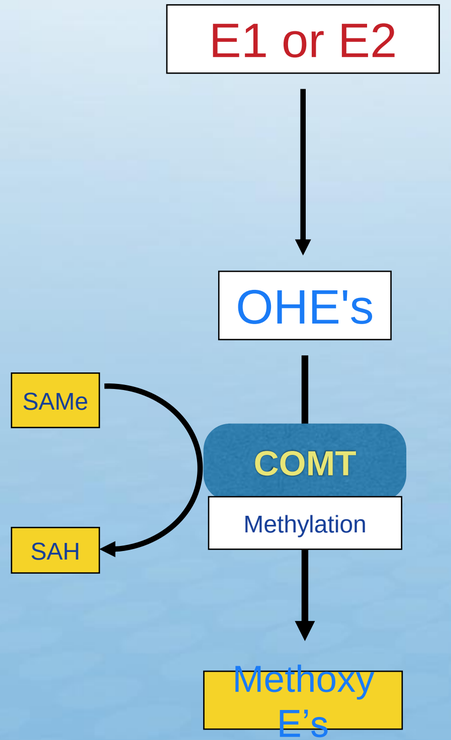
Abstract Go to:

Background

All flour in the USA is fortified with folic acid at a level of 140 μg/100 g which is estimated to supply an extra 100 μg daily to the average diet. Some researchers have advocated that this be increased to double and even four times this amount. Based on previous research these higher levels are likely to lead to the appearance of unmetabolised vitamin in the circulation, which may have safety implications for sub-groups of the population. The UK and the Republic of Ireland will likely introduce mandatory fortification also in the next year or so.

The aim of this study was to capture the short-term effect of folic acid fortification on unmetabolised folic acid in serum after chronic consumption of folic acid.

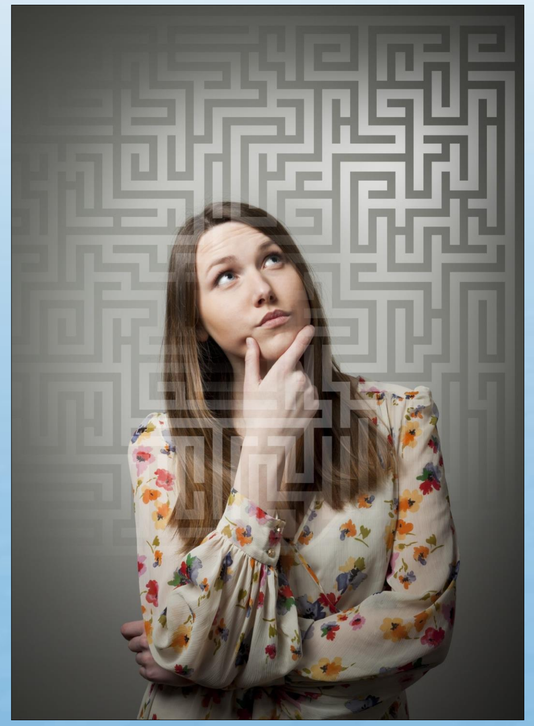
What Else Requires Methylation?
Estrogen



Estrogen Methylation

- Necessary to detoxify/biotransform hydroxy estrogens
- Inactivates potentially harmful estrogens such as 4OHE1 and 4OHE2
- Methoxy estrogens are anti-cell proliferative & possibly therapeutic

Testing Methylation Activity



- Vitamin Diagnostics methylation blood test
- Doctor’s Data methylation blood test
- Whole blood histamine
- Assessment of promoters & cofactors: OAT testing, amino acid testing

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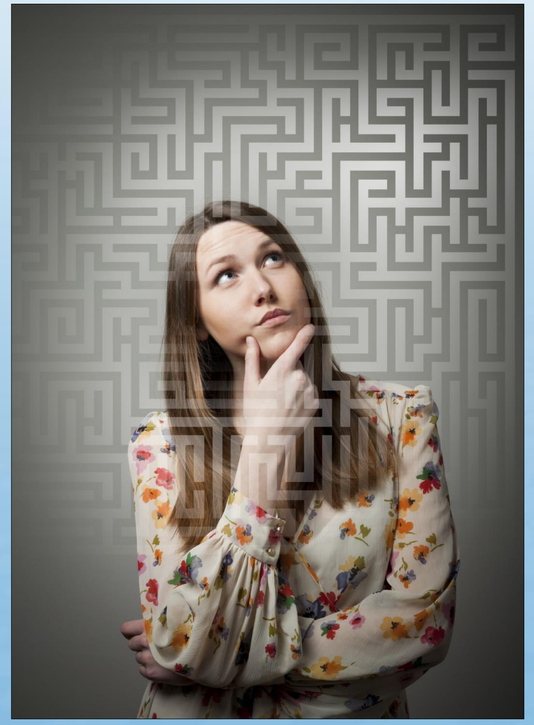
The Problem with "Treating" Methylation & Genetics



- Take this supplement for this gene (doesn't work in practice)
- Inherited genes don't change, but expression can
- Must treat the whole person, not just the individual parts
- There is a time to promote methylation, and certain instances when you don't want to

SOLUTION:

- A comprehensive, individualized & holistic approach

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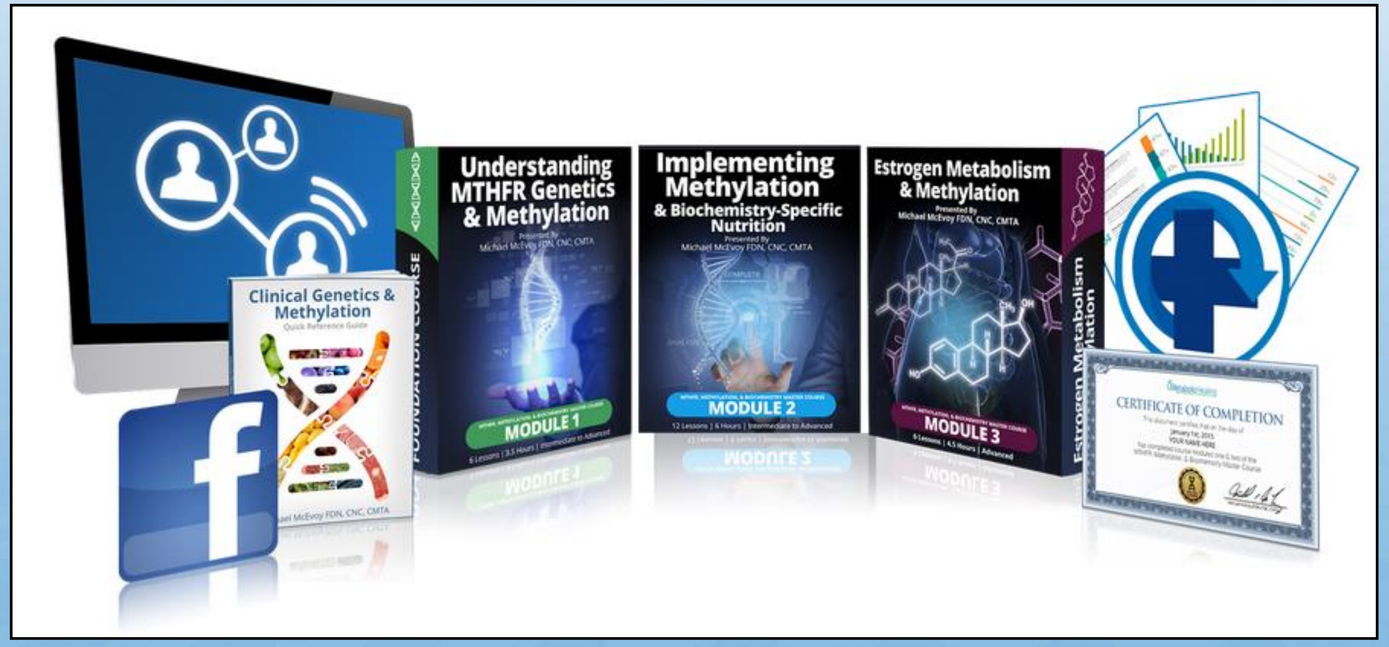
The MTHFR, Methylation & Biochemistry Master Course



- Simplification of complex concepts
- Lab Test interpretation
- Understanding methylation & genetics from a clinical perspective
- 3 modules, 16 hours of in-depth content
- Additional bonuses: talk to us at the table outside

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2,3 & 3,4 Semi-Quinones

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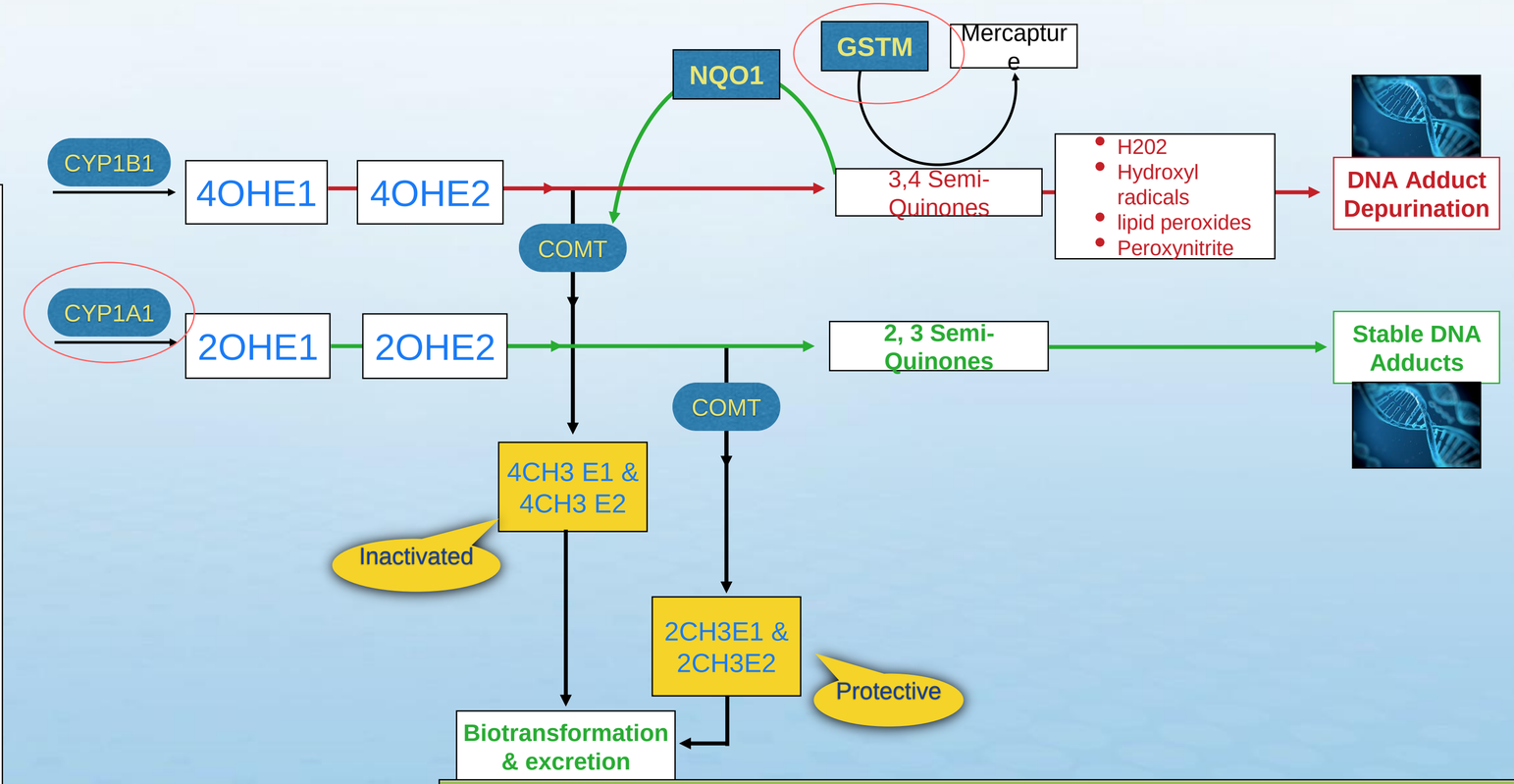
Estrogen Metabolism Genetics

Estrogen Metabolism Genetics analyzes various genetic factors related to the hydroxylation, methylation and metabolism of the estrogens. These genes can be found in association to various estrogen metabolites, such as: 2OHE1, 4OHE1, 16aOHE1, and various methoxy estrogens.

RS#	Call	Risk Allele	Gene	Variation	Result
rs762551	AA	C	CYP1A2	C164A	-/-
rs1799814	GG	T	CYP1A1*4	C2453A	-/-
rs1048943	TT	C	CYP1A1*2C	A4889G	-/-
rs4986883	TT	C	CYP1A1		-/-
rs1056836	CC	C	CYP1B1	L432V	+/+
rs1800440	TT	C	CYP1B1	N453S	-/-
rs10012	CG	C	CYP1B1	R48G	+/-
rs749292	GG	A	CYP19A1		-/-
rs727479	AA	A	CYP19A1		+/+
rs1004982	CC	C	CYP19A1		+/+
rs12248560	CT	T	CYP2C19*17		+/-
rs55785340	AA	G	CYP3A4*2		-/-
rs2740574	CC	C	CYP3A4*1B		+/+
rs4986910	AA	G	CYP3A4*3	M445T	-/-
rs4147565	--	A	GSTM1		NC
rs1056806	CC	T	GSTM1		-/-
rs4680	AA	A	COMT	V158M	+/+
rs4633	TT	T	COMT	H62H	+/+



Androgen Metabolism					
	DHEAS	Within range	535.0	ng/mg	60 - 2000
	Androsterone	Within range	1166.0	ng/mg	640 - 3000
	Etiocholanolone	Above range	2149.0	ng/mg	460 - 1700
	Testosterone	Low end of range	33.3	ng/mg	25 - 100
	5a-DHT	High end of range	15.7	ng/mg	9 - 16.7
	5a-Androstanediol	Low end of range	50.8	ng/mg	49 - 147
	5b-Androstanediol	Within range	114.6	ng/mg	30 - 147
	Epi-Testosterone	Below range	18.8	ng/mg	25 - 100
Estrogen Metabolites					
	Estrone(E1)	Within range	5.8	ng/mg	4 - 12
	Estradiol(E2)	Low end of range	0.6	ng/mg	0.5 - 1.6
	Estriol(E3)	Within range	2.9	ng/mg	2 - 6
	2-OH-E1	Above range	3.6	ng/mg	0 - 3
	4-OH-E1	Above range	0.6	ng/mg	0 - 0.5
	16-OH-E1	Within range	0.6	ng/mg	0 - 0.8
	2-Methoxy-E1	High end of range	1.8	ng/mg	0 - 2
	2-OH-E2	Within range	0.27	ng/mg	0 - 0.5