Summary of Personal Observations:

Steroid-Related Laboratory Patterns Prior to 5α-Reductase Inhibitor Exposure

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Contents

Disclaimer 3

Purpose 4

Phenotypes I Observed 4

Observations 5

Disclaimer

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Informational and Non-Clinical Nature:

This material is shared solely for informational and educational purposes. It reflects personal interpretation of laboratory patterns and biochemical pathways, not medical advice or clinical guidance. It is not intended to influence patient care, guide prescribing, alter treatment plans, or serve as a diagnostic, screening, or risk-assessment tool. No portion of this document is intended to establish a standard of care or to influence clinical decision-making in any jurisdiction.

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Clinicians remain solely responsible for exercising independent professional judgment and for complying with all applicable state, federal, and international laws and regulations, including but not limited to scope-of-practice requirements, diagnostic-testing rules, privacy and data-handling laws, genetic-information protections, informed-consent requirements, and medical-board standards regarding independent decision-making. Nothing in this document should be interpreted as direction, recommendation, or encouragement to act outside those standards or as guidance for ordering, interpreting, or applying any laboratory or genetic test.

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All laboratory information referenced here was shared by individuals who provided explicit permission for their deidentified data to be used for educational discussion for the April 24, 2026, First World Congress on PSSD, PFS, and PAS hosted by Corewell Health and related follow up materials. All data handling complied with applicable state, federal, and international laws governing health information, genetic information, and patient privacy, including but not limited to HIPAA, GINA, and relevant state genetic privacy statutes. No identifiable information is included, and no data was used without consent. These observations do not constitute research and should not be interpreted as research findings.

No Claims of Causation, Mechanism, or Clinical Relevance:

Nothing in this document should be interpreted as implying causation, mechanism, or clinical relevance. These observations are hypothesis-generating only and are not intended to support clinical decision-making.

Purpose

This document summarizes personal observations from a small number of individuals whose laboratory data I have reviewed. The goal is to describe patterns that appeared in these individuals' steroid-related laboratory results prior to exposure to 5α-reductase inhibitors (5ARIs). These observations are informal and exploratory, and their significance is unknown. They are included here solely for descriptive and educational purposes.

The focus of these observations is on situations where serum steroid production appears typical, while urinary steroid metabolite output appears low, absent, distorted, or atypically conjugated. In the individuals whose data I reviewed, this combination sometimes appeared alongside differences in downstream conversion, glucuronidation and other conjugation pathways, transport, or clearance. These patterns raised questions about whether some individuals may have pre-existing differences in androgen or neurosteroid metabolism that influence how their systems respond to metabolic disruption. These questions remain speculative, and their relevance is unknown.

These observations are exploratory and intended to support dialogue among clinicians and researchers; they have not been evaluated for diagnostic, predictive, or screening utility and should be interpreted within that context. These observations are hypothesis-generating only and should not be used to support clinical decision-making.

Phenotypes I Observed

1

Androgenic Signaling Failure

Penile atrophy, libido loss, erectile dysfunction, muscle loss

2

Neurosteroid Disruption

Cognitive effects, depression, psychosis, brain fog (potentially consistent with disruption of neurosteroid pathways)

3

"Melty"

Skin damage, collagen loss, peripheral edema, moon face resembling locally (limited Cushing's disease may be specific to certain tissue types)

Observations

1. Core Urinary Steroid Metabolomics

Tests I Used:

DUTCH Complete / Precision Analytical (reports sex hormones, adrenal hormones, cortisol/cortisone patterns, and hormone metabolites) and ARUP test 0080650 (Total 17-ketosteroids, 24h urine).

Marker	Personal Observations
Urinary testosterone	Key potential flag. Very low urinary testosterone despite typical serum values may suggest impaired urinary appearance, conjugation, transport, or clearance (UGTXXX/SLCO/ABCC genes).
5α-DHT / 5α-DHT metabolites	Provides a view of 5α-reduced androgen output.
Androsterone	Major 5α-reduced 17-ketosteroid. Lower values may indicate downstream androgen disposal through 5α arm.
Etiocholanolone	Major 5β-reduced counterpart. May distinguish 5α-specific impairment from global low androgen output. (normal ratio androsterone: etiocholanolone is 1:1)
5α- and 5β-androstanediol	Downstream DHT/3α-HSD signal and 5β comparator.
5β:5α metabolite ratio	Marked deviation from expected balance may suggest abnormal reductase-branch handling. (5b:5a 2:1)
E1:E2:E3 ratio	Marked deviation from expected male pattern may suggest aromatase diversion or abnormal estrogen metabolism.
Cortisol/cortisone metabolites	Provides a view off HPA-axis stress, 11β-HSD imbalance, cortisol-clearance patterns (potentially useful for identifying the “Melty” phenotype).
Total 17-ketosteroids, 24h urine	Global androgen/adrenal metabolite output; very low values despite normal serum precursors may suggest low androgen disposal (ARUP test 0080650).

Observations

2. Core Serum Steroid and Signal Panel

Tests I Used:

Comprehensive Steroid Panel 90392, Testosterone Panel 14966, and DHT 90567 (Quest Diagnostics)

Marker	Personal Observations
Total T, free/bioavailable T, SHBG, albumin	Production, available androgen signal, binding context, and the broader environment in which androgen activity occurs.
DHT, LC-MS/MS	Direct 5α-reductase product appearing in serum.
3α-androstanediol glucuronide	Peripheral DHT turnover marker. Lower values in the setting of normal or higher DHT may reflect differences in downstream reduction or glucuronidation.
Androstenedione, DHEA, DHEA-S	Upstream androgen/adrenal reserve and sulfation context.
Pregnenolone, progesterone, 17-OHP	Neurosteroid and backdoor-androgen substrate pool; higher 17-OHP with lower urinary androgens may reflect slower downstream disposal or reduced metabolite appearance.
Sensitive estradiol LC-MS/MS + estrone	Provides context for aromatase diversion and estrogen-pathway metabolism.
LH/FSH, prolactin	Gonadal-axis drive and broader hypothalamic–pituitary signaling context.

Observations

3. Conjugation, Export, Bile, Methylation, and Sulfation

Observation Note:

Steroid metabolites, bilirubin behavior, bile-related markers, and conjugated metabolite appearance may raise exploratory questions about how export, conjugation, or transport pathways are functioning.

Tests I Used:	Personal Observations
CMP; AST/ALT; ALP/GGT	Hepatic and biliary/export context; GGT may also reflect general glutathione-related turnover.
Total/direct/indirect bilirubin	Higher bilirubin, especially indirect, may reflect limited glucuronidation capacity or Gilbert-type physiology.
Serum bile acids; urinalysis with bilirubin/urobilinogen	Provides a potential clinical proxy for bile flow, export, and enterohepatic stress.
Creatinine/eGFR + cystatin C	Renal-clearance context, particularly useful in individuals with muscular or atypical body habitus
DHEA-S:DHEA ratio; pregnenolone sulfate if available	Potential view of sulfation/desulfation balance and neurosteroid sulfate context.
Homocysteine; B12 + MMA; folate/RBC folate; B6/PLP	Potential via of methylation and transsulfuration reserve.
2-OH:2-methoxy and 4-OH:4-methoxy estrogens	COMT/methylation output; higher catechol estrogens with lower methoxy-estrogens may reflect a methylation bottleneck.

Observations

4. Neurosteroid Vulnerability

Observation Note:

This pattern may reflect reduced appearance of 5α-neurosteroid products or differences in 5α-conversion. The significance of this observation is unknown.

Marker	Personal Observations
Pregnenolone and pregnenolone sulfate	Upstream substrate and excitatory neurosteroid pool.
Progesterone	Allopregnanolone precursor and backdoor androgen substrate.
Allopregnanolone	5α-reduced GABAergic neurosteroid directly affected by 5α-reductase inhibition.
Pregnanolone	5β-reduced comparator.
DHEA/DHEA-S; cortisol/cortisone metabolites	Neuroactive adrenal pool and HPA-axis/11β-HSD context.

5. Additional Options for Consideration

Tests I Used:

Test 504683 for 11-oxo-androgens panel (Labcorp)

Potential Genome Sequence List:

Self-developed whole genome sequencing list used informally for non-official research exploration.

Potential Add-Ons	Personal Observations
11-oxo-androgens panel	Useful when exploring whether adrenal-origin 11-oxygenated androgens may be contributing to overall androgen context or compensation patterns.
F2-isoprostanes, 8-OHdG, reduced:oxidized glutathione	Provides context for oxidative injury, DNA oxidation, and redox/detox reserve.
Whole-genome sequencing, ≥30x	Research-use review (via Sequencing.com) of genes involved in steroidogenesis, reductases, UGT pathways, SULT/STS pathways, transporters, methylation, and nuclear receptors.

Observations

6. Potential Highest Yield Abnormal Patterns

Marker	Personal Observations
Normal serum T + absent/near-zero urinary T	Production intact; urinary appearance, conjugation, transport, or clearance may be abnormal.
Normal serum androgens + near-absent urinary 5α metabolites	May reflect lower 5α-pathway output or reduced appearance of 5α-reduced metabolites in urine.
Low 17-ketosteroids despite normal serum precursors	Pattern may reflect reduced androgen-metabolite disposal rather than reduced production.
High DHT + low 3α-ADG	DHT present in serum but not appearing downstream as expected; may reflect differences in reduction or conjugation.
High 17-OHP + low urinary androgen metabolites	Upstream substrate accumulation with reduced downstream metabolite appearance.
Elevated bilirubin + low steroid glucuronide output	Possible glucuronidation fragility.
Abnormal DHEA-S:DHEA or steroid sulfate patterns	Possible sulfation/desulfation imbalance.
High estrogen metabolites + low androgen metabolites	May reflect aromatase diversion or rerouting of androgen flux.
Low allopregnanolone despite normal progesterone/pregnenolone	Pattern may reflect reduced 5α-neurosteroid conversion.
Serum/urine mismatch across multiple steroid families	Strongest potential signal that conjugation, transport, or export pathways may differ from expected patterns.
“Absurd value X”	Extremely out-of-range or assay-maxed values may highlight areas worth a closer review. Ratio-based discrepancies can also be informative; for example, a testosterone of 950 ng/dL with a DHT of 35 ng/dL may fall within reference ranges individually, but the ratio between them may be atypical.

Observations

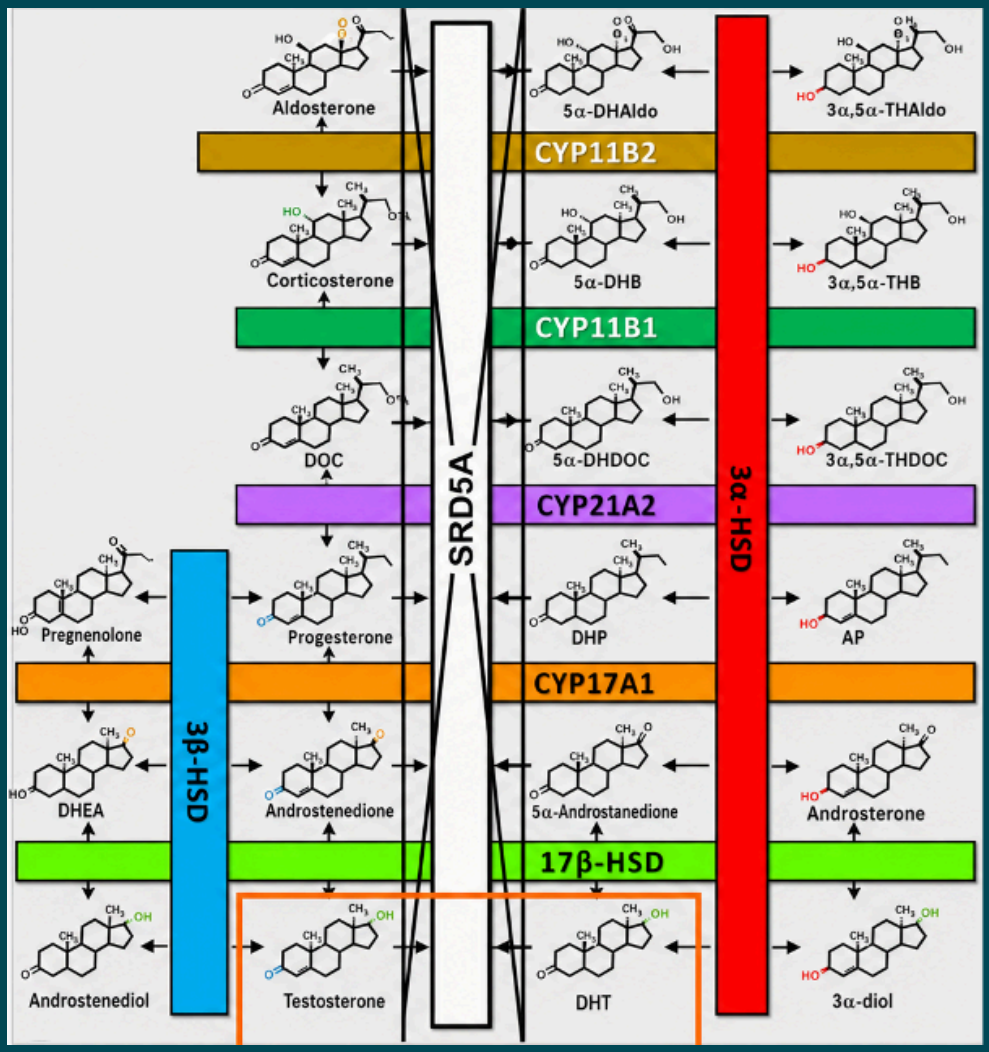
7. Possible Interpretation Domains

Observation Note:

The proposed framework highlights situations where serum steroid production appears typical while urinary steroid metabolite output is absent, reduced, or atypically conjugated. These patterns may raise exploratory questions about how androgen and neurosteroid clearance pathways are functioning.

Domain	Personal Observations
Low urinary androgen appearance	Near-zero urinary T, low total androgen metabolites, low 17-ketosteroids.
5α/5β imbalance	Abnormal androsterone:etiocholanolone ratio, abnormal DHT:T, low 3α-ADG:DHT, low allopregnanolone:progesterone.
Phase-II fragility	Elevated bilirubin, low glucuronide output, abnormal sulfate patterns, abnormal catechol:methoxy estrogen ratios.
Export / enterohepatic stress	Abnormal GGT, ALP, bile acids, direct bilirubin, or recurrent serum-high/urine-low steroid mismatch. History of severe acne may be relevant in the context of transporter-related intracellular retention, though this is speculative.
Aromatase-diversion risk	High E2/E1 relative to T, high SHBG with low free T, high estrogen metabolites with low androgen metabolites.
Neurosteroid vulnerability	Low allopregnanolone, high pregnenolone sulfate relative to allopregnanolone, or progesterone/17-OHP accumulation with low downstream metabolites.

Proposed Interpretation Domains Continued



It is important to remember that SRD5A1 and SRD5A2 participate in the metabolism of many of the compounds shown above. Differences in these metabolites—whether at baseline or after drug exposure—may raise exploratory questions about how an individual's androgen or neurosteroid pathways respond to 5α-reductase inhibition. These observations are speculative and do not identify or confirm any genetic predisposition.

At this time, this framework represents an early, exploratory attempt to organize patterns discussed by clinicians and researchers following the First World Congress for PFS, PSSD, and PAS. It is not validated, not predictive, and not intended to determine whether any individual will or will not develop symptoms after exposure to a 5-ARI.

Any associations described here are speculative and do not identify or confirm genetic susceptibility or pathophysiology. Please see the Disclaimer on page 3 for key details.