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# UNIQUE AAS FEATURES BY DRUG

TYPE-IIX

29 June 2024 – Presented 8:30 PM EST &

6 July 2024 – Presented 11:45 AM EST

## TRIENE (CLASS) – TRENBOLONE

- INSULIN sensitivity
- SATELLITE CELL proliferative response to IGF-I
- TISSUE-LEVEL antiglucocorticoid effects via decreased GR #
- ATHEROSCLEROTIC and pro-inflammatory via MR antagonism

# HETEROCYCLICALLY-RINGED – STANOZOLOL

- MITOGENIC and myogenic effects by increasing *free* IGF-I, the tiny bioavailable fraction ( $t_{1/2} = 12$  min vs. 15 h for ternary complexes (IGF-I + IGFBP-3 + ALS [acid labile subunit]; 10% of total cIGF-I)
- GLUCOCORTICOID modulatory by directly, and indirectly via its 16 $\beta$ -hydroxylated metabolite, 16 $\beta$ -ST, negatively regulating the LAGS (low affinity glucocorticoid-binding site) in liver
- ANTIPROGESTAGENIC effects by PR antagonism
- REMARKABLY long  $t_{1/2} > 24$  h
- *JOINT aching*: Inhibits DNA synthesis in synovial fibroblasts &  $\uparrow$  CI-INH

# ANDROST-4-ENE-3-ONES – FLUOXYMESTERONE

- AMPLIFICATION via  $5\alpha$ -reduction supplants T's role in male sexual function, evidenced by JFK's (Addison's disease) notoriety for sexual prowess
- PARTICULARLY fat mass reductive
- SYSTEMIC antigluocorticoid effect via GR antagonism

## ANDROST-4-ENE-3-ONES – MENT

- POTENTLY suppressive via aromatization to  $7\alpha$ -ME
- ITS  $17\alpha$ -alkylated analogue is mibolerone (Cheque drops®) – potently androgenic and virtually nil anabolism

# ANDROST-4-ENE-3-ONES – TESTOSTERONE

- BIODIDENTICAL
- SUPPLANTS male sexual function via  $5\alpha$ -reduction amplification & all the benefits of estrogens (see [Estrogen Functions in Men \(MesoRx Article\)](#)):
  - Lipid management
  - Sexual function
  - Bone metabolism
  - Male behavior
  - *etc.*

## ANDROST-4-ENE-3-ONES – NANDROLONE

- AROMATIZATION but  $5\alpha$ -reduction diminution causes sexual function problems, in addition to
- INCREASES dopamine metabolism (i.e., net breakdown), causing sexual dysfunction, depression, learning & memory deficits (a la increased HVA)
- JOINT benefits via increased ECM type I collagen deposition but also cardiovascular risks, marked by increased ACE (angiotensin-I converting enzyme)

# OXANDROLONE

- 5  $\alpha$ -ANDROSTAN-3-ONE
- PARTICULARLY lipolytic by enhancing hepatic ketosis



# METHASTERONE

- 5  $\alpha$ -ANDROSTAN-3-ONE
- 2 $\alpha$ -METHYLATION dramatically increases anabolic and decreases androgenic potency
- VIRTUALLY excreted unmetabolized
- THE MOST liver toxic

# I-ENES – I-TESTOSTERONE

- DHB; Dihydroboldenone, is *not* a metabolite of EQ
- INCREASES DHT (perhaps via 17  $\beta$ -HSDI inhibition but *not* 5  $\alpha$ -reduction)
- INCREASES CRP directly and/or by requiring guaiacol to hold in solution

# 1,4-DIENES – METANDIENONE

- SYSTEMIC antigluocorticoid effect by decreasing ACTH
- POTENTLY suppressive
- PARTICULARLY hematopoetic

## 2-ENES – METEPITIOSTANE

- CHEMICALLY remarkable, these AAS lack a 3-keto group (3-deoxysteroids), that acts as an electron acceptor at the AR's N-terminus. Here, a 2,3 alpha-epithio (sulfur atom spanning C-2 and C-3) "claws back" lost affinity
- ACTS *partly* as a prodrug to desoxymethyltestosterone (Madol)
- POTENTLY antigynecomastic, in Japan its nonmethylated analogue epitiostanol is used clinically for gynecomastia treatment, and is more effective at this at 50 mg than 50 mg drostanolone

# QUESTIONS?

THANK YOU FOR JOINING ME FOR THIS SYMPOSIUM

IF additional questions pop up that you'd like answered, after this segment, use the Support Request Form at the bottom of <https://ampouletude.com> to ask away! Or ask on Meso, ProM, etc.