Dr. John Crisler
March 19, 1958 - January 16, 2019
The Dangers of Inhibiting Estrogen In Men

Dr. Rob Kominiarek, DO, FACOFP
Estradiol is a pleiotropic hormone that has instrumental influence over numerous critical functions related to the cardiac and vascular system, bone and mineral metabolism, cognition, memory, mood, balance, age related neurodegenerative disorders and lipolysis of fat.
Evidence suggests that estradiol has neurotrophic and neuroprotective properties and promotes the survival and viability of intimate brain structures. The benefits of estradiol on neuroprotection are attributed to genomic and non-genomic signaling, regulation of mitochondrial energy and antioxidant action.
Damaged neurons increase the production of aromatase, the enzyme that is responsible for the conversion of androgens into estradiol. Our own astrocytes after suffering trauma increase aromatase expression to exert the numerous benefits of estrogen to these intimate structures.
A Quick Search Revealed

• Anastrozole and bone > 16,000 articles
• Anastrozole and cognition > 4000 articles
• Anastrozole and vascular > 10,500 articles
• Anastrozole & sexual function >10,000 articles

• And I read all 40,000 of them....I live in Ohio and it was winter.
So Why Are You Blocking The Aromatization Of Testosterone Into Estradiol?
Benefits Of Estradiol In Men

- Bone health
- Arterial health
- Heart health
- Brain health
- Cognitive health
- Fat reduction
- Colon health
Benefits Of Estradiol In Men

• Multiple neurologic and cerebral benefits

• Multiple vascular benefits

• Improves static balance, preventing falls

• Protects against Alzheimer's, Stokes, Memory Disorders, Macular Degeneration, Cataracts, Heart Disease, Colon Cancer, Osteopenia, Osteoporosis
Estradiol Deficiency Leads To

- Fatigue
- Mood swings
- Depression
- Decreased libido
- Heart disease
- Vascular disease
- Cognitive decline
- Neurodegenerative disorders
- Impaired Bone and Mineral Metabolism
Blocking Estrogen Causes Osteoporosis


Osteoporosis can produce profound morbidity and mortality in men, much as it can in women.

The chief causes of secondary osteoporosis in men are excessive alcohol use, treatment with glucocorticoids, and hypogonadism, including that experienced by men receiving androgen deprivation therapy for prostate cancer.
Blocking Estrogen Causes Osteoporosis


Men steadily lose bone mineral density with aging, and one in five men over 50 will suffer an osteoporotic fracture. Almost 30% of all hip fractures are in men, and the mortality following a fracture is substantially higher in men than women.
Blocking Estrogen Causes Osteoporosis


The role of sex steroids in contributing to osteoporosis in older men, even in the absence of overt hypogonadism. Both testosterone and estradiol are present in the blood; most of the estradiol (85%) is derived from testosterone by peripheral aromatization.

Dr. Rob Kominiarek DO, FACOFP | Hormones Made Simple

For some time we’ve known that bone density, rate of bone loss, and fracture incidence all correlate more closely with estradiol than with testosterone. Furthermore, increased bone turnover was shown to be suppressed by estradiol and not testosterone in older men by very elegant studies.
Blocking Estrogen Causes Osteoporosis


Interestingly, the threshold of bioavailable estradiol below which bone turnover and bone loss are accelerated appears to be similar in men and women, at around 40 pmol/L. (10pg/ml)
Blocking Estrogen Causes Osteoporosis


In symptomatic hypogonadism, testosterone is appropriate, and improves bone density as well as increasing muscle mass and strength.
Blocking Estrogen Causes Osteoporosis


Osteoporosis in men in an important and inadequately appreciated problem. This is partly the result of the earlier disproportionate emphasis osteoporosis in women.
Blocking Estrogen Causes Osteoporosis


Low serum estradiol in men, which is associated with low bone density, increases the risk for hip fractures.
Blocking Estrogen Causes Osteoporosis


Although serum testosterone in men has not been strongly associated with bone density, we found that men with both low estradiol and low testosterone have the greatest risk of hip fracture.
Blocking Estrogen Causes Osteoporosis


Although the key role of estradiol for the bone health of men is increasingly recognized among researchers in bone metabolism, it is not well known in clinical general medicine.
In this population-based sample of elderly men followed longitudinally for 18 years, we found that low estradiol levels are associated with an increased risk of hip fracture.
Blocking Estrogen Causes Osteoporosis


Furthermore, the one year mortality rate after hip fracture has been reported to be 37% higher in men, approximately twofold higher than in women.
Blocking Estrogen Causes Osteoporosis


Conclusion: Men with low estradiol levels are at an increased risk for future hip fracture. Men with both low estradiol and low testosterone levels seem to be at greatest risk for hip fracture.
Bone Study

Effects of aromatase inhibition vs. testosterone in older men with low testosterone: randomized controlled trial. 1J. P. Dias, 1D. Melvin, 2E.M. Simonsick, 1O. Carlson, 2M.D. Shardell, 2L. Ferrucci, 2C. W. Chia, 3S. Basaria and 1J. M. Egan

In summary, this proof-of-concept study confirms that aromatization of T is required for maintaining BMD in older men with low-T levels. The trial also uncovered the novel finding that aromatization of T is required for improvement in fast gait speed.

Estrogen deficiency has recently been implicated in the pathogenesis of male osteoporosis.

Estrogen deficiency is much more prevalent than androgen deficiency in primary male osteoporosis.
Bone Study


Anastrozole therapy increased bioavailable testosterone and DHT compared with placebo. These increases in androgen levels and the associated mild decrease in estradiol were associated with a statistically significant decrease in posterior-anterior spine BMD vs. placebo as measured by DXA.
Bone Study


In the minds of well-informed male medical professionals, more testosterone is clearly “better,” whereas estrogen is largely irrelevant.

The authors chose to test the effects of increasing endogenous testosterone production by blocking aromatization of testosterone to estradiol using a potent orally administered aromatase inhibitor, anastrozole.

Posterior-anterior (PA) spine BMD decreased by 1.7% in the anastrozole group.
Bone Study


The inescapable conclusion from these data is that the 50% increase in testosterone levels was trumped by the much more modest 20% reduction in estradiol levels, leading to negative skeletal effects of anastrozole therapy in aging men.

These findings are not surprising and are perhaps predictable, based on the now extensive body of evidence for a critical role for estrogen in regulating the male skeleton.
Bone Study


In summary, whereas the concept of using an aromatase blocker to enhance endogenous testosterone production was an attractive one, it does not appear to be a viable approach for preventing age-related declines in bone mass or in improving parameters of body composition in men. These findings also suggest that as males, we should perhaps be just as interested in our estradiol levels as we seem to be in our testosterone levels, if not more so.
Bone Study


Although previous studies in men had shown that serum estradiol levels were related to bone density and that estrogen regulated bone turnover and bone loss in elderly men, more recent studies have now also demonstrated that serum estradiol, and not serum testosterone, levels are the most robust hormonal predictors of fracture risk in aging men.
Bone Study


Young adult males who cannot produce or respond to estrogen (E) are osteopenic.

Bone resorption markers increased significantly in the absence of both hormones and were unchanged in men receiving both hormones. By two-factor ANOVA, E played the major role in preventing the increase in the bone resorption markers, whereas T had no significant effect.
Bone Study


We conclude that in aging men, E is the dominant sex steroid regulating bone resorption, whereas both E and T are important in maintaining bone formation.

The subjects were administered a long acting GnRH agonist (leuprolide acetate, Lupron-Depot; Takeda Chemical Industries, Osaka, Japan), 7.5 mg intramuscularly, to suppress endogenous T and E production. They were also started on the aromatase inhibitor letrozole.
Bone Study


Thus, NTx excretion increased significantly in Group A (–T, –E) (by 35%), but remained unchanged in Group D (+T, +E). NTx showed a small (9%), but in this case, significant, increase in Group B (–T, +E) and, as for Dpd, a larger increase in Group C (+T, –E) (22%).
Bone Study


Using the two-factor ANOVA model, we found a highly significant effect of E (P = 0.0002), but a nonsignificant effect of T (P = 0.085) on urinary NTx excretion.
Bone Study


Our study clearly establishes that E regulates both bone formation and resorption in normal elderly men in a direction that would oppose bone loss. By directly manipulating T and E levels and assessing the changes in bone turnover markers, these data thus provide unequivocal proof of the key role that E plays in skeletal metabolism in men.
Bone Study


These data also suggest that elderly men with low bioavailable E levels might benefit from either low dose E replacement or from the use of selective estrogen receptor modulators (SERMs) that have an agonist effect on the skeleton but are not feminizing.

Indeed, in a preliminary study, Taxel et al. (57) treated nine elderly men with either 0.5 mg or 2.0 mg daily of micronized 17b-E2 and found significant reductions in bone resorption markers.

Dr. Rob Kominiarek DO, FACOFP | Hormones Made Simple

Anderson et al. (58) treated 21 eugonadal men with osteoporosis with intramuscular T and found a significant increase in lumbar spine BMD, which was correlated with changes in serum E2, but not T levels.

Finally, our findings would suggest that SERMs might have clinical utility in at least the subset of elderly men with low bioavailable E levels.
Bone Study


In older men, aromatase inhibition increases testosterone levels, decreases estradiol levels, and appears to decrease BMD. Aromatase inhibition does not improve skeletal health in aging men with low or low normal testosterone levels.
Heart Study


A higher serum estradiol level was associated with lower risk for CVD events in older men. The findings are consistent with the hypothesis that endogenous estrogen has vasculoprotective influences in men.
Heart Study


Endogenous estrogens produced by aromatization of androgenic precursors are of physiological importance.

Male patients administered 1 mg of Estradiol daily

Systolic and diastolic blood pressures were reduced. HDL cholesterol levels increased significantly, and vasoconstrictor responses to the NO synthase inhibitor N(G)-monomethyl-L-arginine were enhanced.
Heart Study


We conclude that low-dose estrogen supplementation in hypogonadal men is well tolerated, lowers blood pressure, and may affect vascular reactivity in a manner that is potentially beneficial, through several mechanisms, including enhancement of basal NO release.
Heart Study

Physiological levels of estradiol stimulate plasma high density lipoprotein cholesterol levels in normal men. Bagatell CJ1, Knopp RH, Rivier JE, Bremner WJ. J Clin Endocrinol Metab. 1994 Apr;78(4):855-61.

We then administered testosterone (T) enanthate (100 mg, im, weekly) to restore T levels to the baseline range, and we administered an aromatase inhibitor, testolactone (Teslac), to prevent the normal conversion of T to E2, thereby producing a selective estrogen deficiency state in normal young men.

We found that in men who received Nal-Glu plus T plus Teslac, E2 levels were profoundly suppressed during treatment.
Heart Study

Physiological levels of estradiol stimulate plasma high density lipoprotein2 cholesterol levels in normal men. Bagatell CJ1, Knopp RH, Rivier JE, Bremner WJ. J Clin Endocrinol Metab. 1994 Apr;78(4):855-61.

Plasma HDL cholesterol, particularly, the HDL2 fraction, decreased significantly in response to the low serum E2 level. Plasma apoprotein-AI levels also decreased significantly.

We conclude that in men, physiological levels of E2 are important in maintaining plasma levels of HDL cholesterol, especially the HDL2 fraction.
Heart Study


In healthy men, estradiol level is associated with levels of apolipoprotein E and regulation of systolic and diastolic blood pressure. In addition, it acts along with testosterone to maintain normal levels of insulin sensitivity. The effects of estrogens can also be explained by their action as regulators of nitric oxide.
Heart Study


In healthy men of fertile age subjected to estrogen suppression, a reduction was observed in plasma levels of HDL-C, particularly fraction 2, and a significant reduction in flow-mediated vasodilation.

Estrogen supplementation in healthy men aged more than 65 years reduces the levels of homocysteine, fibrinogen, and plasminogen activator inhibitor (PAI), and has a favorable effect on very low-density lipoprotein (VLDL-C) cholesterol, LDL-C, and HDL-C.
Physiological levels of estradiol stimulate plasma high density lipoprotein2 cholesterol levels in normal men. Bagatell, CJ, Knopp, RH, Rivier, JE, Bremner, WJ. The Journal of Clinical Endocrinology & Metabolism, Volume 78, Issue 4, 1 April 1994, Pages 855–861

We conclude that in men, physiological levels of E2 are important in maintaining plasma levels of HDL cholesterol, especially the HDL2 fraction.

These observations suggest that estrogen, in the amount normally produced in men, may offer some degree of protection against cardiovascular disease in males, as they do in women.
Heart Study


A lower E2 level was associated with higher concentrations of total cholesterol (TC) and low density lipoprotein cholesterol. A lower DHEA-S level was associated with a lower concentration of high density lipoprotein cholesterol (HDLC) and a higher ratio of TC / HDLC.

These results suggest that higher levels of E2 and DHEA-S, at least in physiological concentrations, are related to the favorable lipid and lipoprotein levels in men.

In the community-based sample, a higher serum estradiol level was associated with lower risk for CVD events in older men. The findings are consistent with the hypothesis that endogenous estrogen has vasculoprotective influences in men.

Participants were randomized to receive weekly IM injections of 100 mg Testosterone Enanthate or 100 mg Testosterone Enanthate plus 1 mg daily of Anastrozole to block the conversion of testosterone to estradiol.
Brain Study


Only the group with elevated estradiol levels demonstrated significant verbal memory improvement.

In healthy older men, improvement in verbal memory induced by testosterone administration depends on aromatization of testosterone to estradiol.

Estrogens appear to have significant effects on the male biological system. Favorable effects have been noted on bone, brain and cardiovascular physiology.

Oestrogen effects on the cardiovascular system include those on lipid profiles, fat distribution, endocrine/paracrine factors produced by the vascular wall (such as endothelins, nitric oxide), blood platelets, inflammatory factors and coagulation.

The effect of estrogen on cognition and memory function itself could play a role but the neuroprotective effects of estrogen in the brain may be even more important.

Estrogen may make the neuronal network more resilient against toxic damage as it provides a protective effect against free radical induced cell injury.
Brain Study


In healthy older men, improvement in verbal memory induced by testosterone administration depends on aromatization of testosterone to estradiol.
Sexual Function


The amount of testosterone required to maintain lean mass, fat mass, strength, and sexual function varied widely in men. Androgen deficiency accounted for decreases in lean mass, muscle size, and strength; estrogen deficiency primarily accounted for increases in body fat; and both contributed to the decline in sexual function.
Body Composition


Effects of Testosterone without Aromatase Inhibition on Body Composition

In cohort 1, the percentage of body fat increased significantly in men who received 0 g, 1.25 g, or 2.5 g of testosterone daily, as compared with men who received 5 g daily, and it decreased significantly in men who received 10 g of testosterone daily, as compared with each of the other groups.
Body Composition


Effects of Testosterone with Aromatase Inhibition on Body Composition

In cohort 2, the percentage of body fat increased in all groups when the aromatization of testosterone to estradiol was inhibited. The magnitudes of these increases were similar with doses of 0 g, 1.25 g, 2.5 g, and 5 g of testosterone daily, a finding that suggests a predominantly estrogenic effect.
Testosterone therapy is associated with a significant reduction in obesity and fat mass.

Level of Evidence. 1b
Could It Be That Estradiol Plays A More Significant Role Than Testosterone In Men?
Maybe We (Men) Should Be More Concerned With Our Estradiol Levels Than Testosterone
45 Year Old Male

On testosterone therapy
Anastrozole 1 mg TIW for three years

Trips and falls while walking and fractures hip.
No one questions why.
Common Side Effects of Aromatase Inhibition

• Trouble sleeping (insomnia)
• Depression/anxiety
• Mood changes
• Problems with your fingers while gripping
• High blood pressure
• Blurred vision/visual disturbances
• Stroke
• Osteoporosis/fracture
• Flushing and sweating (hot flashes/hot flushes)
• Breast swelling/tenderness/pain/edema
• Body aches and pains (back pain, bone pain, joint pain, stiffness, worsening arthritis)
• Numbness, tingling, cold feeling, or weakness in your hand or wrist
• Tiredness/weakness
• Vaginal bleeding
• Constipation
• Diarrhea
• Nausea/vomiting
• Upset stomach
• Jaundice
• Loss of appetite
• Headache
• Dry mouth
• Scratchy or sore throat
• Increased cough
• Dizziness
• Hair thinning
• Weight changes
<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Flag</th>
<th>Units</th>
<th>Reference Interval</th>
<th>Lab</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baso (Absolute)</td>
<td>0.0</td>
<td></td>
<td>x10E3/uL</td>
<td>0.0 - 0.2</td>
<td>02</td>
</tr>
<tr>
<td>Immature Granulocytes</td>
<td>0</td>
<td></td>
<td>%</td>
<td></td>
<td>02</td>
</tr>
<tr>
<td>Immature Grans (Abs)</td>
<td>0.0</td>
<td></td>
<td>x10E3/uL</td>
<td>0.0 - 0.1</td>
<td>02</td>
</tr>
</tbody>
</table>

**Comp. Metabolic Panel (14)**

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Flag</th>
<th>Units</th>
<th>Reference Interval</th>
<th>Lab</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, Serum</td>
<td>103</td>
<td>High</td>
<td>mg/dL</td>
<td>65 - 99</td>
<td>02</td>
</tr>
<tr>
<td>BUN</td>
<td>11</td>
<td></td>
<td>mg/dL</td>
<td>6 - 24</td>
<td>02</td>
</tr>
<tr>
<td>Creatinine, Serum</td>
<td>1.19</td>
<td></td>
<td>mg/dL</td>
<td>0.76 - 1.27</td>
<td>02</td>
</tr>
<tr>
<td>eGFR If NonAfrican Am</td>
<td>72</td>
<td></td>
<td>mL/min/1.73</td>
<td>&gt;59</td>
<td></td>
</tr>
<tr>
<td>eGFR If African Am</td>
<td>83</td>
<td></td>
<td>mL/min/1.73</td>
<td>&gt;59</td>
<td></td>
</tr>
<tr>
<td>BUN/Creatinine Ratio</td>
<td>9</td>
<td></td>
<td></td>
<td>9 - 20</td>
<td>02</td>
</tr>
<tr>
<td>Sodium, Serum</td>
<td>143</td>
<td></td>
<td>mmol/L</td>
<td>134 - 144</td>
<td>02</td>
</tr>
<tr>
<td>Potassium, Serum</td>
<td>4.9</td>
<td></td>
<td>mmol/L</td>
<td>3.5 - 5.2</td>
<td>02</td>
</tr>
<tr>
<td>Chloride, Serum</td>
<td>104</td>
<td></td>
<td>mmol/L</td>
<td>97 - 108</td>
<td>02</td>
</tr>
<tr>
<td>Carbon Dioxide, Total</td>
<td>25</td>
<td></td>
<td>mmol/L</td>
<td>18 - 29</td>
<td>02</td>
</tr>
<tr>
<td>Calcium, Serum</td>
<td>9.6</td>
<td></td>
<td>mg/dL</td>
<td>8.7 - 10.2</td>
<td>02</td>
</tr>
<tr>
<td>Protein, Total, Serum</td>
<td>6.8</td>
<td></td>
<td>g/dL</td>
<td>6.0 - 8.5</td>
<td>02</td>
</tr>
<tr>
<td>Albumin, Serum</td>
<td>4.5</td>
<td></td>
<td>g/dL</td>
<td>3.5 - 5.5</td>
<td>02</td>
</tr>
<tr>
<td>Globulin, Total</td>
<td>2.3</td>
<td></td>
<td>g/dL</td>
<td>1.5 - 4.5</td>
<td>02</td>
</tr>
<tr>
<td>A/G Ratio</td>
<td>2.0</td>
<td></td>
<td></td>
<td>1.1 - 2.5</td>
<td>02</td>
</tr>
<tr>
<td>Bilirubin, Total</td>
<td>1.0</td>
<td></td>
<td>mg/dL</td>
<td>0.0 - 1.2</td>
<td>02</td>
</tr>
<tr>
<td>Alkaline Phosphatase, S</td>
<td>52</td>
<td></td>
<td>IU/L</td>
<td>39 - 117</td>
<td>02</td>
</tr>
<tr>
<td>AST (SGOT)</td>
<td>26</td>
<td></td>
<td>IU/L</td>
<td>0 - 40</td>
<td>02</td>
</tr>
<tr>
<td>ALT (SGPT)</td>
<td>24</td>
<td></td>
<td>IU/L</td>
<td>0 - 44</td>
<td>02</td>
</tr>
</tbody>
</table>

**Lipid Panel With LDL/HDL Ratio**

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Flag</th>
<th>Units</th>
<th>Reference Interval</th>
<th>Lab</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol, Total</td>
<td>173</td>
<td></td>
<td>mg/dL</td>
<td>100 - 199</td>
<td>02</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>98</td>
<td></td>
<td>mg/dL</td>
<td>0 - 149</td>
<td>02</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>38</td>
<td>Low</td>
<td>mg/dL</td>
<td>&gt;39</td>
<td>02</td>
</tr>
</tbody>
</table>

**Comment**

According to ATP-III Guidelines, HDL-C >59 mg/dL is considered a negative risk factor for CHD.

**VLDL Cholesterol Cal**

<table>
<thead>
<tr>
<th>Result</th>
<th>mg/dL</th>
<th>5 - 40</th>
</tr>
</thead>
</table>

**LDL Cholesterol Calc**

<table>
<thead>
<tr>
<th>Result</th>
<th>mg/dL</th>
<th>0 - 99</th>
</tr>
</thead>
</table>

**LDL/HDL Ratio**

| Result | ratio units | 0.0 - 3.6 | 02  |

**Please Note:**

<table>
<thead>
<tr>
<th>LDL/HDL Ratio</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avg. Risk</td>
<td>1.0</td>
<td>1.5</td>
</tr>
<tr>
<td>Avg. Risk</td>
<td>3.6</td>
<td>3.2</td>
</tr>
<tr>
<td>2X Avg. Risk</td>
<td>6.2</td>
<td>5.0</td>
</tr>
<tr>
<td>3X Avg. Risk</td>
<td>8.0</td>
<td>6.1</td>
</tr>
</tbody>
</table>

**Testosterone, Serum**

| Result | mg/dL | 348 - 1197 | 02  |

Dr. Rob Kominiarek DO, FACOFP | Hormones Made Simple
<table>
<thead>
<tr>
<th>TESTS</th>
<th>RESULT</th>
<th>FLAG</th>
<th>UNITS</th>
<th>REFERENCE INTERVAL</th>
<th>LAB</th>
</tr>
</thead>
<tbody>
<tr>
<td>HORMONES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free Testosterone (Direct)</td>
<td>43.2</td>
<td>High</td>
<td>pg/mL</td>
<td>6.8 - 21.5</td>
<td>01</td>
</tr>
<tr>
<td>FSH and LH</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LH</td>
<td>0.1</td>
<td>Low</td>
<td>mIU/mL</td>
<td>1.7 - 8.6</td>
<td>02</td>
</tr>
<tr>
<td>FSH</td>
<td>&lt;0.2</td>
<td>Low</td>
<td>mIU/mL</td>
<td>1.5 - 12.4</td>
<td>02</td>
</tr>
<tr>
<td>Sex Hormone Binding Globulin</td>
<td>24.0</td>
<td></td>
<td>nmol/L</td>
<td></td>
<td>03</td>
</tr>
</tbody>
</table>

**Reference Range:**
- Pubertal: 16.0 - 100.0
- 20 - 49y: 16.5 - 55.9
- >49y: 19.3 - 76.4

| DHEA-Sulfate                 | 240.7  |      | ug/dL  | 71.6 - 375.4       | 02  |
| TSH                          | 2.440  |      | uIU/mL | 0.450 - 4.500      | 02  |
| Prolactin                    | 11.3   |      | ng/mL  | 4.0 - 15.2         | 02  |
| Estradiol                    | <5.0   | Low  | pg/mL  | 7.6 - 42.6         | 02  |

**Note:** Roche ECLIA methodology

**Prostate-Specific Ag, Serum**

Prostate Specific Ag, Serum 0.9 ng/mL 0.0 - 4.0 02

According to the American Urological Association, Serum PSA should decrease and remain at undetectable levels after radical prostatectomy. The AUA defines biochemical recurrence as an initial PSA value 0.2 ng/mL or greater followed by a subsequent confirmatory PSA value 0.2 ng/mL or greater. Values obtained with different assay methods or kits cannot be used interchangeably. Results cannot be interpreted as absolute evidence of the presence or absence of malignant disease.

**IGF-1**

Insulin-Like Growth Factor I 363 High ng/mL 67 - 205 01

**Vitamin D, 25-Hydroxy**

Vitamin D deficiency has been defined by the Institute of Medicine and an Endocrine Society practice guideline as a level of serum 25-OH vitamin D less than 20 ng/mL (1,2). The Endocrine Society went on to further define vitamin D insufficiency as a level between 21 and 29 ng/mL (2).

Scan Information:
Scan Date: January 31, 2017    ID: A0131170B
Scan Type: x Left Hip
Analysis: January 31, 2017 11:43 Version 13.0.3
Hip
Operator:
Model: Discovery A (S/N 83116)
Comment:

DXA Results Summary:

<table>
<thead>
<tr>
<th>Region</th>
<th>Area (cm²)</th>
<th>BMC (g)</th>
<th>BMD (g/cm²)</th>
<th>T-score</th>
<th>Z-score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck</td>
<td>4.81</td>
<td>11.18</td>
<td>2.322</td>
<td>10.2</td>
<td>10.9</td>
</tr>
<tr>
<td>Total</td>
<td>40.34</td>
<td>68.00</td>
<td>1.686</td>
<td>4.3</td>
<td>4.6</td>
</tr>
</tbody>
</table>

Total BMD CV 1.0%, ACF = 1.029, BCF = 0.992, TH = 5.662
WHO Classification: Normal
Fracture Risk: Not Increased

Physician's Comment:
Scan Information:
Scan Date: January 31, 2017
ID: A0131170C
Scan Type: Right Hip
Analysis: January 31, 2017 11:45 Version 13.0
Hip
Operator:
Model: Discovery A (S/N 83116)
Comment:

DXA Results Summary:

<table>
<thead>
<tr>
<th>Region</th>
<th>Area (cm²)</th>
<th>BMC (g)</th>
<th>BMD (g/cm²)</th>
<th>T-score</th>
<th>Z-score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck</td>
<td>5.61</td>
<td>3.31</td>
<td>0.590</td>
<td>-2.5</td>
<td>-1.9</td>
</tr>
<tr>
<td>Total</td>
<td>40.33</td>
<td>29.26</td>
<td>0.726</td>
<td>-2.0</td>
<td>-1.8</td>
</tr>
</tbody>
</table>

Total BMD CV 1.0%
WHO Classification: Osteoporosis
Fracture Risk: High

Physician's Comment:
Scan Information:
Scan Date: January 31, 2017  
ID: A0131170A
Scan Type: f Lumbar Spine
Analysis: January 31, 2017 11:41 Version 13.0:3
Spine
Operator:
Model: Discovery A (S/N 83116)
Comment:

DXA Results Summary:

<table>
<thead>
<tr>
<th>Region</th>
<th>Area (cm²)</th>
<th>BMC (g)</th>
<th>BMD (g/cm²)</th>
<th>T-score</th>
<th>Z-score</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1</td>
<td>12.32</td>
<td>8.26</td>
<td>0.671</td>
<td>-3.7</td>
<td>-3.5</td>
</tr>
<tr>
<td>L2</td>
<td>12.93</td>
<td>9.04</td>
<td>0.699</td>
<td>-3.6</td>
<td>-3.4</td>
</tr>
<tr>
<td>L3</td>
<td>13.25</td>
<td>9.59</td>
<td>0.724</td>
<td>-3.4</td>
<td>-3.2</td>
</tr>
<tr>
<td>L4</td>
<td>15.34</td>
<td>10.31</td>
<td>0.672</td>
<td>-3.8</td>
<td>-3.6</td>
</tr>
<tr>
<td>Total</td>
<td><strong>53.84</strong></td>
<td><strong>37.20</strong></td>
<td><strong>0.691</strong></td>
<td>-3.6</td>
<td>-3.4</td>
</tr>
</tbody>
</table>

Total BMD CV 1.0%, ACF = 1.029, BCF = 0.992, TH = 7.853
WHO Classification: Osteoporosis
Fracture Risk: High

Physician’s Comment:
How To Fix

Captain Obvious:
• Stop the AI

Hormone & Supplements
• Vitamin D
• Strontium
• Ipriflavone
• DHEA
• Testosterone
• Estrogen
• Progesterone
• Growth Hormone
DHEA

Substantiated uses of DHEA include:

- Improving bone density
- Treating depression
- Improving mood
- Improving fatigue
- Enhancing immune response
- Decreasing cardiovascular risk factors
- Improving Erectile Dysfunction
- Decreasing cholesterol and triglycerides
Testosterone

Benefits of Testosterone include:

• Improving bone density
• Improve lean muscle mass
• Improving mood
• Decreases fatigue
• Enhancing immune response
• Decreasing cardiovascular risk factors
• Increase sexual performance and libido
• Decreasing cholesterol and triglycerides
• Improves cognition
Growth Hormone

Approach to treating osteoporosis in men:

Hormones studied to promote bone formation.

Both intermittent and continuous HGH led to significant increases in bone mineral density and bone mineral content at 2 and 3 years. The 3 year gains – a 4.6% to 6.6% increase in bone mineral density at the lumbar spine.
Test Question

The way to keep a man’s estrogen in the “sweet spot” while on testosterone therapy is:

A. Administration of an Aromatase Inhibitor daily
B. Administration of an Aromatase Inhibitor BIW
C. Administration of an Aromatase Inhibitor TIW
D. Administration of an Aromatase Inhibitor only after injection or application of scrotal cream or gel
E. Is to allow the natural aromatization of testosterone to occur
A question to ponder while you are here

Is there ever a time or situation where the use of an aromatase inhibitor is warranted in the gridiron of testosterone replacement therapy in men?
References


J Clin Endocrinol Metab, December 2009, 94(12):4665–4667


Effects of aromatase inhibition vs. testosterone in older men with low testosterone: randomized controlled trial 1J. P. Dias, 1D. Melvin, 2E.M. Simonsick, 1O. Carlson, 2M.D. Shardell, 2L. Ferrucci, 2C. W. Chia, 3S. Basaria and 1J. M. Egan


C J Bagatell, R H Knopp, J E Rivier, W J Bremner; Physiological levels of estradiol stimulate plasma high density lipoprotein2 cholesterol levels in normal men, The Journal of Clinical Endocrinology & Metabolism, Volume 78, Issue 4, 1 April 1994, Pages 855–861,

References


Physiological levels of estradiol stimulate plasma high density lipoprotein2 cholesterol levels in normal men. Bagatell, CJ, Knopp, RH, Rivier, JE, Bremner, WJ. The Journal of Clinical Endocrinology & Metabolism, Volume 78, Issue 4, 1 April 1994, Pages 855–861


