

# Topic 000 - Durable Remission for Newly Recurrent Prostate Cancer (PC)

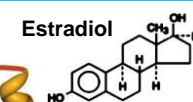
## BOOST



## BOOST

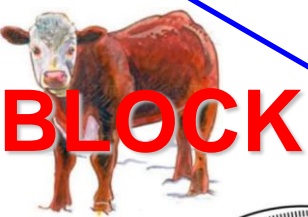
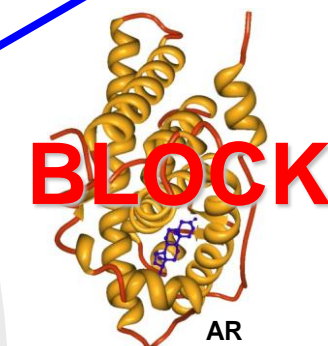
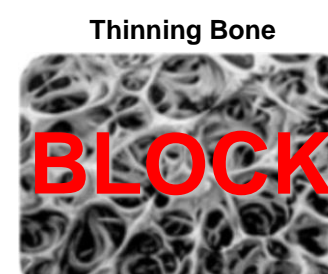
## BOOST

Apoptosis Triggers



ER β

VDR



Heart Healthy Remission Diet

Exercise Program

Increase  
PC Cell  
Death

Limit  
Growth

Limit  
PC Cell  
Change

Increase  
PC Cell  
Death

Limit  
Spread

Bone Density Drugs

Stop  
Division

Intermittent Complete Androgen Blockade

Frequent Detailed Blood Tests Guide Dosing Adjustments and Validate Remission

**Protocol Foundation**

**Example Remission Protocol**

**Supportive Care**

Drugs for Side Effects

Shaded Components Involve One or More Prescription Medications

## Topic 000.a - Durable Remission for *Newly Recurrent* PC

- Remission Protocols for *Newly Recurrent* Prostate Cancer (PC) patients exist that deserve consideration by patients and physicians wishing to force early stage residual PC into a permanent Durable Remission.
- These Remission Protocols are not universally accepted by PC specialists nor are they widely accessible to PC patients at the first sign that their primary prostate cancer treatment procedure may have failed to achieve a “curative outcome.”
- Androgen Deprivation Therapy (ADT) alone fails to consistently achieve a Durable Remission. Although a critical component of many comprehensive protocols, ADT is sadly reserved by many physicians for such a late stage in PC disease progression that a managed Durable Remission may be hard to achieve. Many doctors intentionally DELAY hormone treatment – quite probably CONTRIBUTING to treatment failure!
- This tragic loss of opportunity for *Newly Recurrent* patients to achieve a Durable Remission traces to the pre-2000 belief that Androgen Deprivation can only stall PC disease progression, does not kill the PC cells, is actually believed by many to “induce PC gene mutations,” and thereby claimed as the *cause* of the “hormone refractory” stage of advanced PC.
- This pre-2000 Disease Model MAY QUITE LIKELY be fundamentally wrong in multiple ways.
- The classical PC Disease Model from the 1990’s does not explain the continuing clinical successes reported by prominent Medical Oncologists who practice individualized versions of Remission Protocols.
- A new Disease Model has evolved that explains these clinical successes and provides a much better fit with the entire body of evolving scientific data. Detailed versions of Remission Protocols for PC, as practiced by Dr. Shultz (PCRI) and Dr. Myers (AIDP), can be studied via their websites.

# Topic 000.b - Durable Remission for *Newly Recurrent PC*

One example of a Remission Protocol which integrates multiple PC suppressing components is designed to:

1. **Limit** “nutrients” identified as PC metabolic fuel disabling PC progression through the “cell cycle”
2. **Increase** PC cell death rates as well as overall protocol effectiveness through a program of physical exercise
3. **Limit** both PC cellular “mutations” and “evasive epigenetic changes” to maintain total protocol effectiveness
4. **Increase** PC cell death rates via triggered apoptosis required for any equation driving PC population collapse
5. **Limit** the ability of PC cells to establish tumors in bone by maintaining high bone density with bisphosphonates
6. **Stop** PC cells from growing and dividing with Periodic Intermittent Complete Androgen Blockade (CAB)
7. **Mitigate** side effects by inclusion of *Supportive Care Medications* during Androgen Blockade “on Cycles”

## *The Sub-Topics*

[000.1 How PC cell growth is retarded by elimination from the diet of those foods providing the metabolic fuels needed by PC cells](#)

[000.2 How overall protocol effectiveness is further enhanced by an easily achieved patient-tailored exercise program](#)

[000.3 How protocol effectiveness is maximized by minimizing BOTH PC cellular “mutations” AND “epigenetic changes”](#)

[000.4 How PC cell death is increased through vigorous triggering of multiple programmed cell-death pathways – apoptosis](#)

[000.5 How Bone Density medications not only provide supportive care but also demonstrate direct anti-metastatic activity](#)

[000.6 How PC cell division and proliferation is disabled by “Complete Blockade” of the PC Androgen Receptor Pathway](#)

[000.7 How various protocol side effects can be prevented or substantially reduced with addition of supportive care medications](#)

## **STAY AWAY FROM THE FOLLOWING:**

- Red meat, dairy fat, and egg yolks (Avoid ALL sources of arachidonic acid – a PC primary fuel source)
- Pork is the worst meat you can eat due to what they feed the pigs
- Avoid canola oil at all costs! Avoid all vegetable oils
- King mackerel, shark, and albacore tuna (due to the high mercury content)
- Avoid flax oil at all costs! Flaxseed remains controversial and best left out of the diet until further studies complete.
- No juicing carrots however eating them is OK (this is because of the large amount of beta carotene in the amount of carrots that it would take to make a full glass of juice)
- Avoid peanuts, walnuts, and pecans

## **DO SELECT FROM THE FOLLOWING:**

- Cold water fish 3 to 4 times a week : salmon, haddock, halibut, cod, pink tuna, herring, sardines, arctic char.  
There is no health benefit from flounder, shellfish, grouper or snapper however they are fine to eat.
- White meat of poultry (chicken/turkey)
- Egg whites and egg beaters
- Non-fat dairy (skim milk, nonfat cheese/ice cream)
- Any type of fruits and vegetables. Cabbage family members have been shown to be very beneficial for cancer patients (broccoli, cauliflower, onions, radishes, horseradish)
- Nuts: pistachios, almonds, cashews, macadamia, hazelnuts, pine, and pumpkin
- Use olive oil / avocado oil / hazelnut oil instead of vegetable oil and butter
- Red wine in moderation
- Dark chocolate
- Bread, rice, pasta, legumes (in moderation due to high caloric value)
- Eat stewed or cooked tomatoes
- Drink two to four cups of green tea a day

[Reference: See Dr. Charles Myers – Beating Prostate Cancer](#)

[Reference: See Prostate Cancer Research Foundation – Nutrition Guide](#)

[Reference: Sulforaphane-induced Cell Death in Human Prostate Cancer Cells](#)

## Topic 000.2.1 Physical Exercise Has Multiple Study-Proven Remission Benefits

1. Exercise may directly kill *circulating* PC cells due to the more turbulent environment they experience during exercise as blood flows through the heart chambers and valves. This hypothesis involves increased mechanical damage to circulating PC cells from fluid forces associated with elevated heart rate and blood pressure induced by exercise.
2. Other studies demonstrate that blood taken post-exercise suppresses PC cell growth in test tubes whereas blood taken from sedentary individuals increases PC cell growth in test tubes. These experiments would indicate that PC retardation via exercise has a chemical basis that is systemic and therefore able to retard PC cell growth in tissue.
3. IGF-I is identified as a significant PC growth hormone. Exercise decreases insulin levels which decreases IGF-I produced by the liver. IGFBP-I which binds to IGF-I, removing it from activity, is also measurably increased by exercise.
4. Leptin may or may not be proven to play a role in early PC causation, but it has been shown to drive growth and proliferation of hormone-insensitive PC. Any factors that stimulate the emergence of hormone-insensitive PC cell populations **MUST BE MINIMIZED!** Leptin levels are decreased as a result of physical exercise.
5. “Men on hormonal therapy tend to lose muscle strength and muscle mass. While this obviously limits what you can do physically, it very likely contributes to the weight gain and exacerbates the insulin resistance. If you adopt a program of aerobic exercise and resistance exercise, you can limit your loss of muscle strength and muscle mass and your recovery from hormonal therapy, once it has stopped, will also be more rapid . . . Exercise for 30-40 minutes at least three times a week . . . Incorporate relaxation techniques into your *daily* life.” – Dr. Myers
6. Appropriate exercise also helps maintain healthy bone density indirectly limiting PC growth in bone by limiting empty space.

[Reference: See Dr. Charles Myers – Beating Prostate Cancer](#)

[Reference: UCLA Study – Exercise Lowers PC Growth Stimulant IGF-I by Lowering Insulin & Raising IGFBP-I](#)

[Reference: See Dr. Charles Myers – PCRE – Prostate Cancer Foundation – TellOneFriend](#)

[Reference: Lifestyle therapy for prostate cancer: Does it work – Harvard Family Health Guide](#)

[Reference: Leptin mediates androgen-independent prostate cancer cell proliferation](#)

# Topic 000.3.1 PC Cells Must Be “Protected” So They Can Not Evade

## Examples of ANTIOXIDANTS and other REMISSION PROTOCOL SUPPLEMENTS

<b>POMEGRANATE EXTRACT – take 2 tablets 2 times daily (from LEF)</b> <a href="http://www.lef.org/newshop/items/item00956.html">http://www.lef.org/newshop/items/item00956.html</a>	\$1.76 / day
<b>LYCOPENE 15mg tablets – take 1 tablet 2 times daily</b> <a href="http://www.lef.org/newshop/items/item00455.html">http://www.lef.org/newshop/items/item00455.html</a>	\$ .50 / day
<b>SELENIUM 200 mcg tablets – take 1 tablet daily (from yeast – <u>not</u> Selenomethionine)</b> <a href="http://www.vitaminshoppe.com/store/en/browse/sku_detail.jsp?id=KA-1725">http://www.vitaminshoppe.com/store/en/browse/sku_detail.jsp?id=KA-1725</a> (KAL Vegan)	\$ .13 / day
<b>VITAMIN E (GAMMA TOCOPHEROLS) 200 IU tablets – take 1 tablet daily</b> <a href="http://www.lef.org/newshop/items/item00559.html">http://www.lef.org/newshop/items/item00559.html</a> <a href="http://www.lef.org/newshop/items/item00759.html">http://www.lef.org/newshop/items/item00759.html</a> (NEW Product with sesame lignans )	\$ .47 / day or \$ .32 / day
<b>OMEGA 3 FISH OIL 1000mg tablets – take 2 tablets 2 times daily (total daily dose 4000 mg)</b> <a href="http://www.omega-dha-epa.com/productultimate.html">http://www.omega-dha-epa.com/productultimate.html</a>	\$1.32 / day
<b>SOY ISOFLAVONES – 100 - 135 MG – take 2 tablet 2 times daily</b> <a href="http://www.iherb.com/store/ProductDetails.aspx?c=Herbs&amp;pid=NOW-03288">http://www.iherb.com/store/ProductDetails.aspx?c=Herbs&amp;pid=NOW-03288</a> (NOW Veg)	\$ .65 / day
<b>VITAMIN D3 – 3,000 to 10,000 IU daily</b> <a href="http://www.lef.org/newshop/items/item00713.html">http://www.lef.org/newshop/items/item00713.html</a>	\$ .25 / day
LEF's CAUTION! To be taken only under a physician's supervision. Monthly blood tests are mandated when taking this 5000 IU vitamin D supplement.	
<b>Total</b>	<b>\$5.08 / day</b>

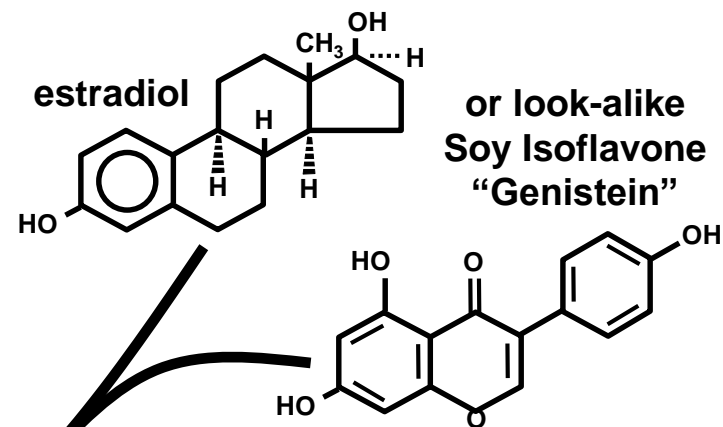
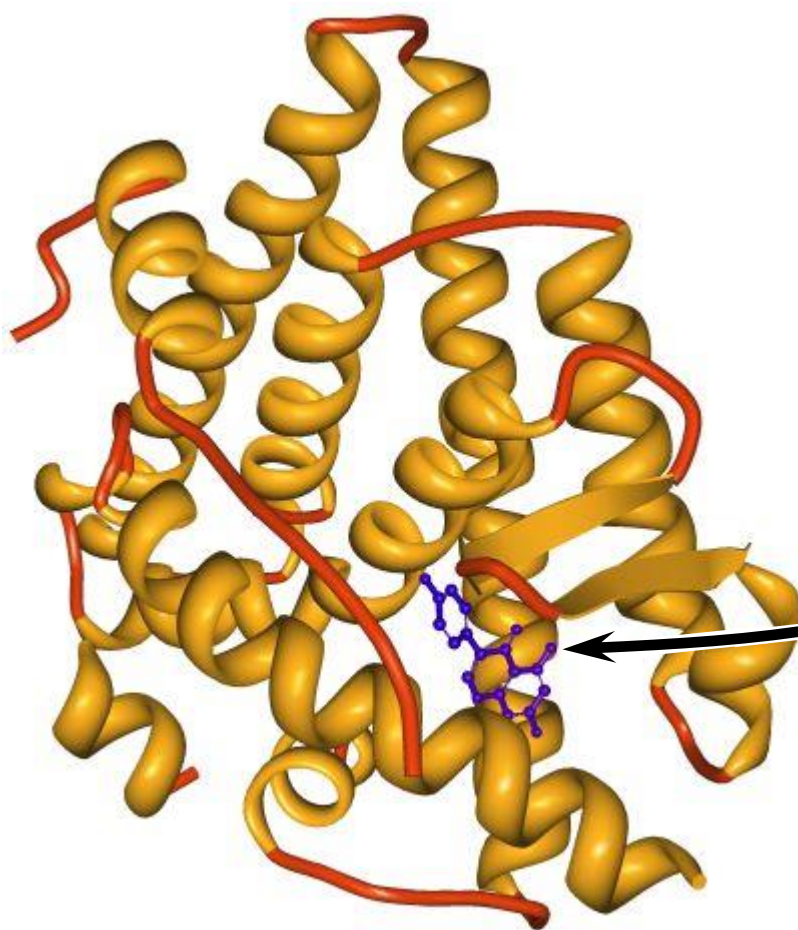
- Free radicals can cause genetic damage (chromosome mutations).
- Antioxidants bind with and otherwise disable free radicals.
- For the Remission Protocol to be sustainable, the cancer must not mutate into the refractory form.
- The PC cells must also be blocked from epigenetic changes that also allow PC cells to evade this protocol.
- Example products listed above have been shown in studies to decrease further genetic change in PC cells, thereby ***maintaining*** the long-term effectiveness of the primary components of the Remission Protocol:  
Block cell division, Interrupt the Cell Cycle, and Trigger Apoptosis.

[Reference: See Dr. Charles Myers – Beating Prostate Cancer](#)

[Reference: See Dr. Jacek Pinski - PCR!](#)

# Topic 000.4.1 Stimulation of Apoptosis via ER $\beta$

## Estrogen Receptor $\beta$ – A “Ribbon View”



Estrogenic Activation of Estrogen Receptor  $\beta$  results from binding of estradiol with ER  $\beta$ 's “Ligand Pocket.”

Soy Isoflavones include look-alike molecules that are also capable of Estrogenic Activation.

When ER  $\beta$  is so activated, the combined molecule enters the PC cell nucleus, interacts with the DNA, and promotes an increased rate of apoptosis, programmed PC cell death.

## The ER $\beta$ with its “Ligand Pocket”

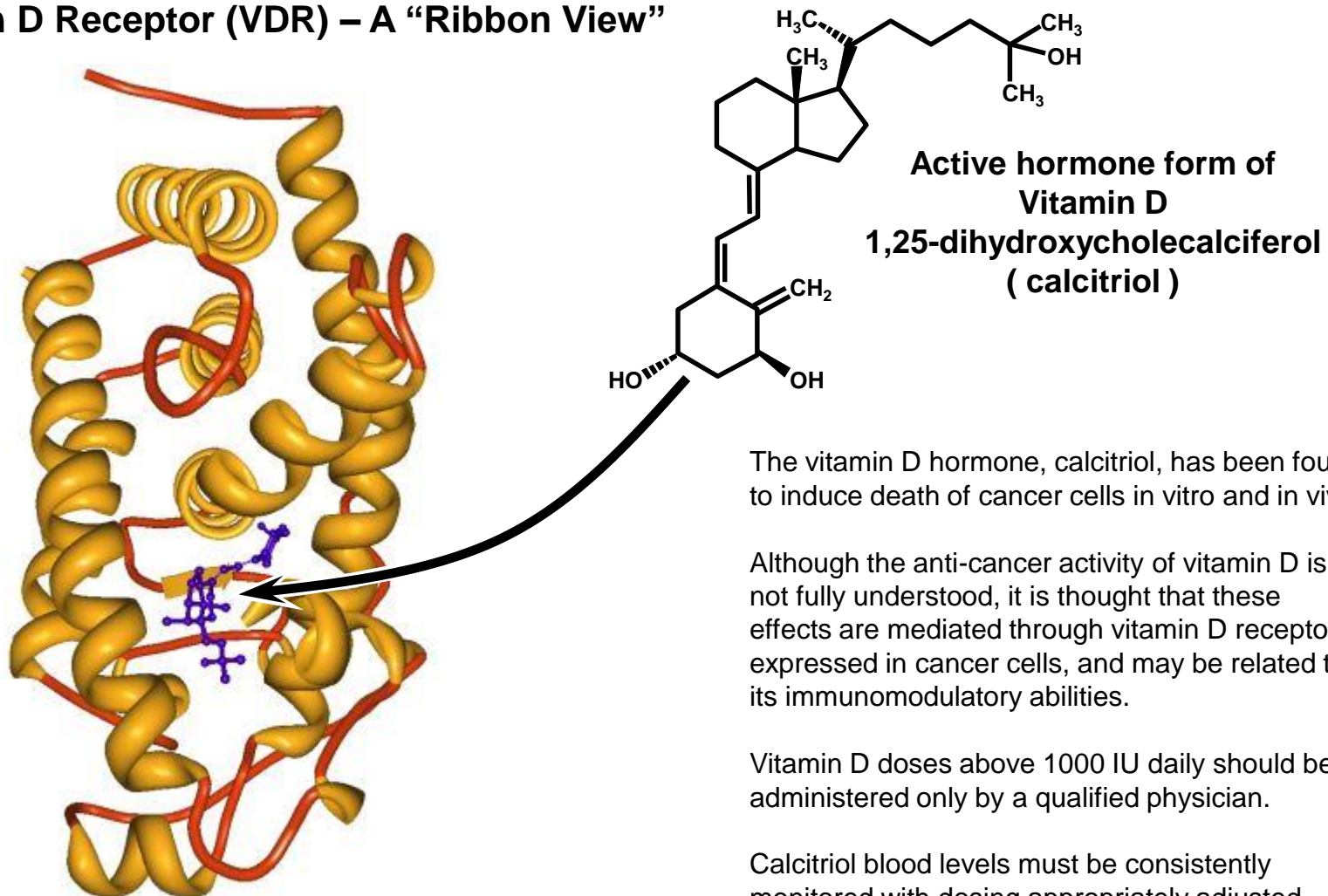
Reference: [See Dr. Charles Myers – Prostate Forums – Back Issue - Volume 6 Number 1](#)

Reference: [See Dr. Charles Myers – Beating Prostate Cancer](#)

Reference: [See Dr. Jacek Pinski - PCRI](#)

## Topic 000.4.2 Stimulation of Apoptosis via VDR

### Vitamin D Receptor (VDR) – A “Ribbon View”



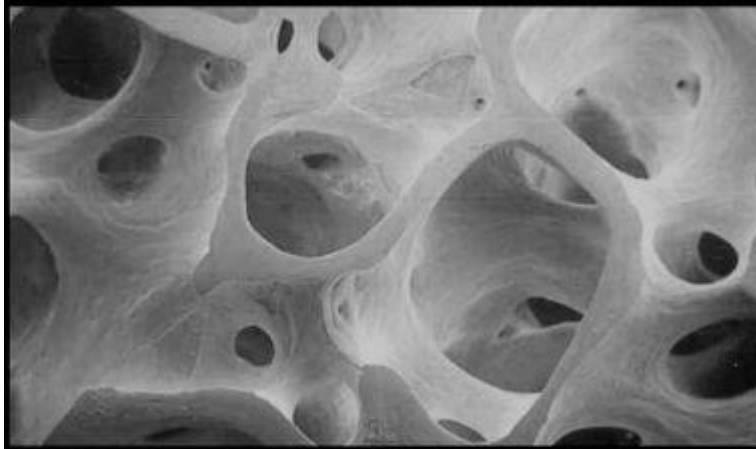
### The VDR with its “Ligand Pocket”

[Reference: See Dr. Charles Myers – Beating Prostate Cancer](#)

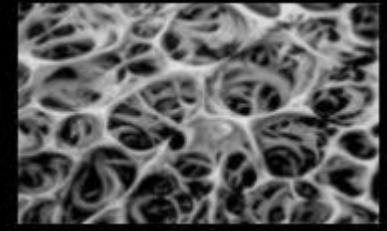
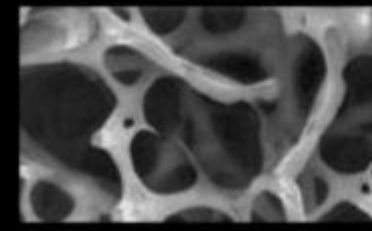
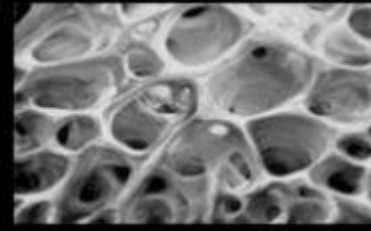
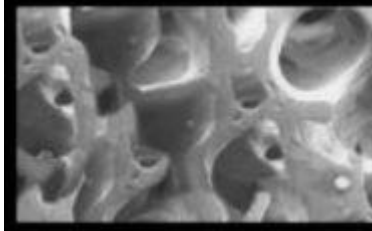
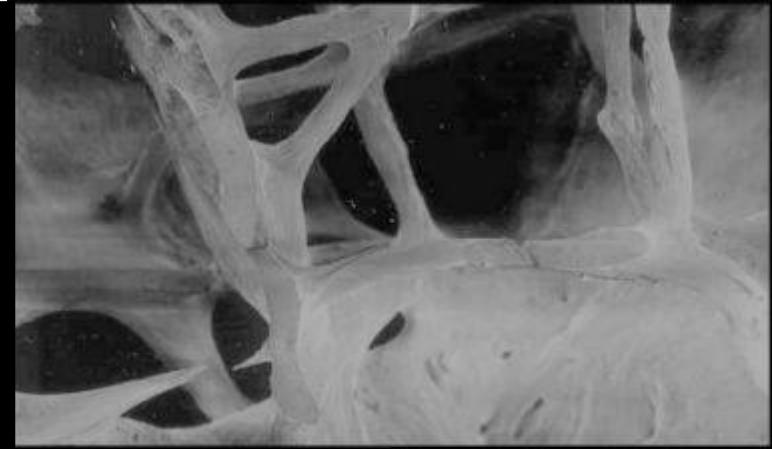
[Reference: See Dr. Jacek Pinski - PCR!](#)

# Topic 000.5.1 High Bone Density May Decrease Ease of Metastatic Colonization

## Healthy Dense Bone Matrix



## “Thin” Bone Matrix Weakened by Osteoporosis



Bisphosphonates (BP) are often prescribed during Hormone Therapy to preclude bone loss caused by low androgen levels. Indirect anti-cancer activity results from maintaining a dense bone matrix which denies PC tumors empty space for growth. Studies now report direct anti-cancer activity in Vitro against metastatic PC through the following mechanism:

- Bisphosphonates have been shown to interfere with the PC cell's ability to adhere to bone matrix surfaces.
- This effect of the studied BP alendronate (Fosamax) on cell invasion was mediated by inhibition of the mevalonate pathway

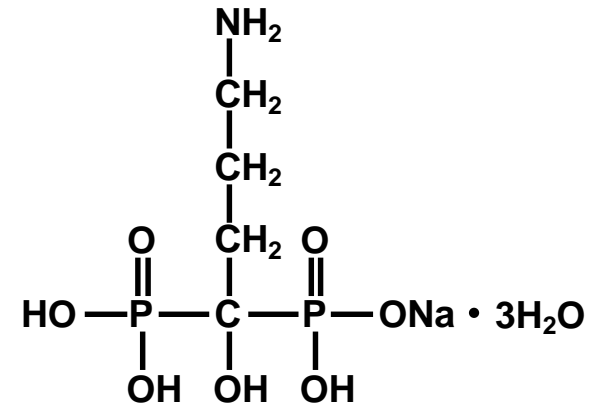
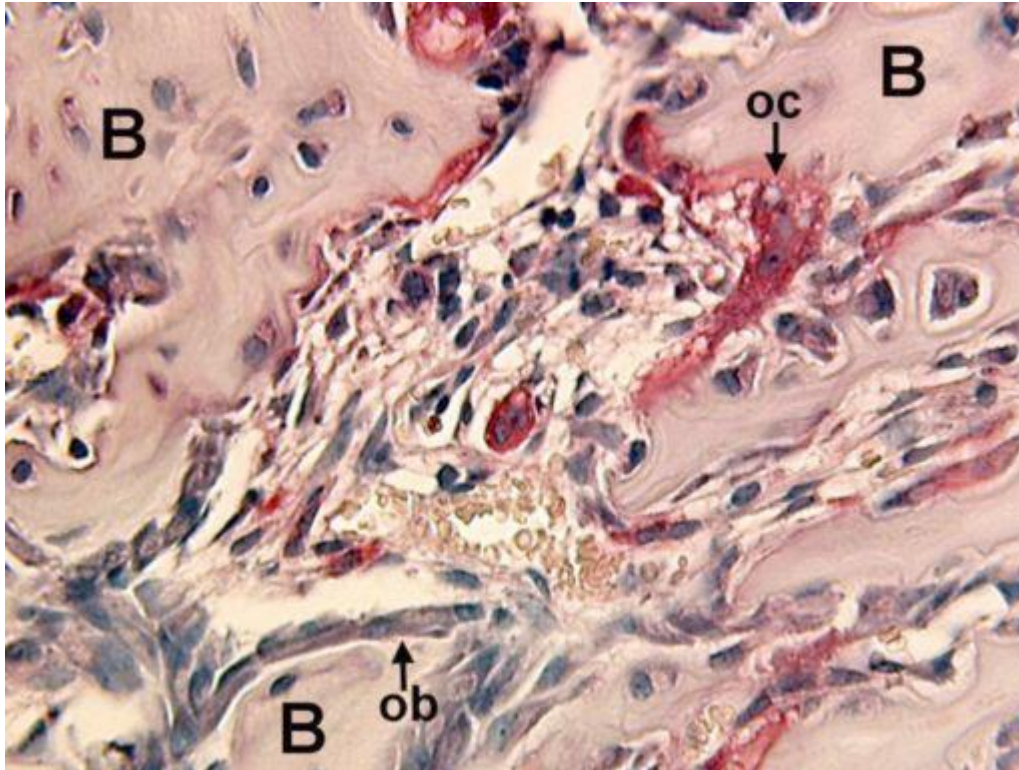
[Reference: Common Treatment for Osteoporosis May Help Men with Prostate Cancer](#)

[Reference: Bisphosphonates Inhibit the Adhesion of Breast Cancer Cells to Bone Matrices In Vitro](#)

[Reference: Reference: Bisphosphonates Inhibit the Adhesion of Prostate Cancer Cells to Bone Matrices In Vitro](#)

[Reference: Alendronate \(Fosamax\) Inhibits Invasion of PC-3 Prostate Cancer](#)

# Topic 000.5.2 Bisphosphonates Maintain Bone Density via Decreased Resorption



Example bisphosphonate  
FOSAMAX® (alendronate sodium)

[MERCK's Datasheet Website](#)

FOSAMAX\* (alendronate sodium) is a bisphosphonate that acts as a specific inhibitor of osteoclast-mediated bone resorption. Bisphosphonates are synthetic analogs of pyrophosphate that bind to the hydroxyapatite found in bone. Osteoclasts (oc), which are stained red in this section of bone (B), are responsible for the degradation of bone and cartilage. Their multiple nuclei appear as darkly stained bodies within the cell. Osteoblasts (ob) are the rectangular-shaped cells, each with a single prominent nucleus, lining the surface of the bone. These cells appear dark in color due to the presence of numerous ribosomes, which are structures in cells that synthesize protein. The catabolic and anabolic activities of osteoclasts and osteoblasts, respectively, are coupled under physiological conditions to maintain bone homeostasis (the balance between bone growth and degradation).

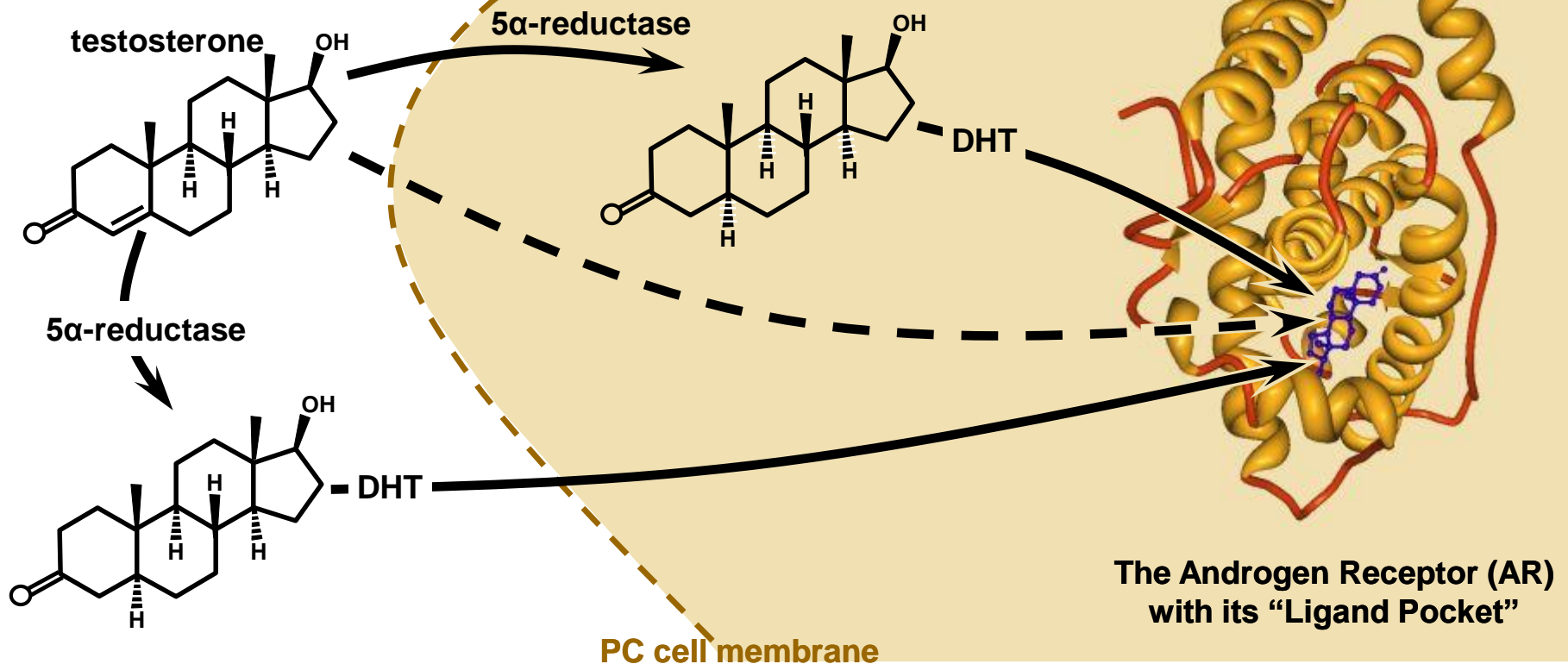
Reference: [Jonathan Lam, Washington University School of Medicine, St. Louis, MO.](#)

# Topic 000.6.1 Complete Androgen Blockade – The AR Pathway

## PC cell division and cell growth usually rely on a functional Androgen Receptor (AR) Pathway

1. Testosterone is produced by the testes and by the adrenal glands
2. Testosterone is converted into 5 $\alpha$ -dihydrotestosterone (DHT) inside various cells by two forms of the enzyme 5 $\alpha$ -reductase
3. Testosterone and DHT easily enter into Prostate Cancer (PC) cells through the outer cell membrane
4. Testosterone is also converted into DHT inside most PC cells by interaction with 5 $\alpha$ -reductase produced within the PC cells
5. Both testosterone and DHT can bind to a protein called the Androgen Receptor (AR), produced in most PC cells
6. Upon binding, the AR+Androgen “combined molecule” migrates into the PC cell nucleus fostering cell growth and division
7. DHT is much more powerful than testosterone as an AR “agonist”
8. PC cell DNA is licensed to replicate by the AR and divides into 2 new PC cells
9. Blocking the “AR Pathway” in prostate cancer can halt proliferation

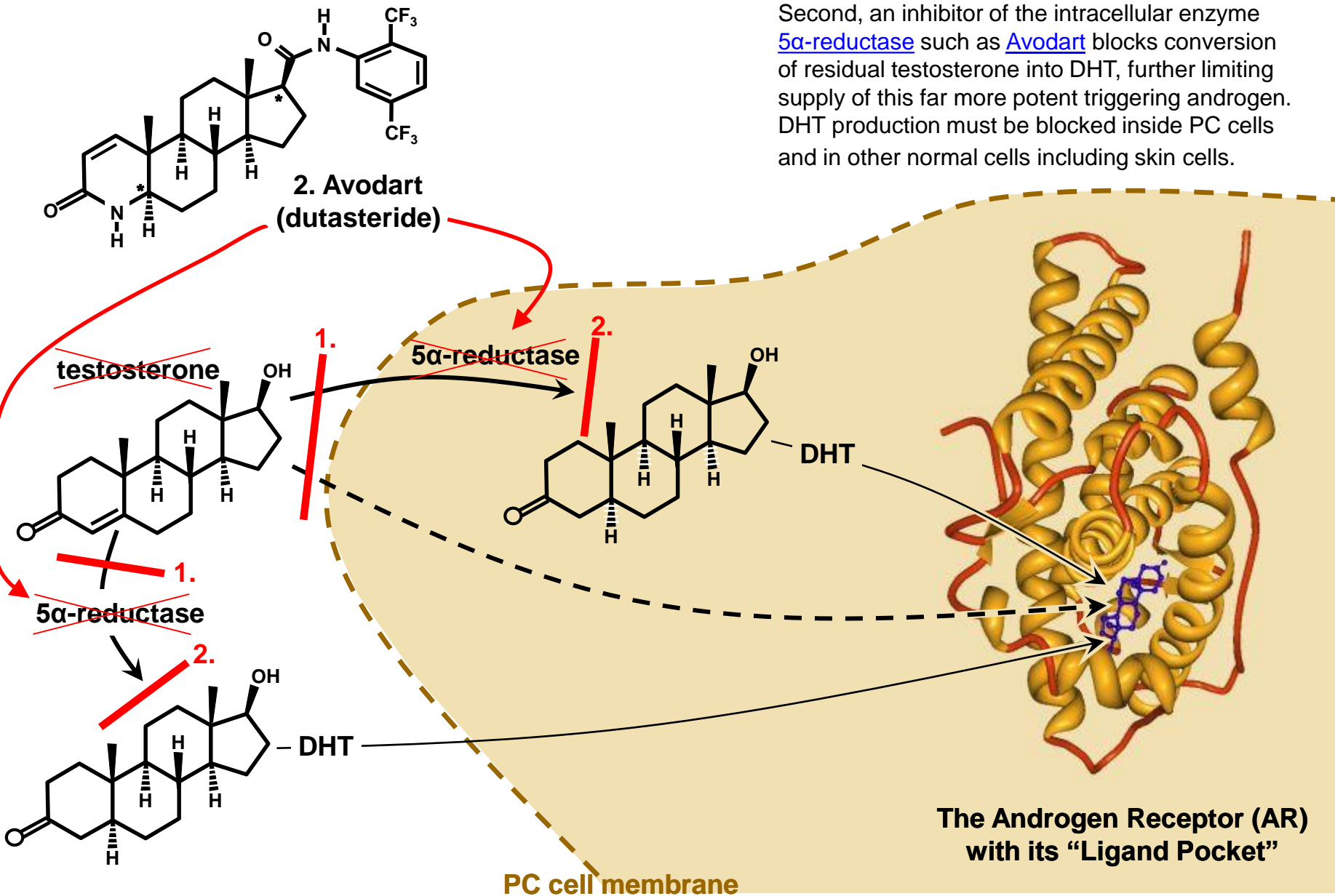
Intermittent Blockade demonstrates the best results and least resistance





# Topic 000.6.3 Complete Androgen Blockade - 5 $\alpha$ -reductase Inhibitors

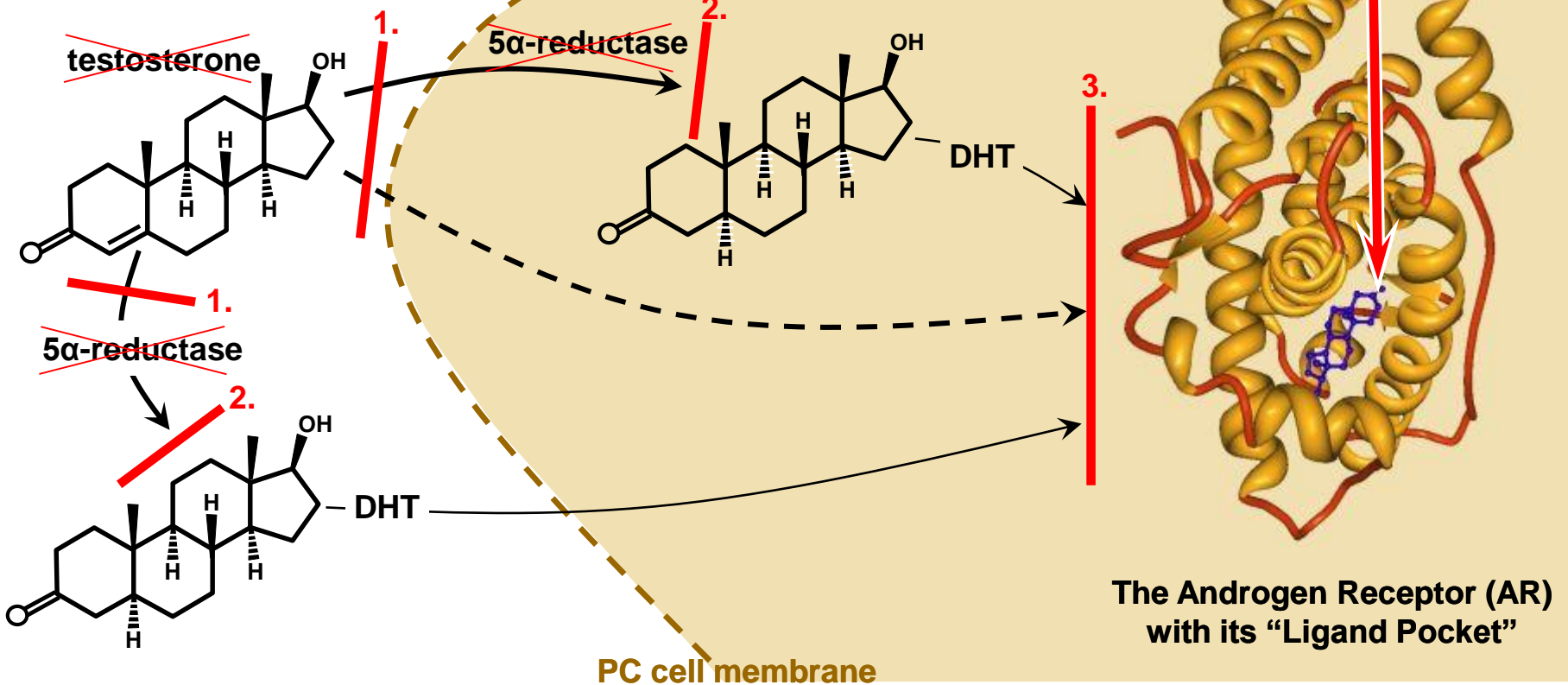
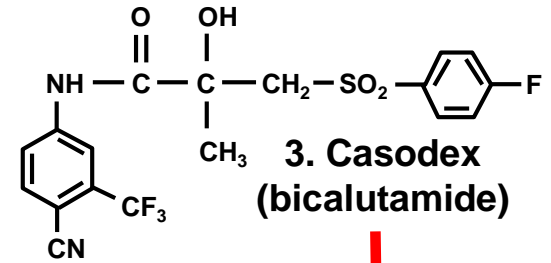
Second, an inhibitor of the intracellular enzyme [5 \$\alpha\$ -reductase](#) such as [Avodart](#) blocks conversion of residual testosterone into DHT, further limiting supply of this far more potent triggering androgen. DHT production must be blocked inside PC cells and in other normal cells including skin cells.



# Topic 000.6.4 Complete Androgen Blockade - Antiandrogens

Third, an [antiandrogen](#) such as [Casodex](#) is used that binds to and blocks the Ligand Pocket of the Androgen Receptor. Residual testosterone and DHT molecules are then blocked because their binding site is occupied – this third level of “Blockade” completes effective interruption of the AR pathway.

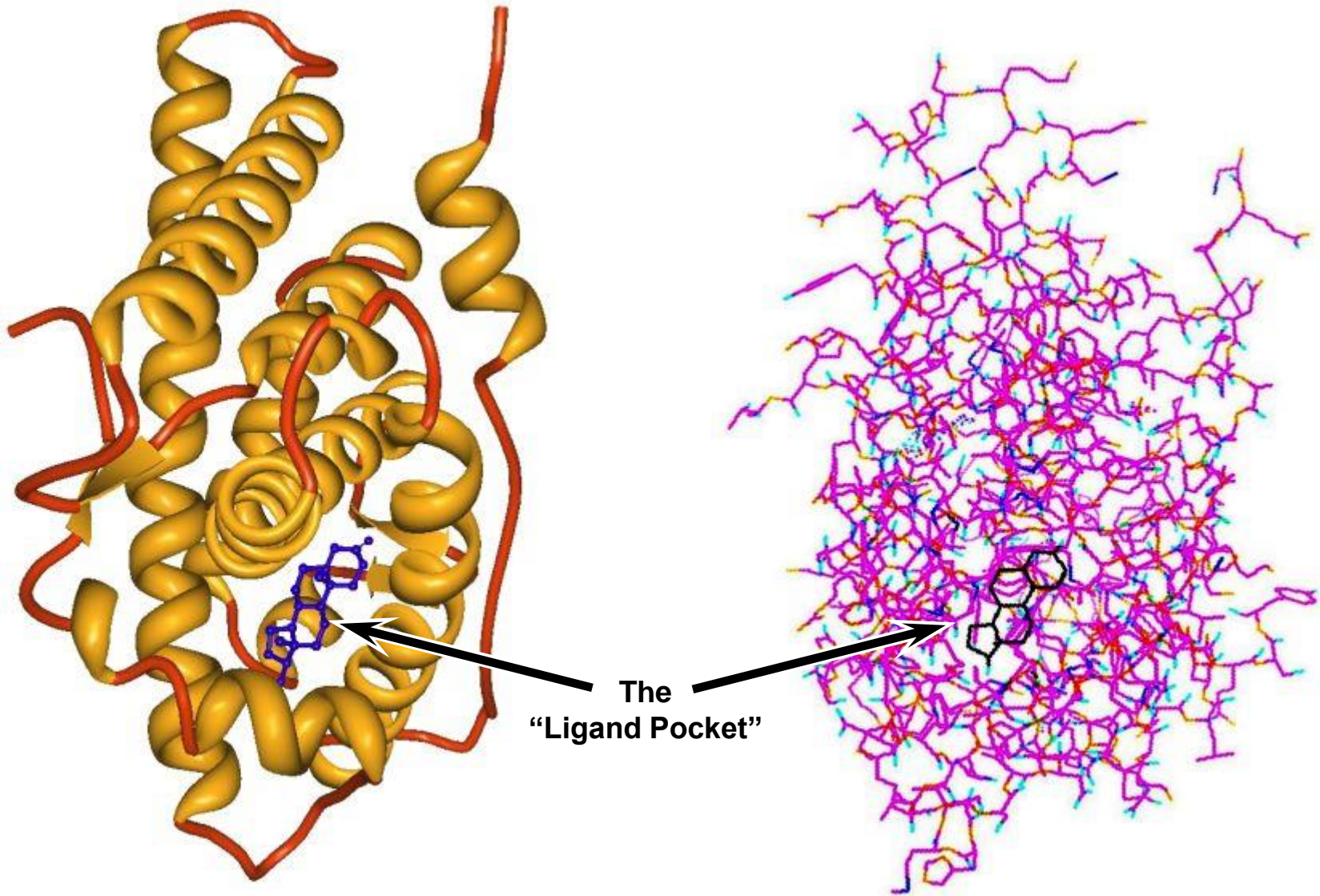
Note: Antiandrogens do not form permanent bonds with the AR. Therefore, a high enough dose of the antiandrogen is key to effectiveness. All Ligand Pockets of all ARs in all PC cells must **almost always** be filled to ensure that residual androgen molecules **very rarely** bind with empty AR Ligand Pockets.



# Topic 000.6.5 Complete Androgen Blockade – The AR Protein

The Androgen Receptor – A “Ribbon View”

The Androgen Receptor – A “Chain View”



Reference: [See Dr. Charles Myers – Prostate Forums – Back Issue - Volume 6 Number 1](#)

## Topic 000.6.6 Complete Androgen Blockade – The AR (cont)

### The Androgen Receptor – The PC Cell Division Enabler

- AR is found in normal prostate cells and most prostate cancer (PC) cell variants
- The Ligand Pocket of the AR is a molecular “Lock,” waiting for a chemical “Key”
- Normal “Keys” for this “Lock” are testosterone and 5 $\alpha$ -dihydrotestosterone (DHT)
- Testosterone or DHT binds to the “Ligand Pocket” creating a combined molecule that enters the PC cell nucleus, interfaces with DNA and enables cell division
- If the Ligand Pocket has been filled with an “antiandrogen,” (example – Casodex) testosterone or DHT can not bind to the receptor.
- Cell division is disabled by reduction of both DHT and testosterone levels AND by filling the Ligand Pocket with a suitable look-alike but non-triggering molecule
- The *AR* gene for the androgen receptor is located on the X chromosome at Xq11-12.

Reference: [See Dr. Charles Myers – Prostate Forums – Back Issue - Volume 6 Number 1](#)

# Topic 000.7.1 Supportive Care Medications Can Mitigate Protocol Side Effects

**Definition:** “[Supportive Care](#)” - Care given to improve the quality of life of patients who have a serious or life-threatening disease.

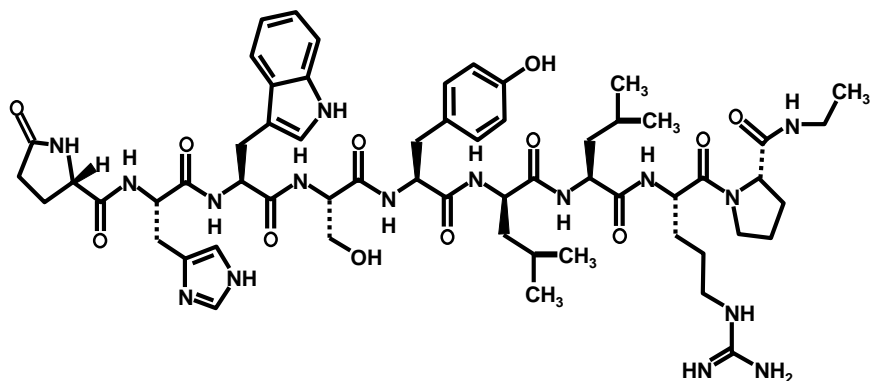
1. [Fosamax](#) or other Bisphosphonates (BP) are prescribed to limit loss of bone density caused by low testosterone levels.
2. [Transdermal estrogen patches \(estradiol\)](#) can limit bone density loss, decrease mental acuity impact, and significantly decrease frequency and/or severity of Hot Flashes caused by low testosterone levels.
3. [Cabergoline](#), a dopamine agonist used to treat high levels of prolactin, can limit breast growth caused by estradiol patch use.
4. [Ursodiol](#), approved as a treatment for gallstones, can reduce liver toxicity caused by high dosage Casodex (150 mg / day).

Reference: [See Dr. Charles Myers – Beating Prostate Cancer](#)

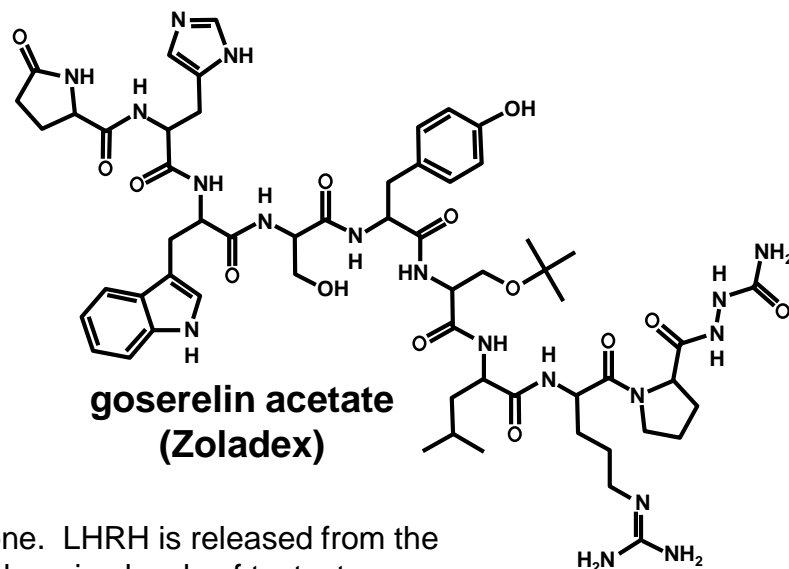
Reference: [Preventing and Treating the \[ADT\] Side Effects – Page 10 - NOVEMBER 2007 VOL 10: NO 4 – PCRI Insights](#)

# Topic 000.6.2.a CAB : LHRH Agonists Details

## LHRH Agonists



**leuprolide acetate**  
(Lupron, Eligard and Viadur)



**goserelin acetate**  
(Zoladex)

- LHRH is shorthand for Luteinizing hormone releasing hormone. LHRH is released from the hypothalamus of the brain when the hypothalamus detects dropping levels of testosterone.
- LHRH is received by the receptors of the pituitary gland which releases luteinizing hormone or LH which travels to the testicles and begins the production of testosterone.
- In prostate cancer hormone therapy, LHRH agonists and antagonists are used to prevent the pituitary gland from releasing LH.
- The levels of testosterone will drop 90 to 95 percent to what is called castrate level. The use of a LHRH agonist or antagonist in prostate cancer hormone therapy therefore is often called chemical castration. LHRH is also referred to as gonadotropin hormone releasing hormone.

• [Lupron \(leuprolide acetate\) – TAP Pharmaceutical Products Inc's Website](#)

• [Zoladex \(goserelin acetate\) – AstraZeneca's Website](#)

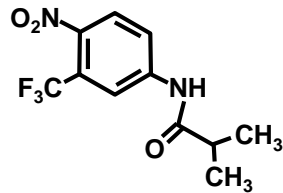
• [Eligard \(leuprolide acetate\) – Sanofi-Aventis U.S. LLC's Website](#)

• [Viadur \(leuprolide acetate\) – Bayer Pharmaceuticals Corporation's Website](#)

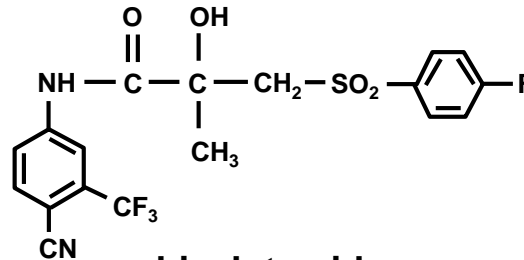


## Topic 000.6.4.a CAB : Antiandrogens - Details

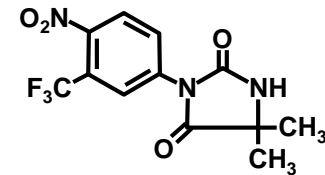
### Antiandrogens



**flutamide**  
**(Eulexin and Euflex)**



**bicalutamide**  
**(Casodex)**



**nilutamide**  
**(Anandron and Nilandron )**

- Antiandrogens are compounds that are capable of inhibiting the effects of androgens, male sex hormones.
- Flutamide (Eulexin, Euflex), nilutamide (Anandron, Nilandron) and bicalutamide (Casodex), are nonsteroidal antiandrogens that work by blocking the Androgen Receptor (AR) and obstructing the androgen pathway.
- flutamide (Eulexin) is the oldest and has more unwanted side effects than the others.
- bicalutamide (Casodex) is the newest and has the least side effects.
- nilutamide (Anandron) is listed by its manufacturer as being used to “complement surgical castration”

- [flutamide \(Eulexin\) – Schering-Plough Pty Limited Website](#)
- [bicalutamide \(Casodex\) – AstraZeneca’s Website](#)
- [nilutamide \(Anandron\) – Sanofi-Aventis U.S. LLC’s Website](#)