

# **HORMONAL DRUGS EXAM 1 PRACTICE (SAMPLE)**

**STUDY GUIDE**



**SAMPLE STUDY GUIDE  
WITH LIMITED QUESTIONS**

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THE FULL VERSION STUDY GUIDE**

<https://hormonaldrugsexam1.examzify.com>



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# Introduction

Preparing for a certification exam can feel overwhelming, but with the right tools, it becomes an opportunity to build confidence, sharpen your skills, and move one step closer to your goals. At Examzify, we believe that effective exam preparation isn't just about memorization, it's about understanding the material, identifying knowledge gaps, and building the test-taking strategies that lead to success.

This guide was designed to help you do exactly that.

Whether you're preparing for a licensing exam, professional certification, or entry-level qualification, this book offers structured practice to reinforce key concepts. You'll find a wide range of multiple-choice questions, each followed by clear explanations to help you understand not just the right answer, but why it's correct.

The content in this guide is based on real-world exam objectives and aligned with the types of questions and topics commonly found on official tests. It's ideal for learners who want to:

- Practice answering questions under realistic conditions,
- Improve accuracy and speed,
- Review explanations to strengthen weak areas, and
- Approach the exam with greater confidence.

We recommend using this book not as a stand-alone study tool, but alongside other resources like flashcards, textbooks, or hands-on training. For best results, we recommend working through each question, reflecting on the explanation provided, and revisiting the topics that challenge you most.

Remember: successful test preparation isn't about getting every question right the first time, it's about learning from your mistakes and improving over time. Stay focused, trust the process, and know that every page you turn brings you closer to success.

Let's begin.

# How to Use This Guide

This guide is designed to help you study more effectively and approach your exam with confidence. Whether you're reviewing for the first time or doing a final refresh, here's how to get the most out of your Examzify study guide:

## 1. Start with a Diagnostic Review

Skim through the questions to get a sense of what you know and what you need to focus on. Your goal is to identify knowledge gaps early.

## 2. Study in Short, Focused Sessions

Break your study time into manageable blocks (e.g. 30 – 45 minutes). Review a handful of questions, reflect on the explanations.

## 3. Learn from the Explanations

After answering a question, always read the explanation, even if you got it right. It reinforces key points, corrects misunderstandings, and teaches subtle distinctions between similar answers.

## 4. Track Your Progress

Use bookmarks or notes (if reading digitally) to mark difficult questions. Revisit these regularly and track improvements over time.

## 5. Simulate the Real Exam

Once you're comfortable, try taking a full set of questions without pausing. Set a timer and simulate test-day conditions to build confidence and time management skills.

## 6. Repeat and Review

Don't just study once, repetition builds retention. Re-attempt questions after a few days and revisit explanations to reinforce learning. Pair this guide with other Examzify tools like flashcards, and digital practice tests to strengthen your preparation across formats.

There's no single right way to study, but consistent, thoughtful effort always wins. Use this guide flexibly, adapt the tips above to fit your pace and learning style. You've got this!

## Questions

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**1. Differentiate finasteride and dutasteride mechanisms and clinical uses.**

- A. Finasteride inhibits type II 5 $\alpha$ -reductase; dutasteride inhibits both type I and II; used for BPH and androgenetic alopecia; teratogenic risk if pregnant.
- B. Finasteride inhibits type II 5 $\alpha$ -reductase; dutasteride inhibits both type I and II; used for BPH and androgenetic alopecia; teratogenic risk if pregnant.
- C. Finasteride inhibits aromatase; dutasteride inhibits estrogen receptors; used for breast cancer.
- D. Finasteride is a glucocorticoid receptor antagonist; dutasteride is a mineralocorticoid receptor antagonist; used for diarrhea.

**2. What is the MOA and clinical use of ketoconazole in hormonal disorders?**

- A. It is a beta-blocker used for tachyarrhythmias.
- B. It inhibits fungal CYP450 enzymes and mammalian steroid synthesis at higher doses; used off-label for Cushing's disease; monitor for hepatotoxicity.
- C. It acts as a glucocorticoid receptor antagonist; used for Cushing's syndrome medically.
- D. It inhibits aromatase; used in breast cancer treatment in premenopausal women.

**3. Which adverse effect is specifically associated with 17 $\alpha$ -alkylated testosterone formulations and requires monitoring?**

- A. Kidney failure
- B. Hypercalcemia
- C. Hypertension
- D. Liver toxicity

**4. Which drug is described as an abortifacient and a progesterone receptor antagonist?**

- A. Norgestimate
- B. Dinoprostone
- C. Misoprostol
- D. Mifepristone (RU-486)

**5. Dienestrol is described with which route of administration in the material?**

- A. Intravenous
- B. Intramuscular
- C. Topical
- D. Oral

**6. Raloxifene belongs to which class?**

- A. Biologic
- B. Small molecule
- C. Selective estrogen receptor modulator (SERM)/Benzothiophene
- D. Calcitonin analog

**7. What is the mechanism of SGLT2 inhibitors and a key risk?**

- A. Inhibit hepatic glucose production; risk lactic acidosis
- B. Inhibit intestinal glucose absorption; risk GI upset
- C. Inhibit glucose reabsorption in proximal tubule; risk genital infections
- D. Increase insulin secretion; risk hypoglycemia

**8. Which statement differentiates GLP-1 receptor agonists from DPP-4 inhibitors?**

- A. GLP-1 receptor agonists mimic incretin and increase glucose-dependent insulin secretion, often with weight loss.
- B. DPP-4 inhibitors mimic incretin and cause weight loss.
- C. GLP-1 receptor agonists are glucose-independent and cause weight gain.
- D. DPP-4 inhibitors boost endogenous incretin by inhibiting degradation and are weight neutral.

**9. Which statement about aromatase inhibitors is true?**

- A. They can contribute to decreased bone mineral density and osteoporosis risk
- B. They increase bone density
- C. They worsen sleep quality only
- D. They have no effect on bones

**10. How do teriparatide differ from denosumab in osteoporosis therapy?**

- A. Teriparatide inhibits osteoclast activity; denosumab stimulates bone formation
- B. Teriparatide is an anabolic PTH analog; denosumab is a RANKL inhibitor that reduces bone resorption
- C. Teriparatide replaces calcitonin; denosumab inhibits calcium absorption
- D. Teriparatide increases bone resorption; denosumab increases bone formation

## **Answers**

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1. B
2. B
3. D
4. D
5. C
6. C
7. C
8. D
9. A
10. B

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## **Explanations**

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## 1. Differentiate finasteride and dutasteride mechanisms and clinical uses.

- A. Finasteride inhibits type II 5 $\alpha$ -reductase; dutasteride inhibits both type I and II; used for BPH and androgenetic alopecia; teratogenic risk if pregnant.
- B. Finasteride inhibits type II 5 $\alpha$ -reductase; dutasteride inhibits both type I and II; used for BPH and androgenetic alopecia; teratogenic risk if pregnant.**
- C. Finasteride inhibits aromatase; dutasteride inhibits estrogen receptors; used for breast cancer.
- D. Finasteride is a glucocorticoid receptor antagonist; dutasteride is a mineralocorticoid receptor antagonist; used for diarrhea.

*The key idea here is how finasteride and dutasteride differ in which 5 $\alpha$ -reductase enzymes they block and what that means for their effects. Finasteride selectively inhibits type II 5 $\alpha$ -reductase, which is the form mainly found in the prostate and hair follicles. Dutasteride inhibits both type I and type II 5 $\alpha$ -reductase, leading to a broader and greater overall reduction of dihydrotestosterone (DHT). Since DHT drives prostate enlargement and androgenetic alopecia, both drugs help with benign prostatic hyperplasia and male-pattern hair loss. Finasteride is particularly associated with hair-loss treatment, while dutasteride provides a stronger, more comprehensive DHT suppression and is widely used for BPH and also employed for hair loss in practice. A key safety point is that both have teratogenic risk if pregnancy occurs, because altering DHT pathways can affect fetal development, so exposure is avoided in pregnancy. The other options describe mechanisms (aromatase, estrogen receptors, glucocorticoid or mineralocorticoid receptors) that these drugs do not target or use, so they don't fit.*

## 2. What is the MOA and clinical use of ketoconazole in hormonal disorders?

- A. It is a beta-blocker used for tachyarrhythmias.
- B. It inhibits fungal CYP450 enzymes and mammalian steroid synthesis at higher doses; used off-label for Cushing's disease; monitor for hepatotoxicity.**
- C. It acts as a glucocorticoid receptor antagonist; used for Cushing's syndrome medically.
- D. It inhibits aromatase; used in breast cancer treatment in premenopausal women.

*Ketoconazole acts as an antifungal by inhibiting fungal cytochrome P450 enzymes, but at higher doses it also blocks several mammalian steroidogenic enzymes. This reduces the synthesis of cortisol and androgens, which is why it can be used off-label to treat Cushing's disease by lowering cortisol production. A key safety point is hepatotoxicity, so liver function monitoring is essential. It is not a beta-blocker, glucocorticoid receptor antagonist, or aromatase inhibitor, which is why the described MOA and use align best with this drug.*

**3. Which adverse effect is specifically associated with 17 $\alpha$ -alkylated testosterone formulations and requires monitoring?**

- A. Kidney failure
- B. Hypercalcemia
- C. Hypertension
- D. Liver toxicity**

*The important idea is that oral 17 $\alpha$ -alkylated testosterone formulations are especially linked to liver toxicity. The 17 $\alpha$ -alkyl group makes these drugs orally active by preventing rapid hepatic breakdown, but that same modification increases their exposure to the liver and can cause hepatic injury, cholestasis, or hepatic adenomas over time. Because of this, monitoring liver health is essential—regular liver function tests (AST/ALT, bilirubin, alkaline phosphatase) and watching for signs such as jaundice, right upper quadrant pain, fatigue, or hepatomegaly are key. Other adverse effects like kidney failure, hypercalcemia, or hypertension aren't specifically tied to this drug class in the same way, so liver toxicity stands out as the adverse effect that warrants routine monitoring.*

**4. Which drug is described as an abortifacient and a progesterone receptor antagonist?**

- A. Norgestimate
- B. Dinoprostone
- C. Misoprostol
- D. Mifepristone (RU-486)**

*The concept being tested is how a drug can terminate a pregnancy by blocking the hormone that maintains it. Mifepristone acts as a progesterone receptor antagonist, binding with high affinity and blocking progesterone signaling. Without progesterone's support, the endometrium breaks down, the pregnancy loses its placental support, and the cervix dilates while the uterus becomes more contractile. This creates a favorable environment for expulsion, especially when followed by a prostaglandin like misoprostol, which stimulates strong uterine contractions to complete the abortion. Among the listed drugs, only this one directly antagonizes the progesterone receptor and thus has abortifacient effects, whereas the others are either progestins used for contraception or prostaglandin agents that promote contractions without blocking progesterone receptors.*

**5. Dienestrol is described with which route of administration in the material?**

- A. Intravenous
- B. Intramuscular
- C. Topical**
- D. Oral

*Topical use is chosen because dienestrol is described as a synthetic estrogen intended to act where it's applied, providing local estrogenic effects on mucosal or skin tissues. Applying it topically delivers the hormone directly to the target area (for example, vaginal or cutaneous tissues) and keeps systemic exposure relatively low, which helps minimize systemic estrogen risks while still giving therapeutic benefit at the site. Oral or parenteral routes would push the drug into systemic circulation and undergo higher hepatic metabolism, increasing the chance of systemic side effects and broader estrogenic effects. Intravenous or intramuscular administration would also produce more widespread effects than the local needs require.*

## 6. Raloxifene belongs to which class?

- A. Biologic
- B. Small molecule
- C. Selective estrogen receptor modulator (SERM)/Benzothiophene**
- D. Calcitonin analog

*Raloxifene is a selective estrogen receptor modulator, a nonpeptide small molecule with a benzothiophene chemical scaffold. It binds estrogen receptors and has tissue-specific effects: it acts like estrogen in bone to help maintain density, while blocking estrogen effects in breast and uterus. This combination—agonist actions in some tissues and antagonist actions in others—defines its SERM classification. It's not a biologic (not a protein), nor a calcitonin analog, and while it is a small molecule, the most precise clinical category is SERM (with benzothiophene as its chemical class).*

## 7. What is the mechanism of SGLT2 inhibitors and a key risk?

- A. Inhibit hepatic glucose production; risk lactic acidosis
- B. Inhibit intestinal glucose absorption; risk GI upset
- C. Inhibit glucose reabsorption in proximal tubule; risk genital infections**
- D. Increase insulin secretion; risk hypoglycemia

*SGLT2 inhibitors work by blocking the sodium-glucose cotransporter 2 in the kidney's proximal tubule, which normally reabsorbs most of the filtered glucose. When this transporter is inhibited, glucose remains in the filtrate and is excreted in the urine (glycosuria), lowering blood glucose without relying on insulin. That urinary glucose creates a growth substrate for microbes in the genitourinary tract, so genital infections (and UTIs) are a well-known risk. This matches the mechanism of action—inhibiting glucose reabsorption in the proximal tubule—and the key risk of genital infections. They do not inhibit hepatic glucose production, nor intestinal glucose absorption, nor do they increase insulin secretion; hypoglycemia is not a typical risk unless combined with other glucose-lowering agents.*

## 8. Which statement differentiates GLP-1 receptor agonists from DPP-4 inhibitors?

- A. GLP-1 receptor agonists mimic incretin and increase glucose-dependent insulin secretion, often with weight loss.
- B. DPP-4 inhibitors mimic incretin and cause weight loss.
- C. GLP-1 receptor agonists are glucose-independent and cause weight gain.
- D. DPP-4 inhibitors boost endogenous incretin by inhibiting degradation and are weight neutral.**

*Understanding how these drugs modulate the incretin system helps explain the difference. GLP-1 receptor agonists provide a direct GLP-1 receptor stimulus, leading to glucose-dependent insulin secretion and commonly associated weight loss due to appetite suppression and slower gastric emptying. DPP-4 inhibitors, in contrast, don't supply a GLP-1 mimic. They inhibit the enzyme that degrades endogenous incretins, so the body's own GLP-1 and GIP levels rise modestly, boosting the incretin effect without the same degree of receptor stimulation. This yields a milder glucose-lowering effect and typically weight neutrality. So the statement that best differentiates them is that DPP-4 inhibitors boost endogenous incretin by inhibiting degradation and are weight neutral.*

**9. Which statement about aromatase inhibitors is true?**

- A. They can contribute to decreased bone mineral density and osteoporosis risk**
- B. They increase bone density
- C. They worsen sleep quality only
- D. They have no effect on bones

*Aromatase inhibitors block the conversion of androgens to estrogen, so estrogen levels drop. Estrogen helps protect bone by keeping the balance between bone formation and resorption; when estrogen falls, osteoclast activity increases and bone resorption rises. This leads to a decrease in bone mineral density and a higher risk of osteoporosis and fractures. That's why the true statement is that these drugs can contribute to reduced bone mineral density and osteoporosis risk. They do not increase bone density, and while they can cause other side effects like hot flashes or sleep disturbances, the bone effects are a key and well-established concern. They also certainly do have effects on bones, so the option claiming no effect on bones isn't correct.*

**10. How do teriparatide differ from denosumab in osteoporosis therapy?**

- A. Teriparatide inhibits osteoclast activity; denosumab stimulates bone formation
- B. Teriparatide is an anabolic PTH analog; denosumab is a RANKL inhibitor that reduces bone resorption**
- C. Teriparatide replaces calcitonin; denosumab inhibits calcium absorption
- D. Teriparatide increases bone resorption; denosumab increases bone formation

*The key idea is the difference between anabolic therapy that builds bone and antiresorptive therapy that slows bone loss. Teriparatide, a recombinant PTH(1-34) analog given intermittently, acts as an anabolic agent by stimulating osteoblast activity and promoting new bone formation, even though bone turnover as a whole increases. Denosumab is a monoclonal antibody against RANKL, blocking the interaction that drives osteoclast formation and activity, so it mainly reduces bone resorption and slows bone loss. The option that describes teriparatide as an anabolic PTH analog and denosumab as a RANKL inhibitor that reduces bone resorption is the accurate one. The other statements mix up the effects—teriparatide does not inhibit osteoclasts directly or stimulate bone formation, and denosumab does not inhibit calcium absorption or replace calcitonin.*

## Next Steps

Congratulations on reaching the final section of this guide. You've taken a meaningful step toward passing your certification exam and advancing your career.

As you continue preparing, remember that consistent practice, review, and self-reflection are key to success. Make time to revisit difficult topics, simulate exam conditions, and track your progress along the way.

If you need help, have suggestions, or want to share feedback, we'd love to hear from you. Reach out to our team at [hello@examzify.com](mailto:hello@examzify.com).

Or visit your dedicated course page for more study tools and resources:

<https://hormonaldrugsexam1.examzify.com>

We wish you the very best on your exam journey. You've got this!

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