ERRATA IN THE PHARMACOTHERAPY SELF-ASSESSMENT PROGRAM FIFTH EDITION (PSAP-V)
(July 17, 2006)

Book 1 (Cardiology)

Front Inside Cover	It should read: If you are completing this module for ACPE credit, you have until **January 31, 2007**, 3 years from the date of publication, to return test sheets to ACCP.
Page 11	Table 1-5 (first page) should be replaced with the enclosed table, which has the correct wording.

Book 2 (Health Care Stakeholders)

Preface	Katherine Chessman's name should be listed as a reviewer for the Medication Safety chapter. Katherine Hammond Chessman, Pharm.D., FCCP, BCPS, BCNSP *Associate Professor* Department of Pharmacy and Clinical Sciences Medical University of South Carolina *Clinical Pharmacy Specialist* Pediatrics/Pediatrics Surgery MUSC Children's Hospital Charleston, South Carolina
Answer Book (Page 46)	Answer 85 should read: **85. Answer: B**

Book 3 (Psychiatry)

Answer Book (Page 27)	Answer 11 should read: **11. Answer: A**

Book 4 (Geriatrics/Special Populations)

Pages 3-4	Figure 1-1 and Figure 1-2 should be replaced with the enclosed figures, which have the corrected subscripts.
Page 208	The fentanyl dose listed in the middle of second column should be changed to the following: **25 mcg** instead of 25 mg

Book 5 (Science and Practice of Pharmacotherapy)

Page 27	Figure 2-3 should be replaced with the enclosed figure, which has the correct arrows.
Page 175	**First column; Line 41.** 17-α-hydroxyprogesterone caproate should be changed to **heparin**.

Book 6 (Infectious Diseases)

Page 169

First column, Line 5. Sentence should read:

Linezolid also is bacteriostatic and also does have activity against both *E. faecium* and *E. faecalis*.

Page 170

First column, Line 24. Cefuroxime in the sentence should be changed to the following drug:

Cefotaxime

Page 259

Figure 2-1 should be replaced with the enclosed figure, which contains wording that dropped out in the printing process.

Page 269

Figure 2-3 should be replaced with the enclosed figure, which has the correct shading that dropped out in the printing process.

Answer Book (Page 57)

Answer 45 should read:

45. Answer: A

Book 7 (Gastroenterology/Nutrition)

Page 1

First column, First paragraph under Introduction, Line 4. Sentence should read:

It was not until the 8th century that hepatitis was though to be infectious in nature.

Page 28

Question 12, Answer D should read:

D. Treatment duration for 48 weeks; 24 weeks of treatment to determine H.E. will benefit from treatment.

Page 29

Question 14, Answer D should read:

D. Administer hepatitis B immune globulin within 7 days of birth and hepatitis B vaccine within 12 hours of birth.

Page 98

Second column, Second paragraph under Acute Phase, Lines 8-9. Sentence should read:

The caloric recommendation for PN is based on whether nutrition is used for maintenance or correction of malnutrition.

Page 109

First column, Second paragraph, Lines 3 and 4. Sentence should read:

Probiotics, such as *Bifidobacterium breve* and *Lactobacillus casei*, may be given to recolonize the intestine.

Page 211

Table 2-5 was not included in the text. See enclosed table.

Page 250

Question 69, Answer A should read:

A. 6.3 mcg.

Answer Book, (Page 63)

Answer 69 should read:

IFE: 10 g = 50 mL IFE 20%
(50 mL IFE) x (0.025 mcg/mL Al) = 1.25 mcg Al
total Al provided by dextrose + amino acids + IFE = 6.3 mcg
(Answer A)

Book 8 (Chronic Illnesses)

Page 105

Equation should read:

Adjusted concentration = observed concentration/(0.25 x albumin) + 0.1

Page 138

Question 33. Question should read:

Based on S.M.'s current situation with menstrual migraine, what is the **best drug** to prevent future attacks?

Page 138

Question 39. Question should read:

What **statement** most accurately makes reference to the International Headache Society (HIS) criteria for associated symptoms?

Page 257

Second column, first paragraph under Calcipotriene, Lines 1-2.
Sentence should read:

Calcipotriene, a vitamine D_3 analog, inhibits keratinocyte differentiation and proliferation.

Second column, first paragraph under Tazarotene, Lines 3-4.
Sentence should read:

"...reverse keratinocyte hyperproliferation, and reduce the influx of inflammation cells in the skin."

Page 286

Question 88, Line 4 should read:

"...receiving oral methotrexate 10 mg once weekly..."

Book 9 (Chronic Illnesses IV/Pediatrics)

Page 176

Question 56, Answer A should read:

Amoxicillin suspension 400 mg/5 mL, 5 mL 2 times/day for 10 days.

Page 299

Question 68, Answer A should read:

A. Anti-seizure pharmacotherapy.

Table 1-5. Pharmacotherapy for Acute Coronary Syndromes (ST-segment Elevation and non-ST-segment Elevation)

Drug Category	Clinical Condition and ACC/AHA Guideline Recommendation	Contraindications[a]	Dose
Aspirin	STE ACS, class I recommendation[b] for all patients NSTE ACS, class I recommendation for all patients	Hypersensitivity Active bleeding Severe bleeding risk	160–325 mg on hospital day 1 75–325 mg/day starting hospital day 2 and continued indefinitely
Clopidogrel	STE ACS, class II recommendation in patients allergic to aspirin NSTE ACS, class I recommendation for all hospitalized patients in whom a noninterventional approach is planned In PCI in STE and NSTE ACS, class I recommendation	Hypersensitivity Active bleeding Severe bleeding risk	300-mg loading dose on hospital day 1 followed by a maintenance dose of 75 mg orally every day starting on hospital day 2 Administer indefinitely in patients with an aspirin allergy (class I recommendation) Administer for at least 9 months in medically managed patients with NSTE ACS (class I recommendation) Administer for at least 30 days to 1 year in patients with STE or NSTE ACS (class I recommendation) undergoing PCI If possible, withhold for at least 5 days in patients whom CABG is planned to decrease bleeding risk (class I recommendation)
Unfractionated heparin	STE ACS, class I recommendation in patients undergoing PCI; class IIa recommendation for patients treated with alteplase, reteplase or tenecteplase; class IIa recommendation for patients not treated with thrombolytic therapy NSTE ACS, class I recommendation in combination with aspirin In PCI in STE and NSTE ACS, class I recommendation	Active bleeding History of heparin-induced thrombocytopenia Severe bleeding risk Recent stroke	60 units/kg intravenous bolus Constant intravenous infusion at 12 units/kg/hour (maximum 1000 units/hour) Titrate to maintain aPTT between 1.5 and 2.5 times control for NSTE ACS and 50–70 seconds in STE ACS The first aPTT should be measured at 4–6 hours for NSTE ACS and STE ACS in patients not treated with thrombolytics The first aPTT should be measured at 3 hours in patients with STE ACS who are treated with thrombolytics
Low-molecular-weight heparin	NSTE ACS, class I recommendation in combination with aspirin; class IIa recommendation over UFH in patients without renal failure who are not anticipated to undergo coronary artery bypass graft surgery within 24 hours	Active bleeding History of heparin-induced thrombocytopenia Severe bleeding risk Recent stroke Dalteparin: creatinine clearance < 30 ml/minute Enoxaparin: creatinine clearance < 10 ml/minute	Enoxaparin 1 mg/kg SC every 12 hours *Adjustment for renal insufficiency*: 1 mg/kg SC every 24 hours for patients with creatinine clearance < 30 ml/minute
Thrombolytic therapy	STE ACS, class I recommendation in patients age < 75 years presenting within 12 hours after the onset of symptoms; class IIa recommendation in patients age 75 years and older; class IIb in patients presenting between 12 and 24 hours after the onset of symptoms with continuing signs of ischemia NSTE ACS, class III recommendation	Absolute and relative contraindications per Table 1-7	Streptokinase: 1.5 MU IV over 60 minutes Alteplase: 15 mg IV bolus followed by 0.75 mg/kg IV over 30 minutes (maximum 50 mg) followed by 0.5 mg/kg (maximum 35 mg) over 60 minutes (maximum dose = 100 mg) Reteplase: Two doses of 10 units IV, 30 minutes apart Tenecteplase < 60 kg = 30 mg IV bolus 60–69.9 kg = 35 mg IV bolus 70–79.9 kg = 40 mg IV bolus 80–89.9 kg = 45 mg IV bolus ≥ 90 kg = 50 mg IV bolus

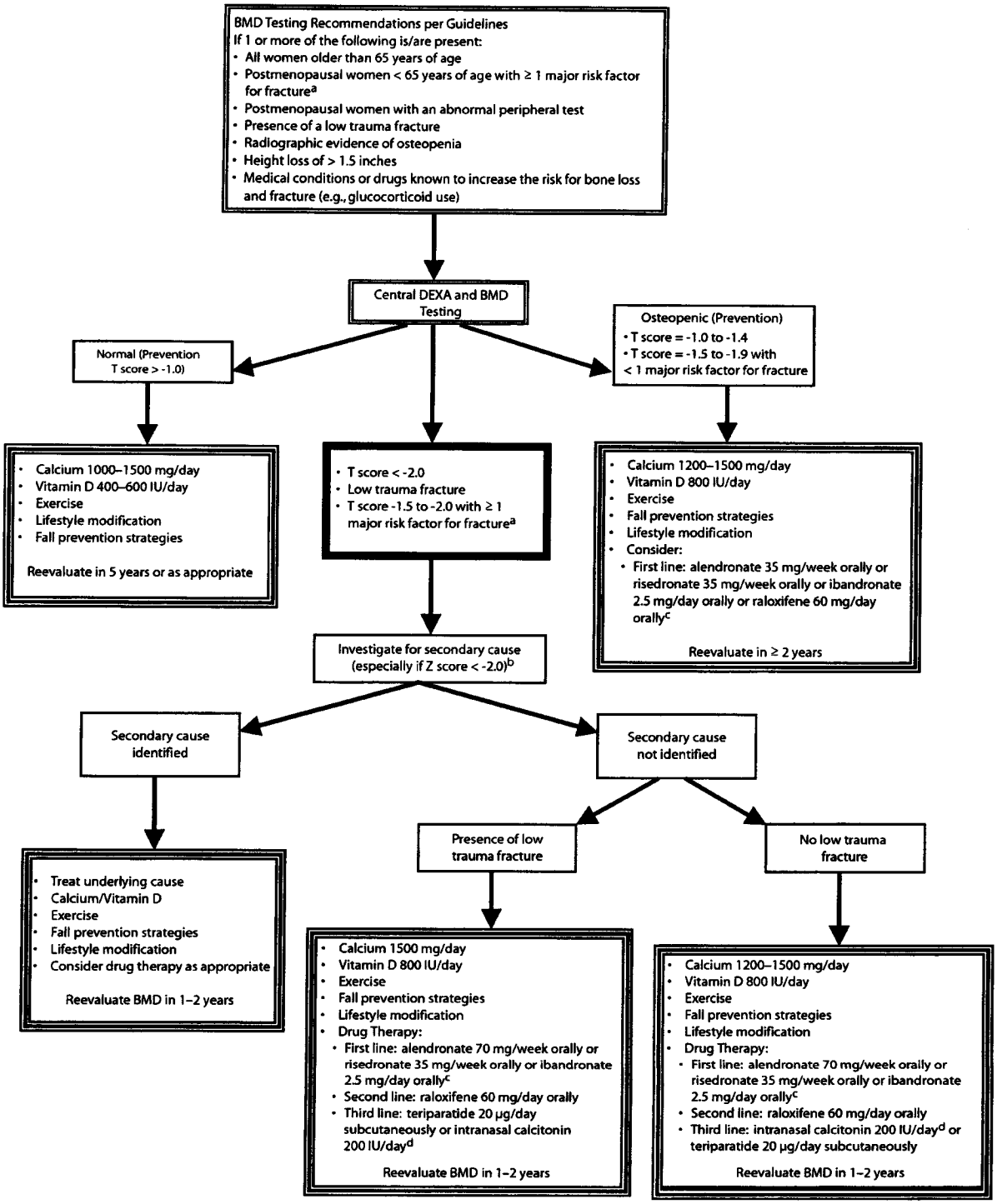

Figure 1-1. Management of osteoporosis in postmenopausal women.

[a]Major risk factors (independent risk factors for fracture): current smoker, low body weight (less than 127 pounds), history of osteoporotic fracture in a first-degree relative, and personal history of fracture as an adult (after 40 years of age)

[b]Examples of secondary causes: (medical conditions: rheumatoid arthritis, multiple myeloma, hyperparathyroidism, Cushing's syndrome, chronic obstructive pulmonary disease, hyperthyroidism, inflammatory bowel disease, chronic kidney disease, and organ transplantation); (drugs: glucocorticoids, anticonvulsants, heparin, neuroleptics, gonadotropin-releasing hormone agonists, and methotrexate).

[c]The biphosphonates (alendronate, risedronate, and ibandronate) should not be used if creatinine clearance is less than 30 ml/minute.

[d]Can be used short term in combination with a first- or second-line therapy for managing pain in patients with an acute vertebral fracture.

BMD = bone mineral density; DEXA = dual-energy x-ray absorptiometry; IU = international units.

Reprinted with permission from American College of Clinical Pharmacy. Follin SL. Update in Osteoporosis. In: Mueller BA, Bertch KE, Dunsworth TS, et al, eds. Pharmacotherapy Self-Assessment Program, 4th ed. Men's Health Module. Kansas City, MO: ACCP, 2003:349.

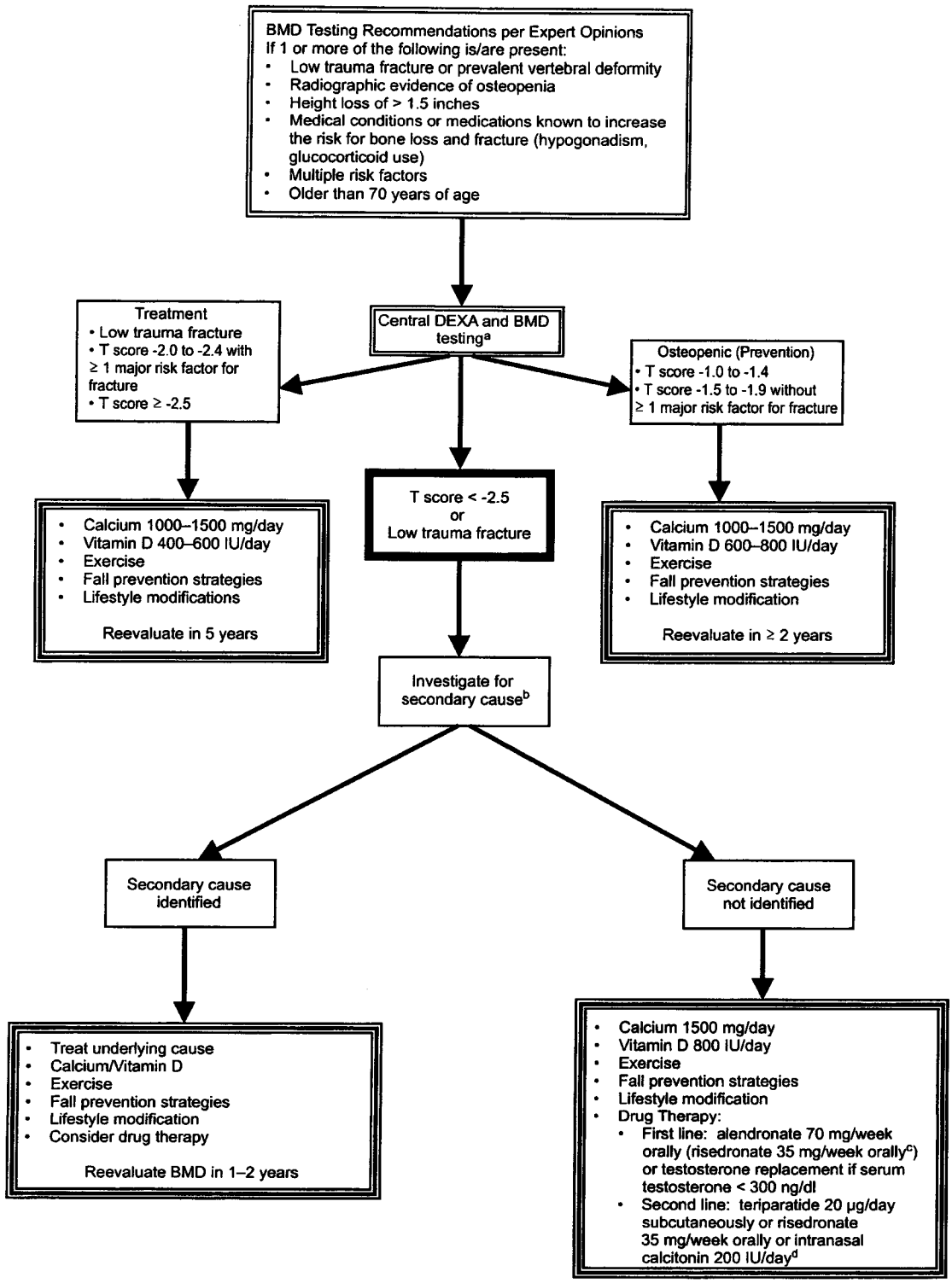

Figure 1-2. Management of osteoporosis in men.
[a]Based on a normal male reference database.
[b]Examples of secondary causes: (medical conditions: rheumatoid arthritis, multiple myeloma, hyperparathyroidism, Cushing's syndrome, chronic obstructive pulmonary disease, hyperthyroidism, inflammatory bowel disease, chronic kidney disease, and organ transplantation); (drugs: glucocorticoids, anticonvulsants, heparin, neuroleptics, gonadotropin-releasing hormone agonists, and methotrexate).
[c]Risedronate may be considered if alendronate is not available or cannot be tolerated.
[d]Can be used short term in combination with a first- or second-line therapy for managing pain in patients with an acute vertebral fracture.
BMD = bone mineral density; DEXA = dual-energy x-ray absorptiometry; IU = international units.
Reprinted with permission from American College of Clinical Pharmacy. Follin SL. Update in Osteoporosis. In: Mueller BA, Bertch KE, Dunsworth TS, et al, eds. Pharmacotherapy Self-Assessment Program, 4th ed. Men's Health Module. Kansas City, MO: ACCP, 2003:350.

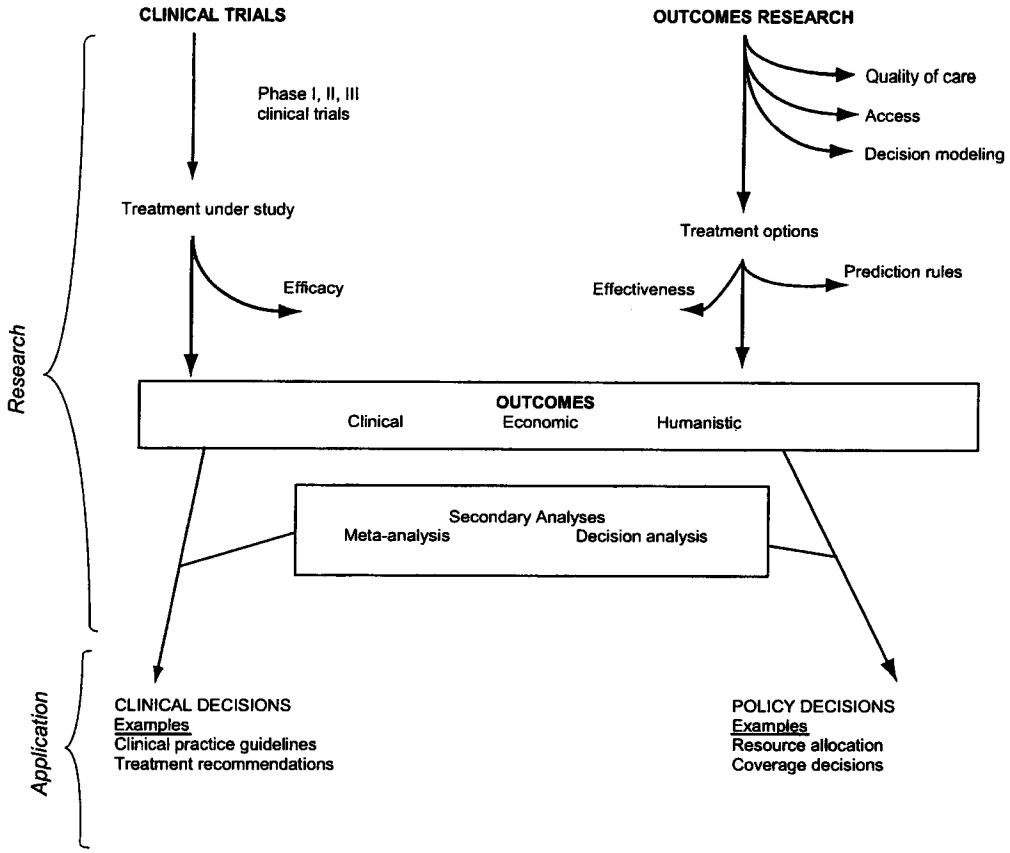

Figure 2-3. Conceptual framework for understanding outcomes research.
Adapted with permission from the National Cancer Institute. Lee SJ, Earle CC, Weeks JC. Outcomes research in oncology: history, conceptual framework, and trends in the literature. J Natl Cancer Inst 2000;92:195–204.

Conceptual Framework of Outcomes Research

As the field of outcomes research has grown, confusion has been created about what exactly constitutes outcomes research versus other more traditional types of clinical research. Several authors have attempted to address this from a conceptual standpoint. In 2000, one such conceptual model was developed to provide an overview of, or conceptual framework for, outcomes research. The model originally was proposed for oncology-related research; however, it applies to all types of outcomes research, regardless of disease state. The model is presented here because it both distinguishes outcomes research from clinical trials and demonstrates the link between research and application.

This conceptual framework compares outcomes research to clinical trials research and attempts to explain the appropriate application of research findings, as shown in Figure 2-3. This model reflects the diverse nature of outcomes research. Unlike other research specialties, there is no unique commonality that defines outcomes research. Instead, outcomes research studies can be identified by a combination of factors, such as research topic, study design, method and source of data, and perhaps most important exclusion. Examples of outcomes research include studies of the quality of health care, studies of access to care provided, and evaluations of medical decision-making and prediction rules. Of interest, clinical trials and outcomes research often have end points that look similar. Thus, such factors as the setting and type of research question become important in differentiating outcomes research from clinical trials. Still, there can be gray areas, such as randomized trials conducted in actual practice settings that are considered a hybrid between clinical trials and outcomes research.

The unique aspect of this model is that it shows the link between outcomes research and application. Clinical decisions are heavily driven by clinical trials, whereas policy decisions primarily are driven by outcomes research. Pharmacists and other health care providers are familiar with using evidence-based medicine to guide their clinical decision-making process. However, nonclinicians, such as administrators and policy-makers, often need help to translate evidence to develop or modify health care policies. Outcomes researchers are in a unique position to help translate research findings for these interested parties.

Outcomes research cannot have an impact unless the results are generalized and acted on. Though not clearly shown or described in this model, it is important to

Lee SJ, Earle CC, Weeks JC. Outcomes research in oncology: history, conceptual framework, and trends in the literature. J Natl Cancer Inst 2000;92:195–204.

Because higher organisms, including mammalian cells lack a cell wall, it is an ideal and selective target for antimicrobial drug therapy in both bacteria and fungi. Bacteria cell walls are composed principally of peptidoglycan, teichoic acids, or lipopolysaccharides, whereas fungi cell walls are composed primarily of glucan and mannan. Consequently, cell-wall active antibacterial drugs, such as penicillins, have no effect on fungal cell wall integrity. In addition, laboratory procedures that rely on staining of the peptidoglycan to broadly identify pathogens in clinical samples (i.e., Gram's stain) are not as useful for identification of fungal organisms and guiding antifungal therapy. Recently, a new class of antifungal drugs has been introduced (echinocandins) that target cell wall synthesis in fungi by inhibiting the synthesis of a key structural glucan polymer, beta-(1,3)-glucan (Figure 2-1). In general, cell membrane-active drugs, such as the echinocandins, have few collateral targets in human cells, which results in antifungal drugs with fewer toxicities and a lower risk for drug-drug interactions.

Laboratory Diagnosis of Fungal Infections

Laboratory diagnosis of invasive mycoses is essential for selecting appropriate antifungal therapy. Fungi typically are classified in the laboratory on the basis of two basic morphologic forms: yeast or mould. Yeast are single-cell fungi that are spherical or oval in shape that multiply by budding or fission—a form of asexual reproduction that gives rise to a daughter cell that is identical to the parent (Figure 2-2). Frequently, buds may fail to detach from the parent cell, forming a chain of elongated cells called pseudohyphae (Figure 2-2). The most common medically important yeast is *Candida albicans*, which exists as part of the normal microbial flora of the gastrointestinal and genitourinary (female) tract. This species can be distinguished within 4 hours from most other common *Candida* species by the formation of germ tubes when the isolate is incubated in serum at 37°C (Figure 2-2). However, a positive germ tube test should be considered

Table 2-1. Common Human Mycoses

Infection Sites
Superficial mycoses
Malassezia furfur
Exophiala werneckii
Trichosporon beigelii
Piedra hortae
Microsporum, *Trichophyton*, and *Epidermophyton floccosuma*
Candida albicans and other *Candida* species[a]
Subcutaneous mycoses
Fonsecaea pedrosoi, Phialophora verrucosa and others
Pseudallescheria boydii, Madurella mycetomatis and others
Sporothrix schenckii
Deep mycoses
Primary (endemic)
Histoplasma capsulatum[a]
Coccidioides immitis[a]
Paracoccidioides brasiliensis
Blastomyces dermatitidis[a]
Opportunistic
Yeast
Candida[a]
Cryptococcus neoformans[a]
Trichosporon species and others
Hylophyphomycetes
Aspergillus fumigatus and other species[a]
Fusarium solani and *Fusarium oxysporum*
Zygomycoses (*Mucor, Absidia, Rhizopus, Cunninghamella, and Rhizomucor*)
Penicillium
Phaeohyphomycetes
Pseudallescheria boydii (*Scedosporium* species)
Bipolaris
Alternaria
Other
Pneumocystis jirovecii (carinii)[a,b]

[a]Most common in United States.
[b]Recently reclassified as a fungus.

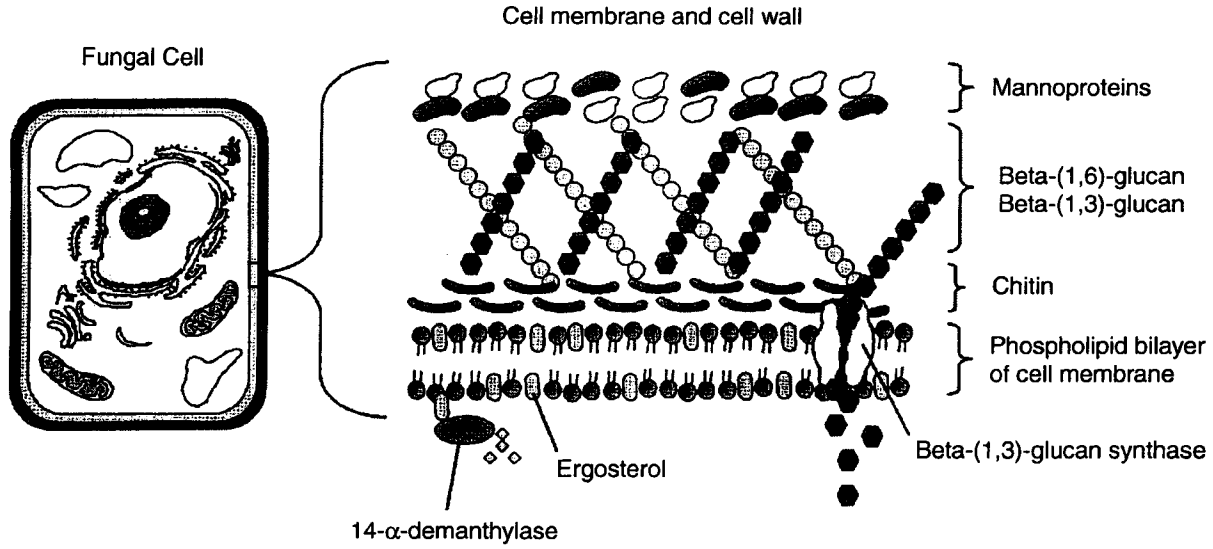

Figure 2-1. Structure of the fungal cell wall and membrane and associated antifungal targets.

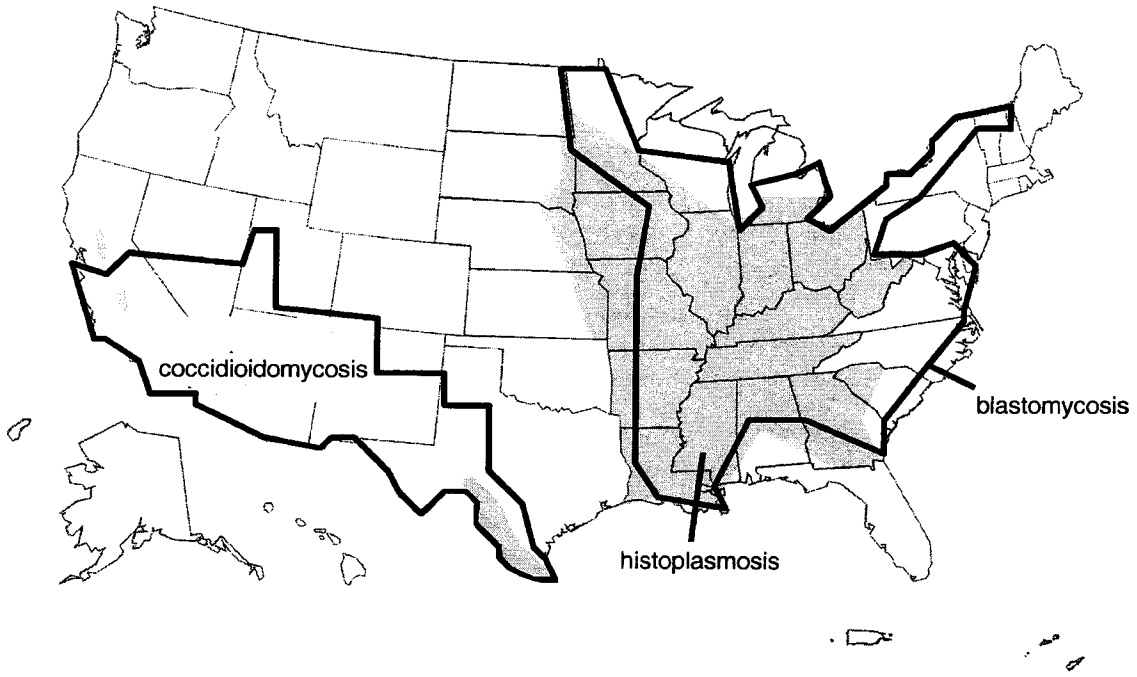

Figure 2-3. Geographic localization of primary (endemic) fungi in the United States. Gray areas represent histoplasmosis.

an antifungal drug regimen through (presumably) synergistic antifungal effects, and 3) to overcome the pharmacokinetic limitations of a single antifungal drug for treating life-threatening mycoses in an anatomically privileged sites, such as the central nervous system (CNS). With the possible exception of cryptococcal meningitis, there are few data from controlled, clinical trials supporting the use of combination therapy for deep mycoses. The majority of data suggesting a possible benefit for combination therapy with echinocandin-based combinations is still derived from animal studies, which can only be considered as hypothesis-generating research and not as support for clinical practices. For these reasons, combination antifungal therapy should not be recommended routinely as a first-line strategy for treating opportunistic mycoses but may be considered as a salvage approach in patients with refractory or breakthrough infections who are receiving antifungal prophylaxis.

Primary Systemic Mycoses

Recommendations for diagnosing and treating common mycoses have been developed by the Infectious Diseases Society of America (IDSA) and currently are in the process of being revised on an individual basis for each mycosis. The guidelines attempt (where possible) to provide evidence-based treatment approaches and recommendations for managing primary and systemic deep mycoses. However, the majority of recommendations provided reflect data generated from nonrandomized, case-controlled studies or are the opinion of respected authorities based on their clinical experience, descriptive studies, or reports from expert consensus committees. Therefore, the guidelines should be considered a general approach for managing deep mycoses that do not take into account patient-specific factors that may guide selection of therapy. Key recommendations of these guidelines and elements of particular importance to pharmacists are discussed in the following sections for the most common primary and opportunistic fungal infections.

Histoplasmosis

Histoplasmosis is restricted to certain areas of North and Latin America in regions noted for high humidity and moderate temperatures (Figure 2-3). *Histoplasma capsulatum* var. *capsulatum*, the causative fungus of histoplasmosis in North America, grows best in soil contaminated with bird or bat droppings, which enhance the sporulation of the fungus. Common activities that lead to significant *H. capsulatum* exposure include cave exploration (bat droppings), working in or demolishing chicken coops, use of avian excreta as fertilizer, demolition of older buildings, or wood cutting in forests with large bird roosts. These activities can aerosolize large numbers of *H. capsulatum* spores that, when inhaled, cause localized or patchy pneumonitis. Although the attack rate may approach 100% in some endemic areas, immunocompromised patients and children are more prone to develop symptoms after primary infection.

Johnson MD, MacDougall C, Ostrosky-Zeichner L, Perfect JR, Rex JH. Combination antifungal therapy. Antimicrob Agents Chemother 2004;48:693–715.

Table 2-5. Purported Activities of Ingredients in Dietary Supplements Marketed for Weight Loss

Increased thermogenesis
Modulation of fat or carbohydrate absorption or metabolism
Appetite suppression
Satiety promotion
Laxation
Diuresis

regarding an association with hemorrhagic stroke, the over-the-counter drug market for weight loss is sparse. Benzocaine, which can produce appetite suppression by a local anesthetic effect, is still available in a limited number of products. The more loosely regulated dietary supplement market discussed in the Dietary Supplements section fills the gap left by the dearth of products marketed as over-the-counter weight loss drugs. Data collected from BRFSS in 1998 (before the removal of phenylpropanolamine from the market) indicated that about 7% of people in five selected states had used either nonprescription or dietary supplement products marketed for weight loss in the previous 2 years. Alarmingly, almost 8% of women of normal weight reported use of these products.

Dietary Supplements

Dietary supplements marketed for weight loss frequently contain several ingredients with a variety of purported mechanisms. Some more common proposed activities of the ingredients included in dietary supplements marketed for weight loss are listed in Table 2-5. There currently are not enough safety and efficacy data available to allow a pharmacist to confidently recommend any of the dietary supplements marketed for weight loss.

In April 2004, it became illegal to sell dietary supplements containing ephedrine alkaloids. The FDA determined that supplements containing ephedrine alkaloids pose an unreasonable health risk to consumers and are adulterated. Ironically, data supporting ephedrine alkaloids either alone or in combination with caffeine for weight loss were probably stronger than data supporting any other dietary supplement for this indication. However, the FDA ruled that the adverse cardiovascular effects from ephedrine alkaloids outweigh the benefits of the modest weight loss. Also banned are country mallow and heartleaf because these contain ephedrine alkaloids. Traditional Chinese herbal remedies containing ephedrine alkaloids are not marketed as dietary supplements and are not covered by the ban. In addition, American species of *Ephedra*, contained in Mormon tea, do not actually contain ephedrine alkaloids and are similarly unaffected by the ban. In April 2005, a federal judge ruled in favor of a company that had distributed ephedra as a product designed to deliver the substance at a low dose (no more than 10 mg/day). The effect of this ruling on the availability of ephedra currently is unclear.

Thyroid-containing and thyroid-stimulating dietary supplements have been promoted for weight loss. The FDA has ordered removal of some thyroid-containing products over the past several years. Bladderwrack is an herbal product believed to possess thyroid-stimulating properties. This product has a high iodine content and should not be recommended for weight loss.

Table 2-6. Prescription Drugs Labeled for Treating Obesity

Drug	Category	Controlled Substance Category
Benzphetamine	Adrenergic	III
Diethylpropion	Adrenergic	IV
Mazindol	Adrenergic	IV
Orlistat	Lipase inhibitor	None
Phendimetrazine	Adrenergic	III
Phentermine	Adrenergic	IV
Sibutramine	Adrenergic/serotonergic	IV

Prescription Therapy

History and Present Usage

Pharmacological therapy of obesity has been fraught with problems for more than a century. Thyroid extract use as early as the 1890s led to problems secondary to hyperthyroidism. Of interest, a century later, the FDA was still dealing with what were deemed to be unsafe dietary supplements containing thyroid extracts. Amphetamines were first promoted as dietary aids in the 1930s but fell into disfavor after a few decades because of the high addiction potential. The serotonergic drug aminorex, an obesity drug marketed in Europe, was withdrawn from the European market in the early 1970s because of reports of pulmonary hypertension. The serotonergic drug fenfluramine was first labeled for weight loss by the FDA in the early 1970s, but its use was limited until the early 1990s when research indicated its efficacy when combined with the adrenergic phentermine, the so-called phen-fen combination. When dexfenfluramine (another serotonergic drug) received label approval by the FDA in 1996, it became the first new prescription drug labeled for use in obesity in more than a decade. Reports of primary pulmonary hypertension, cardiac valvulopathies, and neurotoxicity led to the withdrawal of fenfluramine and dexfenfluramine from the United States market in 1997. Critics have stated that these effects should have been predicted and avoided based on the previous adverse experiences seen with aminorex.

The current prescription drugs available for treating obesity, classified as controlled substance and general categories, are listed in Table 2-6. All of these drugs are indicated only for short-term use (no more than 12 weeks) except for the two newest drugs, orlistat (tetrahydrolipostatin) and sibutramine, which are indicated for long-term use. According to the NHLBI guidelines, orlistat and sibutramine may be useful adjuncts to diet and physical activity. These drugs are only indicated for patients with a BMI of at least 30 kg/m^2 without concomitant obesity-related risk factors, such as hypertension and DM, or a BMI of at least 27 kg/m^2 with concomitant obesity-related risk factors.

Data from the 1998 BRFSS indicated that about 2.5% of United States adults had used prescription weight loss products in the previous 2 years. Alarmingly, 25% of

ASHP therapeutic position statement on the safe use of pharmacotherapy for obesity management in adults. Developed by the ASHP Commission on Therapeutics and approved by the ASHP Board of Directors on April 23, 2001. Am J Health-Syst Pharm 2001;58:1645–55.