When liposomes were initially discovered more than 30 years ago, predictions were largely met with skepticism; however, recent advances have allowed the development of liposomes that are potentially medically useful. Systemic and topical drug delivery by liposomes is possible. Although liposomal amphotericin B, a systemic antifungal agent, is the only medical liposomal product commercially available in the United States, there have been considerable advances in liposomal delivery systems with important applications in dermatology. In this article, the basic chemistry of liposomes will be reviewed as well as their potential uses in dermatology. The basic chemistry and a brief overview of methods of preparation will be discussed first. Then we will examine the kinetics of systemic and topical liposomal therapy, as well as hair follicle targeting. Next, the liposomal advances in skin cancer prevention, cancer therapy, and gene therapy will be discussed. Finally, specific liposomal drugs and drug classes, including topical anesthetics, topical corticosteroids, topical interferon, liposomal amphotericin B, and artificial blood, will be examined. We will not discuss cosmetic applications of liposome preparations.

CHEMISTRY AND PHARMACOKINETICS

What is a Liposome?

Alec Bangham discovered liposomes in 1963, when he demonstrated that phospholipids in water form closed vesicles.¹ The liposome consists of one or more lipid bilayer membranes in which the fatty acid tails are embedded in the interior and the hydrophilic heads are oriented towards the external aqueous phase. The lipid bilayers form concentric lamellae, enveloping a series of aqueous compartments (Fig. 1). Although most liposomes are spherical, the lipid composition and the method of preparation used determine their final shape, size, and the number of lamellations achieved. Hydrophilic drugs are encapsulated within the inner aqueous compartments, whereas hydrophobic drugs are embedded in the phospholipid bilayer that makes up the liposome. Liposomes can be classified as unilamellar or multilamellar, based on the number of concentric lipid bilayers they contain. They can be further classified according to size and composition of the lipid bilayers. The lipid composition and the method of liposome preparation directly affects drug absorption, distribution, metabolism, and elimination, as well as the toxicity profile.

Several methods of liposome preparation can be employed. They include: simple mechanical agitation, sonication, conventional hydration, dehydration-rehydration, and reverse-phase evaporation. Although most liposomes are spherical, the method of preparation used, along with the lipid composition, organic additives, the pH of the aqueous solution, and the temperature of preparation, all determine the size, shape, and number of lamellae in the liposome. Drug incorporation is accomplished by adding hydrophobic drugs to the lipid mixture during preparation or adding hydrophilic drugs to the aqueous salt solution during preparation. In this fashion, hydrophobic drugs are incorporated into the lipid bilayer and hydrophilic drugs are packaged within the interior aqueous compartments.

The simple mechanical agitation method involves mechanical shaking of the component lipids and an aqueous salt solution in a flask. This method produces a heterogeneous mixture of large multilamellar vesicles of varying sizes. Although it is the simplest preparation method, it does not produce a uniform liposome size, so that it is not an ideal method for drug preparation.²

Sonication of these large multilamellar liposomes can be used to make small unilamellar liposomes of uniform size. Once the large multilamellar liposomes are made, they are exposed to prolonged ultrasound radiation that disperses them into small unilamellar liposomes. These small liposomes are then separated from the large multilamellar liposomes by gel-filtration to provide a homogeneous preparation.²

The “film-hydration method” involves dissolving the lipids in an organic solvent which is then allowed to evaporate, leaving a film behind. The film is then hydrated by addition of an aqueous salt solution at a tem-

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The “reverse-phase-evaporation method” is used to prepare large unilamellar liposomes. It involves mixing the lipids, aqueous salt solution, and organic solvents together and sonicating their mixture. After sonication, the solvents and a small quantity of water are removed under nitrogen at a temperature above the phase-transition temperature of the lipids, leaving the large unilamellar liposomes behind.\(^3,4\)

**Systemic Liposomal Kinetics**

The liposomes originally used in medical research were referred to as conventional liposomes. These had limited medical utility because once they were present in the blood stream, they were rapidly phagocytosed by the cells of the reticuloendothelial system (RES).\(^5\) Their therapeutic potential was thus confined to diseases affecting the RES (such as lymphomas or parasitic infestations). Today, liposomes can be engineered to minimize the preferential uptake by the RES. This is accomplished by incorporating glycolipids or ethylene glycol into the lipid bilayer, which forms a steric barrier of highly hydrated groups around the liposome. The steric barrier decreases endocytosis by the RES by inhibiting electrostatic and hydrophobic interactions. Furthermore, incorporation of monosialogangliosides, such as GM1, confers a negative surface charge to the liposome and increases its hydrophilicity. The steric barrier and negative surface charge result in a 100-fold increase in blood circulation time.\(^6\) This results in greater and longer lasting circulating drug levels with lower total doses administered. This, in turn, lowers directly the toxicity for drugs with a narrow toxicity index.

The size of liposomes affects drug deposition and, in this manner, specific tissues may be targeted. A typical large multilamellar liposome can be as large as 20 μm in diameter, whereas a small unilamellar liposome is typically 0.1 μm to 0.15 μm in diameter.\(^9\) In comparison, the typical diameter of an erythrocyte is 7.5 μm, that of a mature melanin granule is 0.4 μm, and an intermediate filament is 0.01 μm (100 Å).\(^10\) Small liposomes extravasate from capillaries more easily and accumulate in tissues, whereas larger liposomes remain in circulation longer and are taken up by reticuloendothelial cells.\(^11\) After endocytosis by the cells of the RES, there is fusion with intracytoplasmic lysosomes with subsequent release of the drug.\(^12\) In this manner, drugs may be targeted to the RES.

Selective targeting of other tissues has been achieved by incorporation of monoclonal antibodies into the outer lipid membrane of small liposomes. In this manner, Heath et al. have been able specifically to target methotrexate-γ-aspartate, an inhibitor of dihydrofolate reductase, to cultured fibroblasts. Although animal studies have not yet been done, one could anticipate introducing intravenous liposomes that extravasate throughout the body, where the antibodies specifically
attach to the target tissue and cause accumulation of liposomes at the target site.\textsuperscript{13,14}

**Topical Liposomal Kinetics**

The stratum corneum is the body's protective layer of skin that prevents the deeper layers from dehydrating and external chemicals and bacteria from entering the skin. Typically, conventional dosage forms, such as solutions, creams, and ointments, deliver drugs in a concentration-dependent manner following Fick's law of passive diffusion across the dead stratum corneum. Multilamellar liposomes can deliver drugs within 30 minutes to the stratum corneum, epidermis, and dermis in significantly higher concentrations than conventional preparations. For example, dermal drug levels delivered by liposomal hydrocortisone are 9 to 14 times higher than after exposure to conventional hydrocortisone preparations.\textsuperscript{15,16} Observations of penetration of radio-labeled liposomes through pig and mouse skin have shown that the ratio of radiolabeled lipids changes from the stratum corneum to the deeper epidermis. This suggests that the liposomal lipid assembly aggregates in the stratum corneum and the component lipids diffuse independently into the deeper layers of the skin. Independent diffusion would be anticipated when the lipids are released from the liposome, because of the variable polarity of lipids.\textsuperscript{15}

The drug is delivered to the deeper epidermal layers through dehydration of the liposomes within the stratum corneum. Paradoxically, when larger quantities or higher concentrations of liposomes are applied, there is a decrease in the amount of drug that penetrates to the deeper layers of skin. A possible explanation for this is, that liposome uptake is driven by the hydration gradient that exists across the epidermis, stratum corneum, and ambient atmosphere.\textsuperscript{17} In this manner, a thinly applied film with a lower concentration of liposomes may penetrate faster and to a greater extent than a thicker, more concentrated, layer. The less concentrated liposome film will dehydrate faster and thus be driven across the hydration gradient faster than the more concentrated preparation.\textsuperscript{3,18} In support of this explanation is the observation that occlusion results in less drug absorption due to abolition of the hydration gradient.\textsuperscript{18}

The melting point of the lipids contained in the liposome also affects the depth of penetration. Higher concentrations of long-chain alcohols in the hydrophobic-hydrophobic interface decrease the transition temperature of the lipid chain-melting phase by partitioning the head group region and altering the phospholipid interdigitation.\textsuperscript{19} Liposomal formulations, using glycerol dilaurate in the lipid bilayer, are able to deposit ten times as much drug in the deeper layers of the epidermis than the standard topical liposomal preparations. The glycerol dilaurate has a melting temperature of 30°C, which is below the skin temperature. The increased penetration may be due to a phase transition of the lipid bilayer, in which there is partial melting of the liposome. This would put the drug in direct contact with the stratum corneum, where it penetrates through to the epidermis.\textsuperscript{20}

The hydrogen ion content of the liposomal preparation also affects penetration. Basic solutions, with a higher pH, cause deprotonation of the lipid molecules. This increases the interbilayer repulsion and the water solubility of the lipids that, in turn, decreases the chain-melting phase transition temperature. The deprotonated lipids also increase the stability of the liposome in solution. There is less liposomal aggregation and the liposomes may be maintained with a smaller diameter; however, the increased stability results in a lower efficiency in membrane fusion and decreases the ability of the liposome to penetrate the stratum corneum.\textsuperscript{21}

The manner in which the drug is encapsulated into the liposome also seems to affect the positioning of the drug and alters the efficiency of drug delivery to the skin. Studies indicate that drug loading by dehydration and subsequent rehydration of the liposome creates a liposome that delivers its drug more efficiently than liposomes prepared by other methods.\textsuperscript{22,23}

An advantage to topical drug delivery by liposomes is an increased uptake by the skin without a proportionate increase in systemic absorption. Less than 0.1% of the applied dose is absorbed into the systemic circulation despite the rapid penetration of liposomally prepared drugs into the epidermis. This potentially limits systemic toxicity.\textsuperscript{24}

**Hair Follicle and Pilosebaceous Targeting**

Many drugs are absorbed to a greater extent from areas of skin with large or great numbers of hair follicles because the drugs are shunted through the pilosebaceous unit (Fig. 2).\textsuperscript{25–27} Multilamellar liposomes have an even greater ability to penetrate the skin through these follicular orifices.\textsuperscript{28} The lipids that form the bilayer membrane do not show any increased penetration when they are applied as a simple emulsion, but only when they are organized as lamellar assemblies. Liposomal preparations enhance drug delivery to the follicle five to eight times over standard aqueous preparations.\textsuperscript{29} Variations in the size and lipid composition of the liposome can affect whether absorption remains at the upper layers of the skin or in the liposome when it penetrates to the base of the hair follicle.\textsuperscript{30}

The preferential absorption of multilamellar liposomes into the pilosebaceous unit can be exploited to target hair follicles and sebaceous glands. Recently, the delivery of intact DNA to the hair follicle and evidence for active gene transfer into the hair shaft and follicles have been demonstrated in tissue cultures of skin.\textsuperscript{31} Melanin can also be delivered specifically to hair follicles. When melanin is entrapped in a phosphatidylin
choline liposome, the liposome penetrates the pilosebaceous unit and deposits the melanin both at the periphery of the follicles and within the follicle cells themselves. Microscopic examination of the hair shaft of white-haired mice treated with liposomal melanin demonstrated a band-like melanin-distribution pattern, similar to the melanin-distribution pattern that is seen in hair colored by endogenously produced melanin. Thus, in addition to deposition of the melanin within the hair follicle and shaft, the normal pattern of melanin distribution is replicated by liposomal delivery of melanin.

SELECTED CLINICAL APPLICATIONS

Prevention of Skin Cancer

Exposure to ultraviolet (UV) light has clearly been linked to the development of skin cancer in humans. Ultraviolet radiation causes the formation of cyclobutane-pyrimidine dimers in the DNA. These dimers are the initial molecular change that ultimately leads to the carcinogenic transformation of the cell. Most people are able to repair most of the DNA damage with excision-repair endonucleases and, therefore, develop only very few skin cancers.

Patients with xeroderma pigmentosum and animal models have deficiencies in excision-repair endonuclease. Therefore, they are unable to excise the cyclobutane-pyrimidine dimers that accumulate with ultraviolet light exposure. These patients develop many cutaneous, ocular, and neurologic abnormalities of a higher incidence than the rest of the population. One half of the patients with xeroderma pigmentosum will develop a skin cancer, predominantly in sun-exposed areas, by age 10 years. This is 50 years before the remainder of the population will do so.

Endonuclease V is an enzyme produced by the denV gene of bacteriophage T4. The purified product, T4-endonuclease V, produces single-stranded breaks in DNA at cyclobutane dimers by cleaving the N-glycosyl bond on the 5' side of the dimer. The phosphodiester bond between the two pyrimidines is then cleaved and the DNA strand is returned to the state before ultraviolet irradiation. The T4-endonuclease V can be entrapped in both positively and negatively charged liposomes and delivered in its active form to normal persons and to keratinocytes in culture of patients with xeroderma pigmentosum, to human skin explants, and to live mice.

In the recipients of liposomal T4-endonuclease V, there is an increase in DNA synthesis and increased excision and repair of cyclobutane-pyrimidine dimers. Liposomal delivery of the enzyme is far more efficient than cell transfection or permeabilization. The liposome applied topically penetrates the stratum corneum and the epidermis of murine skin. Both the liposome and the enzyme can be found in basal keratinocytes. When it is applied to the skin of mice irradiated with UV rays, there is a dose-dependent decrease in the number of cyclobutane-pyrimidine dimers and a subsequent 45% decrease in the incidence of skin cancer.

Cancer Therapy

Liposomes have had a significant impact on research in cancer treatment. Liposomes can be used to alter the molecular makeup of tumor cells to make them more accessible to the body's own immune system. They can be used to target tumors for systemic chemotherapeutic agents preferentially, increasing their therapeutic index. Thus, efficacy can be enhanced, whereas toxicity is decreased by using liposomal drug preparations, such as liposomal vincristine. Finally, liposomes can be used to protect against common side effects of chemotherapy.

Genes for human transplantation-antigens can be packaged within liposomes and injected directly into malignant melanoma nodules, where the genes are taken up and actively expressed by the local tumor cells. In a human clinical trial, five HLA-B7-negative patients with stage IV melanoma, refractory to conventional treatment, had one to three of their melanoma nodules injected with liposomes containing the gene encoding the HLA-B7 major histocompatibility-complex protein. Immunohistologic staining of injected nodules confirmed that the HLA-B7 protein was actively ex-
pressed by cells of the injected melanoma nodule. Analysis using PCR sensitive to < 2 pg per mL, failed to detect any uptake of the liposomal DNA into the systemic circulation. No anti-DNA antibodies or evidence of systemic toxicity were observed. One of the five patients treated, had a complete regression of the treated nodules as well as regression of several distant, untreated nodules. A possible explanation is that the transplantation antigens expressed on the tumor cells allow the patient's immune system to recognize them as foreign and to attack the tumor successfully. The regression of untreated nodules suggests that the immune response triggered by the treated nodule also enhances the immune response to other antigens of the melanoma tumor.

Early attempts at developing tumor vaccines were largely unsuccessful, because tumor antigens are processed in association with MHC class II molecules. Thus, they generate an antibody response rather than activate cytotoxic T lymphocytes that can attack the tumor more aggressively. Tumor specific antigens prepared in liposomes are engulfed by macrophages and processed with MHC class I that activates cytotoxic T lymphocytes. In studies on murine thymoma, injection of liposomes containing thymoma proteins resulted in the regression or elimination of the tumors with the corresponding surface antigens.

Tumors tend to have an increased vascularity compared with normal surrounding tissue and the capillaries within tumors tend to be "leakier" than normal capillaries. This altered vascularity of tumors allows increased transcytosis and extravasation of small liposomes from the blood stream causing the liposomes to localize in skin tumors as well as in the surrounding normal skin. The liposomes are found both free in normal skin, in the tumor, and in the endosomes and lysosomes of perivascular macrophages. The localization of liposomes within tumors allows a lower administered dose to generate an equal therapeutic effect. Studies in transgenic mice that develop a Kaposi's sarcoma-like dermal lesion have confirmed that sterically stabilized liposomes localize in early and mature Kaposi-like lesions as well as in the surrounding tissue.

Using a microwave device, topical hyperthermia can enhance the release of drugs encapsulated in liposomes in the skin or subcutaneous tissue. When solid phase phospholipids and cholesterol constitute more than half of the liposomal membrane, the resulting liposomes release significantly more drug at 42°C than at 37°C. The local hyperthermia also increases vascular permeability, allowing for increased extravasation and deposition of the drug in the heated tissue. Furthermore, tumors, especially those in skin and muscle, tend to heat more rapidly than the surrounding normal tissues. These characteristics of local hyperthermia and liposomes lead to an increased accumulation of liposomes at the targeted tissue as well as an enhanced release of the drug at that site.

Anagen effluvium is a common and unpleasant side effect of many cancer chemotherapeutic drugs. A monoclonal antibody to doxorubicin, when packaged in a multilamellar liposome and applied to hairy areas, delivers active antibody to the hair follicle and the surrounding epidermis. The antibody acts locally to bind doxorubicin and inhibit its effect on the hair follicle. The presence of the antidoxorubicin antibody, surrounding the hair follicle at the time of peak systemic doxorubicin levels, prevents drug-induced alopecia in animal models.

**Gene Therapy**

Liposomes can be used for topical and systemic gene therapy; they can be designed to transfer active, intact DNA specifically to hair follicles and can deliver genes to correct the deficiency in respiratory epithelial chloride transport in cystic fibrosis. The reticuloendothelial system and hematopoietic cells of the bone marrow can also be targeted for gene therapy.

Topical gene transfer to hair follicles also has been accomplished. Lecithin-containing liposomes can be used to entrap high-molecular-weight DNA. The entrapment within the liposome protects the DNA from naturally occurring DNase present in the skin. This allows the DNA to penetrate to its target and be incorporated without degradation. In histocultured skin, the lecithin liposomal DNA preparations are preferentially shunted down the pilosebaceous unit, where they actively and specifically transfer the genes to the hair follicles.

Topical gene transfer to respiratory epithelial cells has also been accomplished. Patients with cystic fibrosis have a defect in CAMP-regulated Cl- channels on the apical aspect of respiratory and intestinal epithelial cells as well as in the sweat glands. Respiratory complications are the major cause of fatality in these patients. The gene CFTR encodes the proteins that comprise the CAMP-regulated Cl- channels. The CFTR DNA can be incorporated into a liposome and be actively transferred to respiratory epithelial cells. When applied to the nasal epithelium of patients with cystic fibrosis, there is a 20% restoration of function of the CAMP-regulated Cl- channels without any toxicity; however, the effect is confined to the area of spray application in the airway. Further studies are needed to assess whether the entire respiratory epithelium can be treated.

The immune system can be targeted for systemic gene therapy. Liposomes containing DNA can be injected intraperitoneally. Following drainage by the lymphatic capillaries, the liposomes are delivered to T lymphocytes located in lymph nodes and spleen that phagocytose the liposomes and incorporate the active DNA. This delivery system also transfects active DNA into the hematopoietic cells of the bone marrow. There is no systemic toxicity associated with this procedure. This has been accomplished in a murine model. Thus,
liposomal DNA potentially provides an effective and safe mechanism for the delivery of genes to the reticuloendothelial system.52

SELECTED THERAPEUTIC AGENTS

Topical Anesthesia

Local anesthetics are commonly used in dermatologic surgery, but the potent anesthetic effect is preceded by the pain of injection. The eutectic mixture of lidocaine and prilocaine is the only effective transcutaneously absorbed local anesthetic currently available in the United States. It requires application with occlusion 1 hour prior to the procedure with only a mild anesthetic effect. An injection of local lidocaine through the mildly anesthetized area is usually required before the procedure. Thus, it is not very practical in an outpatient dermatologic practice.

Liposomal anesthetics can provide reliable, deep anesthesia without injection by needle. Liposomal tetracaine can be applied 1 hour prior to a procedure without occlusion and will provide sufficient, local anesthesia at the site of removal of a skin lesion or for minor plastic surgery. Although the onset is slow, there is deep anesthesia that lasts for more than 4 hours.53 A second effective topical anesthetic is a lipid-detergent liposomal variation encapsulating lidocaine. This preparation provides reliable, deep, local anesthesia at the site of application within 15 minutes after application; however, the effect is not persistent and diminishes after about 30 minutes. This may be sufficiently long to perform punch and shave biopsies or for venipuncture. For longer procedures, repeated drug applications would be required.54

Topical Corticosteroids

Hydrocortisone and other corticosteroids are the therapeutic workhorses of dermatology. High-potency corticosteroids are very effective in modulating inflammatory responses; however, their long-term application is limited by their local and systemic side effects such as atrophy of the skin,55,56 formation of striae,57 purpura,58,59 glaucoma, cataracts,60 suppression of the hypothalamic-pituitary-adrenal axis,51,62 and even avascular necrosis of the femur.60 Although hydrocortisone is safer than fluorinated corticosteroids for long-term therapy, its low potency is often inadequate to treat moderate or severe inflammatory reactions. The penetration kinetics of hydrocortisone are greatly enhanced by encapsulating it in liposomes.63

Liposomal hydrocortisone preparations provide drug concentrations that are 8 to 14 times higher in the epidermis and dermis than supplied by conventional topical hydrocortisone preparations.64 This effect is even more dramatic than those of hydrocortisone creams employing penetration enhancers such as urea.63,65 The increased epidermal and dermal concentrations delivered by liposomal hydrocortisone preparations have the potential advantage of lower toxicity with enhanced efficacy.64

Topical Interferon

The incidence of herpes simplex infection in the United States is 10% per year.66 Potentially, interferon is able to prevent herpes simplex from occurring. In a randomized, double-blind, placebo-controlled study of 76 patients with severe recurrent herpes simplex infections, those treated with interferon-α2b, 3 × 106 IU subcutaneously three times per week, had 33% fewer occurrences, milder symptoms, a 25% faster resolution of an episode, and an 81% longer interval between episodes. A lower dose of 1 × 106 IU subcutaneously, three times per week, was not significantly effective. Unfortunately, the higher, effective dose caused severe adverse effects in 35% of patients and moderately adverse effects in 48% of patients. The adverse effects included malaise, fever, myalgia, arthralgia, and fatigue, despite administration of acetaminophen, 650 mg every 4 hours. The patients also developed a mild reversible leukopenia with a 33% decrease in granulocyte counts and a 20% decrease in lymphocyte counts.67 A second study of 69 women with recurrent herpes simplex infection confirmed the above results. The women treated with 5 × 106 IU per kg subcutaneously for 12 doses over 14 days had a decrease in the size of their lesions, but also had a moderate but reversible neutropenia.68 The relatively severe adverse effects of subcutaneously injected interferon make it unsuitable for the general treatment of herpes simplex infections.

The aim of topical therapy is to achieve adequate drug levels in the stratum basale of the epidermis where the active herpetic infection takes place. Topical application of standard interferon solution or emulsion does not offer any benefits in the treatment of herpes simplex due to the low extent of absorption. Interferon encapsulated in liposomes, consisting of lipids in concentrations similar to those found in the stratum corneum, is delivered in substantial quantities to the epidermis. This results in a reduction in the number of herpetic vesicles that develop in hamsters inoculated with the herpes simplex virus.22 This may provide a safe and effective mechanism for topical treatment of herpes simplex.

Liposomal Amphotericin B

Amphotericin B preferentially binds to ergosterol found in fungal membranes and disrupts the integrity of cells. This potent fungal cytotoxic effect makes it the drug of choice for most serious fungal infections. Its major limitations are its toxicity, including chills, fever, and nephrotoxicity that frequently necessitate early discontinuation of the drug; however, amphotericin B can be
incorporated into small unilamellar liposomes. In this form, it is better tolerated by patients with very few of them developing fever and chills. Liposomal amphotericin B provides sustained serum concentrations as much as five times higher with a longer half-life than conventional amphotericin B. Despite daily doses ranging from 4 to 6 mg per kg and higher serum concentrations, the toxicity to brain, kidney, and heart is significantly lower. The liposomal form remains highly effective in treating systemic fungal infections with response rates of nearly 60% in treating invasive aspergillosis and systemic candidosis.

Artificial Blood

A novel application of liposomes is their encapsulating hemoglobin to form neoehemocytes. These are made large enough that they do not extravasate, but small enough that they can pass through normal and slightly constricted capillaries. In animal experiments, nearly 50% of animals survived a 95% exchange transfusion replacing their blood with neoehemocytes. The neoehemocytes do not cause any acute toxicity and have the obvious advantages of prolonged shelf-life and a decreased risk of viral transmission. These appear to be an ideal substitute for red blood cells in the treatment of trauma.

CONCLUSIONS

Liposomal technology is advancing rapidly and promises to deliver major advances in many medical fields. In dermatology, we can anticipate drugs to help prevent and treat skin cancer more effectively. We may see drugs specifically targeted to hair follicles and sebaceous glands. Gene therapy may be used to alter hair growth. Effective topical anesthetics have been developed, as well as corticosteroids with enhanced efficacy and reduced toxicity. We may have a safe, effective topical treatment for herpes simplex. Although liposomal amphotericin B is the only commercially available product in the United States, the future clearly holds many applications for liposomes in dermatology.

DRUG NAMES

The eutectic mixture of lidocaine and prilocaine is EMLA cream.
Liposomal Amphotericin B is Ambisone.

REFERENCES


