Volume XIV No. 2

# THE JOURNAL OF THE SOCIETY OF COSMETIC CHEMISTS

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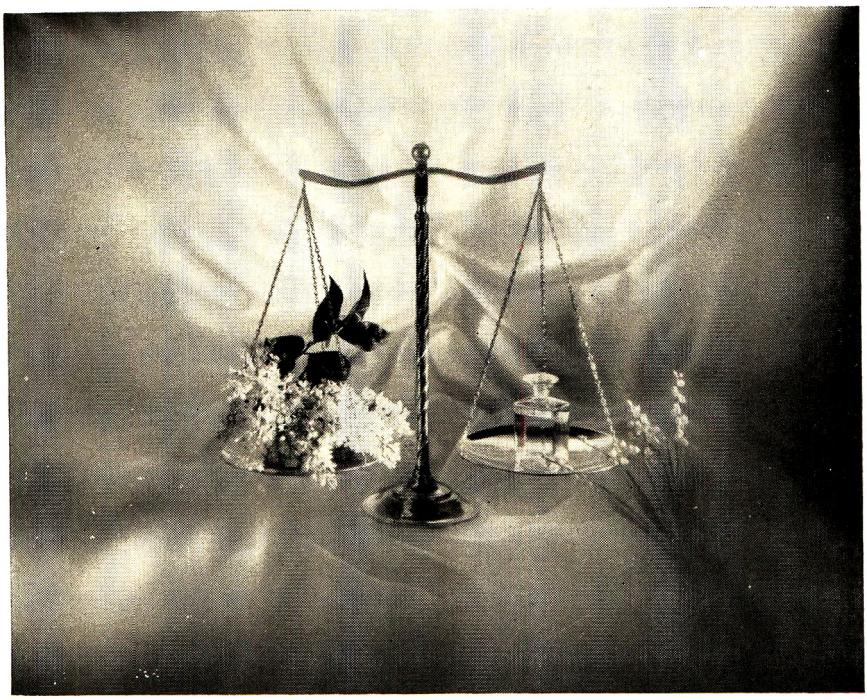

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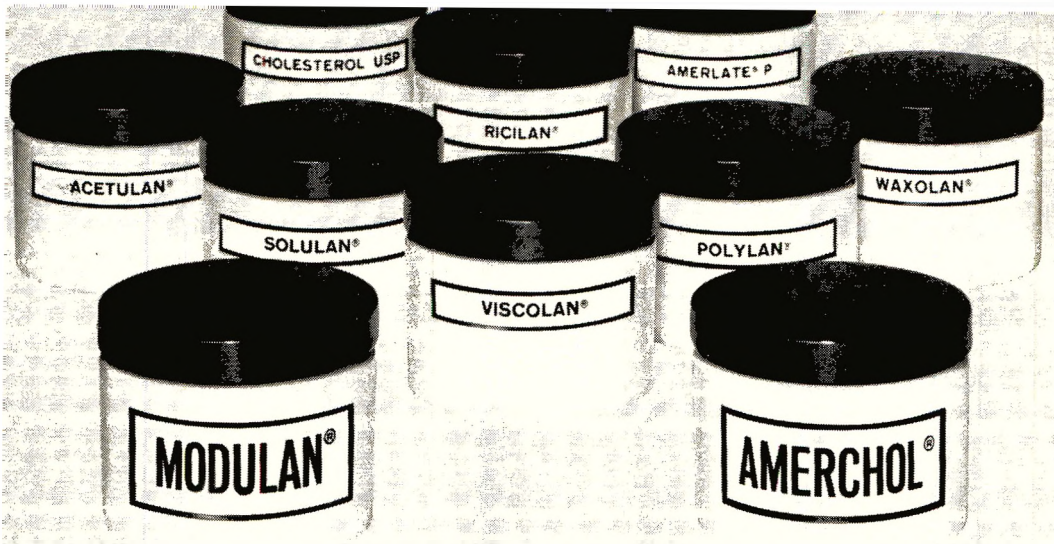


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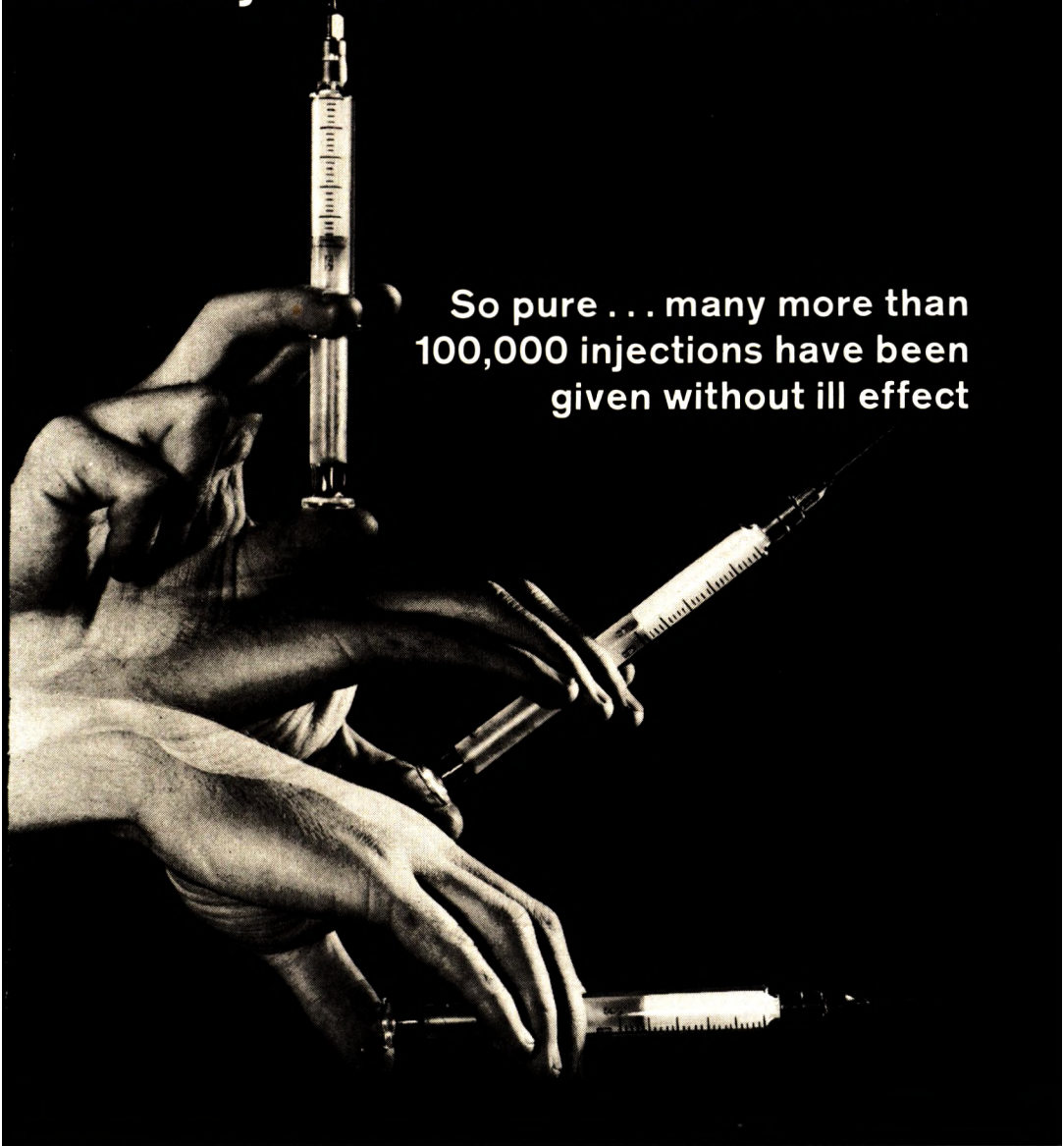


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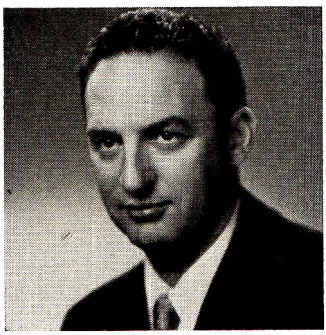

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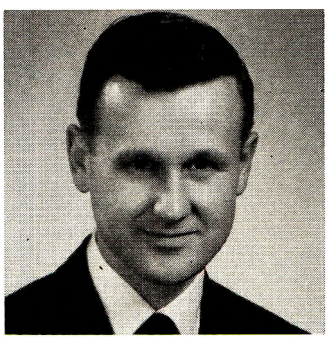

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# PRESIDENT'S REPORT

WARREN B. DENNIS

**December 4, 1962**

THIS YEAR'S Report of your President to the membership has proven to be a very complex affair, since 1962 seems to have been a year of decision in many areas of our interest.

It was always my belief that the work of our organization was in the hands of the Committee Chairmen and of course this is still true to a great degree. On the loyal and dedicated efforts of your Standing Committee Chairmen the annual march of our affairs has become an accepted fixture of events.

Walter Wynne as Arrangement Chairman brings his many years of experience to the smooth running of the Annual and Semi-Annual Meetings and Seminar as well as the Medal Award Presentation and Dinner Dance.

By this time you are all acquainted with the decision of Mr. Raymond Reed's Medal Award Committee that the 1962 Medalist is Dr. Paul G. I. Lauffer. This Committee was not content to choose only a Medalist. It went even further and has made proposals whereby the public relations aspect of the Medal Award function can be extended to serve the public image of our SOCIETY even better.

The Library Committee has been Chaired by Dr. Frank J. Steele: a dedicated bibliophile as well as an educator in our field. Frank Steele has strengthened the library through purchases of new as well as historical reference items and has contributed valuable volumes from his own collection.

The Special Award which proved so brilliant an occasion last May brought great credit to Dr. Herman Jass, the Special Award Committee Chairman, and to Robert L. Goldemberg, Chairman of the Literature Review Committee. Both have been persuaded to continue another year in these assignments. We are grateful for their continued willingness to serve the Society in this important way.

Harry Isacoff, after five years as Library Chairman and Historian, agreed to undertake the important post of Membership Chairman. Together with his Committee, the Chapter Membership Chairmen, he has brought 81 new members into our society which now totals 916.

Edward Silkin undertook the difficult Public Relations Committee

Chairmanship. His efforts have resulted in distinctly better advance reporting of our meetings, and he has succeeded in placing our notices in several journals which previously failed to print any of our releases.

Dr. Barry Dash mounted a Seminar Program, under very difficult conditions, which many have told me was one of the finest Seminars we have ever held.

The Seminar is assuming a unique significance in the area of the SOCIETY's service to the membership. The Chapters, led by Chicago, have persuaded the Board of Directors to change the locale of this meeting periodically to a major city other than New York. This has the happy effect of removing the geographical barrier which exists for many of our members when meetings are confined to New York. The 1963 Seminar will be in Boston. In 1965 present plans call for Los Angeles.

Dr. Hyman Henkin has twice been responsible for programming of exceptional quality. The May meeting was the subject of much laudatory comment, as I am sure today's meeting will be.

Perhaps the event with the greatest potential impact during 1962 was Maison G. deNavarre's request to be relieved of his responsibility as Editor and Publications Chairman for THE JOURNAL OF THE SOCIETY OF COSMETIC CHEMISTS. This was a grievous blow indeed. What can one add to the evidences of the esteem in which we have held Ed deNavarre over the years. All of you know his zeal for the science of cosmetics, author, editor, columnist, founder of our SOCIETY, an honorary member, its Medalist in 1951, an instigator and first President of the I.F.S.C.C., the encourager of individual Societies of Cosmetic Chemists the world over. We were indeed faced with a crisis as his retirement as Editor became imminent.

Your President-Elect, Lester Conrad, acted as Chairman of a Special Committee on Editorial Policy to review present policy, suggest our future course of action and search for a successor to Ed deNavarre as Editor and Publication Committee Chairman.

The English Society had entered into a contractual arrangement with Pergamon Press to handle the printing and business affairs of their two issues of the Journal. We reviewed their arrangement and thoroughly explored offers by Pergamon Press and others to do the same for us. It was finally recommended that we continue our present control of our six issues of the JOURNAL, reduce the load on the Editor by the appointment of a subcommittee for advertising matters and arrange for Mrs. McGillivray to continue her present role in our JOURNAL.

These recommendations were accepted along with the nomination of Dr. Martin Rieger to succeed Ed deNavarre as Editor and Publication Committee Chairman.

The Board is very pleased that Dr. Rieger has accepted this assignment.

His years of service to the SOCIETY at the Chapter level as well as for the past three years as a Director and his often expressed concern for the caliber of all phases of our JOURNAL enable him to bring to his new task all the attributes necessary for the task of increasing the prestige of the JOURNAL which I referred to above.

The area of International Affairs, this year under the Chairmanship of Robert A. Kramer, began to loom large in our thinking as the World's Fair and the 1964 International Congress of the International Federation of Societies of Cosmetic Chemists approached.

On June 29, 42 members and friends of the SOCIETY under the tour directorship of Sam Cohen left New York on Irish Airlines for our biennial European tour.

The Congress in London was at such a fine academic level, the social and tour arrangements so well worked out that a very high standard has been set to challenge our own efforts for 1964.

The members of the tour participated in activities sponsored by I.F.S.C.C. affiliates in Hamburg, Paris, Milan and Rome.

Eight of us were your SOCIETY's delegates at the I.F.S.C.C. Council meeting which took up the better part of two days. New officers of the I.F.S.C.C. were elected, including Sabbat J. Strianse. The Council also elected the Dutch Society to membership, and the possibility of a Mexican and a Czechoslovakian Society was mentioned.

The main item of interest to your delegates was, of course, the acceptance of New York as the 1964 site for the third International Congress of the International Federation of Cosmetic Chemists. This was finally accepted by the Council, thanks to the ground work done by Robert A. Kramer.

In 1964 the May Scientific Meeting and the Seminar will be absorbed into the Congress. It will be held in New York City during the week of June 21, 1964. Present plans call for the use of Columbia University facilities to house overseas visitors and to provide facilities for scientific and social events.

Apropos of International Congresses, the present schedule calls for Paris in 1966. You may be interested to know that the I.F.S.C.C. has tentatively accepted the invitation of the Japanese Society to hold the 1968 Congress in Tokyo.

As I prepare to turn over this office to my successor, I wish to thank all these Committee Chairmen and their Committees for the loyal and dedicated service they have given to our Society during 1962. I also want to thank the membership at large for their trust in honoring me with this supreme opportunity to serve our common interests in our chosen profession as cosmetic chemists. I shall long treasure the memory of the moments which you set in train for me when I became President-Elect.

And now there remains only the results of the activities of the Nominat-

ing Committee under Sabbat J. Strianse. Yesterday the Board of Directors accepted the formal report of the tellers as follows:

*President-Elect*—Robert A. Kramer
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*Directors* for two years

John M. Longfellow, 1964
Harry Isacoff, 1964

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John E. Garizio, 1963
William H. Mueller, 1963

Mr. Conrad, you have served as President-Elect since your election and installation at the Annual Meeting in 1961. By-Laws and custom provide that I shall now present you with this gavel, the tangible symbol of the office you are about to attain, and shall now install you as President. You will assume this high office at the close of the Medal Award Dinner held in conjunction with this meeting.

As President you shall have all the duties of a Chief Executive Officer as well as those of presiding at all meetings. You are further charged to use the influence of the SOCIETY to enhance the professional status of cosmetic chemists.

I congratulate you on your installation as President. I hope you shall be able to use this gavel with good fortune and good health.

# THE EFFECT OF U. V. IRRADIATION ON ENZYME SYSTEMS IN THE EPIDERMIS

By W. Coffey, P. Finkelstein, and K. Laden*

*Presented September 19, 1962, Seminar, New York City*

The physiological effect of ultraviolet light irradiation upon the epidermis and dermis has been extensively studied from many points of view. Thus the effect of U. V. light on erythema production, melanin formation, long-term changes in dermal collagen, induction of cancer and histological changes in the epidermis have been, and continue to be, important areas of research (1–6).

It is interesting to note, however, that attempts to identify the initial biochemical alterations induced by U. V. light have been in general unsuccessful. Indeed most of the changes brought about by exposure to U. V. light do not manifest themselves until at least several hours after exposure. Thus, for example, erythema begins to develop two to three hours after irradiation (1) and histopathological changes, twenty-four hours later (7).

Recently, Daniels *et al.* (8) using histochemical procedures have examined the histochemical, enzymatic and cellular changes occurring in the epidermis after U. V. exposure. While histological and some histochemical changes were observed as soon as four hours after irradiation, no histologically demonstrable enzymatic changes were noted until extensive cellular damage was observed.

It was the purpose of this investigation to see if any signs of damage resulting from U. V. irradiation could be detected at fairly short periods of time after exposure. Since one might expect enzymatic changes to precede any marked histological changes, our investigation has centered around the effect of U. V. irradiation on some of the enzyme systems present in the skin. The enzymes we chose to investigate were those involved in glucose metabolism and two transaminase enzyme systems.

## Experimental

*Preparation of Tissue Homogenates*—Male rats (200 ± 25 gm.) of the Sprague-Dawley strain were used in all experiments. Four days prior to

* The Toni Company, Div. of Gillette, Chicago 54, Ill.

the day of the experiment, the animals were anesthetized with ether and the hair plucked from the dorsal area. The plucking was done four days before the experiment to allow the skin flare reaction caused by the hair removal to subside. Both control and treated animals were sacrificed by cervical fracture and their pelts quickly removed. After scraping away the subcutaneous fat, the pelt was stretched over a chilled drum. The epidermis was then scraped from the dermis with a scalpel, and after weighing on a Roller-Smith precision balance, placed into a Potter-Elvehjem homogenizing tube. A small pair of scissors was next used to mince the tissue, and then a calculated amount of KCl-$KHCO_3$ homogenizing fluid (sufficient fluid to prepare a 5 per cent homogenate) was added and the contents homogenized for three to five minutes in the cold. For spectrophotometric studies, the homogenate was centrifuged at 5000 r.p.m. in a Precision Vari-Hi Speed Centricone centrifuge for fifteen minutes and the resultant supernatant liquid was used.

*Injury Inducing Procedure*—Four days after plucking, the animals were exposed to U. V. light (Westinghouse R. S. 275W Sun Lamp) for twenty-four minutes at a distance of $10^1/_2$ in. This exposure was sufficient to produce a reaction comparable to a first-degree burn. The animals were then sacrificed at varying time periods after exposure. Control animals were plucked four days prior to the experiment, but were not exposed to U. V. light.

*Glucose Oxidation*—Glucose oxidation was determined manometrically in a Warburg apparatus by measuring oxygen uptake. The complete system contained homogenate, buffer (phosphate pH 7.4), $Mg^{++}$, ATP, TPN and glucose with KOH in the center well.

*TPN Reduction*—Glucose oxidation *via* the hexose monophosphate shunt was determined spectrophotometrically by following the rate of TPN·H formation at 340 mμ. The system contained epidermal extract, buffer (phosphate pH 8.0), $Mg^{++}$, ATP, TPN and glucose.

*Transaminase Activity*—Transaminase activity was followed spectrophotometrically *via* the oxidation of DPN·H. Both glutamic-oxalacetic transaminase and glutamic-pyruvic transaminase were studied. The complete system contained: epidermal extract, buffer (phosphate pH 7.4), aspartic acid or alanine, DPN·H, lactic dehydrogenase and alpha keto glutaric acid.

## Results

Initial experiments in this investigation were aimed at studying the effect of U. V. light irradiation on glucose metabolism by rat skin. Rats were irradiated in the manner described and sacrificed at one-half hour, one hour, one and one-half hours and two hours after exposure. Epidermal homogenates were prepared from the exposed sites, and glucose metabolism

was evaluated *via* oxygen consumption and compared to homogenates prepared from nonexposed animals. The results indicated that at the one-half hour interval, the rate of $O_2$ consumption was often comparable or slightly elevated from that of unexposed skin. However, at all other time intervals, the $O_2$ consumption of the homogenates prepared from the exposed animals was markedly depressed. In some cases this depression in oxygen consumption was as great as 75 per cent.

Since previous work in our laboratories and others (9–11) indicated the presence of the hexose monophosphate shunt in glucose metabolism in skin, it was decided to investigate further the effect of U. V. irradiation on glucose oxidation by following the rate of TPN reduction in epidermal extracts prepared from exposed and nonexposed animals.

Animals were irradiated and sacrificed at intervals of one-half hour and one hour after exposure. Epidermal extracts were prepared from their skins as well as from the skin of a nonexposed animal and the early stages of glucose oxidation followed spectrophotometrically *via* TPN reduction. The results are presented in Table 1.

TABLE 1—THE EFFECT OF U. V. IRRADIATION ON GLUCOSE METABOLISM*

| Time, min. | Control Nonirradiated | Irradiated Sacrificed | |
|---|---|---|---|
| | | ½ hr. later | 1 hr. later |
| 1 | 0.258 | 0.295 | 0.090 |
| 2 | 0.300 | 0.350 | 0.150 |

* Glucose metabolism measured as rate of TPN reduction (increase in O.D. at 340 mμ).

TABLE 2—GOT AND GPT TRANSAMINASE ACTIVITY OF IRRADIATED *vs.* NONIRRADIATED RAT SKIN

| | GOT Activity | GPT Activity |
|---|---|---|
| Nonirradiated | $15 \times 10^3$ units* | $3 \times 10^3$ units* |
| Irradiated (sacrificed one hr. after irradiation) | 0 | 0 |

* One unit equals a decrease in optical density of 0.001 units per minute per gram wet weight of tissue.

These results suggest an increase in glucose metabolism at the one-half hour interval and a depression at the one hour interval.

Since a marked depression in glucose oxidation was consistently noted at time intervals from one hour to four hours after U. V. irradiation, further attempts were made to find the cause for this inhibition.

Rats were irradiated in the usual manner and sacrificed one hour after exposure. Epidermal extracts were prepared and glucose oxidation studied *via* TPN reduction. As usual, a marked reduction in glucose oxidation by irradiated skin was noted (see Fig. 1). When, however, glucose-6-phosphate was used as a substrate in place of glucose, no dif-

ference was observed between the rate of glucose metabolism in exposed *vs.* nonexposed animals (Fig. 2).

These results suggested that the cause for the decreased rate of glucose metabolism in the irradiated animals' skin was related to some inactivation or inhibition of the glucose phosphorylating mechanism.

The supposition was further confirmed by showing that when exogenous hexokinase (the enzyme which converts glucose to glucose-6-phosphate) was added to the epidermal homogenate system, no difference in the rate of glucose metabolism could be observed between irradiated and nonirradiated skin (Fig. 3).

The effect of U. V. irradiation on one other enzyme system was investigated. In earlier work in this laboratory it was shown that the rat epidermis contains at least two transaminase systems. These are glutamic-oxalacetic transaminase (GOT) and glutamic-pyruvic transaminase (GPT). The activities of these two transaminase systems were compared in epidermal extracts of normal rats *vs.* epidermal extracts of U. V. irradiated animals. The results (Table 2) indicated a complete inhibition of transaminase activity in the extract from the irradiated animals.

## Discussion

The time delay between exposure to U. V. irradiation, and the onset of clinical or histological evidence of damage has been explained by a number

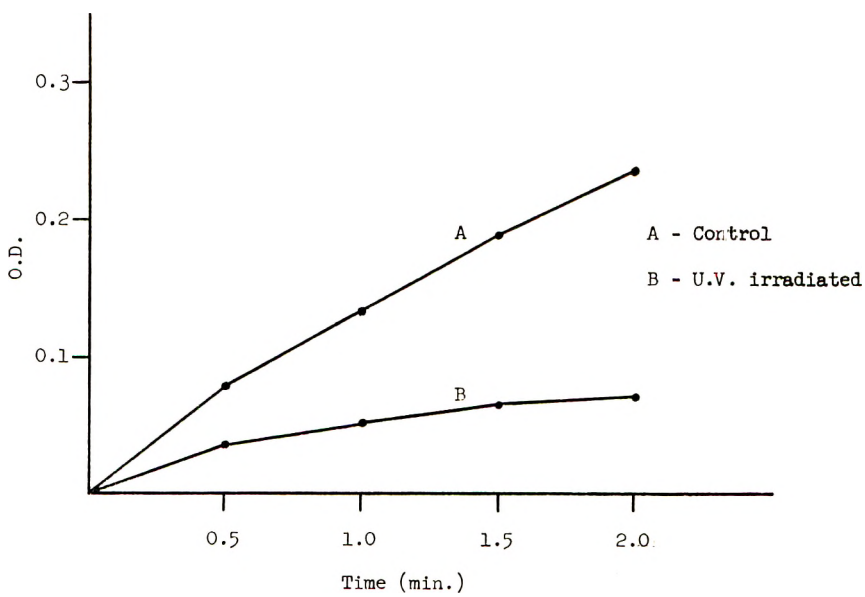


Figure 1.—Effect of U. V. irradiation on glucose metabolism by rat epidermis. Rate of TPN reduction (increase in O.D. at 340 mμ) *vs.* Time (min.).

of different theories (12). Perhaps the most widely accepted theory speculates that sunlight sets up some type of free radical reaction which in turn results in the release of some noxious agent thus setting the stage for the classical symptoms of sunburn. While the nature of these initial reactions is unknown, it appears that it takes several hours for the body to respond *via* gross clinical or histologic changes.

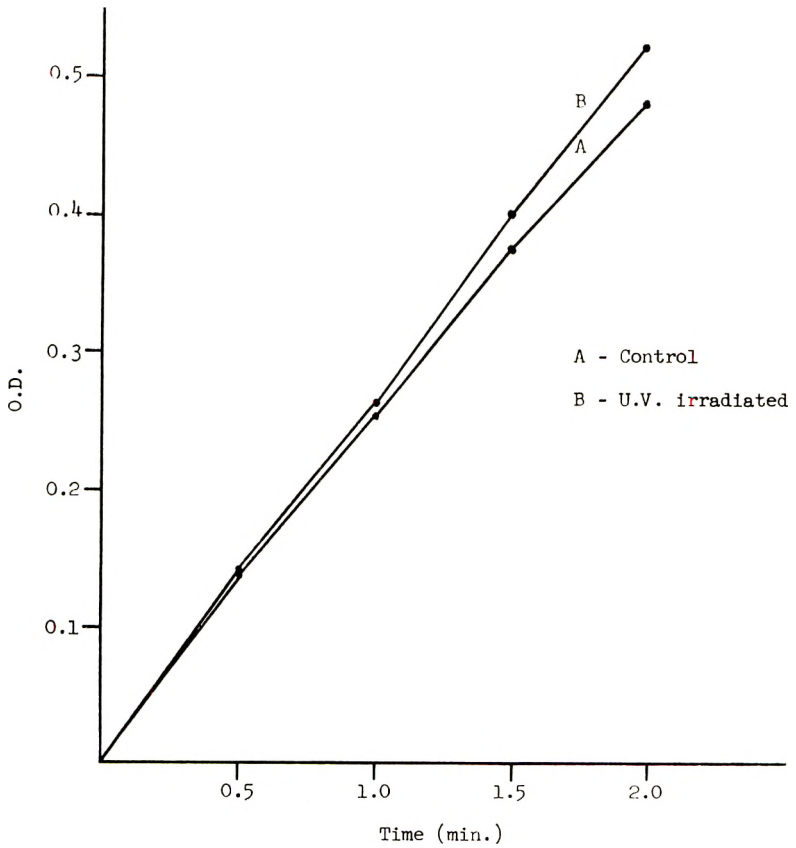


Figure 2.—Effect of U. V. irradiation on metabolism of glucose-6-phosphate by rat epidermis. Rate of TPN reduction (increase in O.D. at 340 mμ) *vs.* Time (min.).

It is only reasonable to expect that during this time lapse, enzymatic changes are occurring which initiate some of the cellular responses seen. While the work reported in this paper does not clarify the mechanism of the initial damaging effects of U. V. irradiation, it does point out some of the early enzymatic changes occurring.

The damaging effects of U. V. irradiation on many enzyme systems is well known (13). Usually these effects have been studied with *in vitro* systems

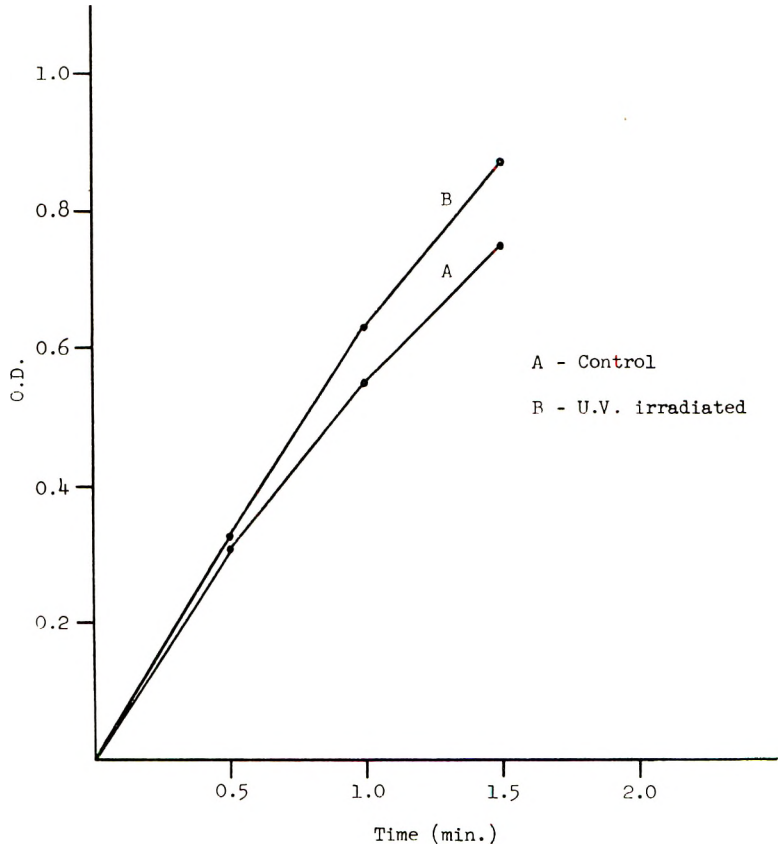


Figure 3.—Effect of U. V. irradiation on epidermal metabolism of glucose with added exogenous hexokinase. Rate of TPN reduction (increase in O.D. at 340 mμ) *vs.* Time (min.).

and are often related to denaturation of the enzyme protein itself or inactivation of some co-factor needed for enzyme activity. Thus, for example, it has been reported that U. V. light can inactivate transaminase systems *in vitro* (14). Exposure of the under side of mouse skin to U. V. irradiation has been reported to cause a decrease in succinic dehydrogenase activity as estimated histologically (15). Exposure of skin or skin extracts to U. V. causes an increase in phosphorylase activity (16, 17).

In our studies, the most marked effects observed as a result of U. V. irradiation of rat skin *in vivo* were a decrease in the rate of glucose oxidation and a complete inhibition of two transaminase systems present in the epidermis. Both of these enzymatic effects manifested themselves within one hour after irradiation and continued up to four hours after irradiation. At time intervals of one-half hour after irradiation, this inhibition of glucose oxidation was not noted, and indeed in several experiments a slight

increase in rate was noted. Measurements of glucose oxidation and transaminase activity were not measured at periods of time longer than four hours after irradiation.

While the mechanism for the inactivation of transaminase activity is unknown, it appears that the mechanism by which the decrease in glucose oxidation occurs is related to some malfunction in the glucose phosphorylating system. Thus it has been shown that in the presence of glucose-6-phosphate as substrate or with the addition of exogenous hexokinase, no difference can be observed between the rate of glucose oxidation of extracts prepared from normal animal skin and those from irradiated animal skin.

It is interesting to note that one of the early histological changes seen in irradiated skin is a build-up in glycogen (8). This glycogen build-up has also been observed after X-irradiation, and it is speculated to result from an enzymatic disbalance between glucose and glycogen metabolism (18). Perhaps the enzymatic changes in glucose metabolism reported herein are related to this imbalance.

## Summary

The effect of U. V. light on enzyme systems present in the epidermis of the rat has been investigated. Exposure of rats to U. V. light has resulted in a decreased rate of glucose metabolism in homogenates prepared from the epidermis of the irradiated animals. This marked decrease in glucose oxidation can be observed within one hour after irradiation. Inactivation of the glucose phosphorylating mechanism is believed to be the cause for this decreased glucose metabolism. Complete inhibition of two transaminase enzyme systems was also noted in the epidermis of rats, one hour after U. V. irradiation.

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# BIOCHEMISTRY OF INFLAMMATION

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Inflammation ranks next to pain as the greatest symptom of body difficulty or pathology. Menkin (1) defines inflammation as the "complex vascular, lymphatic and local tissue reaction elicited in higher animals by the presence of viable or of nonviable irritants." More simply, it is the local response of small blood vessels to injury. Inflammation is characterized to the naked eye by swelling, increased heat, redness, pain and disturbance of function. The swelling is due to the edema and congestion in the area. The inflamed area feels hot in comparison with the surrounding areas because the dilated vessels bring a large amount of warm blood to the area. The redness results from the dilatation and congestion of the arterioles and capillaries. Pain is due to the swelling and tension on tissues with pressure on sensory nerves. The disturbance of body function is linked to the pain and destruction of the affected cells and tissue.

Inflammation is not a static single event but a sequence of constantly changing interdependent reactions, each triggered by a previous alteration, a dynamic process by means of which cells and exudate infiltrate, accumulate and finally destroy the integrity of connective tissue. Intimately and inseparably related to the inflammation is the repair process, whereby the tissues are protected from further injury. The agents causing the injury and hence leading to inflammation, may be of bacteriologic, physical, chemical or traumatic nature. Following a local acute injury there is disturbance in the flow through small blood vessels. A momentary constriction of the capillaries is rapidly replaced by dilatation with an increase in blood flow. This dilation is also transitory and the blood flow slows down to almost stagnation. These local changes result chiefly in an increase in the permeability of the capillaries to the plasma proteins. Fluid plasma and white blood cells escape through the capillary walls into the surrounding tissues. This accumulation of fluid and cells is called an "exudate." Exudation is the primary and pivotal response on which all subsequent inflammatory responses depend (2). The fluid, or serous part of the exudate is, as noted, largely plasma and, when abundant, may be referred to as inflammatory edema.

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The fluid plasma in the accumulated exudate coagulates in the area of injury with the precipitation of an abundant network of fibrin. This "walls off" the inflamed area, localizing the injurious agent and shielding adjacent tissues. The early fixation of, e.g., a bacterial irritant, allows time in which leukocytes can assemble to attempt to destroy the invader by phagocytosis. The first white cells to migrate through the vessel walls are the polymorphonuclear leukocytes, aided in some cases by a chemical stimulus, a process referred to as chemotaxis. Bacteria and products of injured tissue act as chemotactic agents, intensifying the emigration of the leukocytes from the blood vessels and directing them toward the injurious agent, leading them to actual contact with the foreign particle which makes phagocytosis possible.

Acidosis develops in the area of inflammation (3), injuring the cells. These local changes in the hydrogen ion concentration of the exudate appear to govern the cellular sequence in inflammation, consisting of polymorphonuclear leukocytes followed by mononuclear phagocytes (4–6). When the pH falls below 6, all types of white cells are injured, and pus results. Pus formation in acute inflammation is virtually a function of the hydrogen ion concentration (7). The mechanism of acidosis appears primarily referrable to a developing glycolysis (1), with the cellular sequence at the site of inflammation conditioned by the local pH, which in turn is determined by disturbance in the intermediary carbohydrate metabolism (7).

## Connective Tissue

Inflammation can be considered to begin as a change in small blood vessels, or rather, as a change in the state of connective tissue components which determine the physiological properties of the small blood vessels. Whatever the organ affected, it is alteration in the connective tissue of the blood vessels which is fundamental to the development of inflammation. Connective and skeletal tissues are concerned throughout the body with the formation and maintenance of structure. They have as a common origin the embryonic mesenchymal cell, which in the course of differentiation forms the connective tissue proper, cartilage and bone (8). The texture of these tissues depends upon the orientation of the cells, their physical and chemical properties, the spatial organization of the various constituents with respect to each other and the relative amount of each substance present. The connective tissue cells are required to produce a wide variety of extracellular materials which determine to a large extent the processes of growth, regeneration and repair.

The chief connective tissue cells are the fibroblasts and mast cells. The mast cells synthesize and release several substances that are important in the metabolism of connective tissue (9). Fluctuations in the rate of forma-

tion, infiltration and lysis of these cells provide cellular mechanisms for several systems of balances and counterbalances that control activities of interstitial fluids and connective tissues (10). Mast cells synthesize, store and release histamine which increases capillary permeability, which in turn increases the plasma proteins in the interstitial fluid (11). Increased interstitial proteins stimulate formation of the stem cells of mast cells, and further synthesis of histamine. Mast cells are also able to synthesize and release mucopolysaccharides which appear to be necessary for the deposition of different collagen fibrils (12, 13) and play a role in the maintenance of normal cell permeability. Mast cells are the only cells in the connective tissues that contain acid mucopolysaccharides and they are able to release these substances to the ground substance. The concentration of tissue water is a stimulation to mucopolysaccharide release. Mast cells may be able to release histamine independently of simultaneous mucopolysaccharide release. Histamine may induce an increase in capillary permeability and produce edema; the edema provokes release of mucopolysaccharides that bind the water, changing it into a hydrated gel. The presence of acid mucopolysaccharides stimulates the deposition of collagen fibrils and thus connective tissue growth. It appears that fibroblasts as well as mast cells are actively and indispensably involved (14). Mast cells are present in increased numbers in some chronic skin inflammations, such as urticaria pigmentosa (15).

The fibroblasts are the precursor cells, or origin, of collagen. The collagen molecule is synthesized on the surface of the fibroblast (16) and released to the extracellular compartment, where the molecules become polymerized and oriented to collagenous fibers (17). The fibroblasts probably also secrete the ground substance (18).

The ground substance is a gelatinous material permeating loose connective tissue, acting as a substrate through which salts, water and a variety of proteins, as well as neutral sugars and mucopolysaccharides are transported to various cells of the body. The capacity to accumulate and bind water is one of the most important functions of the ground substance. Hyaluronic acid, one of the main mucopolysaccharides, lends to the ground substance its viscosity, gelatinous character and water-binding capacity (17).

The mucopolysaccharides of connective tissue ground substance are similar, but nevertheless, chemically quite distinct and probably are synthesized by different enzymes. It seems likely that these polysaccharides have different functions because they are not evenly distributed in connective tissue (19) and their proportions change with age, although they are apparently all linear unbranched polymers containing hexosamine and another sugar, usually uronic acid, arranged alternately. Apart from hyaluronic acid and chondroitin of cornea the connective tissue polysac-

charides are all sulfate esters and are therefore highly charged polyanions, which show considerable interaction with themselves and with proteins and which also bind salts and water to a marked degree. In the native state the polysaccharides of connective tissue are probably all combined with noncollagenous protein (20), forming very large complex molecules with molecular weights of several millions (21).

Efforts to delineate the mechanism of action of acid mucopolysaccharides (AMP) have been severely hampered because procedures for extraction are not quantitative, methods of separating different AMP from one another are insufficiently refined, and the knowledge of the mechanisms of their biosynthesis is still sketchy (22).

## Chemical Mediators

The similarity of the inflammatory cycle in many different species in response to many diverse types of injury has led to the now generally accepted view that the vascular events of inflammation are due at least in part to the release of local hormones or mediators (23, 24). At some time or other, almost every active material extracted from blood or tissue has been incriminated as a causative factor in inflammation. These include potassium ions, acetylcholine, serotonin, catecholamines, adenyl derivatives (adenosine, adenylic acid, ATP), histamine, proteins, varied peptides, including particularly bradykinin, etc. (25). These substances display the most varied physiological actions. Only histamine, the kinin peptides, globulin proteins and catecholamines satisfy, in large part, the criteria for true mediators.

The beginning of modern mediation theory can be considered to begin with Lewis' (26) studies on wheal formation or hives. Possibly the outstanding dermatological example of the leakage of blood plasma from dilated small blood vessels into extracellular spaces is represented by wheal formation. Whealing represents circumscribed, superficial edema of the skin as it develops in response to various chemical, mechanical, thermal and actinic stimuli. There are three distinct steps in the development of wheals: local vasodilatation of the capillaries, causing local reddening; local increased capillary permeability causing local wheal formation; and a vasodilator axon-reflex complex with arteriolar dilatation, causing the red flare. This is known as Lewis' "triple response" of the skin to injury (26). It was Lewis who argued that these reactions, which appear quite uniformly in response to diverse physical and chemical inflammatory stimuli, are so uniform because all the stimuli eliciting the response do not act directly on vascular and nervous elements, but by liberating substances from the injured cells (24). This has led to the now generally accepted view that the release of local hormones or mediators by the injured cells reasonably explains some of the biological manifestations of inflammation. This

hypothesis has gained support from the discovery of naturally occurring substances with effects on small blood vessels similar to those seen in inflammation. In recent years the theory has been strengthened further by the demonstration of active forms of such compounds at the site of injury at the time when they should be exerting their effect (23).

*A. Histamine.* Lewis drew attention to the similarity of the action of histamine and the vascular events of early inflammation and postulated the release, by injury, of histamine or a histamine-like substance. Histamine is a diamine derived from the amino acid histidine. It is very widely distributed in the tissues of all mammals. It is formed by the enzyme histidine decarboxylase and destroyed by the diamine oxidase, histaminase (27). Mast cells synthesize, store and release histamine. Endogenous and exogenous histamine are potent inductors of hyperemia and of increased capillary and tissue permeability (10, 28, 29, 30, 31). Pathological tissues rich in mast cells contain very high concentrations of histamine. Analysis of tissue homogenates indicates that histamine is loosely held in the mast cells within definite granules in the mitochondrial fraction of the cell. It is evidently held in a readily diffusible form. Histamine is readily released from suspensions of these mitochondrial particles by freezing, hypotonic media, surface-active agents and a large variety of organic bases (24). The release of histamine from mast cells speedily induces dilatation of capillaries and increased permeability and reduces the viscosity of hyaluronic acid in interstitial fluid. Usually these changes result in increased passage of plasma proteins with the formation of protein rich edema (10, 32). This edema has been considered a factor in initiating and continuing collagen degeneration. Swelling of collagen fibers is one of the early changes in collagen diseases (33, 34). The release of histamine from the granules of mast cells primes the mast cell-histamine chain and possibly indicates a contributing factor in the predilection of the following tissues or organs to lesions in the collagen diseases (35): (a) abundance of mast cells in the pleura in pleurisy of rheumatoid arthritis (36); (b) "cuffing" of mast cells around arterioles in periarteritis nodosa (37); (c) foreign protein release of mast cell-histamine in serum sickness. Edema appears in skeletal muscle also in dermatomyositis (38) and in degeneration of muscle fibers.

The discovery of a group of drugs, the antihistamines, that antagonized more or less specifically the effects of histamine led to further advances. Treatment with these compounds greatly reduced the inflammation caused by antigen-antibody reactions in certain diseases such as allergic rhinitis and urticaria. However, the majority of inflammatory lesions were not affected by the antihistamine drugs (39). As a result it seemed that histamine played a minor part in the total inflammatory reaction. Another weakness of the "generalized" histamine theory is the case of the sunburn reaction (15), which has a latent period of one to several hours. Sub-

sequently, reddening develops which is sharply limited to the area of irradiation. This limitation cannot be compared to the widespread, suddenly appearing flare of Lewis' triple response (26). In burn reactions, eczematous types of dermatitis and tuberculin-type hypersensitivities, the role of histamine in the inflammation is also negligible. There are hardly ever any transitions between the urticarial and eczematous type of inflammations. Such transitions should be expected if the reactions depended only on the rate of histamine liberation and on its concentration in the tissue. The same lack of histamine liberation is found in the case of erythematous-edematous blistering reactions and other diseases with prevailing blister formation, such as smallpox, chicken pox, herpes simplex, herpes zoster and persistent papules (15).

The consensus of results (24, 40, 41) appears to indicate that the role of histamine in inflammation is to initiate the vascular changes, especially increased capillary permeability, and the subsequent sustenance of these changes is due to other mechanisms independent of histamine release (23). The rapidity with which the effects of histamine occur after injury may be explained by assuming that the histamine is rapidly released from the mast cells, which disrupt and liberate their granules in response to injury. The precise mechanism of the release is a complicated process not yet completely clarified, but appears to involve formation of ATP, dependent on the glycolytic cycle of carbohydrate metabolism. There is a possible final activation of lytic enzymes capable of lysing the structure of the mast cells (25, 42), and injury activates the enzymes. It has been suggested that the release of histamine follows from the rupture of a peptide or polar bond linking histamine to a protein, or an ion exchange reaction, releasing histamine from loose combination with an acidic body compound (43). There is also evidence that injury may cause increased activity of histidine decarboxylase and this leads to increased synthesis of histamine (44).

*B. Serotonin.* Serotonin, 5-hydroxy tryptamine, is a monoamine derived from the amino acid tryptophane. The primary role of serotonin is unknown but it is believed to have a part in the transmission of nerve impulses (45). In very low concentrations serotonin increases capillary permeability (46) and will cause local progressive collagenous and fibrous proliferation within the dermis on long-term injection, in the rat (47). However, it does not produce these effects in most other species and even in the rat the evidence of an important role in inflammation is meagre (48). Compounds exist which are more or less specific antagonists to serotonin, such as 1-methyl-d-lysergic acid butanolamide and cyproheptadine. Dosage with such substances considerably reduces the inflammatory reaction caused by serotonin in rats. These studies (48) however, did not prove that the observed effects were due to the inhibition of serotonin or related compounds. It is possible that some unknown pharmacologic action was responsible.

*C. Peptides.* The possible role of peptides in inflammation has been suspected for over a hundred years, but the study of the effect of peptides on capillaries really dates from the observations of Menkin in the middle 1930's (49, 50). His early studies indicated that injured cells at the site of inflammation release a permeability and chemotactic factor which was called "leukotaxine," and which appears to be a polypeptide. Leukotaxine is capable of essentially reproducing the effect of the whole exudate as far as increasing local capillary permeability and inducing the emigration of polymorphonuclear leukocytes (51). Unfortunately, leukotaxine has never been purified, its detailed structure is unknown and its significance not adequately defined. It might also be noted that leukotaxine appears after the inflammation is established.

Later work demonstrated that large numbers of different peptides from various sources were able to increase capillary permeability and that the most likely requisite for this peptide property was a molecule containing eight to twelve amino acids (52). These observations culminated in the discovery (53), purification (54), and synthesis (55) of one of the most important members of this group, bradykinin, a peptide derived by proteolysis from plasma globulin. It is released by proteolytic and coagulating venoms and by trypsin from the globulin fraction of normal plasma. The name "bradykinin" was given to indicate the slow movement of the guinea pig ileum produced by it, differing from the more rapid contractions of the ileum by histamine or acetyl choline. It is now customary to refer to all inflammatory peptides by the term "kinins."

In addition to stimulating smooth muscle, bradykinin causes all the inflammatory reactions of vasodilatation, increased capillary permeability, accumulation and migration of leukocytes, and especially pain (56, 57). Bradykinin is also known to have some effect on the nervous system, but precisely what it does is still not known. It will cause pain when injected under the skin and at the same time there will be a flushing of the skin in the region, but if the area is denervated the flushing will not take place. This appears to indicate that the redness is due to a specific effect of the bradykinin on the nerves.

When tissues are damaged by severe burns, or animal skins are experimentally heated, there is a marked increase in bradykinin concentration in the interstitial fluid and urine (58). Unfortunately, there is, as yet, little quantitative data to correlate the amount of cell damage. These findings, however, provide substantial evidence that a plasma kinin, possibly bradykinin, takes part in the inflammatory response.

Fortunately, there exists an efficient bodily system for the destruction of excess bradykinin and other kinins (58). If there were no such system, the kinins would keep building up in the blood stream and continue to enlarge the entire system of blood vessels, continually increasing their permeability.

This would result in general vascular collapse. A bradykinin-inactivating enzyme, kininase, is present in blood and extracts of renal tissue. In addition, an inactivator of kallikrein, mentioned as the bradykinin-forming enzyme, is present in high concentration in lymph nodes. This destruction by peptidases may in fact be a homeostatic mechanism for inactivating these powerful kinin substances once they escape from the site of inflammatory reactions.

Natural bradykinin was isolated in pure form (59, 60) after the release by trypsin from bovine plasma globulins. It was actually synthesized (61, 62) before its structure was known. The structure is now known to be a linear nonapeptide of the following amino acid sequence (58, 63, 64):

(H—Arg—Pro—Pro—Gly—Phe—Ser—Pro—Phe—Arg—OH)

However, bradykinin is not the only active peptide formed by the action of enzymes on the plasma globulins. A decapeptide, called kallidin, differing from bradykinin only by the addition of one amino acid residue, lysine, at one end, has been isolated and characterized from blood (65). The structure of kallidin is:

(H—Lys—Arg—Pro—Pro—Gly—Phe—Ser—Pro—Phe—Arg—OH)

Kallidin is less active than bradykinin on most tissues but has a similar spectrum of activity. Another potentially important kinin is the recently isolated peptide known so far only as "substance P" (66). The purified material has many of the properties of bradykinin, but its structure contains at least six amino acids not found in bradykinin. Since different proteolytic enzymes not only give rise to bradykinin, but to other peptides with similar properties, it has greatly complicated the problems of relating the various "kinins" to body function and particularly to inflammatory function.

*D. Proteins.* Kallikrein, the bradykinin-forming enzyme and certain globulin fractions of plasma have been shown to be capable of increasing capillary permeability, in very low concentration (41, 67). The plasma globulin is present normally as an inert precursor that can be activated by a variety of procedures, including dilution with saline, contact with organic solvents and incubation with minced tissues (23). It is thus an obvious candidate for the role of mediator of the vascular changes of acute inflammation. The protein could act directly on vessels (possibly enzymically) or could be either a protease, or less likely, a substrate in formation of kinin peptides. Kallikrein obviously exerts its effects on capillaries and smooth muscle by catalyzing the formation of kinins from globulin substrates (24).

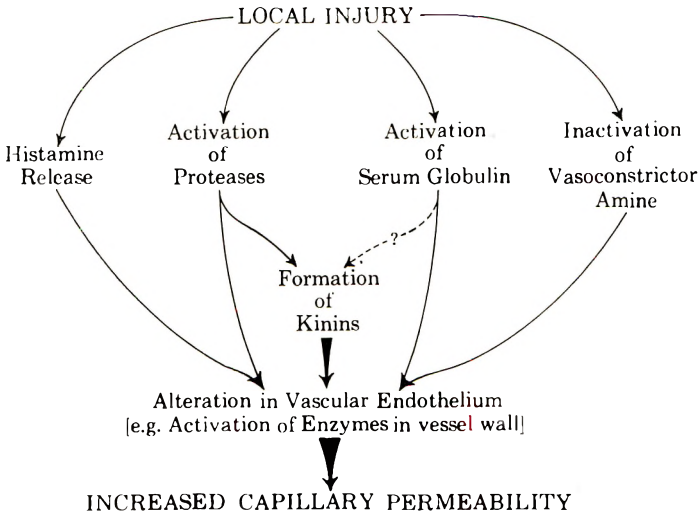
*E. Vasoconstrictor Amines.* The previously discussed mediators of

inflammation have all been active dilatators of blood vessels, increasing capillary permeability. Vasoconstrictor forces have been ignored. Yet the first vascular event following an acute injury is a momentary and transitory contraction of local blood vessels. A newer concept has recently arisen explaining initial vasoconstrictor forces, following the observation that the manifestations of rheumatoid arthritis and related diseases could be altered by the administration of small doses of iproniazid, an amine oxidase inhibitor (68, 69). This appeared to indicate that the vascular changes of acute inflammation are partly due to the destruction of an amine that would otherwise constrict and reduce the permeability of capillaries and oppose the action of compounds such as histamine and kinins. The most important endogenous compounds with these "antipermeability" actions on blood vessels are the catechol monoamines: adrenalin and noradrenalin. They are present in platelets, leukocytes and vessel walls, and at least two enzymes destroy them in the body, monoamine oxidase and catechol-O-methyl transferase. Administration of specific monoamine oxidase inhibitors greatly reduces the increased capillary permeability consequent to thermal or chemical injury. This result is explicable on the basis that inflammatory phenomena are partly due to inactivation of vasoconstrictor amines by monoamine oxidase. Competitive inhibitors of catechol-O-methyl transferase failed to modify the inflammatory reaction.

Recent evidence (70) dealing with the genesis of tissue destruction following locally administered bacterial extracts suggests that epinephrine is an important factor in mediating the subsequent inflammation. The mechanism by which catechol amines accelerate tissue damage is not well understood. Epinephrine does not seem to be involved in the local inflammatory response *per se* (71), but rather in the subsequent development of endothelial damage. While the evidence is not yet well substantiated, Cameron and Spector (23) postulated that, following injury, an adrenalin-like substance and an amine oxidase may be brought into contact. As a result the vasoconstrictor amine may be destroyed, and inflammation allowed to proceed. It seems possible that in the wall of the normal small blood vessel, adrenalin-like and histamine-like compounds compete for receptor sites, the interplay of their actions making for normal vascular reactions. In inflammation, not only are the vasodilator forces greatly augmented but the vasoconstrictor forces may be inactivated. The enzymic inactivation of the adrenalin-like substance could be precipitated either by local activation of monoamine oxidase or release of the amine from a site inaccessible to the enzyme (23).

## Mechanism of Increased Capillary Permeability

Cameron and Spector (23) attempt to illustrate schematically the sequence of events of increased capillary permeability as follows:




It is suggested that the initial event appears to be a release of histamine from mast cells by a mechanism not yet fully understood but possibly involving activation of lytic enzymes which break down the structure of the mast cells. Histamine appears to exert its effects within a minute or two of injury, but continues to dilate capillaries and increase their permeability for some time afterward, for at least one to two hours.

At the same time as histamine is released, it is postulated that an adrenalin-like substance is brought into contact with the enzyme monamine oxidase which destroys it. The hypothetical destruction of the adrenalin-like substance leads to dilatation and increased permeability of small vessels that begins rapidly and may last for twenty-four hours or even longer. Soon after these initial events there may be an activation of globulins and peptides that increase capillary permeability.

Little to nothing is known of the intimate mechanism whereby histamine, globulins, and peptides increase capillary permeability. There is some evidence that all endogenous mediators of increased capillary permeability may exert their effects by activating an enzyme of the esterase-protease group in or near the vessel wall (72). The substrate of this enzyme could be a protein or phospholipid in the capillary wall of the precursor of yet another mediator substance which then acts on the blood vessel (73).

It is of interest that high concentrations of some antihistamine drugs not only exert a general antagonism to increased capillary permeability but also cause a general inhibition of electrolyte movements in damaged cells and mitochondria (23). This may possibly mean that increased capillary permeability to protein is in some way secondary to, or at least associated with, electrolyte disturbance in the vascular epithelium and that capillary

permeability factors such as histamine may act by altering the electrolyte and water balance of these cells. These speculations have gained some support by electron microscopy which has indicated that protein appears to leave histamine-treated vessels by transport through the endothelial cytoplasm rather than by passage through channels of molecular dimensions between the cells (74).

A new point of view has related the inflammatory processes and blood coagulation (75). There is no experimental proof that all the aforementioned mediators exert their effects by a simple direct action on the vascular wall. It is suggested that the intervention of a coagulating mechanism or fibrinogen production is a prerequisite to typical inflammatory vascular reaction. The assumed coagulation process in the vascular wall may be initiated by the inflammatory agent itself or by endogenous thromboplastic factors produced under the influence of these agents.

## Anti-Inflammatory Drugs

*A. Corticosteroids.* Clinical anti-inflammatory agents, in order of their importance, are the corticosteroids, salicylates, phenylbutazone-antipyrine type, gold salts, antimalarial aminoquinoline compounds and enzymes. This review shall only discuss the corticosteroids since mechanistic data is sadly lacking in all the others. The corticosteroids represent the most successful anti-inflammatory drugs yet found. There are few physiologic processes which are not influenced directly or indirectly by the corticosteroids. Yet they do not appear to start any physiologic activity, but merely influence rates. Apparently, corticosteroids are not consumed in the process of exerting their physiologic effects, but rather they act as catalysts.

Corticoids tend to suppress the whole process of inflammation, inhibiting to various degrees the maximum development of any of the stages of inflammation (76). Unlike other inhibitors of inflammation they can check the inflammatory response at any stage from the initial swelling and increased capillary permeability to final dissolution of connective tissue (77). Hydrocortisone acts in a dose-response fashion, inhibiting to various degrees the maximum development of any of the stages of inflammation, depending on the nature and amount of the inflammatory stimulus (78–80).

The processes of regeneration and growth depend on well regulated processes in connective tissues. The mast cells and fibroblasts are the major active components. These processes of regeneration and growth are inhibited by the glucocorticoids. Mast cells diminish, become vacuolated and acquire irregular outlines. The number of demonstrable cells is diminished (81). The most consistent effect of the steroids appears to be an inhibition of mucopolysaccharide synthesis by the connective tissue. At a metabolic level, several investigators have demonstrated an inhibition of the incorporation of radioactive sulfate into the mucopolysaccharides of

connective tissue under the influence of cortisone or hydrocortisone (82–85). Inhibition of mucopolysaccharide synthesis by steroids was also demonstrated, following depletion of cartilage matrix by papain (85). When animals are intravenously injected with crystalline papain protease, all of the basophilic and metachromatic components of cartilage disappear within a few hours. This is associated with a loss of chondroitin sulfate from the cartilage and its appearance in the circulating blood. Reconstitution of cartilage matrix begins two days after the injection of papain, and chondromucoprotein is completely restored within the next three or four days. When cortisone is administered after papain, reconstitution of matrix is completely prevented. This inhibition seems to involve a direct action of cortisone on the cartilage. This inhibition of chondroitin sulfuric acid synthesis has been demonstrated in delaying the healing of wounds (86).

Cortisone inhibits the acccumulation of liberated histamine in the connective tissue (87). The steroid is a very effective inhibitor of the mast cell-histamine chain. Cortisone appears to maintain the tonus of small arterioles which are injured by histamine (10) and decreases permeability of existing capillaries and interstitial substances, possibly by inhibiting the action of hyaluronidases (10, 33), of histidine decarboxylase (88), and of histaminase (89).

Early in the process of inflammation, degenerative changes occur in the fibroblasts. The administration of hydrocortisone also produces morphological changes in many of the fibroblasts of loose connective tissue (90). Fibroblasts are among the first cells showing degeneration when connective tissue becomes inflamed. It appears that one essential action of the corticosteroids is the inhibition of the progressive destruction of fibroblasts in an area of potential inflammation (77, 91–93). The fibroblast appears to be the most common and dominating cell in connective tissue which sequesters and metabolizes hydrocortisone (94). It has been suggested (77, 95) that the degree of inflammation is enhanced by the autocatalytic destruction of fibroblasts, in which the destruction of one cell adds to the amount of inflammatory substances such as histamine or a kinin and leads to the destruction of other inflammatory substances. This chain reaction of cell breakage is interrupted by the action of hydrocortisone, which increases the resistance of some fibroblasts. While different research groups may differ upon the importance of steroidal effects on fibroblasts, it is clear that hydrocortisone is metabolized by fibroblasts. Even if a sufficient amount of hydrocortisone is produced by the adrenal cortex in individuals having inflamed tissue, if the hormone is inactivated at the fibroblastic site of action more rapidly than under usual normal conditions, anti-inflammatory influence could be insufficient to inhibit the inflammation (96). According to this theory, the peripheral cells may influence the disease process by their own capacities to metabolize the

steroid. Thus, the fundamental defects in chronic inflammatory diseases may not be at the hormone supply level but may be due to altered hormone metabolism at the fibroblastic level. Perhaps it is through the preservation of cellular integrity that hydrocortisone tends to minimize the subsequent phases of the inflammatory reaction.

The adrenal steroid actions contrast markedly with the effect of the estrogenic sex hormones. The sex hormones have been shown to increase the amount of intracellular water in the skin of mice, probably by increasing the amount of ground substance, while the corticoids appear to have an opposite effect (97). Hydrocortisone ointment causes progressive atrophy of collagen fibers, disappearance of interfibrillar mucopolysaccharides, dissociation of elastic fibers and atrophy of fibroblasts (98).

A most interesting observation has been made that corticosteroids can chelate potassium ions and are capable of binding copper (99). Cortisone causes a redistribution of copper in the body, with an increased renal and urinary concentration and a decreased concentration in other tissues and in serum. It was suggested that the anti-inflammatory effects of the steroids may occur because of the chelation of an essential metal activator of an undefined enzyme (99). A recent study (100) suggests that the ability to form complexes or chelates in or across a lipid phase is of anti-inflammatory importance. The potency of a number of anti-inflammatory drugs could be correlated with at least two physical properties: ability to form complexes with metal ions and the lipophilic character, favoring partition into the lipid rather than aqueous phase. As an index of potency, the investigator used the ability of the compounds to inhibit incorporation of inorganic sulfate into cartilage and corneal polysaccharides.

It has been well demonstrated that administration of cortisone will be followed by a diminished amount of circulating antibody (101). Cortisone inhibits the incorporation of labeled amino acids into tissue protein (102). Under most circumstances, it appears that cortisone has a distinct antianabolic effect on proteins, generally inhibiting the synthesis of protein. On a cellular level, corticosteroids inhibit the inflammation, which can readily be observed during the course of immunization with adjuvants such as alum, killed tubercle bacilli, and various vehicles and irritants (77). This may also be regarded as a kind of anti-inflammatory action of cortisone which results in inhibition of antibody synthesis.

While the corticosteroids are used principally for their anti-inflammatory effects, the mass of diverse effects cause additional actions which have come to be regarded as "side effects." While this paper is concerned chiefly with mechanisms, it will be concluded with a brief review of the broad side effects resulting from excess levels of corticosteroids.

Hydrocortisone indirectly controls adrenocortical secretion by restraining the secretion of adrenocorticotropic hormone (ACTH) by the pituitary.

It is becoming increasingly evident that the pituitary secretes ACTH in response to influences reaching it from the central nervous system. Hydrocortisone may suppress ACTH secretion by altering the rate at which corticotropin-releasing factors are elaborated by the central nervous system or by diminishing the responsiveness of the adenohypophysis to corticotropin-releasing factors. Whatever the mechanism, the higher the level of hydrocortisone, the greater is the restraint on ACTH secretion. Thus, when supraphysiologic doses of hydrocortisone are used in the treatment of inflammatory conditions, ACTH secretion is suppressed, and this leads to cessation of adrenocortical secretory activity, diminished responsiveness to exogenous ACTH, and progressive atrophy of the adrenal cortex. These changes in adrenocortical function are generally reversible if exogenous ACTH is administered or if hydrocortisone administration is discontinued, permitting recovery of endogenous ACTH secretion (103).

There are so many other diverse manifestations of supraphysiologic levels of hydrocortisone that they cannot all be treated here. It should be mentioned that the corticoids affect protein metabolism in a variety of ways. Which of the effects will ultimately come to be regarded as primary and which secondary cannot be judged with certainty. There is both an anabolic effect by uptake of amino acids by the liver (104) and catabolic interference with cellular uptake of amino acids (105). As a consequence of these actions, hydrocortisone causes clinical manifestations of protein wasting.

Corticosteroids, in excess dosage, promote the deposition of adipose tissue in the facial, abdominal and shoulder areas, as well as promoting sodium retention and potassium excretion by stimulating cation exchange by the renal tubule. Opposing this effect is the tendency of hydrocortisone to increase glomerular filtration rate which promotes sodium excretion.

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# THE HUMAN SCALP AS A HABITAT FOR YEASTS*

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## Introduction and Background

Almost from the very moment when Rivolta (1) in Italy in 1873 first isolated and described the yeast organism, *Pityrosporum ovale* from the human scalp, the etiological role of this organism in seborrheic dermatitis and pityriasis capitis has been a matter of much dispute. On the one hand, its universal presence in dandruff scales in numbers proportional to the severity of the condition has been verified by every worker interested in the problem. On the other hand, the lack of convincing evidence to prove pathogenicity through animal or human inoculation has led to the modern supposition that *P. ovale* is an inoffensive saprophyte of man.

Some investigators, however, argue that this organism more nearly behaves as a true parasite. It has been isolated nowhere but from the animal skin. It is extremely fastidious in its growth habits, requiring naturally occurring fatty acids together with a source of nitrogen and glucose. Each of these nutrients is supplied by the skin. Stained smears of the scalp usually reveal many actively budding forms.

In addition to the possible role of *Pityrosporum ovale* in seborrheic dermatitis and pityriasis capitis, an etiological relationship to seborrheic blepharitis and dermatitis of the eyelids has been suggested by Gots (2) and his co-workers. In their studies budding yeast forms morphologically similar to *P. ovale* were found in 100 per cent of 143 cases of seborrheic blepharitis. They were also able to demonstrate sensitization of the organism by intradermal skin tests and have indicated that inflammatory lesions of the conjunctiva are allergic in character, irrespective of the role of the organism in seborrheic dermatitis.

More recently, Gordon (3) has described a new species of *Pityrosporum* which he has named *P. orbiculare* because of its spherical shape. The organism was isolated from 15 of 18 cases diagnosed as tinea versicolor. Because of its lipophilic nature it seems to be related to *P. ovale*. Attempts

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at experimental inoculation have failed to produce tinea versicolor in humans. It is interesting to note that the organism described by Gordon is morphologically identical to the so-called spherical forms of *P. ovale* found by Gots in lid-margin scrapings from cases of seborrheic blepharitis.

The authors of this paper became interested in the yeast flora of the human scalp while working with improved culture media for isolating and growing *P. ovale*. Isolation studies showed that *P. ovale*, while prevalent, is not the only yeast organism of the scalp. The possibility occurred to us that since *P. ovale* could not definitely be linked to dandruff, perhaps some other, as yet undiscovered, yeast could be a contributing factor.

We therefore decided to survey a large number of humans in the hope of relating the scalp yeast flora to possible pathogenicity.

The literature makes many references to the isolation and identification of yeasts from various areas of the body in both normal and pathological conditions but only an occasional reference to the human scalp. Surveys on the yeast flora of the external surface, the nails, the orifices and the alimentary canal have been carried out. At least three factors seem to be responsible for this interest.

1. New and improved media have been formulated for the isolation and study of yeasts, which have resulted, for example, in the realization of the obligate lipophilic nature of *Pityrosporum ovale* (4). The introduction of certain antibiotics into culture media has facilitated the isolation of yeasts without interference by bacterial overgrowth.

2. The monograph of Lodder and Kreger-Van Rij (5) and the contributions of Wickerham (6) have created a better understanding of the taxonomy of yeasts.

3. A number of infections attributed to members of the yeast group have been reported. Many species of *Candida* and *Cryptococcus* are being isolated with increasing frequency from a variety of lesions in man, although *Candida albicans* and *Cryptococcus neoformans* have been considered to be the only pathogenic members of the group. The widespread use of antibiotics is responsible in no small part for the increase in candidiasis and cryptococcosis.

Huxley and Hurd (7) isolated from the skin and described a number of pink yeasts belonging to the genus *Rhodotorula* and *Sporobolomyces*. The skin areas selected for study were those between the toes, between the fingers, and from the lumbar region, the axilla, the inframammary region and the navel. They found the predominant organism to be *Rhodotorula mucilaginosa*. Connell and Skinner (8) determined the occurrence of nonfermenting, nonpigmented yeasts on 250 human subjects. Of 784 isolates of yeasts and yeast-like fungi the following species are recorded as having been isolated. They are given in the order of their frequency: *Cryptococcus diffluens*, *Cr. aerius*, *Cr. albidus*, *Cr. laurentii*, var. *flavescens*, *Cr. rotundatus*,

*Candida spp.*, *Lipomyces starkeyi*, *Candida zeylanoides*, *C. lipolytica* and *C. mesenterica*. Their studies show that qualitatively and quantitatively the yeasts on the body are different from those in the air or from surfaces with which the body comes in contact. They concluded that the skin surface may well be a true habitat for yeasts.

Croft and Black (9) also concluded that yeasts live saprophytically on normal skin. They isolated 29 yeasts from the fingertips of 22 per cent of the persons surveyed. *Candida parapsilosis* was the predominating organism, appearing in 12 cases. Other yeast-like organisms found were *Endomyces spp.*, in four cases; *Monilia nigra*, in three; *Cryptococcus*, in two; *Mycoderma*, in one; *Schizosaccharomyces hominis*, in one; unidentified *Monilia*, in three; and unknown colonies, in three.

DiMenna (10) examined several areas of the body. She found that the following nonpathogenic species occurred in approximately equal numbers in the alimentary canal and in the oral cavity: *Saccharomyces spp.*, *Candida krusei*, *C. parapsilosis*, and *Torulopsis glabrata*. The pathogenic *C. albicans* was isolated from the oral cavity in 83 per cent of the cases and from the alimentary canal in 67 per cent of the cases. The areas of the skin surveyed by DiMenna were those of the fingernails and of the arms of 120 isolates from 381 subjects. The predominating yeast-like organisms were *Debaryomyces kloekeri*, in 34 cases; *Cryptococcus spp.*, in 20 cases; *Rhodotorula spp.*, in 27 cases; and the yeast-like phase of *Cladosporium spp.*, in 22 cases. In addition, direct smears were made of the scalp to determine the presence of *P. ovale*. This organism was found to be present in 82 of 87 subjects examined. In only one instance was a cultural examination made of the scalp, and this was found to be negative for *P. ovale*. DiMenna considers that there are three possible categories of yeasts, with respect to their habit, isolated from man: 1) those that are contaminants, picked up from their surroundings and incapable of reproduction or of prolonged survival upon the human body; 2) those that are capable of multiplying upon the body as well as apart from it; and 3) those that are obligate parasites upon the human body. *Candida albicans*, *P. ovale*, and very possibly *Torulopsis glabrata* belong in the third category. Contrary to the findings of Connell and Skinner, DiMenna concluded that the yeasts found on the skin (exclusive of *P. ovale*) and in the air are similar, both in proportions of different genera and in kinds of species. Skinner *et al.* (11) stated that yeasts were consistently isolated from the skin of about 50 per cent of the 275 persons who were examined. In another survey Benham and Hopkins (12) isolated 57 strains from 100 fingernail cultures, 62 from toe nails and 41 from toe webs.

Sturde (13) in Germany examined the infected fingernails of 50 patients for the presence of yeasts. In each instance one or more yeasts were isolated. Those predominating were *C. albicans*, *C. parapsilosis*, and *Toru-*

*lopsis famata.* The most complete study to date on the distribution of yeasts in the human body has been made by Rieth (14) also in Germany. Over 15,000 patients were studied during a four year period. From the skin, hair and nails almost 2000 yeasts were identified. Those predominating were *C. albicans*, *C. parapsilosis*, *Torulopsis famata*, *Trichosporon cutaneum*, *Rhodotorula mucilaginosa* and *Rh. rubra.* Kapica and Blank (15), Fischer (16), Nino *et al.* (17) all claim repeated isolation of *C. parapsilosis* from infected nails showing typical signs of moniliasis. Kapica and Blank furthermore have carried out studies to prove that *C. albicans* (18) and *C. parapsilosis* (15) are both capable of utilizing nitrogen obtained solely from keratin, provided glucose is present as an initial stimulus. These workers admit that while such a biochemical investigation cannot provide an answer to the question of pathogenicity of *C. albicans* and *C. parapsilosis* its results indicate a reason for the pathogenicity. They found that this keratin breakdown occurs between 40 and 56 days.

As far as the references to the scalp flora are concerned Ota and Huang (19) simply report "ordinary" yeasts in addition to the organism they believed to be *P. ovale.* MacKee and Lewis (20) showed that the scalp yields numerous fungi and yeasts as well as bacteria but made no attempt to identify them. Benham (21) isolated a number of yeast-like organisms which she placed in the genus *Cryptococcus.* In several instances, moreover, isolates from the scalp reported to be *P. ovale* were later shown by her also to be members of the genus *Cryptococcus.* Similarly, Spoor *et al.* (22) in their isolation studies of *P. ovale* from normal and seborrheic subjects have isolated not only this organism in approximately 60 per cent of the cases but speak also of unidentified strains which are classified as "yeast-like" types.

## EXPERIMENTAL METHODS

Two groups of individuals were studied. They consisted of 98 members of the senior class of the Massachusetts College of Pharmacy from September, 1958, to June, 1959, and 91 members of the senior class from September, 1959, to January, 1960. Included in these groups were 17 females. The members of both groups ranged in age between twenty and thirty years.

The material used in this study from which the isolation of yeasts was made is commonly known as "scurf." Scurf represents desquamated epithelial cells from the scalp together with accumulated secretions, acquired soil, and a mixed microbiological flora. With all subjects it was possible to obtain a sample of scurf by instructing each one to brush his scalp vigorously and to collect the material on appropriate culture media. No attempt was made to record the degree of sloughing off or subsequently to correlate the amount of scurf with the type of organism

isolated. The groups surveyed were considered to be composed of normal young adults.

The culture media used for primary isolation were Littman Oxgall Agar (Difco-pH 7), Littman Oxgall Agar with 2 per cent sesame oil (pH 6.8) and Yeast Morphology Agar (Difco-pH 5). Streptomycin sulfate (30 micrograms per ml.) was added to each of the three media after sterilization and cooling to 45°C. Prior to pouring the plates which were to contain 2 per cent sesame oil, the oil was shaken vigorously with melted Littman Oxgall Agar in order to form an emulsion. Each culture was incubated for three days at 35°C. and then for two weeks at 25°C. The techniques and media used in the determination of the morphological and physiological properties of each yeast isolate are those described by Lodder and Kreger-Van Rij (5).

## RESULTS

Yeasts capable of growing on one or more of the media used in this study were isolated from 122 of the 189 individuals participating in the survey. This represents 65 per cent of the total. In some instances more than one yeast was present in the scalp of the same person, resulting in a total of 145 identifiable yeasts which could be maintained in subculture. Three members of the *Dermatiatiae* were also isolated. These are sometimes called the "Black Yeasts" because of their color. Since they are believed to represent a yeast-like phase of certain molds they were not included in this survey.

All the yeasts identified in this survey were found to be members of the *Cryptococcaceae*. No member of the ascospore forming family *Endomycetaceae* and the ballistospore forming family *Sporobolomycetaceae* was found. It was also noted that the two yeasts most commonly identified with pathological conditions in man are not scalp inhabitants. These are *C. albicans* and *Cr. neoformans*.

The predominant yeast was *P. ovale* which was found in 46 individuals. Other workers have reported the occurrence of *P. ovale* on the scalp as being between 70 and 94 per cent; but this percentage was reached by a direct smear from the scalp and not by growth on isolation media. The other two most common yeasts were *C. parapsilosis* in 32 individuals and *Rh. mucilaginosa* in 26 individuals. In all, there were fourteen different species of yeasts identified. These are listed in Table 1.

Each of the 14 species of yeasts isolated in this survey were studied to determine which of them had been isolated from the human skin by previous workers and if possible to draw inferences concerning their possible role as scalp pathogens. Table 2 represents a compilation of the work of previous investigators.

It would appear, based upon the frequency of occurrence that only, *P.*

TABLE 1—IDENTIFIABLE YEASTS ISOLATED FROM THE SCALPS OF 189 HUMANS

| Organism | Number of Isolates | Frequency Percentage |
|---|---|---|
| *Pityrosporum ovale* | 46 | 31.72 |
| *Candida parapsilosis* | 32 | 22.07 |
| *Rhodotorula mucilaginosa* | 26 | 17.93 |
| *Cryptococcus diffluens* | 9 | 6.20 |
| *Torulopsis famata* | 8 | 5.52 |
| *Cryptococcus albidus* | 7 | 4.83 |
| *Rhodotorula minuta* | 4 | 2.76 |
| *Candida mycoderma* | 3 | 2.07 |
| *Trichosporon cutaneum* | 3 | 2.07 |
| *Torulopsis inconspicua* | 2 | 1.38 |
| *Cryptococcus laurentii* | 2 | 1.38 |
| *Rhodotorula flava* | 1 | 0.69 |
| *Rhodotorula rubra* | 1 | 0.69 |
| *Candida scotti* | 1 | 0.69 |
| | 145 | 100.00 |

*ovale*, *C. parapsilosis*, and *Rh. mucilaginosa* are prevalent enough in the human scalp to warrant serious consideration. The case for and against *P. ovale* has already been discussed. In view of the frequency with which *C. parapsilosis* has been isolated from onychomycotic infections and because of its proven keratinolytic nature this organism could be added to the list of possible pathogens. *Rh. mucilaginosa*, on the other hand, has not been recorded as being associated with any skin pathological condition except in one instance. The monograph of Lodder and Kreger-Van Rij (5) makes a single reference to its isolation by Wolfram and Zach from diseased nails in 1934. Because of the higher incidence of *Rh. mucilaginosa* in the air than upon human skin Connell and Skinner (8) refer to this organism as an adventitious saprophyte.

TABLE 2—COMPARATIVE STUDIES OF YEASTS ISOLATED FROM VARIOUS SKIN SURFACES

| Organism | A | B | C | D | E | F | G |
|---|---|---|---|---|---|---|---|
| *P. ovale* | * | | | | * | | |
| *C. parapsilosis* | * | | | * | * | * | * |
| *Rh. mucilaginosa* | * | * | * | | | * | * |
| *Cr. diffluens* | * | | * | | | | * |
| *T. famata* | * | | | | | * | * |
| *Cr. albidus* | * | | * | | | | |
| *Rh. minuta* | | * | | | | | * |
| *C. mycoderma* | * | | | | | | * |
| *Trich. cutaneum* | * | | | | | * | * |
| *T. inconspicua* | * | | | | | * | * |
| *Cr. laurentii* | * | | * | | | | |
| *Rh. flava* | | | | | | | |
| *Rh. rubra* | * | * | | | | | * |
| *C. scotti* | | | | | | | * |

A—Lodder and Kreger-Van Rij. B—Huxley and Hurd. C—Connell and Skinner. D—Croft and Black. E—DiMenna. F—Sturde. G—Rieth.

Preliminary studies undertaken by the authors indicate the possibility that both *P. ovale* and *Rh. mucilaginosa* are capable of utilizing keratin as a source of nitrogen following the technique of Kapica and Blank (15, 18). If such is the case the parasitic nature of these organisms would be established.

## SUMMARY

1. The yeast flora of the scalps of 189 college seniors has been investigated.

2. From 122 of these individuals (65%) a total of 145 yeasts were isolated by culture and were identified according to the monograph of Lodder and Kreger-Van Rij.

3. The 145 yeasts were represented by 14 species.

4. The three most prevalent yeasts were *Pityrosporum ovale*, *Candida parapsilosis*, and *Rhodotorula mucilaginosa*.

5. A comparative study was made with other surveys of yeasts of skin surfaces, which also showed that *C. parapsilosis* and *Rh. mucilaginosa* were common yeasts found on the scalp and on other skin surfaces.

6. Because of the frequency with which *C. parapsilosis* has been isolated from onychomycotic infections and because of its proved keratinolytic activity an association with pathological scalp conditions is suggested. Preliminary studies indicate a similar activity for *P. ovale* and *Rh. mucilaginosa*.

7. There is no evidence from this paper or from a survey of the literature that any of the other yeasts isolated could be causative of a pathological condition of the scalp.

8. This survey shows that the human scalp harbors yeasts in greater abundance and variety than other areas of the body.

9. The prevalence of *P. ovale* as reported by other workers has been substantiated by this survey.

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# THE THIRTEENTH MEDAL AWARD

## December 4, 1962

**Hotel Biltmore, New York City**

DR. PAUL G. I. LAUFFER, Head of Research, Stamford Division, Chesebrough-Pond's, was honored by the SOCIETY OF COSMETIC CHEMISTS for his scientific achievements and for his services to the cosmetic industry. Dr. Lauffer was awarded the Medal of the Society during its annual dinner dance. Dr. Everett G. McDonough acted as toastmaster for the presentation. H. Goulden was eulogist, and W. Dennis, as President of the SOCIETY, presented the Medal with the following citation:

DR. PAUL G. I. LAUFFER

In recognition of your many achievements during your devoted career in the field of cosmetic chemistry;

In recognition of your personal integrity and detailed efforts that have so greatly contributed to elevating the professional status of the cosmetic chemist;

In recognition of your outstanding services which you have performed on behalf of all cosmetic scientists for the cosmetic industry;

In gratitude for your many outstanding contributions to cosmetic technology and the scientific literature relating to cosmetics;

In appreciation of the thirty-six years you have served, not only as a chemist and director of research but also as mentor to young and new chemists entering our field;

Paul Lauffer, on behalf of the SOCIETY OF COSMETIC CHEMISTS, I hereby present to you the Medal of the SOCIETY which reads:

Awarded to
Paul G. I. Lauffer
by the
SOCIETY OF COSMETIC CHEMISTS
for Outstanding Contributions
to the Art and Science of Cosmetics

Dr. Paul Lauffer, (l.) receiving Medal from President Warren Dennis (r.) at the Society's 1962 Medal Award Dinner.

# FRIEND OF MAN—PAUL GIDEON ISAAC LAUFFER

EULOGY by EVERETT G. MCDONOUGH, PH.D.*

MY FRIEND Carey P. McCord, the father of industrial medicine, tells why he selected "Blind Hog's Acorn" as the title of one of his very interesting books. It seems that his father, as a minister, obtained additional remuneration by boarding traveling salesmen and other wandering men. One such man was a phrenologist who agreed to pay for his board by the reading of the lumps on the heads of the two sons of the minister. Dr. McCord's older brother was examined first and it was foreseen for him that he would have a brilliant future. When it came to Carey P. McCord's time, the man's face began to take on a doubtful look, and he began to shake his head. Carey's mother became truly worried and downcast as the phrenologist admitted that he could only see a most dismal future for this son. In his attempt to add some blue sky to the gloomy clouds ahead, the phrenologist attempted to cheer Mrs. McCord with the statement, "even a blind hog gets an occasional acorn."

Despite the fact that our Medalist, like Dr. McCord, is a minister's son, I hasten to correct the impression that this story refers to him. Rather I believe that *we* have been the blind hog in overlooking for so long this excellent candidate. I hope I can do justice to my part of this presentation as I feel we have, not an acorn, but a full grown oak tree.

I have known our Medalist for over thirty-five years; so I know you and he will forgive me if I refer to him as Paul.

My first encounter with Paul was an enriching one, for in part he contributed to my discovery of a way to obtain considerable free space while traveling even on the most crowded subway. The formula for this great discovery is as follows: work for several months on the study of autooxidation of aldehydes so that your clothes, skin and hair take on an odor which is a cross between a tub of rancid butter and a herd of wild goats. This will assure that the noses of all those who surround you will direct their eyes to your presence. Then be certain that you are reading the then outstanding book on chemotherapy, a very large volume whose title was clearly visible in large type, "Principles and Practices of Chemotherapy with Special References to the Specific and General Treatment of Syphilis."

* Evans Chemetics, Inc., New York 17, N. Y.

I can assure you that if you follow this process, that by the time those around you read that last word you will have armlength room in all directions.

Paul's contribution to this discovery was that he had just obtained his Doctor of Philosophy degree at Columbia University under Marston Taylor Bogert who incidentally was our first Medalist. His research was concerned with certain acridine dyes whose greatest potential use was in the field of chemotherapy.

Paul's dissertation was such a masterful presentation of the subject that Professor Bogert selected this general area for my first seminar. In order to be competent to understand the dissertation, I had to read Kolmer's authoritative book with the very effective title.

My second contact with Paul was a more direct one, and I believe it is worth relating as it indicated a trait which I feel that you will all agree that Paul has carried through his life. . . this is his consideration of others.

As a part of the initiation into the honorary chemical fraternity, Phi Lambda Upsilon, I had to obtain the signatures of all members at Columbia University in ink on an unglazed porcelain plate. You can imagine the damage that this abrasive plate did to the old-fashioned fountain pen. All of the signers, excepting Paul, used my pen. As he signed with his own pen, he said he guessed my pen had taken enough abuse.

In his thought-provoking book, "The Tao's of Science," Dr. Siu points out that America is a country that lives by the motto: "If you do not know what to do, do something." To adapt this philosophy to fit our Medalist, we need only add two words so that it becomes, "If you do not know what to do, do something for somebody."

This lending of the helping hand has been with Paul all of his life and it has earned him the title of "Professor" from his discerning high school math teacher and the title of "Mayor" from the appreciative citizens of the Village of Hastings-on-the-Hudson. This latter was not easily won and it took a lot of doing for a good-sized predominantly Republican community to elect a man running on a Democratic ticket. In Hastings, Paul is known as a dedicated man and has held almost all of the more important civic offices.

Another story will serve to illustrate this "Friend of Man" trait of Paul's. It took place as our country entered the First World War, which was just at the close of Paul's college freshman year. Paul had entered Washington-Jefferson College largely because his father had graduated from there. He had intended to study electrical engineering but, fortunately for all of us, as a freshman, he got a job in the chemistry stockroom and was converted to the idea of becoming a chemist. The chemistry department consisted of one professor who patriotically entered the service before commencement. However, he left a sad senior who had failed in

chemistry and could not graduate until he had passed a supplementary examination in general chemistry. Since Paul was the only person left in the college with any semblance of connection with the chemistry department, he had to make up an examination for the senior to take. Needless to say, the senior passed and graduated—thanks to Paul's help.

To be able to bring help to so many in such a wide variety of ways, a man must be capable not only mentally but also physically and also must have had a wide variety of experiences. Paul qualifies on all of these accounts.

Listen to the following for an accumulation of varied experiences. As I mentioned before, his father was a minister, and this unique environment was made even more varied by the fact that his father moved so frequently that Paul attended seven different grade schools. Yet, he was such a good scholar that he was valedictorian of his high school graduating class. In college, after the experience with the failing senior, he became, in his sophomore year, the laboratory assistant to the new professor of chemistry and held this job until he graduated. Washington-Jefferson was unwilling to let him go so he stayed on for two more years as a graduate assistant in the chemistry department. During this time he took sufficient courses to obtain a Master of Science degree.

Undoubtedly, the two greatest names in chemistry in the early 20's were Stieglitz of the University of Chicago and Bogert of Columbia University. Paul studied for two summers under Stieglitz and, in addition, had some side courses under the also very famous scientist, Morris Cohn. At Columbia, as I have already mentioned, he worked on the acridine dyes, specifically the isomers of proflavine and acriflavine under the sponsorship of Bogert. In addition, he was a graduate assistant in organic chemistry, a DuPont Fellow, a Fritzsche Fellow and, on the side, minored in metallography.

I should not forget to stress that his study of four foreign languages allows him to keep us all up-to-date on what is happening elsewhere in the cosmetic world.

Paul's life was not an easy one. His father died when he was eleven years old and it was necessary for him to do all types of odd jobs. At the ages of fifteen and sixteen he was on piece work in a glass factory. At seventeen, we find him selling books in rural communities. At eighteen, he was working in a rolling mill and, at nineteen and twenty, we have him working "down on the farm."

To do all of this work, a man must have a pretty sturdy body and it is noted that Paul played basketball, baseball, and was on the track team and despite his lack of height was fullback on the high school football team. His most important recreational activity in recent years centers around mountain climbing, a sport which in my opinion demands more physical stamina than any other. He has been an officer and a member of the

Board of Governors of the Adirondack Mountain Club. His other hobby is the conservation of our beautiful forests and water supplies.

On the more personal side, Paul has been a devoted family man. I know that he considers as his greatest accomplishment his daughter and son and four lovely grandchildren.

In this much too brief review, I think I should mention at least one other outstanding characteristic of Paul's. Perhaps I do so to excuse our lack of recognition for so long. Paul is a modest man and a quiet man; nevertheless he does serve as a living evidence to support the Greek historian Thucydides' statement that "of all the manifestations of power, restraint impresses men the most."

It seems to me that it is most appropriate that Paul was born and raised in Pennsylvania, a state long renowned for its friendly people, and in closing I ask that you ponder the appropriateness of his given names, two of them being the names of his two grandfathers, and the title I have tried to use as a summation of him—"Friend of Man—Paul Gideon Isaac Lauffer."

# PAUL G. I. LAUFFER, MAN AND SCIENTIST

EULOGY BY H. D. GOULDEN*

I AM to tell you something about Paul Lauffer, Scientist. It is not my intention to discuss in detail or to enumerate his many contributions to scientific literature but to cite instances and examples which, I believe, will interest you and indicate in some measure why he is so esteemed by his colleagues. I shall be brief, because I am under admonition from our honored guest to keep this brief, the briefer the better.

Twenty years ago, shortly after I came to The Toilet Goods Association, your toastmaster introduced me to Paul during luncheon. I became thoroughly convinced that here was one of the great minds, a true scientist. Quite naturally, I cultivated his acquaintance. He has been friend and counselor, and, since he has always had the industry point of view, a much appreciated ally.

Whenever in the course of my activities it becomes necessary to form a committee for any purpose, I always try to place Paul on the committee. Why? Because he will sit quietly listening to what all the other members of the committee have said; when I ask for his opinion, there is complete silence, and then he will in a few well-chosen sentences summarize all the facts developed, the conclusions to be drawn and the indicated actions, if any. Invariably he is right, and all agree. I doubt very much that Paul is aware of this situation; however, I have seen it happen time and time again. Committees of which he is a member frequently tell me they feel they accomplished something and acknowledge that it was largely through Paul's efforts that they did. He is truly a scientist's scientist.

During the war years, while Paul was at the George W. Luft Co., he was involved in making gas detection kits for the government. As you all know, the purpose of these kits was to detect extremely small concentrations of poisonous gases in the atmosphere. The chemicals used for this purpose were quite sensitive and often would detect traces of other chemically similar substances; hence, misleading conclusions would often result. Paul, I am informed by those who worked with him, devised tests for identifying these contaminants and methods for eliminating these unwanted reactions during production. I understand that his co-workers were dumbfounded at the speed with which this problem was solved. The

* The Toilet Goods Association, New York 20, N. Y.

production problems which arose were many and complicated enough to rattle ordinary men, but not Paul. This ability to remain calm and objective under all circumstances is characteristic of Paul and most frequently mentioned by his many admirers. I can tell you, because I visited him at his plant, that he is not only a great "theoretical" scientist but a very practical one too, for that production line for turning out those gas tester kits was as sweet and ingenious a job as I have ever seen, and I have seen many.

In the laboratory, Paul is engrossed in the task at hand, so much so that he may not speak more than two or three words to the man alongside of him. I am told that on one occasion a rather talkative chemist, who was working in the same laboratory with our honored guest, became rather annoyed with this habit and decided not to talk to Paul at all. After a month of this treatment, the talkative one had to break the silence and admit that this was concentration the like of which he had never seen. Paul was completely unaware of what had been going on. When he is concentrating on a problem, he is oblivious of everything about him.

Paul is an avid reader of all scientific literature. He amazes all of us. We can't understand where he gets the time to do it—but he does. A former co-worker, and most of these refer to themselves as his students, informed me that Paul has, since his early days in the industry, kept chronological abstracts of scientific data pertaining to cosmetic science and related fields. It didn't take these co-workers long to discover that when assigned a new research problem, or when in need of up-to-date information on a cosmetic problem, the initial action was a search of Paul's files, and frequently one did not need to go further.

I wonder how many of you know that Paul, since receiving his doctor's degree, has devoted his life to the cosmetic industry. He has taken an active interest in the industry, not confining his activities to the particular company by whom he may have been employed. Organizing the first scientific organization in the domestic cosmetic industry was quite a task. Paul was of very great help to me, chiefly by encouraging me to keep at it and backing me up when needed. That was back in the days when *your* chemist, if seen talking with *my* chemist, would result in both being fired. Paul, I guess that more or less reveals how old we are, and I'm sorry—but don't you forget that I'm eleven days your senior and shall expect the respect and deference due your senior in years. His scientific contributions to the literature are many and cover a wide range of subjects, including amine soaps, emulsions, odor and olfaction, acridine, lipsticks, chapters on cosmetics in books and encyclopedias. His annual literature review under the title "Some New Keys to Cosmetic Chemistry" is most often cited as a major contribution by cosmetic chemists here and abroad. I do not need to tell you that cosmetic scientists face the almost impossible

task of keeping up with the various scientific disciplines which impinge on cosmetic technology. Paul's excellent reviews have annually brought a beautiful condensation of this literature. This has been of immeasurable benefit to all of us and, together with his other writings, must exert a tremendous influence on the young men who come into this very complex field of cosmetic science.

"Some New Keys to Cosmetic Chemistry—1962" is now in press. Let me quote the eleven printed lines which Paul uses to describe this major contribution: "As in previous years, a condensed summary has been prepared setting forth the past year's yield of new data, hypotheses, and concepts which appear to bear unusual interest for the cosmetic chemist. The reports have been selected from periodicals serving a wide variety of scientific and technological areas, and the emphasis has been on basic investigations, rather than upon new applications of old data. No attempt has been made to present a comprehensive treatment of any of the topics included in this summary. Material has been selected on the basis of its adjudged potential for changing the concepts or practices of the cosmetic chemist." This, ladies and gentlemen, is the author's modest description of a 15-page major contribution covering 322 articles in the scientific literature!

I trust that you will pardon a more detailed discussion of this contribution, especially since I am the editor of the "Proceedings" in which it appears. However, since it is gratefully and so well received throughout the world, such a discussion is in order, especially since it will reveal the many scientific disciplines in which our Medalist is interested. The subjects covered are: composition of the skin; metabolism of skin; keratinization; effect of chemicals on skin; hair and nails; sweat; sebum; ultrastructure of cells; metabolism of cells; mechanisms of the synthesis of proteins; structure of proteins; binding of proteins; structure and composition of collagen and elastin; collagen synthesis and fibrogenesis; effect of chemicals on collagen and elastin; structure and composition, formation and function of connective tissue; enzymes; permeability of skin; mechanism of permeability; effects of chemicals on permeability; antibodies and allergy; inflammation; germicides; pigmentation; aging of skin; effect of chemicals on aging skin; biosynthesis of cholesterol; structure and activity; gels; emulsions, etc. Our Medalist states, "During the past year, the literature of chemistry and allied disciplines has presented a galaxy of stimulating and useful new ideas," and concludes with this gross understatement, "A few of them have been presented here for their possible value to fellow cosmetic chemists."

In discussing Paul's contributions to the scientific literature, I would indeed be remiss if I did not mention his several contributions in the field of olfaction. He has made many contributions to this field that have

stimulated others to experimentation that has resulted in major advances in our knowledge of the mechanism of olfaction. Recently, when I was introduced to a European authority in this field, he said, "Oh yes, you're an American, aren't you? Do you know Dr. Lauffer?" and then he mentioned various articles Paul had written. He assured me he was very well thought of by others in olfaction. Paul is, heart and soul, a cosmetic chemist. He has always had the industry point of view. In my capacity as Scientific Director of T.G.A., I have called on Paul repeatedly during the past twenty years. His work on our Scientific Advisory Committee in developing raw material standards has been outstanding. He has been a very able chairman of our Scientific Section and, has on many occasions, presented erudite papers such as: Odor and Olfaction; The Sphere of Research; Olfaction and Cholinesterase, etc. He has appeared before Committees of Congress to present an industry point of view, and he has done so ably and effectively. He ably represented the cosmetic industry at the hearings on an order by F.D.A., seeking to delist certain certified colors of great importance to the industry. During this "show," which went on for days, he remained objective and effective and, wonder of wonders, really impressed the attorneys. He serves as a member of our Color Additives Scientific Advisory Committee which has done such a tremendous job, one not fully appreciated, for the entire cosmetic industry. The cosmetic industry may be particularly proud of the job done by Paul and his fellow committee members. It warms the cockles of my heart, and I am proud of them and of this industry effort. You are well aware that the industry is sponsoring the pharmacological evaluation of some 25 certified colors. Paul is one of four who periodically review, with me, the data developed. I value greatly his counsel and advice.

Paul Lauffer is more than just a dedicated scientist working in our industry. Morally and ethically, he has the respect of his many friends. No man, during the thirty years I have been in this industry, is more highly regarded and esteemed by his colleagues than is Paul. No greater praise can there be. For thirty-five years he has been contributing to cosmetic science, serving the Society of Cosmetic Chemists and T.G.A. Paul has always been an excellent, thorough and capable researcher. The cosmetic industry needs more men like Paul, "who avoid the spectacular and promotional and who constantly add to the fundamental basic knowledge on which the industry should be based." The Society of Cosmetic Chemists is to be congratulated for selecting Paul Lauffer as the recipient of its medal. A more popular decision could not have been made. Paul, may we offer our sincerest congratulations.

# A NEW ERA FOR COSMETIC CHEMISTRY

By Paul G. I. Lauffer*

In the past thirty-five years I have seen the cosmetic industry make tremendous progress in the volume of its business, in the variety of products offered, and in the quality of its merchandise. The very size of the Society of Cosmetic Chemists at the present time attests recognition on the part of cosmetic management that technical services are indispensable in developing and producing outstanding goods. The few small laboratories maintained by leaders in the industry thirty-five years ago have grown into scores of cosmetic laboratories, and technical staffs counted in the scores are no longer exceptional.

Still, as remarkable as such progress has been, I believe we are at the dawn of a new era in cosmetic technology. Great as has been the increase in volume of technical effort concentrated in our industry, most of that effort continues to be expended on control, development, and applied research. Very little time is spent in activity which by any stretch of the imagination can be called basic research.

The cosmetic industry is by no means alone in this situation. The United States pharmaceutical industry is spending about $268 million on research in 1962 (1), and the Federal government is spending about $850 million for research on medical and health problems (2). However, just this year a new drug came perilously close to being released for nationwide distribution before it was realized that it could produce serious deformities in the offspring of women who had used the drug in the early months of pregnancy. This near catastrophe had wide repercussions, and one result was the adoption of legislation which made more cumbersome the conditions for acceptance of a new drug. However, informed persons agree that the failure to predict the side-effects of this drug was not due to inadequate regulations nor to noncompliance with the regulations. It was due to the fact that nobody knows in sufficient detail how the differentiation of fetal tissues is controlled. In spite of the billion dollars a year spent on health research, this fundamental problem, and many other problems concerning the mechanisms of drug action, are getting little study and remain unsolved.

The general paucity of information on the effects of chemicals on physio-

* Chesebrough-Pond's, Stamford, Conn.

logical processes and structures was recently brought forcefully to the attention of the cosmetic industry. Manufacturers were directed to prove that the dyes they have been using, under the supervision of the U. S. Food and Drug Administration, for the last twenty-five years, are harmless and suitable for use in cosmetics. To prove this, they have been directed to use cumbersome, lengthy, and costly methods involving mass feeding of animals. Everyone agrees that such methods are primitive, and that the results are inconclusive. However, in the absence of any sound theory there is no alternative to such an empirical approach.

The drug and cosmetic industries are not alone in suffering the results of too little basic research. The federal budget for research and development was $9.6 billion in fiscal 1962, 11.4 billion in 1963, but there was increasing evidence that the very small percentage of this effort that had been expended on basic research was inadequate. The Department of Defense, for instance, tended to see its task as that of developing hardware and to leave basic research to the National Science Foundation. In general, short-term urgent needs tended to win out over possible gains to be expected over the long haul from basic research. The Office of Science and Technology was therefore set up in May, 1962, to coordinate and evaluate the research programs of all Government agencies. This Office is expected to increase the program of basic research (3).

Comment was recently made on the inadequacy of current Congressional machinery to determine the optimal allocation of funds for research (4). A move for reform of the legislative procedures governing such appropriations was said to have fairly substantial bipartisan support.

Each recent year has seen a rise in the total outlay for research in the United States. In fiscal 1961, public plus private expenditure for research and development was 2.78 per cent of the 1960 gross national product. Many leaders in the national research effort have pled for allocation of more funds for basic research, but it has been difficult to convince budget makers, public or private, that the long waits involved in reaping results from such research are justified. However, as National Science Foundation Director Alan Waterman pointed out (5), the money spent on basic research will in the long run lessen the total expenditure necessary to reach certain goals, for it will lessen the waste of diffuse and aimless exploration, unguided by adequate theory or principle.

Estimates of expenditures on basic research vary, since there is no generally accepted dividing line to determine which projects are basic. One report held that the chemical industry led all others in this respect, with basic research accounting for 11 per cent of the total 1960 expenditures of $1.067 billion for research and development (6). Another report, however, indicated that of the $1.2 billion R. and D. outlay by chemical industries in 1961, only about 4 per cent went for basic research (7). Total

R. and D. by all industrial firms in 1960 was $10.5 billion, of which 3.6 per cent went for basic research, according to the National Science Foundation (8).

Putting together various estimates, we may be justified in setting a figure of roughly $100 million a year as the current expenditure for basic research in the life sciences and another $100 million a year for other basic chemical research. The indications are that the volume of such basic research will continue to rise, as it has during the entire post-war period. As the volume of scientific literature expands, the problem of keeping abreast of it will become more acute. Every laboratory will need a staff of literature experts to screen out and condense the pertinent reports.

It is fortunate that the cosmetic industry can benefit from much of the data uncovered in today's large-scale research effort, for the $2 billion a year cosmetic business can support only a modest expenditure for research, in comparison with the stupendous figures mentioned above. We can, by alert attention to work in neighboring fields, uncover many ideas for use in solving our own problems.

Many signs point, however, to the likelihood of expanded programs of basic research within the laboratories of the cosmetic industry in the coming years. The continual demand for products of unique efficacy will best be met by intensive investigation to uncover new data not available from other sources. Many firms are learning that the best way to arrive at something really new is *via* serious work in their own laboratories. I predict that thirty-five years hence, barring nuclear catastrophe, research in the cosmetic industry will be on a scale surpassing that now in vogue in the drug and chemical industries.

Legislation which appears to be imminent threatens to seal the doom of the small cosmetic manufacturer. Such legislation would demand pretesting, control, and record keeping too formidable for any small company. Such increased technical overhead will be burdensome to manufacturers of all sizes, and may tempt some to seek relief by cutting their research budgets. The press of competition will, however, insure that research effort will soon resume its upward trend. Research personnel in the drug industry rose by 18 per cent during the two-year period 1960-1961 (9).

Possibly cooperative research into more scientific means of safety testing may be undertaken under the auspices of the Toilet Goods Association. This would be a natural extension of the services now rendered by the Scientific Director of T.G.A. in supervising the pharmacological testing of dyes and in issuing standards for methods of testing ingredients. Many other problems of common interest to cosmetic manufacturers could well be studied by similar cooperative methods, rather than by the duplicated efforts of many laboratories.

Most prophets agree that thirty-five years hence the population will include a much larger proportion of people in higher age groups than now. This should be the almost inevitable result of the billion-dollar a year study of health problems. One major goal of the cosmetic chemist is to develop products which will more effectively combat, counteract, or disguise the effects of age on the individual's appearance. When the problems of prolonging life have been largely solved, more effort can be concentrated on the means of preserving attractiveness in the later years.

The cosmetic chemist therefore has every reason to look upon the future with optimism. The trend appears to be toward more demand for cosmetic products, more insistence upon the novel and exclusive features which can best be perfected by basic research, and a general need for more technical services. A new era for cosmetic chemistry may well be dawning.

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CALIFORNIA CHAPTER OFFICERS

*Left to right*: 1st row: Warren Dennis, National President, 1962; and Ken Walker, California Chapter Chairman; 2nd row: Benjamin Kapp, Treasurer; Harold Jackson, Secretary; and E. Jeff Karolyi, Chairman-Elect.

## CALIFORNIA CHAPTER OFFICERS FOR 1963

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| Government Information | Dr. Paul W. Jewel |
| Membership | David S. Lozano |
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| Publicity | Oscar L. Scherr |
| 1965 Symposium | Clive N. Ross |
| University Relations | James Murray |

CHICAGO CHAPTER OFFICERS FOR 1963
*Left to right:* Warren J. Hintz, Chairman; James G. Atherton, Secretary; William D. Ackley, Chairman-Elect; Dennis H. Savoie, Treasurer.

## CHICAGO CHAPTER OFFICERS FOR 1963

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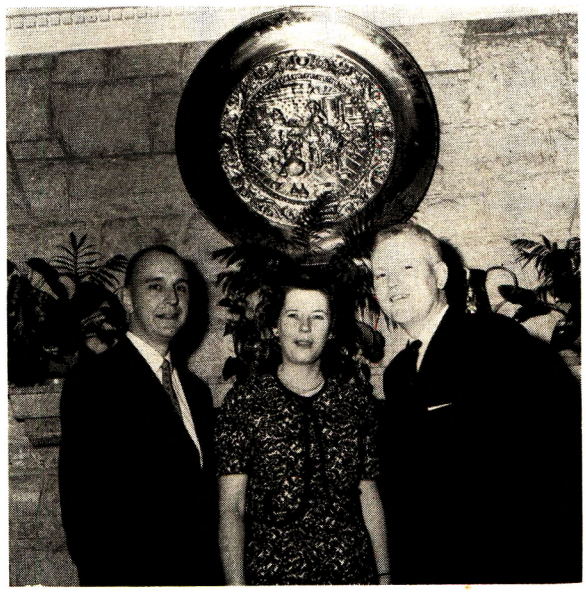
| Hospitality | Blaine Crouch |
| House | Helen E. Oberstar |
| Publicity | David A. Krochok |
| Inter-Professional Relations | Louis L. Lerner |
| Education | Marvin Kraft |
| Membership | Marshall Sorkin |
| Policy and By-Laws | Dr. Robert West |
| Ladies' Night | Barton R. Owells |
| Nominating | Stillman R. Goff |



NEW ENGLAND CHAPTER OFFICERS

*Left to right:* Dr. Winthrop Lange, Chairman-Elect; Pamela Low, Secretary; and Richard Reavey, Chairman. Vincent Beck, Treasurer not present when photo was taken.

## NEW ENGLAND CHAPTER OFFICERS FOR 1963

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| Chairman-Elect | Dr. Winthrop Lange |
| Secretary | Pamela Low |
| Treasurer | Vincent Beck |

Committee Chairmen:

| | |
|---|---|
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| House | Natalie Collins |
| Program | Dr. Winthrop Lange |
| Publicity | Donald Kirby |
| Inter-Professional Relations | Robert Schuler |
| Special Events | Hart Harris |
| News Letter | Myron Slotsky |

NEW YORK CHAPTER OFFICERS

*Left to right:* Chairman Elect, Henry Maso; Secretary, Shirley de Ragon; Chairman, Arthur Cohane; Treasurer, Herbert Levetown. The above officers were installed at the November 1962 meeting of the New York Chapter at the Chemists' Club, New York City.

## NEW YORK CHAPTER OFFICERS FOR 1963

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| House | Sam Cohen |
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| Education | Morton Scott |
| Hospitality | Bertram W. Ahrens |
| Inter-Professional Relations | Mitch Schlossman and George Fioto |
| Ex-Officio | Martin Katz |

## NEW YORK CHAPTER
## 1963 PROGRAM SCHEDULE

*January 9, 1963*
The Potential of Enzymes for Topical Application by Dr. T. Cayle

*February 6, 1963*
Makeup by L. Fishbach

*March 6, 1963*
Transparent Emulsions by L. Osipow

*April 3, 1963*
Quantitative Evaluation of Irritating Substances by Dr. L. Kligman

*October 2, 1963*
The Use of the Microscope in Cosmetics by Dr. P. Bartels

*November 6, 1963*
The Application of Polyglycerol and Polyglycerol Esters in Cosmetics by Victor Babayan

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## ELECTED OFFICERS OF I.F.S.C.C.

| | |
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| President | Prof. J. Artigas (Spain) |
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| | Dr. P. Velon (France) |
| | E. Thomsen (Denmark) |

# BOOK REVIEWS

POLYOLEFINES, by A. V. Topchiev and B. A. Krentsel, translated by A. D. Norris. Pergamon Press, Inc., New York 22, N. Y. 1962. 92 pages, illustrated. Price $3.50.

This little book is a translation from the Russian by a British lecturer in physical chemistry. If nothing more, it is interesting to see how a Russian technical book is put together. There are no references, either to the literature or to patents although this field has literally hundreds of each.

One is pleased to learn that a Russian, Gustavson, by name, produced polyethylene in 1884, but it was a liquid of low molecular weight.

In a condensation as this is, the material is quite well put together but with a strong reference to Russian technology.

Polyethylene of the three types, polypropylene, the chlorinated, and sulfo-chlorinated polyethylenes are all discussed. Interesting is the introduction which mentions polymers of branched chain unsaturated hydrocarbons such as isoamylene which gives polymers with melting points as high as 240°C. The book is a useful supplement to Kresser's "Polyethylene."—M. G. DENAVARRE, BEAUTY COUNSELORS, INC.

PHYSICAL PHARMACY, by A. N. Martin. Lea and Febiger, Philadelphia, Pa. 692 pages, illustrated and indexed. Price $15.

The author aims his book at pharmacy students following the new five-year program in pharmacy. However, the present edition does not require a knowledge of physical chemistry or of mathematics beyond the beginning college courses, according to the author.

Twenty-two chapters, starting with a review of mathematical principles through thermodynamics, comprise the book. The mathematical approach to chemistry, particularly slanted to pharmacy, is the dominating method of presentation. Rheology, colloid and interfacial phenomena are given considerable attention. Thirty-three pages are devoted to thermodynamics, sufficient for the student to get to know the fundamentals and some applications.

This is an excellent review book for chemists who do not get too much opportunity to work with higher sciences relating to cosmetic practices and who want to continue to be "brushed up" on the subject.—M. G. DENAVARRE, BEAUTY COUNSELORS, INC.

SYSTEMATIC ANALYSIS OF SURFACE ACTIVE AGENTS, by M. Rosen and H. Goldsmith. Interscience Publishers, Inc., New York 1, N. Y. 422 pages, illustrated and indexed. Price $13.50.

The present work is Volume XII in the series of "Chemical Analysis" which hopes to fill the need for a comprehensive treatise on the analysis of surface-active agents, based on a classification of the title compounds worked out by the authors.

The authors do succeed in their purpose in five chapters and almost forty pages of an appendix. The latter consists of three tables classi-

fying commercially available surfactants along with their properties.

In today's cosmetic laboratory it is becoming increasingly more important to know how much of which surfactant is contained in a product. This book will help you find out.—M. G. deNAVARRE, BEAUTY COUNSELORS, INC.

LIPIDE METABOLISM, edited by Konrad Bloch. John Wiley and Sons, Inc., New York 16, N. Y. 411 pages, illustrated and indexed. Price $10.50.

The present volume is intended to be a companion to Hanahan's "Lipide Chemistry." It deals "with the transformation of lipides by living systems and by isolated enzymes," according to the author's preface. Thirteen contributors have written the eight chapters comprising this work; they have been selected from four different countries, the U. S. A., Canada, Sweden and France.

One of the most interesting chapters is that discussing the chemistry of bacterial lipides. Here one encounters some of the more unusual fatty substances, such as corinnic alcohol, dihydroxyoctadecanoic acid, oleotetracosanopalmitin and phosphatidyl inositodimannoside to name only a few. The chapter on the metabolism and functions of phosphatides is equally well done.

This book supplements practically any existing tome on lipides, regardless of type.—M. G. deNAVARRE, BEAUTY, COUNSELORS INC.

PRACTICAL AND INDUSTRIAL FORMULARY, by M. Freeman. Chemical Publishing Co., Inc., New York 10, N. Y. 1962. 297 pages, indexed. Price $7.95.

One would think that the day of formularies was coming to a close, but it is not, apparently.

The source of the formulas is not given. So one assumes the author has developed them, ranging from adhesives, cosmetics, perfumes, foods, inks, insecticides through wood preservatives. Few chemists are this clever.

The astringent on page 15 seems pretty strong—and oily. Brilliantine No. 1 (page 16) will not be homogeneous unless a particular sulfonated castor oil is used. The bleaching cream on the same page will also need milling to be smooth. The cream mascara on page 23 will smudge easily. Deodorant No. 1 may crystallize in the bottle if indeed the bottle does not explode.

No attention is given to possible patent infringement in the cosmetic section hence it is probably the same in the balance of the book.

This formulary is no worse or no better than others. If you want a lot of formulas for many things and are not satisfied with the one you have, here is another you can buy.—M. G. deNAVARRE, BEAUTY COUNSELORS, INC.

COSMETIC CHEMISTRY FOR DERMATOLOGISTS, by Emil G. Klarmann. Charles C. Thomas Publishers, Springfield, Ill. 1962. 126 pages, indexed. Price $5.75.

This fascinating book is the published record of a lecture series on cosmetic chemistry for dermatologists within the course on industrial dermatoses given by the author for several years at New York University, Post Graduate Medical School.

It was this reviewer's intent to read a chapter at a time, take notes and prepare this review. But this was not possible, for one finds it difficult to put the book down once you start reading it.

Most books require the name of the author on the cover. This book does not. For starting with the

dedication "To Piccina" and the word "didactic" on line 14 of the first page of the text, the book is obviously authored by no one but Emil Klarmann. One can literally hear him deliver every word.

Others require hundreds of pages or multiple volumes to tell their story, yet all this is boiled down in a manner dramatically smooth in the present text. Author Klarmann introduces his text with a quick résumé of history, sales, safety, rationale of formulation, legal aspects of labeling and advertising, color additives and practical use of cosmetics. In the author's introductory remarks it is stated "the several formulas given in the text may not always represents the ultimate in technical refinement; however, they are not only workable, but also more directly illustrative of the formulation principles involved, precisely because of their simplicity."

Eighteen chapters equivalent to the same number of lectures, comprise this monograph. Although aimed at the dermatologist, there will be many others who can profit from this work. Cosmetic executives, students with term papers, beginners in the cosmetic industry, medical practitioners, beauty editors, copy writers, suppliers and pharmacy teachers can all find the world's oldest art and science explained in readily understandable language.

The author treads with uncommon sure-footedness in the areas of hormone cosmetics and cutaneous reactions from cosmetics. These subjects in particular, to this reviewer's mind, are discussed with deep understanding and tactfulness not usually seen in cosmetic writing. Subjects as artful as perfumery and as scientifically involved as hair dyes or hair waving are discussed with masterly facility.

It is proper that this work at last is found in print under the author's sole name. For Author Klarmann has contributed to many joint publications during his carrer, but this is his heart's love, under his sole authorship.

It is a good book. Anyone interested in cosmetics in any way will find it a valuable and useful reference.—M. G. deNAVARRE, BEAUTY COUNSELORS, INC.

PHARMACEUTICAL MANUFACTURERS U. S. A. Noyes Development Corp., Pearl River, N. Y. 1962. 61 pages. $12.00.

This is a guide to pharmaceutical producers in the United States, Canada, United Kingdom and India.

The 500 major pharmaceutical manufacturers in the United States are listed with the following information: name, address, ownership, principal executives, subsidiaries, plant locations, products, annual sales and number of employees. Distributors, wholesalers and repackagers are not included.

Also included are 200 Canadian, 200 British and 125 Indian pharmaceutical manufacturers.—M. G. deNAVARRE, BEAUTY COUNSELORS, INC.

RADIOACTIVITY FOR PHARMACEUTICAL AND ALLIED RESEARCH LABORATORIES, edited by A. Edelmann. Academic Press, Inc., New York 3, N. Y. 171 pages, illustrated and indexed. Price $6.00.

This book is the published version of a symposium sponsored by Nuclear Science and Engineering Corporation. As a result, there are no chapters. However eleven contributions comprise the text.

Of particular interest to this industry is Nelson's paper entitled "Use of Radioisotopes in Soap, Detergent and Cosmetic Research."

This author has presented a good and what appears to be, a thorough survey of work done to date. It would be more meaningful if the word "many" was used less often.

Bogner's contribution "Product Development and Product Evaluation—Areas of Radioisotope Applications," is unfortunately too brief. For example, only 18 lines (in a small book) are devoted to percutaneous absorption.

The main weakness of this otherwise well presented publication of a symposium is the brevity of a number of the contributions. Perhaps the sponsors of this symposium will hold another one bringing the material up to date and overcoming any faults in the previous one.—M. G. deNAVARRE, BEAUTY COUNSELORS, INC.

# THE 1964 I.F.S.C.C CONGRESS

Dear Members:

The International Federation of Societies of Cosmetic Chemists has held two Congresses in the past for the members of its affiliated societies. The first such meeting was held in Munich in 1960, the second in London in 1962. The United States Society of Cosmetic Chemists will have the honor and privilege of being the host society for the third Congress, which will be held on the campus of Columbia University in New York City during the week of June 21, 1964. The S.C.C. was started in 1945 in the United States with twelve charter members. It has grown to the point where it now has 930 active members, while the I.F.S.C.C., which was organized in Brussels in 1959, has a total membership of approximately 2000 cosmetic chemists affiliated with societies chartered in eleven countries.

New York City was chosen for the 1964 Congress because the World's Fair will be held there at that time and will no doubt be a factor in inducing chemists from the U. S. and from all over the world to participate in the Congress. The World's Fair officials are honoring us by designating June 25, 1964, as Cosmetic Chemists Day. Columbia University, a school of world-wide reputation, located in the heart of New York City, has really gone overboard to make it possible for us to use their campus, hotel facilities and meeting rooms during the week of June 21, 1964, at extremely reasonable rates.

## Purpose of Congress

The primary purpose of a 1964 I.F.S.C.C. Congress is scientific. It is designed to advance the science of cosmetics and the prestige of the cosmetic chemist as a scientist throughout the world. To accomplish this end and to effect an exchange of scientific ideas which will benefit all, we must do everything possible to induce as many members as possible to participate. We are looking for a large attendance from overseas as well as from the U. S., Canada and Mexico.

## Technical Meetings

The scientific aspect of the Congress will consist of seminars held on four different subjects, each occupying a half day. All will be morning sessions, from Tuesday through Friday. We have learned from the two

previous congresses, held in Munich and London, that half-day seminars allowed visiting members more time to participate in other activities.

Cosmetic chemists everywhere are invited to submit papers on scientific subjects which might be of interest to the research, development and manufacturing of cosmetics. Although the scientific fields which will be covered have yet to be decided upon, suggestions are invited. Submission of papers should be made as soon as possible to allow for sufficient time to select the best papers for presentation at the Congress.

In order to guarantee the international character of the meeting and to establish the sound basic scientific atmosphere in which the Congress will operate, well-known scientists, authorities in their fields which will be related to cosmetic science, will be invited to present papers before the I.F.S.C.C. A fund is being raised to pay for the basic expenses of such invited speakers who will be honoring us by their attendance.

## Accommodations

Considering how costly and scarce accommodations will be during the period of the World's Fair in 1964, we are indeed fortunate to have the splendid cooperation of Columbia University and the use of the facilities on its campus. Ferris Booth Hall, in which we will have our four seminars, has a seating capacity of 750 for lectures and 500 for banquets. It is air-conditioned and only two years old. It is of the most modern accoustical design, having built-in facilities for all types of films and slides. The lounge is available to us for receptions and cocktail hours. New Hall, the newest residence hall on the campus, is built like a "hotel" and will be operated like one for a large conference such as ours.

Our Congress will be held midway between the regular college year and the summer sessions. Nevertheless, we can count on ample dining facilities during the week of June 21, 1964. All of our meals will be in campus cafeterias, with the exception of the banquet which will be in air-conditioned Ferris Booth Hall. With 2000 members in all I.F.S.C.C. affiliated societies, we expect to use the facilities at Columbia University to the fullest!

## Fees

Those of us who were fortunate enough to attend the Munich and London conferences and to have visited cosmetic societies in different parts of the world have been impressed by the fact that cosmetic chemists, outside of the United States, earn comparatively low salaries in terms of the U. S. dollar. Not many of them can afford the trip and other necessary expenses which will be incurred while attending the New York Congress. For this reason, our Board of Directors is undertaking a substantial fund-raising campaign within the two-billion-dollar U. S. cosmetic industry to render

financial assistance to members of affiliates of the I.F.S.C.C. other than the U. S. Society. It is hoped that sufficient funds will be raised to allow these members a free ticket to all official functions plus room and board at Columbia University for six days. It is only by this type of activity that we can hope to make the Congress a truly International one. We all must interest our companies in supporting this effort which, in the long run, will benefit both the cosmetic chemists and the cosmetic industry by advancing our science to a new level of competence.

REGISTRATION FEES

| | Package Deal Note (1) | | Meetings, etc. Note (2) |
|---|---|---|---|
| Members of S.C.C.-U.S.A. | $ 80.00 | per person | $35.00 |
| Members of other Societies affiliated with the I.F.S.C.C. | Free Note (3) | | Free Note (3) |
| Members of a registrant's family | 80.00 | " " | 35.00 |
| Nonmembers | 100.00 | " " | 55.00 |
| Children under 12 | 60.00 | " " | Excluded |
| Extended Stay at Columbia University | Note (4) | | ... |

*Notes:*

(1) The package deal includes room from Sunday evening, June 21, through breakfast Saturday, June 27; all meals at Columbia; banquet and cocktail party; and all official functions and meetings in connection with the seminars. The "Package Deals" are for parties of three or four in a suite of two rooms (each with two beds) and one bath. Separate keys are provided for each room. The bath is off the hallway entrance to the two rooms. There is a telephone in this hallway serving the two rooms. All rooms are in New Hall, which is adjacent to Ferris Booth Auditorium.

The cost per person is the same, whether four or three people occupy the suite. If only two people occupy it, the cost of the "Package" will be $10 more per person. Entire suites will be reserved only for two or more people. What few "singles" are available must be held for invited speakers.

(2) Meetings, etc., includes all official functions in connection with the seminars, banquets and cocktail party; also luncheons at Columbia following each seminar session.

(3) The Package Deal and meetings are free to members of societies affiliated with the I.F.S.C.C., other than U.S.A. members, provided reservations are made before June, *1963*. After June, *1963*, they will only be accepted provided funds are still available for this purpose. Early registrants will be certain of receiving this benefit which applies to members only—not their families. All registrants are expected to pay a deposit of $25 (or its equivalent in foreign currency) each when making reservations. This deposit, which will be returned when they arrive at the Congress in 1964, is necessary to guarantee that the members will use the accommodations which we must purchase for them.

(4) Anyone may make arrangements to arrive at Columbia University as early as June 15, 1964, and/or remain as late as July 3, 1964. For the cost of such additional accommodations beyond our basic six-day "Package," please consult our Arrangements Chairman, Walter Wynne.

## PRIORITY

We have reserved rooms for 280 at Columbia University, and these rooms will be assigned, upon receipt of a deposit of $25 per person, in the following order of priority until June 15, *1963:*

1. Members of societies affiliated with the I.F.S.C.C., other than U. S. members.
2. U.S.A. members of S.C.C.
3. Nonmembers

Because of the Teachers' Conference being held at Columbia University at the same time as our Congress in 1964, it is necessary for us to get our reservations in before June, *1963*, in order to obtain more rooms than the 280 now held for us. First come, first served!

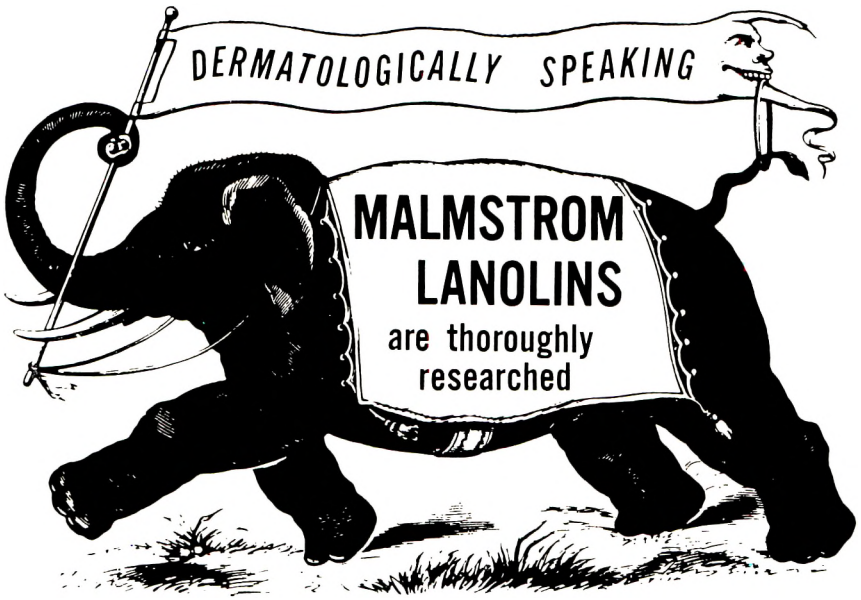
## Budget

To accomplish our objective of making the 1964 Congress a scientific success, we must raise at least $25,000. Approximately 80 per cent of this is required to underwrite the expenses of invited speakers and to give some financial assistance to members of visiting societies. We plan to have sixteen speakers, of whom we would like to invite at least half from abroad. We have been guided by Mrs. Eunice Miner, Executive Director of the New York Academy of Sciences, in preparing our budget, based on the Academy's experiences with many similar scientific conferences. We must look to manufacturers of cosmetics and raw materials suppliers to underwrite a substantial part of our budget. Plans are already under way for raising this money, with one of our former presidents, George Kolar, heading the fund-raising committee.

## Reservations

To assist us in the organization of the meeting, it is essential that an estimate be made of the number of members, their families and friends who will attend. While it is appreciated that some members may not be able to give a firm commitment so far ahead, they are, nevertheless, asked to send the following information if they have a reasonable expectation of participating: number of adults and number of children in party and number of rooms desired. Please write to the Arrangements Chairman, Walter Wynne, Room 700, 321 West 44th Street, New York 36, N. Y., if there is any possibility that you will attend the 1964 I.F.S.C.C. Congress. Checks should be made payable to the Society of Cosmetic Chemists.

Robert A. Kramer
Chairman—International Affairs

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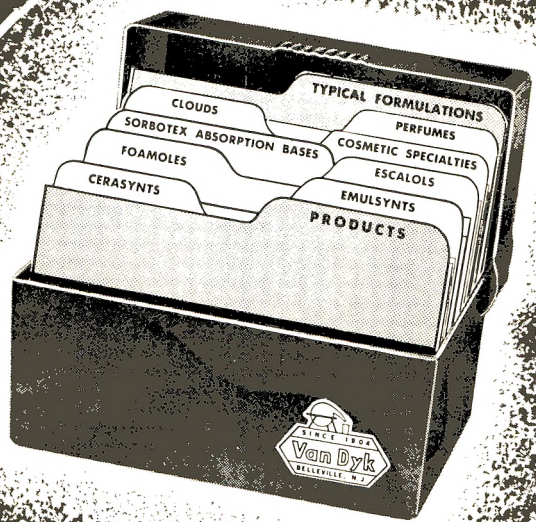


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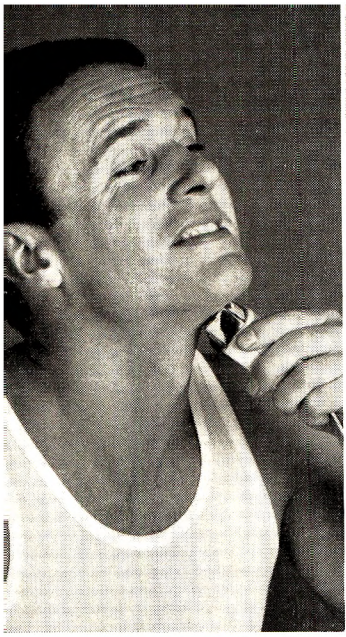

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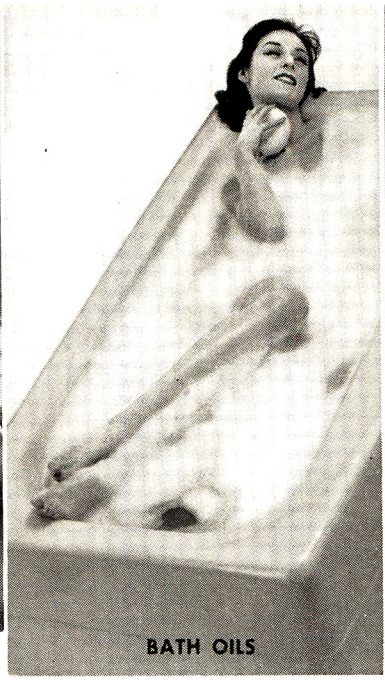

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## CORRECTION

THE FOLLOWING index was omitted from the Volume XIII, 1962 index which appeared in the December issue (No. 9). These articles were originally published in the May issue (No. 4).

# AUTHOR INDEX



# SUBJECT INDEX

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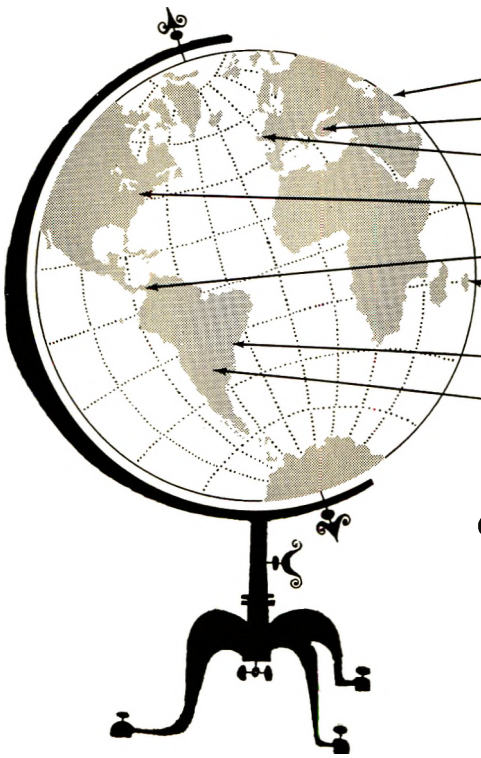

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