XI. SYNOPSIS OF PHARMACO-CHEMICAL AND PHARMACO-PHARMACOLOGIC INTERRELATIONS

In analyzing the mechanisms through which hormones can influence resistance, we must consider first the various forms of drug interactions in general and second the structural prerequisites for the protective actions of hormones. It is obvious that drugs and hormones may interact by influencing each other's absorption, excretion, distribution within the organism, metabolism, as well as by direct chemical or pharmacologic interactions that occur between drugs in vivo. However, these mechanisms are the subject of general pharmacology and hence they will only receive cursory attention here.

The principal objective of this section is to correlate the many experiments which we performed to obtain material for a glimpse into the structural (chemical) or pharmacologic prerequisites for the ability of steroids to protect the body against toxicants.

VARIOUS FORMS OF DRUG INTERACTIONS

In discussing the various forms of interactions between drugs (including hormones), several mechanisms must be considered, for example:

1. The gastrointestinal **absorption** of one drug can be affected by another which alters the pH of the alimentary tract, the permeability of its epithelium or peristalsis.

2. Drugs may affect the renal, biliary, or intestinal **excretion** of other drugs.

3. One drug may alter the **distribution** of another drug within the organism, e.g.:

a) by altering the permeability of the hemato-encephalic barrier;

b) by competing for shared protein-binding sites in the plasma, thus augmenting the biologic activity of the displaced drug;

c) by competing for receptor sites in tissues, thus diminishing the activity of the displaced drug.

4. Drugs may affect the synthesis, degradation or activity of microsomal and extramicrosomal **drug-metabolizing enzymes.**

5. Direct **chemical interactions between drugs** (e.g., chelation) may lead to their inactivation in vivo. Addition of a pharmacologically inert radical to a potent drug may also alter the susceptibility of the latter to microsomal enzyme activity (e.g., morphine and barbital are resistant, whereas ethylmorphine and phenobarbital are highly susceptible to degradation by microsomal enzymes). Such an "opsonization" by addition of a radical may increase the liposolubility of a drug and hence, its capacity to penetrate through membranes. This type of synthesis has not been definitely proven to occur in vivo, but it may play a role in drug interactions.

6. Direct **pharmacologic interactions between drugs** (e.g., between a vasoconstrictor and a vasodilator, or between two vasodilators) may lead to an increase or a decrease in their activity.

H. Selye, *Hormones and Resistance*

Drug Interactions in General

Fouts G77,566/64: Review (5 pp., 18 refs.) on the effect of drugs upon drug metabolism.

McIver G78,391/65: Review (4 pp., 43 refs.) on drug incompatibilities.

Remmer G78,385/65: Review (24 pp., 156 refs.) on the mechanism of drug interactions.

Gillette G78,390/67: Review (19 pp., 96 refs.) on theoretical aspects of drug interaction.

McIver G77,708/67: Review (6 pp., 41 refs.) on drug incompatibilities.

Block & Lamy G81,054/68: Review (6 pp., 82 refs.) on therapeutic incompatibilities of importance to pharmacists.

Hartshorn G78,178/68: Greatly simplified but excellent revue (8 pp., about 35 refs.) enumerating various ways in which drugs can interact and illustrating each type with a few examples.

Hartshorn G81,056/68: Review (4 pp., 17 refs.) with practical hints on drug interactions useful to the pharmacist.

Hartshorn G81,057/68: Review (7 pp., 3 refs.) consisting mainly of tables concerning drug interactions.

Hussar G81,059/68: Review (11 pp., 82 refs.) on drug incompatibilities.

Pelissier & Burgee, Jr. G79,655/68: A brief guide to drug incompatibilities irrespective of the underlying mechanism.

Block & Lamy G81,052/69: Review (5 pp., 27 refs.) on drug interactions with tabular summaries on changes in metabolism, binding to plasma and urinary pH changes produced by one substance which may alter the activity of another compound.

Dunphy G77,712/69: Review (11 pp., about 30 refs.) on drug interactions with an outline of an extremely simplified index listing a few examples of these.

Hartshorn G81,055/69: Review (7 pp., 33 refs.) on interactions among anti-infective agents.

Hussar G81,053/69: Practical hints concerning drug interactions of interest to the pharmacist.

Visconti G77,709/69: Review (10 pp., no refs.) on how drug interaction information may be used preventively.

Ariëns G79,943/70: Review (48 pp., about 160 refs.) on drug interactions with special reference to antagonisms, metabolic inactivation and metabolic activation. [Excellent summary, but the poor English is difficult to follow (H.S.).]

Neumann G80,018/70; G80,550/70: Review (5 pp., no refs.) on drug interactions in the style of a postgraduate lecture.

Mannering G74,881/71: Review of the literature on drug interactions in general leads to the conclusion that "a drug may alter the action of another drug in several ways: a) by producing one or more effects similar or opposite to those of the drug in question, b) by direct chemical or physical interaction of drugs, c) by displacement of drugs bound to plasma or other proteins, d) by altered renal clearance of drugs, e) through conditioning by previous drug effects, f) by interaction of drugs with receptor sites, and g) by inhibition or stimulation of the metabolic site. Examples of all of these interactions are given . . ."

Lipid Solubility

Brodie G55,013/62: "Metabolism of the microsomal enzymes seems to be governed by lipid solubility, for only substances that are highly lipid soluble are metabolized by microsomes."

Mannering G71,818/68 (p. 74): A survey of the literature shows that compounds with low lipid solubility are poor inducers of drug-metabolizing microsomal enzymes, whereas potent inducers are, in general, highly soluble in organic solvents. [Yet all steroids are lipid soluble but are not good inducers (H.S.).]

Carriers

Baird & Reid G51,827/67: Pancuronium bromide produces neuromuscular blockade in man.

Lewis et al. F95,691/67: In various test procedures, steroidal monoquaternary ammonium salts exhibited non-depolarizing neuromuscular blocking activity.

Bonta & Goorissen G75,052/68; Buckett G75, 531/68: Pharmacologic studies on the neuromuscular-blocking effect of pancuronium.

Buckett et al. G56,175/68: "Pancuronium possesses up to ten times the potency of tubocurarine according to the species used for testing, while possessing similar duration of action."

Reyes et al. G71,233/69: In rats phenobarbital "enhanced hepatic uptake of an organic anion, bromsulphalein, in vivo and simultaneously increased the amount of Y, a hepatic cytoplasmic organic anion-binding protein. This study supports the postulate that Y is a major determinant in the selective hepatic uptake of certain organic anions from

plasma." Induction of Y may enhance hepatic uptake and metabolism of various substrates following treatment with phenobarbital and other inducers.

Wall et al. G69,969/69: A series of steroid esters of p-[N,N-bis(2-chloroethyl)amino] phenylacetic acid (BCAPAA), steroidal sulfides of p-(N,N-bis-2-chloroethylamino)thiophenol, and a variety of steroidal ethylenimine derivatives were synthesized and tested for antitumor activity. "Activity was found only in those instances in which the steroid and potential oncolytic agent were connected by ester or heterocyclic ether linkages. The steroidal BCAPAA esters were of particular interest showing excellent inhibition of a DMBA-induced and transplantable mammary adenocarcinoma, and marked increase in survival when tested on a variety of rat leukemias. . . . The steroidal BCAPAA esters were judged to be less toxic than some of the well-known nitrogen mustards in general use."

Chaouki et al. G77,223/70: In 50 patients, pancuronium bromide was found to be an active non-depolarizing neuromuscular blocking agent, about 5 times as active as curare and 25 times more potent than gallamine triethiodide. Its advantages are rapid onset of action, no release of histamine and little disturbance in blood pressure, because of a weak ganglioplegic action.

Dick et al. G77,777/70: Favorable clinical experiences with pancuronium as a muscle relaxant in anesthetized patients.

Feldman & Tyrrell G76,666/70: Dacuronium bromide (2β, 16β-dipiperidino-5x-androstane-17β-diol-3x-acetate dimethobromide) is closely related to pancuronium bromide but the acetoxy group at C_{17} is replaced by a hydroxyl group. It has proven its usefulness as a non-depolarizing muscle relaxant in man. Its effect is fully reversible by edrophonium or neostigmine.

PREREQUISITES FOR THE PROTECTIVE ACTIONS OF HORMONES

Looking back upon research on hormones and resistance, as outlined in this monograph, it may be instructive to reexamine the justification of the path followed and to summarize the main points.

It is not without hesitation that we embarked upon this project some 35 years ago; we realized, to start with, that this would be a life-long undertaking with virtually no background data for logical planning. On the basis of what we had learned in 1936 about the role of the adrenal cortex in defense against stress, no other course seemed to be open to us but that of a purely empirical, large-scale screening of many steroids (more or less closely related to the corticoids) for possible protective effects against many toxicants.

However, the possibility of finding highly potent and comparatively nontoxic protective steroids appeared to hold considerable promise of practical applicability and the screening did not have to rely on chance alone. To some extent, we could be guided by the pharmacologic and chemical characteristics of compounds previously shown to have protective potency against certain substrates. We used the same guide lines for the identification of toxicants amenable to prophylaxis by steroids. It is on the basis of this kind of empirical research that we are now beginning to see outlines of a classification which permits us to predict, with some degree of probability, what compounds are likely to possess protective effects against what types of toxicants.

Because of the large number of experiments required to explore the many possible combinations of such interactions, we had to rely on simple in vivo observations in which directly visible (functional or structural) changes and mortality rates were our principal indices of activity. Yet, in the early days we were encouraged by the knowledge that similar screening efforts did prove to be eminently successful in many other fields. The classification of bacteria on the basis of their ability to grow on

certain media or to take up the Gram stain, the screening of antibiotics on plates inoculated with various bacteria, the blind testing of 606 chemotherapeutic agents that finally led Ehrlich to the discovery of "Salvarsan," are but a few examples to illustrate this point.

In our own work we could demonstrate the nonspecificity of the pituitary-adrenal response only by countless in vivo tests with many stressors; only the screening of numerous calciphylactic sensitizers and challengers permitted us eventually to induce localized tissue calcification in a predictable and highly specific manner.

Naturally, as soon as any new protective phenomenon was discovered, in vitro studies in depth became necessary to clarify the underlying mechanisms; for example, after we noted the prevention by spironolactone of digitoxin and mercurial intoxication, or the extraordinary degree and spectrum of protective effects that can be induced in steroids by the introduction of a nitrile group. Much of this work is still to be done, but before we could even think about elucidating the manner in which a protective phenomenon works, we had to know first that the phenomenon exists.

So much for self-justification.

To begin with, we had to develop an economic procedure which permitted large scale experimentation with a minimum waste of steroids, many of which are difficult to synthesize. Then we had to design a technique for the correlative evaluation of the many individual observations made.

Our assay procedure consisted of the following three steps:

1. Establishment of possible protective potency against two standard toxicants: digitoxin and indomethacin.

2. Determination of the "protective spectrum" of the steroids found to be active in step 1.

3. Identification of damaging agents amenable to prophylaxis by steroids which have revealed interesting activities in step 2.

Before describing these steps in detail, we shall have to say a few words about the evaluation of the data obtained. The results of the **first screening** have been tabulated in the conventional way (Table 135) by listing the mean severity of the actual changes as previously described for comparable studies on the effect of 304 steroids upon indomethacin and digitoxin poisoning (*Selye G70,421/70*).

In addition, we constructed **Synoptic Tables** (Tables 136, 137, 138) in which these and many additional results are summarized on the basis of the degree of the significance ratings (*cf.* "Statistical Evaluation," p. IX) according to a system developed by Mrs. I. Mécs of this Institute. The figures indicate the means of the statistical significance grades of the changes (functional or structural) used as indicators, plus that of the mortality rates divided by 2. Thus, in an experiment in which the protection against intestinal ulcers had a significance rating of "0" (no protection), and the significance of the protection against mortality was 3 "***" (perfect protection), the figure given in the tables would be 1.5. Only in the case of compounds which normally cause no mortality (e.g., anesthetics, muscle relaxants) do the grades correspond to structural or functional lesions alone.

The Synoptic Tables (136, 137, 138) also list the **"Mean Overall Protective Index"** computed according to a procedure closely related to the "Simplified Activity Grading" system previously described (*Selye G70,421/70*). This index represents the allgebraic sum of all the individual activity gradings for a certain protective substance divided by the number of toxicants against which it was tested.

In addition, we computed the **"Total Overall Protective Index"** which represents the arithmetic sum of all the individual activity gradings (disregarding negative grades).

Furthermore, we computed the **"Protective Spectrum Index"** which is the percentage of

those toxicants tested against which significant protection is obtained (irrespective of the degree of significance). Thus, if a steroid offers significant protection against 6 out of 10 toxicants examined, its "Protective Spectrum Index" is 60%.

In the Synoptic Tables, these Indexes have also been computed for the amenability to protection of the various toxicants (three bottom horizontal lines). In other words, here the figures indicate the mean degree or the percentile frequency of protection offered by the entire series of conditioners against any one toxicant.

Finally, to further facilitate the overview of this complex field we constructed a **"Diagram Table"** (Table 139) which graphically summarizes the highlights of Table 138, except that the results are registered only for one dose level of the toxicants and are expressed in a scale in which intermediates between the four grades are not recognized (for details *cf.* footnote on Table 139). Furthermore, in Table 138, the toxicants are listed in alphabetical order for easy identification, whereas in Table 139, they are enumerated according to decreasing "Total Overall-Protective Index" values.

Since the large number of experiments to be reported here was performed over a considerable period of time, in each case, a group of unpretreated controls received the same toxicant, simultaneously with the rats that had been pretreated with potentially protective substances. The statistical significance of the resulting changes in the pretreated animals was always calculated in comparison with the corresponding group of unpretreated controls handled under identical circumstances, at the same time and by the same technician.

Using these procedures for the evaluation of our data, we examined the steroids available for this work according to the above mentioned three-step procedure.

First Step: Protection Against Digitoxin and Indomethacin

The first systematic investigations designed to identify protective steroids consisted of a series of bioassays in which 304 natural and synthetic steroidal compounds were tested for their ability to protect the rat against digitoxin and indomethacin, under the experimental conditions outlined in the preceding section. Since these results have been the subject of an extensive review *(Selye G70,421/70)*, we shall limit ourselves here to a brief description of the principal conclusions derived from them.

Protective activity was widespread among the steroids of this first series; it was demonstrable among gonanes, estranes, androstanes, androstenes, 5β- and 5α-pregnanes as well as among pregnenes, with one or more double bonds, and with or without halogen substitution in the ring system. On the other hand, cholanes, cholestanes and genins were uniformly inactive, with the sole exception of methylnordeoxycholanate (3α,12α-dihydroxy-24-nor-5β-cholan-23-oic acid methyl ester).

Because of this widespread distribution of anti-indomethacin and antidigitoxin activity throughout various classes of steroids, it was difficult to formulate any clear-cut rules about pharmaco-chemical correlations in this field. It does appear, however, that although catatoxic activity is not strictly dependent upon any single structural prerequisite, in general the 17α-propionic acid-γ-lactone side-chain is advantageous for both antidigitoxin and anti-indomethacin activity. It is perhaps also not purely coincidental that a very large number of active catatoxic steroids is found among the 1,4-androstadienes as well as among halogenated androstene and pregnene derivatives. It is likewise noteworthy that several of the most active catatoxic steroids are 19-nor compounds, hence the angular methyl group at C_{10} is not only dispensable but often advantageous. The most striking observation in this series of tests was that among all 304 steroids tested the most active against both substrates proved to be a cyano-compound, namely PCN.

This first systematic screening series also revealed that the catatoxic activity is not strictly dependent upon any other known pharmacologic property, although most of the highly potent antidigitoxin and anti-indomethacin steroids also exhibit anti-mineralocorticoid or anabolic properties.

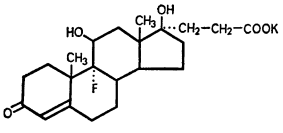
Because of the comparatively small number of animals that could be used for the bioassay of the many, not readily available, steroids, only the lowest and the highest activity grades were given serious consideration. However, even on this rigid basis of appraisal, we found that at a 10 mg dose level, among 304 steroids tested, there were:

Active only against indomethacin: 42
Active only against digitoxin: 32
Active against both substrates: 24
the remainder being inactive or of doubtful activity.

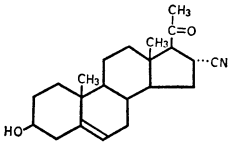
At the 0.5 mg dose level, we found:

Active only against indomethacin: 1
(Cpd. 277 and betamethasone acetate)
Active only against digitoxin: 1
(CS-1)
Active against both substrates: 1
(PCN)

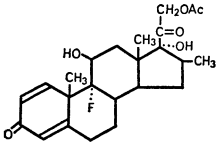
These compounds correspond to the following structures:



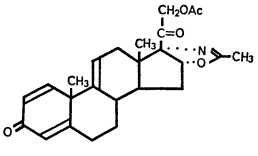
CS-1
9α-Fluoro-11β-,17-dihydroxy-3-oxo-4-androstene-17α-propionic acid potassium salt (SC-11927)



PCN
3β-Hydroxy-2-oxo-5-pregnene-16α-carbonitrile (SC-4674)



Betamethasone acetate
9α-Fluoro-16β-methyl-11β,17,21-trihydroxy-1,4-pregnadiene-3,20-dione 21-acetate



"Cpd. 277"
21-Hydroxy-3-oxo-1,4,9(11)-pregnatrieno-[17α,16α-d]-2'-methyl-oxazoline acetate

It is especially noteworthy that several of the active catatoxic steroids are naturally occurring hormones, hormone precursors or hormone metabolites such as: progesterone, 17α-hydroxyprogesterone, 5-pregnenolone, dehydroisoandrosterone.

Encouraged by these first observations, we then proceeded to repeat some of the key observations at lower dose levels. We also performed similar tests on many additional steroids, especially carbonitriles and other compounds related to the most active members in the preliminary series. This work (Selye G 70,480/71) was done under experimental conditions exactly corresponding to those of the first screening tests, and the results are summarized in Table 135.

However, before describing these most recent experiments, we must say a few words about the **SSS steroid terminology.** Up to now, in discussing the literature, we used essentially the same terms as did the authors quoted. This was far from satisfactory, but unfortunately unavoidable, because we could not have taken the responsibility of translating into any accepted steroid nomenclature the often ambiguous mixtures of trivial and systematic designations that are sometimes employed even by the most reputable pharmacologists and clinicians. However, in the following description of our own experiments with fully characterized compounds, these will be identified not only by their structure formulas and the official designations accepted by the International Union for Pure and Applied Chemistry (IUPAC Commission G82,068/69), but also by the much more easily understandable and simpler SSS terms. The SSS (Symbolic Shorthand System of steroid terminology) (Selye et al. G79,034/71) can be briefly summarized as follows:

Basic Principles

The main advantages of the SSS nomenclature are: **shortness** of code terms, **logical consistency,** and **simplicity.** The principles which guided its design are:

Rigid observance of left-to-right precedence order in the coining of symbols. The parent hydrocarbon comes first, then its substituents in a strictly determined sequence. Each substituent is named by a symbol followed by a positional number according to the principle of: "first what, then where" (e.g., 17β-Hydroxyandrostan-3-one = $\mathbf{AOl}_{17\beta}\mathbf{on}_3$).

We deviate from this rule only in the case of "secondary parent hydrocarbons" in which the main parent hydrocarbons (e.g., estrane, androstane) are provided with side-chains attached by C—C linkages (e.g., methylandrostane or cyanopregnane). In such cases, substituents of the side-chains are mentioned immediately after the latter. E.g., an androstane with a hydroxylated (ol) methyl group (I) attached at 16β is written $\mathbf{AIol}_{16\beta}$ and the whole expression is underlined (or boldface) to distinguish it from substituents on the main parent hydrocarbon itself which follow according to the order of precedence described below. For example, if the methylandrostane just mentioned also contains a ketone group at 3 and a fluorine in the 9α-position, we write $\mathbf{AIol}_{16\beta}\mathbf{on}_3\mathbf{F}_{9\alpha}$.

No signs to indicate two or more identical characteristics (e.g., diene, triol, dione). The sign Δ and the suffix "-ene" are also omitted since appropriate position numbers make them superfluous. After the symbol of the parent hydrocarbon (e.g., **A** = Androstane) superscript numerals suffice to designate the degree of unsaturation, while subscripts qualify the position and number of functional groups (e.g.: $\mathbf{A}^{4,6}\mathbf{ol}_{11\beta,17\alpha}\mathbf{on}_3$ = 11β,17α-Dihydroxy-4,6-androstadien-3-one).

No silent "e" (e.g., in "one"). This is always omitted, not only when the next syllable begins with a vowel. The final "e" does not change either the meaning or the pronunciation of the syllable and, in many languages, it is not used in any case. Besides, SSS (like IUPAC) is not meant for oral communication.

Since esters are always formed with alcohols, the **esterified OH groups are not separately indicated.** E.g., instead of 5α-Androstane-3β,17α-diol 3-acetate 17-propionic acid ester, we merely write $\mathbf{A5\alpha{*}II''}_{3\beta}\mathbf{{*}III''}_{17\alpha}$ (* = ester; '' = acid), since the 3β-acetate or 17α-propionate can only be formed with the corresponding alcohols (*cf. also* p. 775 and 776). Furthermore, we employ short symbols instead of writing out the full names of the esterified acids and, instead of the usual "-", we separate ester functions from the parent hydrocarbon by an asterisk which uses no more space than the dash and yet further characterizes the following symbols as an ester.

No prefixes. It is superfluous to burden a codifier with a complex dictionary of rules according to which he must write a given symbol as a prefix or suffix. In SSS, the parent hydrocarbon is the first part of any designation; all other symbols are added as suffixes.

No synonyms. Every structural feature receives one name or symbol. This procedure is greatly facilitated by the omission of prefixes which complicate other systems. For example in IUPAC:

Carboxylic acids are named "Carboxy-" as prefix, and "-oic", "-ic" or "carboxylic acid" as suffix.

Alcohols are "Hydroxy-" as prefix and "-ol" as suffix.

Aldehydes are "Oxo-" as prefix and "-al" as suffix.

Most confusingly, ketones are also "Oxo-" as prefix, but "-one" as suffix.

Typesetting and punctuation are used to specify chemical characteristics without lengthening names. Instead of separating all parts of a term by dashes (-), SSS uses no separating sign or (where there may be ambiguity) the shorter comma (,). The latter is replaced by an asterisk (*) to indicate ester function. Commas also separate the position numerals of identical functions (e.g., $ol_{3\beta,9\beta,17\alpha}$). No punctuation is used to separate the constituents of a single functional group. E.g., *III″$_{3\beta}$, fully characterizes the location on C_3 and the steric position of an alcohol group esterified with propionic acid; the three ingredients (3, β, III″) need not be separated by dashes or any other sign which would only tend to break up what is actually one unit of expression.

The simplest symbolic code terms are used for the most common expressions. Thus, the main parent hydrocarbons have symbols consisting only of the first letter of their full name (e.g., **G** = Gonane, **E** = Estrane, **A** = Androstane) although, in some instances, one or two additional letters are added to avoid ambiguity (e.g., **CH** = Cholane, **CHT** = Cholestane, etc.). For emphasis, the symbols of the main and secondary parent hydrocarbons — including signs of intrinsic modifications such as: isomerism, unsaturation, nor, homo, abeo — are underlined and always capitalized (in printing, italicized or set in boldface).

Whenever possible, simple, selfexplanatory **mnemonic abbreviations** are used for all substituent radicals, e.g., for normal alkyls roman numerals, indicating the number of carbon atoms contained:

I = Methyl II = Ethyl III = Propyl
IV = Butyl V = Pentyl.

Longer and more complex radicals are represented by letter symbols, usually contractions of the IUPAC names (*cf.* below).

When **alkyls appear as side chains** (secondary parent hydrocarbons), they follow the name of the parent hydrocarbon (on the same line) and, like the latter, are boldface (e.g., **AI** = methylandrostane).

Whenever the chemical formula characterizing a substituent is shorter than a meaningful corresponding word or abbreviation could be, we use the former (e.g., $=CH_2$, $=NH$, $-CN$, K, F respectively for methylene, imino, carbonitrile, potassium or fluorine). In order to avoid three-line symbols, the positional numbers of such formulas as $=CH_2$ are appended to a bracketed symbol e.g. $(CH_2)_{16\beta}$.

The Main Parent Hydrocarbons (in order of precedence)

Gonane	= **G**	Ergostane	= **ER**
Estrane	= **E**	Stigmastan	= **ST**
Androstane	= **A**	Cardanolide	= **CAR**
Pregnane	= **P**	Bufanolide	= **BUF**
Cholane	= **CH**	Spirostane	= **SP**
Cholestane	= **CHT**	Other nuclei	

All ring modifications, other than those recognized above as parent hydrocarbons, are listed as derivatives at the end of each main category in this order:

Heterocyclic steroids, *cf.* p. 777
Nor-ring steroids = **n** (e.g., **AnA** = A-nor-androstane, **PnB** = B-nor-pregnane).
Homo = **ho** (e.g., PhoD=D-homo-pregnane)
Abeo = **ab**
Cyclo = **cyc**
Retro = **ret**
Seco = **sec**

Derivatives (in order of precedence)

With each parent hydrocarbon, its immediate derivatives are classified in the following order:

Isomerism: Isomerism at C_5 is indicated by **5α** or **5β** following the symbol of the parent hydrocarbon (e.g., **A5α** = 5α-androstane). Isomers are listed next to each other, α before β. For ring-isomers, *cf.* below.

Unsaturation: —C—C— (an), e.g., **A** (for Androstane),

—C=C— (en), e.g., A^4 (for Androst-4-ene), $A^{4,6}$ (for Androsta-4,6-diene).

The number of unsaturations is not indicated as it is evident from the positional number(s) in the superscript. Thus, $A^{4,6}$ suffices to indicate that we are dealing with a diene. [Not $\Delta^{1,2,etc.}$]. Cyclosteroids are also identified by superscripts, e.g., $A^{3(5)}$. The superscript numbers of triple bonds (-yn) are repeated (e.g., P^{20-20}).

Alcohols: —OH (ol), e.g., $Aol_{17\alpha}$ (for Androstan-17α-ol),

$Aol_{3\beta,17\alpha}$ (for Androstane-3β,17α-diol). [Not hydroxy, dihydroxy, etc.]

Esters: —CO[OHH]OC— (*), e.g., $P^*Bz''_{3\alpha}{}^*II''_{17\beta}$ (for 3α,17β-Dihydroxypregnane 3-benzoate 17-acetate).

Esters are identified by an asterisk preceding the symbol of the acid, with which the steroid hydroxy group is esterified; they are listed immediately following the corresponding free alcohols. Since ester formation is possible only with alcohol groups, it is redundant to identify the position of each (the alcohol and acid) separately (*cf. also* below). [Not oxy (e.g., acetoxy) or "acid ester" (e.g., acetic acid ester)]. Esters of steroid acids are distinguished by placing the symbol for acid (″) immediately after that of the steroid, before the asterisk (e.g., $P''{}^*IIol_{21}$ for 21-pregnanoic acid esterified with ethanol).

Ketones: C=O (-on), e.g., $E^{1,3,5(10)}ol_3on_{17\beta}$ (for 3-Hydroxy-1,3,5(10)-estratrien-17-one or estrone). [Not oxo, keto, one (with terminal "e") and the redundant indication of the number of ketone groups by dione, trione, etc.]

Carboxylic acids: —COOH (″), e.g., P''_{21} (for Pregnan-21-oic acid). Here the carboxyl group itself is considered to be part of the steroid skeleton, not an oxidized methyl side-chain on 21-norpregnane. However, a carboxyl which is not part of a recognized main parent hydrocarbon, but is attached to it by a C—C linkage, is written as an oxidized methyl side-chain (e.g., $AI''_{16\alpha}$ for androstane 16α-carboxylic acid). Carboxylic acids not attached to the steroid skeleton by C—C linkages, e.g., those of esters, are similarly identified (by ″), e.g., I″ = formic, III″ = propionic, V″ = pentanoic. Bz″ = benzoic acid (*cf.* p. 778) for alkane symbols). [Not carboxylic acid or -oic acid.]

Steroid acid esterified (e.g., $P''{}^*IIIol_{16\alpha}$).

Steroid alcohol esterified (e.g., $P^*III''_{16\alpha}$).

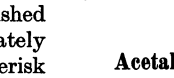
Ethers: —CO[HHO]C— (θ), e.g., θI, θII, θPIII (for methyl, ethyl or propyl ethers). [Not methoxy or methyl ether.]

Lactones:  (-lac, -γlac), e.g., $A^4III''\gamma lac_{17\alpha}ol_{11\beta,17}on_3F_{9\alpha}$ (for 9α-Fluoro-11β,17-dihydroxy-3-oxo-4-androstene-17α-propionic acid γ-lactone).

Aldehydes: —CHO (-al), e.g., $P^4ol_{11\beta,21}on_{3,20}al_{18}$ (for 11β,21-Dihydroxy-3,20-dioxo-4-pregnen-18-al or aldosterone). As with the carboxylic acids, here the carbon bearing the aldehyde function is considered part of the parent hydrocarbon and is not derived from a methyl side chain. Otherwise, we would have the grotesque situation of deriving aldosterone from an 18nor, but 18 methylated pregnane.

Hemiacetals, acetals, hemiketals and ketals: The symbols used for hydroxy and other groups (e.g., ol, θI) will be applied.

Hemiacetals:  ($^1/_2$acetal) reaction product of an aldehyde with one alcohol group.

Acetals:  (acetal, not abbreviated) reaction product of an aldehyde with two alcohol groups.

Hemiketals:  ($^1/_2$ketal) combination of a ketone with one alcohol group.

Ketals:  (ketal, not abbreviated) combination of a ketone with two alcohol groups.

Epoxy:  (regarded as ethers). An oxygen attached to two vicinal carbons, e.g., $A\theta_{1\alpha-2}$ (for 1,2α-epoxy-androstane).

Halo compounds: Br, Cl, F, i (-bromo, -chloro, -fluoro, -iodo), e.g., $P^4Cl_{9\alpha}$ (for 9α-Chloro-4-pregnene). Instead of "I" for iodine write "i" (e.g., $P^4i_{9\alpha}$) to avoid confusion with methyl, whose symbol is roman one.

Nitrogen containing steroids: NH_2 (for amino), NH (for imino), CN (for carbonitrile or

cyano). These symbols are used as such in SSS terms. However, symbols with subscript numbers (NH_2) are bracketed.

e.g., ANH_{17} (for 17-imino-androstane),

$P^5CN_{16\alpha}ol_{3\beta}on_{20}$ (for 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile or PCN). All other N-containing steroids, including the alkaloids, belong to this group. The aza-steroids are listed last, in the heterocyclic category (*cf.* below).

Sulfosteroids: All thio compounds, e.g., —SH (-thiol),  epithio, (θS), thioether.

Heterocyclic steroids: These are listed separately for each element replacing carbon in the ring structure. They are indicated by the chemical symbol of the substituent in brackets, followed by a subscript number identifying the position of the replaced ring carbon after the symbol of the parent hydrocarbon (e.g., $A(O_2)$ for 2-Oxa-androstane, $P(N_3)$ for 3-Aza-pregnane).

Ring modifications: (*cf.* p. 775).

Other non alkyl-substituted compounds: Here, we list all those non alkyl-substituted compounds for which no category has been foreseen above.

Secondary Parent Hydrocarbons

All radicals attached to the parent hydrocarbon by a **carbon-to-carbon linkage** are considered to form "secondary parent hydrocarbons." They are written by attaching the symbol for the substituent following that of the parent hydrocarbon, both being underlined (e.g., $A5\alpha I_{17\alpha}$ = 17α-methyl-5α-androstane).

All alkyl derivatives, except those listed themselves as main parent hydrocarbons (e.g., 17β-ethyl-androstane = pregnane), are regarded as alkyl derivatives of the latter. This includes even 17α-ethyl-androstane whose essential difference from pregnane is further emphasized by attaching the side chain to the structure formula of androstane, not only by an alpha (...) bond, but horizontally.

The alkyl side-chains are enumerated: 1. according to the increasing numbers of the carbon atoms to which they are attached on the skeleton of the main parent hydrocarbon and 2. according to increasing numbers of the carbon atoms they contain (methyl, ethyl, propyl, butyl, etc.). Saturated radicals are mentioned first, and those with double bonds in the order of increasing unsaturation. Triple bonds (superscript position numbers boldface e.g.: $P^{\mathbf{20}}$) are mentioned after all multiple double bonds. Branched and ring-substituents are listed in the order of increasing complexity. (For corresponding symbols, *cf.* p. 778.)

Among the aliphatic 17-alkyl-androstanes, only pregnanes, cholanes, cholestanes, ergostane and stigmastan are regarded as special parent hydrocarbons because of their common occurrence.

Order of Precedence in General

The order of precedence for the above mentioned substituents is rigidly observed except in the first (underlined or boldface) portion of the SSS terms of secondary parent hydrocarbons. As previously stated, here all substituents attached to a side-chain are listed immediately after the positional number of the latter, to avoid subsequent repetition of the locant, e.g., $A^5I''_{16\beta}III_{17\alpha/on//1}ol_{3\beta}$.

When two or more identical substituents appear in the same molecule, all these must be enumerated before considering compounds possessing additional substituents mentioned later in the above list (e.g., all -enes before -dienes, before -trienes, etc.; all -ols before -diols, before -triols, etc.). Only after this do we proceed to compounds having an additional type of substituent (e.g., only after all mono- and polyols do we procede to -ones).

All unsaturations (double, triple and cyclizing bonds) and substitution products are listed according to the **ascending order of the affected carbons** (e.g., in pregnanes 1—21). Ring modifications, such as contractions (nor), expansions (homo), openings (sec), etc. are listed in the **order of the ring lettering** (A—D).

Computerization

Subscript and superscript notations are unsuitable for computerization but SSS names can be written in a single line without loss of precision. E.g., normally we write dehydroepiandrosterone thus: $A^5ol_{17\beta}on_3$ and the corresponding 5α saturated compound thus: $A5\alpha ol_{17\beta}on_3$. For computer use, these two steroids can be written respectively A5 ol 17β on 3 and A5α ol 17β on 3.

The superscript position of a number following the symbol for the parent hydrocarbon (e.g., A^5) merely facilitates its immediate recognition as a double bond. Similarly the subscript position of numbers helps to identify them as locants. But actually, this style is not indispensable if the constituent symbols are properly separated. The rule of "first what,

then where" makes it obvious that the "5" belongs to the "**A**", the "17β" to the "ol", and the "3" to the "on". The number 5 could only indicate a double bond; it would have to be followed by α, β, or ξ if it were to denote the steric position of the 5-hydrogen. Even the omission of underlining (or boldface printing) of the parent hydrocarbon would not introduce any ambiguity. Greek letters such as α, β, or ξ, can be written out as alpha, beta or xi, and the roman numerals can be composed by capitals on computers, just as on typewriters that do not have corresponding signs. The use of capital and small letters is likewise not necessary, but it helps rapid reading and is recommended for those who have the appropriate modern computer equipment.

Chemical Symbols

, = sign of separation. To be used at discretion of codifier, wherever two adjacent symbols might be read as one (e.g., $on_{4,6}$).

* = ester (precedes symbol of acid. E.g.: *III = propyl ester).

θ = ether (precedes symbol of alkyl. E.g.: θI = methoxy or methyl ether).

A = androstan (for symbols of other parent hydrocarbons, *cf.* p. 775). Only parent hydrocarbons and all alkyls attached to them by carbon-to-carbon linkages are bold face (E.g.: $\mathbf{AIII}_{17\beta}\mathbf{I}_{2\alpha}\mathbf{ol}_{4\beta}\mathbf{on}_{11}$ = 17β-propyl-2α-methyl androstane with 4β-alcohol and 11-keto groups.)

/,// = successive levels (subdivisions) in a code, obviating the need for more than one subscript line (e.g., 2-methylbutane = $IV_{I/2}$). The successive levels of more complex branchings coded according to the general formula: $R_{R'/2//R''///3}$ (e.g., 2(3-methylbutyl)-heptane = $VII_{IV/2//I///3}$.)

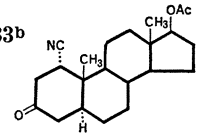
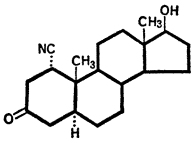
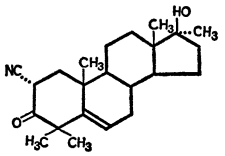
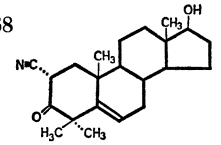
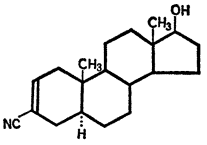
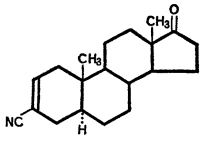
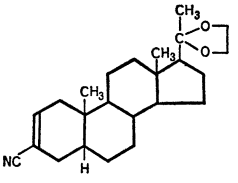
( ) = brackets are used to enclose symbols with subscript numbers which might be confused with locants, e.g., (NH_2), (CH_2).

Substituents	Methyl Formic	Ethyl Acetic	Propyl Propionic	Butyl	
Alkanes, normal	I	II	III	IV	
Alkanes, branched	—	—	$III_{I/2}$	$IV_{I/2,3}$	
			(2-methyl-propane)	(2,3-dimethyl-butane)	
Alcohols	I ol	II ol	III ol	IV ol	Symbols of side chains bound to ring by carbon-to-carbon linkages, follow roman numerals.
Acids	I″	II″	III″	IV″	
Aldehydes	Ial	IIal	IIIal	IVal	
Esters	*I	*II	*III	*IV	Symbols of groups bound to ring by oxygen, precede roman numerals.
Ethers	θI	θII	θIII	θIV	

The 92 carbonitriles of Table 135 have been arranged according to the increasing number of the skeletal carbon atoms to which the —CN groups or —CN-bearing side-chains are attached. A few additional steroids, other than nitriles (Cpds. 93—99) are listed in arbitrary order.

The most potent catatoxic steroids against both substrates of this test were those bearing a 2α- or 16α-carbonitrile group. Among these, several showed potency against one or both substrates at individual dose levels as low as 100 μg or even 30 μg. In general, protection against indomethacin was more readily obtained than against digitoxin.

Table 135. *First step: Screening of steroids against digitoxin and indomethacin intoxication*

Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
$CN_{1,2,3}$ Steroid Carbonitriles				
1 IM 433[b] 	17β-Hydroxy-3-oxo-5α-androstane-1α-carbonitrile acetate (SC-16027)	0.5	0	0
	A 5 α $CN_{1\alpha}$ *II″$_{17\beta}$on$_3$			
2 IM 427 	17β-Hydroxy-3-oxo-5α-androstane-1α-carbonitrile (SC-16026)	0.5	0	0
	A 5 α $CN_{1\alpha}ol_{17\beta}on_3$			
3 IM 364 	17β-Hydroxy-4,4,17-trimethyl-3-oxo-5-androstene-2α-carbonitrile. *Trimethylandrostenolone carbonitrile*, "TMACN" (Winthrop)	10 0.5 0.1 0.03	3 2.5 2 0	3 3 2 0
	$A^5I_{4,4,17\alpha}CN_{2\alpha}ol_{17}on_3$			
4 IM 568 	17β-Hydroxy-4,4-dimethyl-3-oxo-5-pregnene-2α-carbonitrile (U-26854)	0.5 0.03	2 0	0.5 —
	$A^5I_{4,4}CN_{2\alpha}ol_{17\beta}on_3$			
5 IM 368 	17β-Hydroxy-5α-androst-2-ene-3-carbonitrile (Lepetit)	10 0.5	0.5 0	2 0
	A 5 $\ddot{\alpha}^2CN_3ol_{17\beta}$			
6 IM 365 	17-Oxo-5α-androst-2-ene-3-carbonitrile (Lepetit)	10 0.5	2 0	2 0
	A 5 α$^2CN_3on_{17}$			
7 IM 367 	20,20-(Ethylenedioxy)-5β-pregn-2-ene-3-carbonitrile (Lepetit)	10	0	0
	P 5 β$^2CN_3\theta\theta II_{20}$			

[a] For details, see Digitoxin and Indomethacin in the list of techniques used to produce and appraise various types of damage.

[b] Underneath the serial number of the compounds, the "IM" numbers (for Institut de Médecine et de Chirurgie expérimentales) are mentioned. These identify steroids in our collection and remain the same in all Tables as well as in other publications from this Institute.

Table 135 (continued)

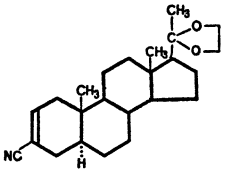
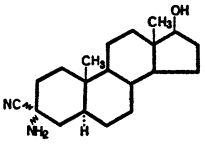
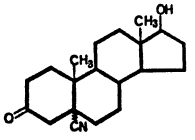
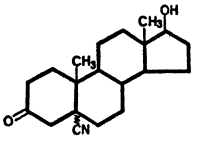
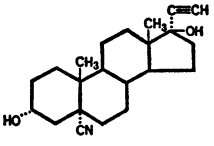
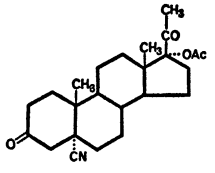
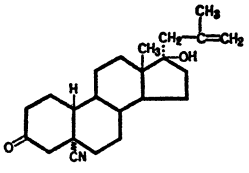
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
8 IM 366 	20,20-(Ethylenedioxy)-5α-pregn-2-ene-3-carbonitrile (Lepetit) **P5α²CN₃θ,θII₂₀**	10 0.5 0.03	0.5 0 —	2 2 0
9 IM 424 	17β-Hydroxy-3ξ-amino-5α-androstane-3-carbonitrile (SC-13265) **A5αCN$_{3\xi}$ol$_{17\beta}$(NH$_2$)$_3$**	0.5	0	0
CN$_5$				
10 IM 420 	17β-Hydroxy-3-oxo-5β-androstane-5-carbonitrile (SC-13389) **A5βCN$_5$ol$_{17\beta}$on$_3$**	10 0.03 0.015 0.005 0.001	0 0 0 0 0	0 0 0 0 0
11 IM 434 	17β-Hydroxy-3-oxo-5α-androstane-5-carbonitrile (SC-13269) **A5αCN$_5$ol$_{17\beta}$on$_3$**	0.5	0	0
12 IM 432 	3α,17-Dihydroxy-5α,17α-pregn-20-yne-5-carbonitrile (SC-13675) **A5αCN$_5$II$^{1-1}{}_{17\alpha}$ol$_{3\alpha,17}$**	0.5	0	0
13 IM 431 	17-Hydroxy-3,20-dioxo-5α-pregnane-5-carbonitrile acetate (SC-13795) **P5αCN$_5$*II″$_{17}$on$_{3,20}$**	0.5	0	0
14 IM 430 	5ξ-Cyano-17-hydroxy-17α-(2-methylallyl)-estran-3-one (SC-13969) **E5ξCN$_5$III$^2{}_{17\alpha/I//2}$ol$_{17}$on$_3$**	0.5	0	0

Table 135 (continued)

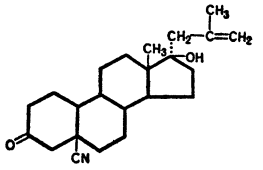
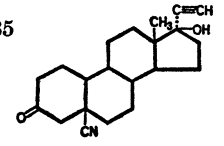
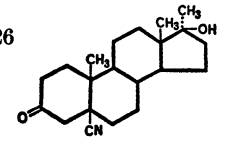
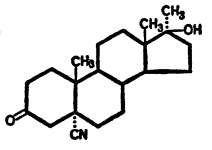
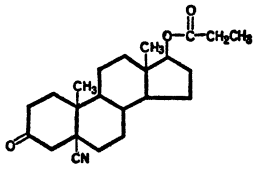
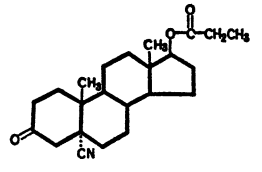
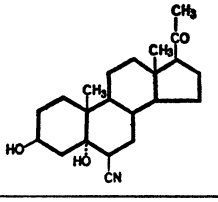
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
15 IM 428 	5β-Cyano-17-hydroxy-17α-(2-methyl-allyl)-estran-3-one (SC-14373) **E5βCN$_5$III^2$_{17\alpha/I//2}$ol$_{17}$on$_3$**	0.5	0	0
16 IM 435 	17-Hydroxy-3-oxo-19-nor-5β,17α-pregn-20-yne-5-carbonitrile (SC-13823) **E5βCN$_5$II$^{1-1}$$_{17\alpha}ol_{17}on_3$**	0.5	0	0
17 IM 426 	17β-Hydroxy-17-methyl-3-oxo-5β-androstane-5-carbonitrile (SC-13754) **A5βI$_{17\alpha}$CN$_5$ol$_{17}$on$_3$**	0.5	0	0
18 IM 425 	17β-Hydroxy-17-methyl-3-oxo-5α-androstane-5-carbonitrile (SC-13503) **A5αI$_{17\alpha}$CN$_5$ol$_{17}$on$_3$**	0.5	0	0
19 IM 422 	17β-Hydroxy-3-oxo-5β-androstane-5-carbonitrile propionate (SC-14175) **A5βCN$_5$*III″$_{17\beta}$on$_3$**	0.5	0	0
20 IM 421 	17β-Hydroxy-3-oxo-5α-androstane-5-carbonitrile propionate (SC-14174) **A5αCN$_5$*III″$_{17\beta}$on$_3$**	0.5	0	0
CN$_6$				
21 IM 449 	3β,5α-Dihydroxy-20-oxopregnane-6β-carbonitrile (Syntex) **P5αCN$_{6\beta}$ol$_{3\beta,5}$on$_{20}$**	0.5	0	0

Table 135 (continued)

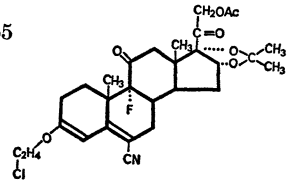
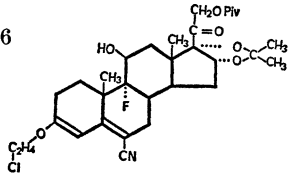
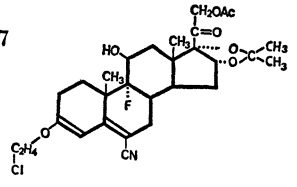
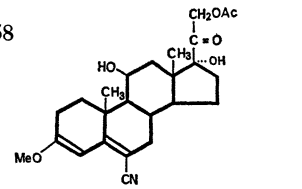
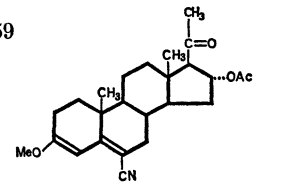
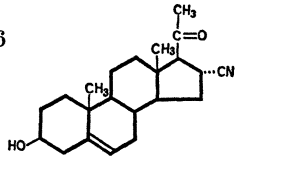
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
22 IM 455 	3-(2′-Chloroethoxy)-6-cyano-9α-fluoro-3,5-pregnadiene-16α, 17α,21-triol-11, 20-dion-21-acetate-16,17-acetonide (Farmitalia)	0.5	0	1
	$P^{3,5}CN_6{*}II''_{21}on_{11,20}\theta II_{3\beta/Cl//2}\theta\theta I_{16\alpha-17/I,I}F_{9\alpha}$			
23 IM 456 	3-(2′-Chloroethoxy)-6-cyano-9α-fluoro-3,5-pregnadiene-11β,16α,17α, 21-tetrol-20-one-21-pivalat-16,17-acetonide (Farmitalia)	0.5	0	
	$P^{3,5}CN_6ol_{11\beta}{*}III''_{21/I//2,2}on_{20}\theta II_{3/Cl//2}\theta\theta_{16\alpha-17/I,I}F_{9\alpha}$			
24 IM 457 	3-(2′-Chloroethoxy)-6-cyano-9α-fluoro-3,5-pregnadiene-11β,16α,17α, 21-tetrol-20-one-21-acetate-16,17-acetonide (Farmitalia)	0.5	0	0
	$P^{3,5}CN_6ol_{11\beta}{*}II''_{21}on_{20}\theta II_{3/Cl//2}\theta\theta I_{16\alpha-17/I,I}F_{9\alpha}$			
25 IM 458 	3β,11β,17α,21-Tetrahydroxy-20-oxo-3,5-pregnadiene-6-carbonitrile 3-methyl ether, 21-acetate (BDH)	0.5	0	0
	$P^{3,5}CN_6ol_{11\beta,17\alpha}{*}II''_{21}on_{20}\theta I_3$			
26 IM 459 	3,16α-Dihydroxy-20-oxo-3,5-pregnadiene-6-carbonitrile 3-methyl ether, 16-acetate (Glaxo Canada Ltd.)	0.5	0	0
	$P^{3,5}CN_6{*}II''_{16\alpha}on_{20}\theta I_3$			
CN_{16}				
27 IM 346 	3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile (SC-4674), (U-14975) *Pregnenolone carbonitrile "PCN"*	10 1 0.5 0.2 0.1 0.03 0.015	3 3 3 3 3 2 0	3 3 3 3 3 3 0
	$P^5CN_{16\alpha}ol_{3\beta}on_{20}$			

Table 135 (continued)

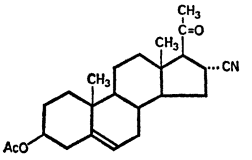
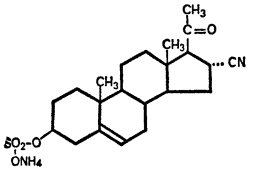
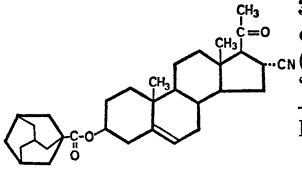
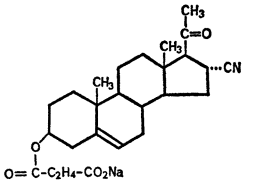
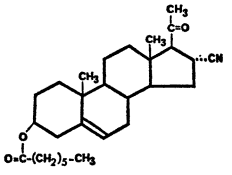
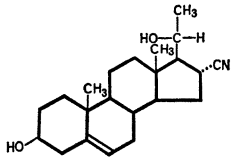
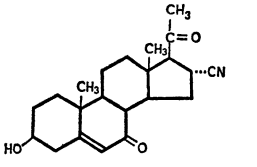
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
28 IM 413	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile acetate (U-34889, Syntex) *Pregnenolone carbonitrile acetate* "*PCN-ac*" $P^5CN_{16\alpha}$*II''$_{3\beta}$on$_{20}$	0.5 0.1 0.03 0.015	2.5 0.5 1 0	2 2 1.5 0
29 IM 483	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3-sulfate ammonium salt (U-37863C) "*PCN-ammonium sulfate*" $P^5CN_{16\alpha}$*(S''NH$_4$)$_{3\beta}$on$_{20}$	0.5 0.03 0.015	3 3 1	3 3 0.5
30 IM 478	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3(1'adamantoate) (U-36789) "*PCN-adamantoate*" $P^5CN_{16\alpha}$*adamantoate$_{3\beta}$ on$_{20}$	0.5 0.03	0.5 —	3 0
31 IM 476	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3-hemisuccinate sodium salt (U-36278A) "*PCN-hemisuccinate sodium*" $P^5CN_{16\alpha}$*$^1/_2$suc''Na$_{3\beta}$on$_{20}$	0.5 0.03 0.015 0.005	3 2.5 2 0	3 3 1.5 2
32 IM 479	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3-heptanoate (U-37001) "*PCN-heptanoate*" $P^5CN_{16\alpha}$*VII''$_{3\beta}$on$_{20}$	0.5 0.03 0.015	3 1 0	3 1.5 0
33 IM 444	 3β,20-Dihydroxy-5-pregnene-16α-carbonitrile (Syntex) $P^5CN_{16\alpha}ol_{3\beta,20}$	0.5 0.1 0.03	1.5 1 0	3 1.5 0
34 IM 423	 3β-Hydroxy-7,20-dioxo-5-pregnene-16α-carbonitrile (SC-6813) $P^5CN_{16\alpha}ol_{3\beta}on_{7,20}$	0.5 0.03	3 0	1.5 0.5

Table 135 (continued)

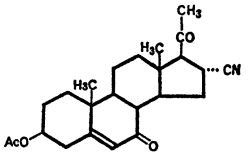
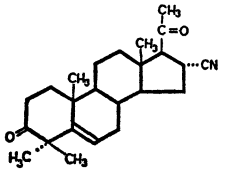
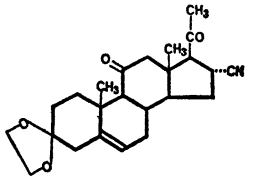
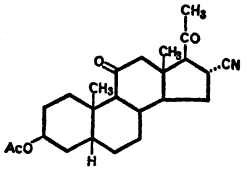
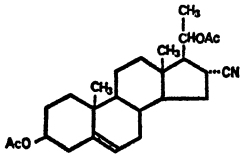
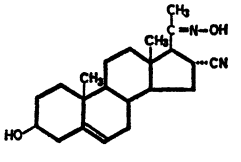
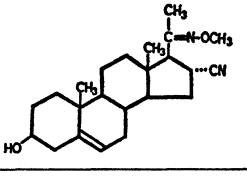
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
35 IM 429	 3β-Hydroxy-7,20-dioxo-5-pregnene-16α-carbonitrile acetate (SC-6703) $P^5CN_{16\alpha}{}^*II''_{3\beta}on_{7,20}$	0.5 0.03	3 0	2 1.5
36 IM 474	 4,4-Dimethyl-3,20-dioxo-5-pregnene-16α-carbonitrile (U-35641) $P^5I_{4,4}CN_{16\alpha}on_{3,20}$	0.5 0.03	0.5 —	3 0
37 IM 411	 3,3-(Ethylenedioxy)-11,20-dioxo-5-pregnene-16α-carbonitrile (U-35006) $P^5CN_{16\alpha}on_{11,20}\theta\theta II_{3,3}$	0.5 0.1 0.03 0.015	2.5 2.5 0 0	2 2 1.5 1.5
38 IM 418	 3β-Hydroxy-11,20-dioxo-5β-pregnane-16α-carbonitrile acetate (U-34575) $P5\beta CN_{16\alpha}{}^*II''_{3\beta}on_{11,20}$	0.5 0.1 0.03 0.015	2.5 2.5 0 0	2 2 1.5 1.5
39 IM 464	 16α-Cyano-5-pregnene-3β,20β-diol diacetate (SC-5482) $P^5CN_{16\alpha}{}^*II''_{3\beta,20\beta}$	0.1 0.03	0 —	2 0
40 IM 462	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile,20-oxime (U-37722) $P^5CN_{16\alpha}ol_{3\beta}NOH_{20}$	0.5 0.03 0.015 0.010	3 2.5 0.5 —	3 3 1 0
41 IM 482	 3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile,20-methyloxime (U-37694) $P^5CN_{16\alpha}ol_{3\beta}(NOCH_3)_{20}$	0.5 0.03 0.015	1 1.5 0	1.5 0 —

Table 135 (continued)

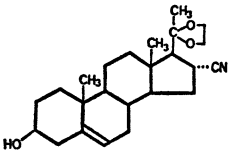
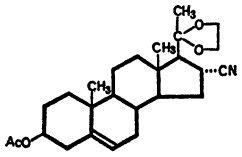
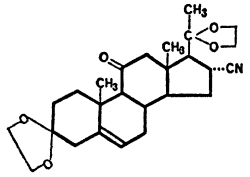
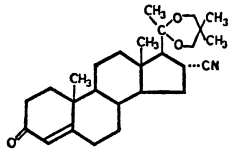
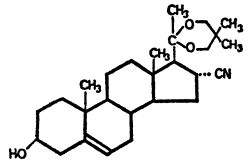
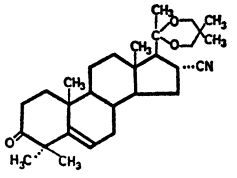
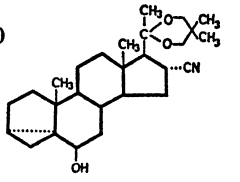
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
42 IM 414 	20,20-(Ethylenedioxy)-3β-hydroxy-5-pregnene-16α-carbonitrile (U-19553) $P^5CN_{16\alpha}ol_{3\beta}\theta\theta II_{20}$	0.5 0.1 0.03 0.015	2.5 2 0.5 0	2 2 1.5 0
43 IM 445 	3β-Hydroxy-20,20-ethylene-dioxy-5-pregnene-16α-carbonitrile acetate (Syntex) $P^5CN_{16\alpha}{*}II''_{3\beta}\theta\theta II_{20}$	0.5 0.1 0.03	1.5 0 0	3 1.5 0
44 IM 412 	3,3,20,20-Bis-ethylenedioxy-11-oxo-5-pregnene-16α-carbonitrile (U-35910) $P^5CN_{16\alpha}on_{11}\theta\theta II_{3,20}$	0.5 0.1 0.03 0.015	2.5 0 0 0	2 2 1.5 0
45 IM 475 	3,20-Dioxo-4-pregnene-16α-carbonitrile,20-cyclic(2′,2′-dimethyltrimethylene acetal) (U-35655) $P^4CN_{16\alpha}on_3\theta\theta III_{20/I//2,2}$	0.5 0.03	1.5 0.5	3 0
46 IM 461 	3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile, cyclic(2,2-dimethyltrimethylene acetal) (U-36961) $P^5CN_{16\alpha}ol_{3\beta}\theta\theta III_{20/I//2,2}$	0.5 0.03 0.015	2 2 0	3 0 —
47 IM 481 	4,4-Dimethyl-3,20-dioxo-5-pregnene-16α-carbonitrile,20-cyclic(2′2′-dimethyltrimethylene acetal) (U-37542) $P^5I_{4,4}CN_{16\alpha}on_3\theta\theta III_{20/I//2,2}$	0.5	0	0
48 IM 480 	6β-Hydroxy-20-oxo-3α,5α-cyclopregnane-16α-carbonitrile, cyclic(2′,2′-dimethyltrimethylene acetal) (U-37483) $P^{3\alpha(5)}5\alpha CN_{16\alpha}ol_{6\beta}\theta\theta III_{20/I//2,2}$	0.5 0.03	1 0	3 0

Table 135 (continued)

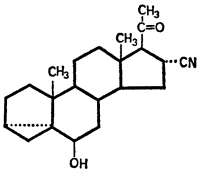
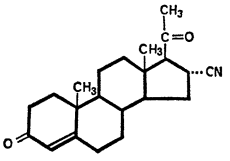
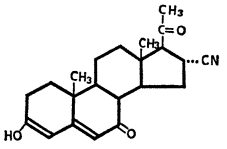
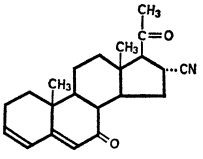
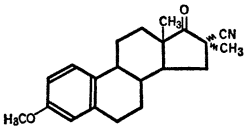
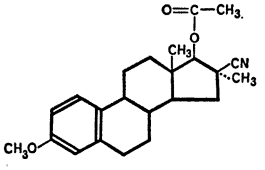
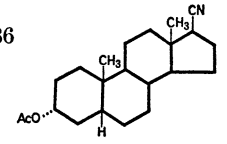
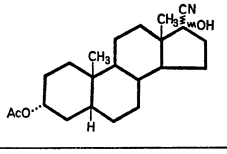
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
49 IM 477 	6β-Hydroxy-20-oxo-3α,5α-cyclopregnane-16α-carbonitrile (U-36710) **$P^{3\alpha(5)}5\alpha CN_{16\alpha}ol_{6\beta}on_{20}$**	0.5 0.03	2 0	3 0
50 IM 466 	16α-Cyano-4-pregnene-3,20-dione (SC-4688) **$P^{4}CN_{16\alpha}on_{3,20}$**	0.1 0.015	3 0	0 0
51 IM 471 	16α-Cyano-3-hydroxy-3,5-pregnadiene-7,20-dione (SC-6963) **$P^{3,5}CN_{16\alpha}ol_{3}on_{7,20}$**	0.1	0	—
52 IM 470 	16α-Cyano-3,5-pregnadiene-7,20-dione (SC-6786) **$P^{3,5}CN_{16\alpha}on_{7,20}$**	0.1	0	0
53 IM 395 	3-Methoxy-16-methyl-17-oxo-estra-1,3,5(10)-triene-16ξ-carbonitrile (Roussel) **$E^{1,3,5(10)}I_{16\xi}CN_{16}on_{17}\theta I_3$**	0.5	0	0
54 IM 392 	17β-Hydroxy-3-methoxy-16-methylestra-1,3,5(10)-triene-16β-carbonitrile acetate (Roussel) **$E^{1,3,5(10)}I_{16\alpha}CN_{16}{*}II''_{17\beta}\theta I_3$**	0.5	0	0
CN_{17}				
55 IM 386 	3α-Hydroxy-5β-androstane-17β-carbonitrile acetate (Roussel) **$A5\beta CN_{17\beta}{*}II''_{3\alpha}$**	0.5	0	0
56 IM 377 	3α,17-Dihydroxy-5β-androstane-17ξ-carbonitrile 3-acetate (Roussel) **$A5\beta CN_{17}ol_{17\xi}{*}II''_{3\alpha}$**	0.5	0	0

Table 135 (continued)

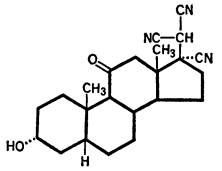
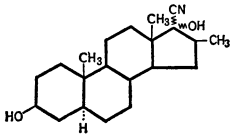
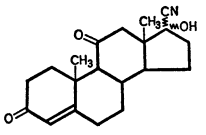
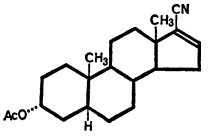
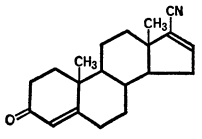
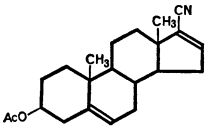
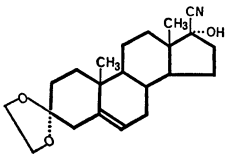
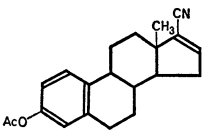
Group		Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
57 IM 369		17-Cyano-3α-hydroxy-11-oxo-5β-androstane-17β-malononitrile (Roussel)	0.5	0	0
		$A5\beta I_{17\beta/CN//1,1}CN_{17}ol_{3\alpha}on_{11}$			
58 IM 382		3β,17-Dihydroxy-16β-methyl-5α-androstane-17ξ-carbonitrile (Roussel)	0.5	0	0
		$A5\alpha I_{16\beta}CN_{17}ol_{3\beta,17\xi}$			
59 IM 374		17-Hydroxy-3,11-dioxo-4-androstene-17ξ-carbonitrile (Roussel)	0.5	0	0
		$A^4CN_{17}ol_{17\xi}on_{3,11}$			
60 IM 378		3α-Hydroxy-5β-androst-16-ene-17-carbonitrile acetate (Roussel)	0.5	0	0
		$A5\beta^{16}CN_{17}{*}II''_{3\alpha}$			
61 IM 389		3-Oxo-4,16-androstadiene-17-carbonitrile (Roussel)	0.5	0	0
		$A^{4,16}CN_{17}on_3$			
62 IM 396		3β-Hydroxy-5,16-androstadiene-17-carbonitrile acetate (Roussel)	0.5	0	0
		$A^{5,16}CN_{17}{*}II''_{3\beta}$			
63 IM 390		3,3-(Ethylenedioxy)-17-hydroxy-5-pregnene-17β-carbonitrile (Roussel)	0.5	0	0
		$A^5CN_{17\alpha}ol_{17}\vartheta\vartheta II_3$			
64 IM 380		3-Hydroxy-1,3,5(10),16-estratetraene-17-carbonitrile acetate (Roussel)	0.5	0	0
		$E^{1,3,5(10)16}CN_{17}{*}II''_3$			

Table 135 (continued)

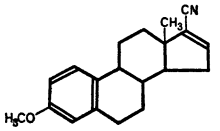
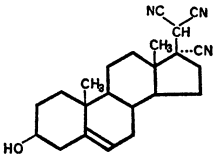
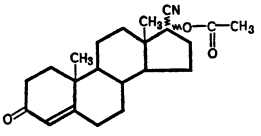
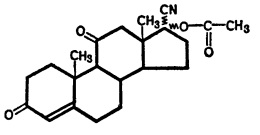
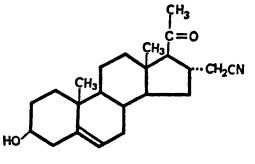
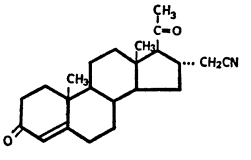
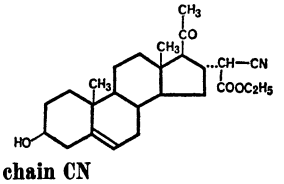
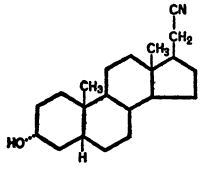
Group		Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
65 IM 400		3-Methoxy-1,3,5(10),16-estratetraene-17-carbonitrile (Roussel) $E^{1,3,5(10)16}CN_{17}\theta I_3$	0.5	0	0
66 IM 417		17-Cyano-3β-hydroxy-5-androstene-17β-malononitrile (U-28406) $A^5I_{17\beta/CN//1,1}CN_{17}ol_{3\beta}$	0.5	0	0
67 IM 391		17-Hydroxy-3-oxo-4-androstene-17ξ-carbonitrile acetate (Roussel) $A^4CN_{17}{*}II''_{17\xi}on_3$	0.5	0	0
68 IM 375		17-Hydroxy-3,11-dioxo-4-androstene 17ξ-carbonitrile acetate (Roussel) $A^4CN_{17}{*}II''_{17\xi}on_{3,11}$	0.5	0	0
16-Side chain CN					
69 IM 469		16α-Cyanomethyl-3β-hydroxy-5-pregnen-20-one (SC-6939) $P^5ICN_{16\alpha}ol_{3\beta}on_{20}$	0.5	0	0
70 IM 472		16α-Cyanomethyl-4-pregnene-3,20-dione (SC-7097) $P^4ICN_{16\alpha}on_{3,20}$	0.5	0	0
71 IM 409		α-Cyano-3β-hydroxy-20-oxo-5-pregnen-16α-acetic acid ethyl ester (SK & F) $P^5II''_{16\alpha/CN//2}{*}IIol_{///1}ol_{3\beta}on_{20}$	10	0	0
17-Side chain CN					
72 IM 381		3α-Hydroxy-5β-androstan-17β-acetonitrile (Roussel) $A5\beta ICN_{17\beta}ol_{3\alpha}$	0.5	0	0

Table 135 (continued)

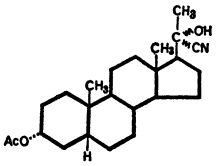
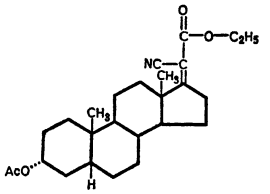
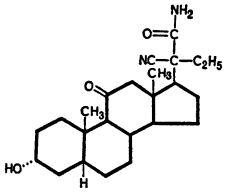
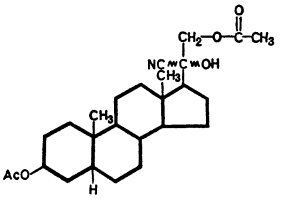
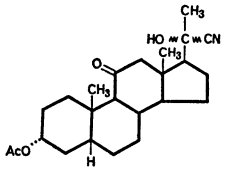
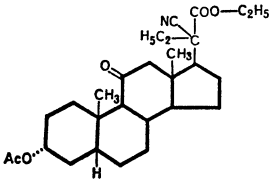
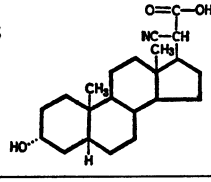
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
73 IM 383 	3α,20-Dihydroxy-5β-pregnane-20ξ-carbonitrile 3-acetate (Roussel) **P5βCN$_{20}$ol$_{20\xi}$*II″$_{3\alpha}$**	0.5	0	0
74 IM 371 	20-Cyano-3α-hydroxy-5β-pregn-17(20)-en-21-oic acid acetate ethyl ester (Roussel) **P5β$^{17(20)″}$CN$_{20}$*II″$_{3\beta}$*IIol$_{21}$**	0.5	0	0
75 IM 376 	20-Cyano-3α-hydroxy-11-oxo-24-nor-5β-cholan-21-oic acid amide (Roussel) **CHn(24)5βCN$_{20}$ol$_{3\alpha}$on$_{11}$(CONH$_2$)$_{20}$**	0.5	0	0
76 IM 388 	3β,20,21-Trihydroxy-5β-pregnane-20ξ-carbonitrile 3,21-diacetate (Roussel) **P5βCN$_{20}$ol$_{20\xi}$*II″$_{3\beta,21}$**	0.5	0	0
77 IM 403 	3α,20ξ-Dihydroxy-11-oxo-5β-pregnane-20ξ-carbonitrile 3-acetate (Roussel) **P5βCN$_{20}$ol$_{20\xi}$*II″$_{3\alpha}$on$_{11}$**	0.5	0	0
78 IM 401 	20-Cyano-3α-acetoxy-11-oxo-24-norcholan-21-oic acid ethyl ester (Roussel) **CHn(24)5β″CN$_{20}$*II″$_{3\alpha}$*IIol$_{21}$on$_{11}$**	0.5	0	0
79 IM 398 	20-Cyano-3α-hydroxy-5β-pregnan-21-oic acid (Roussel) **P5β″$_{21}$CN$_{20}$ol$_{3\alpha}$**	0.5	0	0

Table 135 (continued)

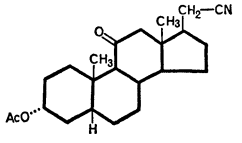
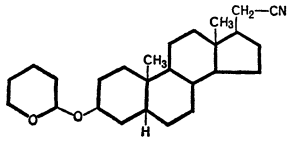
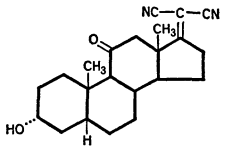
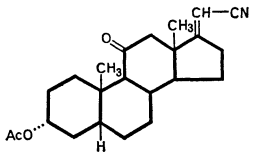
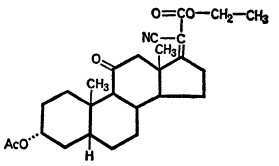
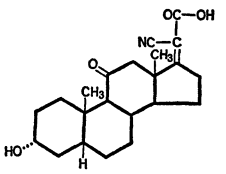
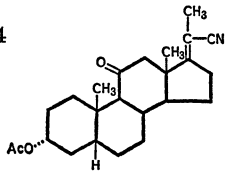
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
80 IM 385 	3α-Hydroxy-11-oxo-5β-androstan-17β-acetonitrile acetate (Roussel) **A5βICN$_{17\beta}$*II″$_{3\alpha}$on$_{11}$**	0.5	0	0
81 IM 384 	3β-(Tetrahydropyran-2-yloxy)-5β-androstan-17β-acetonitrile (Roussel) **A5βICN$_{17\beta}$θtetrahydropyranyl$_{3\beta}$ol$_{//2}$**	0.5	0	0
82 IM 406 	3α-Hydroxy-11-oxo-5β-androstane-Δ^{17}-malononitrile (Roussel) **A5β$^{17(20)}$I$_{17/CN//1,1}$ol$_{3\alpha}$on$_{11}$**	0.5	0	0
83 IM 393 	3α-Hydroxy-11-oxo-5β-androstan-Δ^{17}-acetonitrile acetate (Roussel) **A5β$^{17(20)}$ICN$_{17\alpha}$*II″$_{3\alpha}$on$_{11}$**	0.5	0	1.5
84 IM 408 	20-Cyano-3α-hydroxy-11-oxo-5β-pregn-17(20)-en-21-oic acid acetate ethyl ester (Roussel) **P5β$^{17(20)}$″CN$_{20}$*II$_{3\alpha}$*IIol$_{21}$on$_{11}$**	0.5	0	0
85 IM 405 	20-Cyano-3α-hydroxy-11-oxo-5β-pregn-17(20)-en-21-oic acid (Roussel) **P5β$^{17(20)}$″$_{21}$CN$_{20}$ol$_{3\alpha}$on$_{11}$**	0.5	0	0
86 IM 394 	3α-Hydroxy-11-oxo-5β-pregn-17(20)-ene-20-carbonitrile acetate (Roussel) **P5β$^{17(20)}$CN$_{20}$*II″$_{3\alpha}$on$_{11}$**	0.5	0.5	1.5

Table 135 (continued)

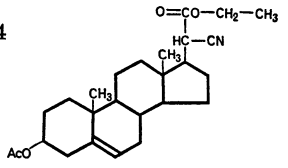
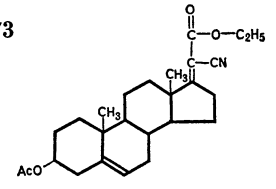
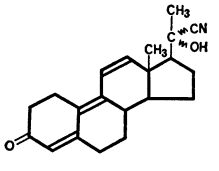
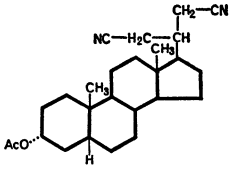
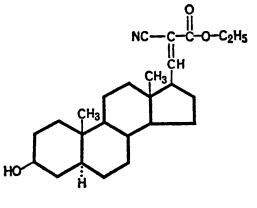
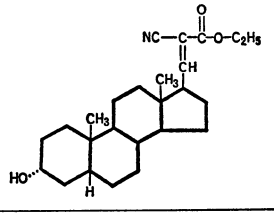
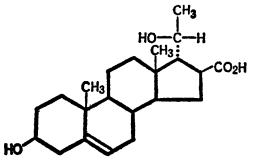
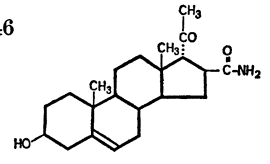
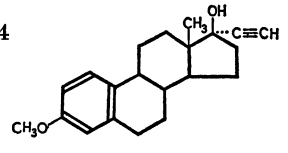
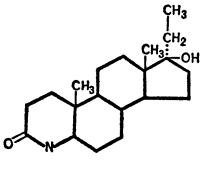
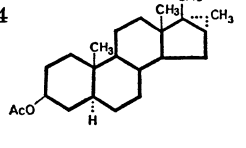
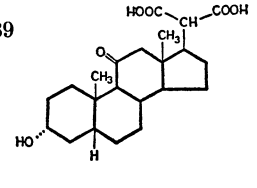
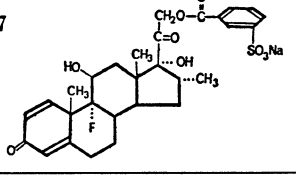
Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
87 IM 404 	20-Cyano-3β-hydroxy-5-pregnen-21-oic acid acetate ethyl ester (Roussel)	0.5	0	0
	$P^{5''}CN_{20}{*}II{ol}_{21}{*}II''_{3\beta}$			
88 IM 373 	20-Cyano-3β-hydroxy-5,17(20)-pregnadien-21-oic acid 3-acetate ethyl ester (Roussel)	0.5	0	0
	$P^{5,17(20)''}CN_{20}{*}II{ol}_{21}{*}II''_{3\beta}$			
89 IM 407 	20-Hydroxy-3-oxo-19-norpregna-4,9,11-triene-20ξ-carbonitrile (Roussel)	0.5	0	0
	$Pn\,19^{4,9,11}CN_{20\xi}{ol}_{20}{on}_3$			
90 IM 399 	3α-Hydroxy-5β-androstan-17β-glutaronitrile acetate (Roussel)	0.5	0	0
	$P\,5\,\beta\,CN_{21}ICN_{20}{*}II''_{3\alpha}$			
91 IM 402 	21-Cyano-3β-hydroxy-5α-pregn-20-ene-21-carboxylic acid ethyl ester (Roussel)	0.5	0	0
	$P\,5\,\alpha^{20}I''_{21}{/}{*}_{II\,ol}CN_{21}{ol}_{3\beta}$			
92 IM 370 	21-Cyano-3α-hydroxy-5β-pregn-20-ene-21-carboxylic acid ethyl ester (Roussel)	0.5	0	0
	$P\,5\,\beta^{20}I''_{21}{/}{*}_{II\,ol}CN_{21}{ol}_{3\alpha}$			

Table 135 (continued)

Group	Steroids	Dose (mg)	Digitoxin[a]	Indomethacin[a]
Other Active Steroids				
93 IM 448	 3β,20-Dihydroxy-17α-pregn-5-ene-16β-carboxylic acid (Syntex) **$A^5I''_{16\beta}II_{17\alpha/ol//1}ol_{3\beta}$**	0.5 0.1 0.03	1.5 0.5 0	3 0 0
94 IM 446	 3β-Hydroxy-20-oxo-17α-pregn-5-ene-16β-carboxamide (Syntex) **$A^5II_{17\alpha/on//1}(CONH_2)_{16\alpha}ol_{3\beta}$**	0.5 0.1	0 0	1.5 0
95 IM 304	 3-Methoxy-19-nor-17α-pregna-1,3,5(10)-trien-20-yn-17-ol (Lilly) *Mestranol* **$E^{1,3,5(10)}II^{1-1}{}_{17\alpha}ol_{17}\theta I_3$**	10 0.5	0 —	3 0
96 IM 410	 17-Hydroxy-4-aza-17α-pregnan-3-one (Organon) **$A(N_4)II_{17\alpha}ol_{17}on_3$**	10 0.5	1.5 0	3 0
97 IM 454	 16α,17-Dihydro-3'H-cyclopropa(16,17)-5α-androstane-3β,17β-diol diacetate (SC-21940) **$A5\alpha I_{16\alpha-17}{}^*II''_{3\beta,17}$**	10 0.5	3 0	— 0
98 IM 489	 3α-Hydroxy-11-oxo-5β-androstane-17β-malonic acid (Roussel) **$A5\beta I_{17\beta/I''//1,1}ol_{3\alpha}on_{11}$**	0.5 0.03	0 —	1.5 0
99 IM 527	 9α-Fluoro-16α-methyl-11β,17,21-trihydroxy-1,4-pregnadiene-3,20-dione 21-sodium m-sulfobenzoate *Dexamethasone sodium m-sulfobenzoate* (B.R.L.) **$P^{1,4}I_{16\alpha}ol_{11\beta,17}$m-sulfobenzoate $Na_{21}on_{3,20}F_{9\alpha}$**	0.5 0.03	0 —	1.5 0

Cpd. 54, the only 16β-carbonitrile of our series, as well as Cpd. 53 in which the steric position of the 16-carbonitrile is unknown, were inactive in protecting against either substrate, even at the dose level of 500 µg. Cpds. 69—71 in which the —CN group is attached to a 16α-side-chain (rather than to the C_{16} carbon of the steroid skeleton itself) showed no protective activity, even at the dose of 10 mg. On the other hand, it is hardly coincidental that among 28 16α-carbonitriles tested (Cpds. 27—54) all but three (Cpds. 47, 51, 52) were active, and most of them even at very low dose levels. This suggests that the attachment of a —CN group in the 16α-position directly to the steroid skeleton is very favorable for this type of protective effect; the configuration of the rest of the steroid molecule, though capable of influencing the degree of activity, is of much lesser importance.

It is known from our previous work that a carbonitrile group in position 2α (e.g., Cpd. 3, TMACN) is also compatible with high catatoxic activity against a variety of substrates; additional evidence justifying this conclusion is given in Tables 135, 136.

Carbonitrile groups in position 3, may or may not convey some potency (Cpds. 5—9), but steroids with carbonitriles attached to C_1 (Cpds. 1, 2), C_5 (Cpds. 10—20) or C_6 (Cpds. 21—26) were uniformly inactive at all dose levels tested.

Carbonitrile substitution at C_{17}, C_{20}, C_{21} or in side-chains resulted in no remarkable catatoxic potency at the dose levels tested, with the exception of Cpds. 83 and 86 which were moderately effective in this respect at the dose level of 500 µg.

Among the steroids of Table 135 other than carbonitriles (Cpds. 93—99), special interest is attached to Cpd. 96 (an aza-steroid), Cpd. 95 (mestranol), a strong folliculoid used in anticonceptional pills, and Cpd. 93 (a 16β-carboxylic acid) all of which showed some catatoxic activity at comparatively high dose levels. This degree of activity is of little practical significance, but it is interesting that a heterocyclic aza-compound, a 16β-carboxylic acid and a folliculoid can possess some catatoxic potency.

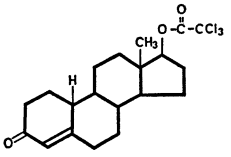
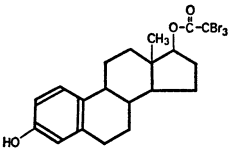
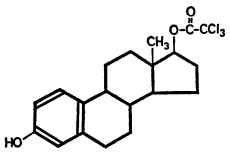
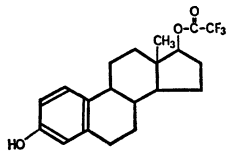
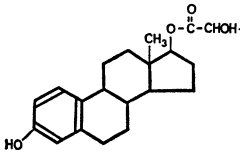
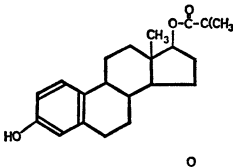
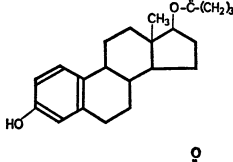
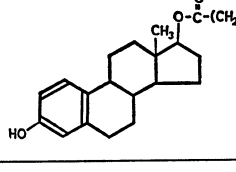
Finally, it is noteworthy that (except for the moderate potency of Cpd. 93) all 16-carboxylic acids (Cpds. 66, 67 and 69 in Table 135A) are devoid of catatoxic potency against both substrates. A priori, the possibility could not have been excluded that nitriles are metabolized in vivo into the corresponding carboxylic acids and that the latter would be responsible for catatoxic activity, but this does not appear to be the case.

Additional inactive steroids are listed in Table 135A *cf.*, p. 794.

First Step: Synopsis of all 500 Steroids Tested for Their Ability to Prevent Digitoxin and Indomethacin Intoxication

The preceding tables summarize our hitherto unpublished data on the protection by steroids, and particularly by carbonitriles, against digitoxin and indomethacin intoxication. However, in order to obtain a proper overview of this field, the following list summarizes the results obtained with *all 500 steroids tested up to now, cf.* Table 135B, p. 807. It will be kept in mind that all these experiments were performed under essentially identical conditions as outlined on p. VIII and in earlier publications (Selye G 70,421/70, G 70,480/71). Since no other laboratory has published comparable data on the detoxication of digitoxin and indomethacin by steroids, the list is assumed to be a reasonably complete inventory of all steroids tested for this effect and

Table 135 A. *First step (Ctd.): Other inactive steroids.* (Tested at 0.5 mg dose level unless otherwise stated)

1 IM 522[a]		17β-Hydroxy-4-estren-3-one 17-trichloroacetate *19-Nortestosterone trichloroacetate* (Bio. Research Lab.) (B.R.L.) E^4 *II″$_{17\beta/Cl//2,2,2}$ on$_3$
2 IM 505		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-tribromoacetate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *II″$_{17\beta/Br//2,2,2}$
3 IM 504		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-trichloroacetate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *II″$_{17\beta/Cl//2,2,2}$
4 IM 506		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-trifluoroacetate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *II″$_{17\beta/F//2,2,2}$
5 IM 513		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-[2′-hydroxy]propionate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *III″$_{17\beta/ol//2}$
6 IM 507		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-[2′-hydroxy-2′-methyl]propionate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *III″$_{17\beta/Iol//2}$
7 IM 509		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-pentanoate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *V″$_{17\beta}$
8 IM 510		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-heptanoate (B.R.L.) $E^{1,3,5(10)}$ ol$_3$ *VII″$_{17\beta}$

[a] Underneath the serial number of the compounds, the "IM" numbers (for Institut de Médecine et de Chirurgie expérimentales) are mentioned. These identify steroids in our collection and remain the same in all Tables as well as in other publications from this Institute.

Table 135 A (continued)

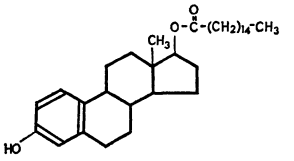
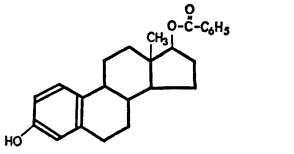
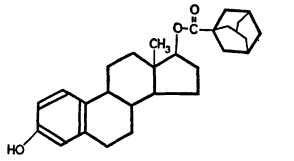
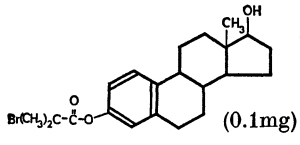
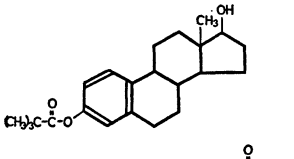
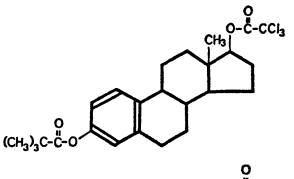
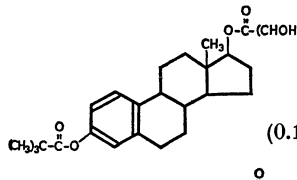
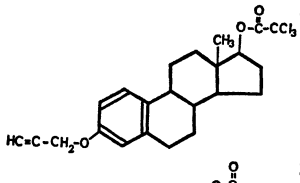
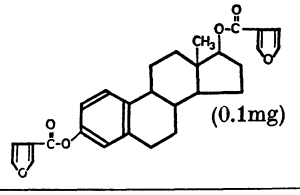
9 IM 511		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-palmitate (B.R.L.) $E^{1,3,5(10)}$ ol_3 *XVI″$_{17\beta}$
10 IM 512		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-benzoate (B.R.L.) $E^{1,3,5(10)}$ ol_3 *Bz″$_{17\beta}$
11 IM 508		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-adamantoate (B.R.L.) $E^{1,3,5(10)}$ ol_3 *adamantoate$_{17\beta}$
12 IM 519	 (0.1mg)	3,17β-Dihydroxy-1,3,5(10)-estratriene 3-[2′-bromo-2′-methyl]propionate (B.R.L.) $E^{1,3,5(10)}$ $ol_{17\beta}$ *III″$_{3/I,\ Br//2}$
13 IM 518		3,17β-Dihydroxy-1,3,5(10)-estratriene 3-trimethylacetate (B.R.L.) $E^{1,3,5(10)}$ $ol_{17\beta}$ *II″$_{3/I//2,2,2}$
14 IM 514		3,17β-Dihydroxy-1,3,5(10)-estratriene 17-trichloroacetate 3-trimethylacetate (B.R.L.) $E^{1,3,5(10)}$ *II″$_{3/I//2,2,2}$ *II″$_{17\beta/Cl//2,2,2}$
15 IM 516	 (0.1mg)	3,17β-Dihydroxy-1,3,5(10)-estratriene 3-trimethylacetate 17-methyltartrate (B.R.L.) $E^{1,3,5(10)}$ *II″$_{3/I//2,2,2}$ *methyltartrate$_{17\beta}$
16 IM 515		3,17β-Dihydroxy-1,3,5(10)-estratriene 3-propargyl ether 17-trichloroacetate (B.R.L.) $E^{1,3,5(10)}$ *II″$_{17\beta/Cl//2,2,2}$ $\theta III^2{}_3$
17 IM 517	 (0.1mg)	3,17β-Dihydroxy-1,3,5(10)-estratriene 3,17-difuroate (B.R.L.) $E^{1,3,5(10)}$ *furoate$_{3,17\beta}$

Table 135 A (continued)

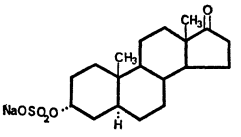
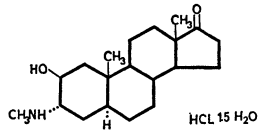
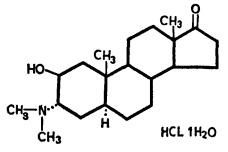
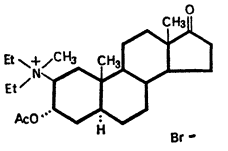
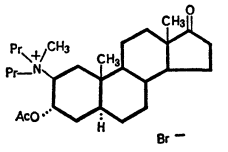
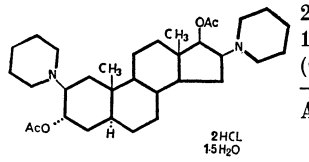
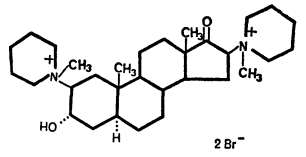
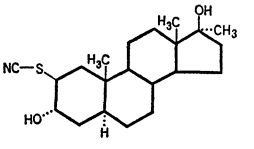
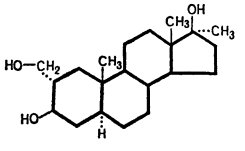
18 IM 191		3α-Hydroxy-5α-androstan-17-one 3-sodium sulphate (Organon) **A5α** *S″$Na_{3\alpha}$ on_{17}
19 IM 313		2β-Hydroxy-3α-methylamino- 5α-androstan-17-one hydrochloride hydrate (Organon) **A5α** $ol_{2\beta}on_{17}NHI_{3\alpha}$ · HCl 15 H_2O
20 IM 140		3α-Dimethylamino-2β-hydroxy- 5α-androstan-17-one hydrochloride monohydrate (Organon) **A5α** $ol_{2\beta}on_{17}N_{3\alpha/I//1,1}$ · HCl H_2O
21 IM 284		2β-Diethylamino-3α-hydroxy-5α-androstan -17-one methobromide 3-acetate (Organon) [**A5α** *II″$_{3\alpha}$ $on_{17}N_{2\beta/I,II,II}$] · Br^-
22 IM 260		2β-Dipropylamino-3α-hydroxy-5α-androstan- 17-one methobromide 3-acetate (Organon) [**A5α** *II″$_{3\alpha}$ $on_{17}N_{2\beta/I,III,III}$] · Br^-
23 IM 453		2β,16β-Dipiperidino-5α-androstane-3α, 17β-diol diacetate dihydrochloride (Organon) **A5α** *II″$_{3\alpha,17\beta}$ $piperidino_{2\beta,16\beta}$ · 2HCl 15 H_2O
24 IM 185		2β, 16β-Dipiperidino-3α-hydroxy-5α-androstan- 17-one dimethobromide (Organon) [**A5α** $ol_{3\alpha}$ on_{17} $piperidino_{2\beta,16\beta/I//1,1}$] · $2Br^-$
25 IM 419		Thiocyanic acid 3α, 17β-dihydroxy-17-methyl-5α- androstan-2-yl-ester (SC-12697) **A5α** $I_{17\alpha}$ $ol_{3\alpha,17}SCN_{2\beta}$
26 IM 442		17α-Methyl-3β, 17-dihydroxy-5α-androstane-2α- hydroxymethyl (Syntex) **A5α** $I_{17\alpha}Iol_{2\alpha}$ $ol_{3\beta,17}$

Table 135 A (continued)

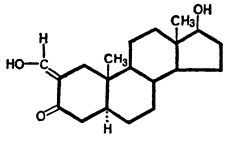
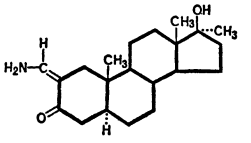
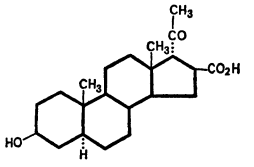
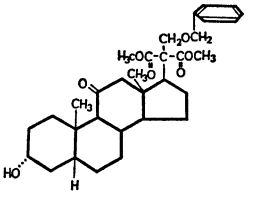
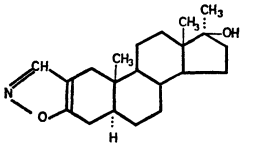
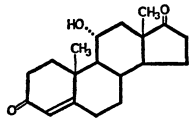
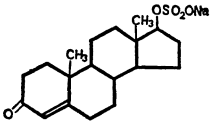
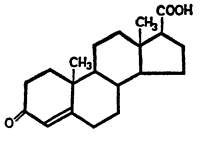
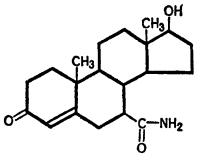
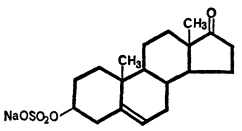
27 IM 451		17β-Hydroxy-3-oxo-5α-androstane-2-hydroxy-methylene (Syntex) **A5αCHOH$_2$ol$_{17\beta}$on$_3$**
28 IM 443		17α-Methyl-17-hydroxy-3-oxo-5α-androstan-2-aminomethylene (Syntex) **A5αI$_{17\alpha}$(CH NH$_2$)$_2$ol$_{17}$on$_3$**
29 IM 441		3β-Hydroxy-20-oxo-5α,17α-pregnane-16β-carboxylic acid (Syntex) **A5αI″$_{16\beta}$II$_{17\alpha/on//1}$ol$_3$**
30 IM 495		3α-Hydroxy-11-oxo-5β-androstane-17β[α-benzyloxymethyl]-dimethyl-malonate (Roussel) **A5β benzyloxy methyl-dimethyl malonate** $_{17\beta}$ol$_{3\alpha}$on$_{11}$
31 IM 150		17β-Hydroxy-17-methyl-5α-androstan-[2,3-d]isoxazol (Sterling Winthrop) **A5α^{2}I$_{17\alpha}$isoxazolyl$_{2-3/3,4}$ol$_{17}$**
32 IM 563		11α-Hydroxy-4-androstene-3,17-dione (U-1680) **A^{4}ol$_{11\alpha}$on$_{3,17}$**
33 IM 199		17β-Hydroxy-4-androsten-3-one 17-sodium sulphate (Organon) **A^4 *S″Na$_{17\beta}$on$_3$**
34 IM 502		3-Oxo-4-androstene-17β-carboxylic acid (Roussel) **A^{4}I″$_{17\beta}$on$_3$**
35 IM 450		17β-Hydroxy-3-oxo-4-androstene-7β-carboxamide (Syntex) **A^4(CONH$_2$)$_{7\beta}$ol$_{17\beta}$on$_3$**

Table 135 A (continued)

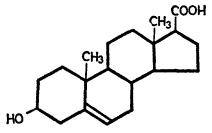
36
IM 362



3β-Hydroxy-5-androsten-17-one
3-sodium sulfate
(Organon)

A^{5} *S″Na$_{3\beta}$on$_{17}$

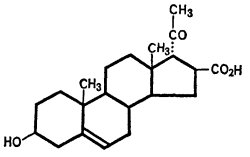
37
IM 501



3β-Hydroxy-5-androstene-17β-carboxylic acid
(Roussel)

A^{5}**I**″$_{17\beta}$ol$_{3\beta}$

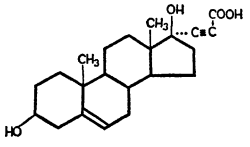
38
IM 440



3β-Hydroxy-20-oxo-17α-pregn-5-ene-16β-carboxylic acid
(Syntex)

A^{5}**I**″$_{16\beta}$**II**$_{17\alpha/}$on$_{//1}$ol$_{3\beta}$

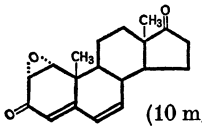
39
IM 500



3β,17β-Dihydroxy-5-pregnen-20-yne-21-carboxylic acid
(Roussel)

A^{5}**III**$^{2,2''}{}_{17\alpha}$ol$_{3\beta,17}$

40
IM 363

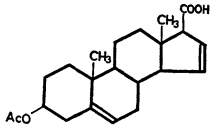


(10 mg)

1α,2α-Epoxy-4,6-androstadiene-3,17-dione
(Linet)

A^{4,6}on$_{3,17}$ $\theta_{1\alpha-2}$

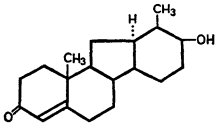
41
IM 498



3β-Acetoxy-5,15-androstadiene-17-carboxylic acid
(Roussel)

A^{5,15}**I**″$_{17\beta}$ *II″$_{3\beta}$

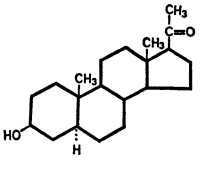
42
IM 452



17β-Hydroxy-12α-13β-etiojerv-4-en-3-one
(SC-19886)

An18,nC,hoD^{4}**I**$_{17\alpha}$ol$_{17\beta}$on$_{3}$

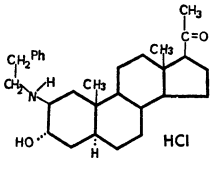
43
IM 447



3β-Hydroxy-5α-pregnan-20-one
(Steraloids)

P 5α ol$_{3\beta}$on$_{20}$

44
IM 135

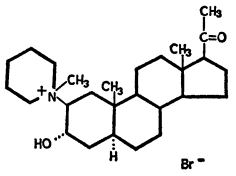


2β-(2′-Phenyl-ethylamino)-3α-hydroxy-5α-pregnan-20-one hydrochloride
(Organon)

P 5α ol$_{3\alpha}$on$_{20}$NH$_{2\beta/II//Ph///2}$ · HCl

Table 135 A (continued)

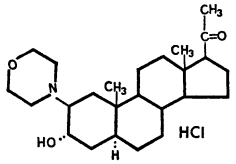
45
IM 82



2β-Piperidino-3α-hydroxy-5α-pregnan-20-one methobromide
(Organon)

[**P5α** $ol_{3\alpha}on_{20}$piperidino$_{2\beta/I//1}$] · Br^-

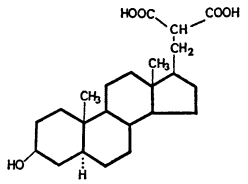
46
IM 460



2β-Morpholino-3α-hydroxy-5α-pregnan-20-one hydrochloride
(Organon)

P5α $ol_{3\alpha}on_{20}$morpholino$_{2\beta}$ · HCl

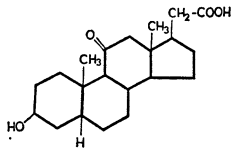
47
IM 496



3β-Hydroxy-5α-pregnane-21,21-dicarboxylic acid
(Roussel)

P5αI″$_{21,21}ol_{3\beta}$

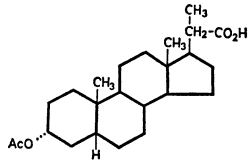
48
IM 497



3β-Hydroxy-11-oxo-5β-pregnan-21-oic acid
(Roussel)

P5β″$_{21}ol_{3\alpha}on_{11}$

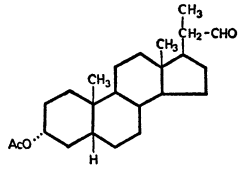
49
IM 545



3α-Acetoxy-23,24-bisnor-5β-cholan-22-oic acid
(B.R.L.)

P5βI″$_{20}$ *II″$_{3\alpha}$

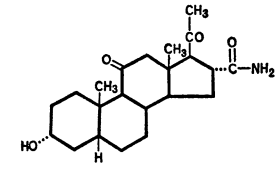
50
IM 544



3α-Acetoxy-23,24-bisnor-5β-cholan-22-al
(B.R.L.)

P5βI al_{20} *II″$_{3\alpha}$

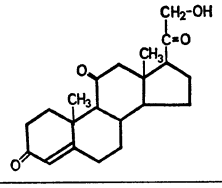
51
IM 436



3α-Hydroxy-11,20-dioxo-5β-pregnane-16α-carboxamide
(U-35827)

P5β $(CONH_2)_{16\alpha}ol_{3\alpha}on_{11,20}$

52
IM 557



21-Hydroxy-4-pregnene-3,11,20-trione
(U-0569)

P$^4ol_{21}on_{3,11,20}$

Table 135 A (continued)

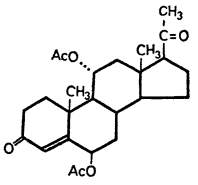
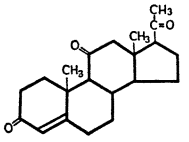
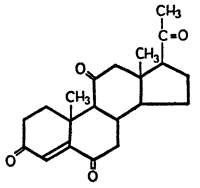
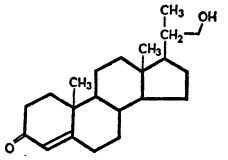
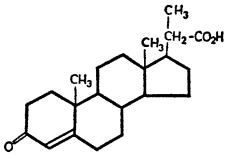
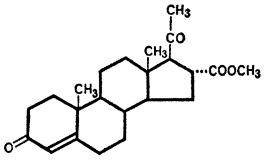
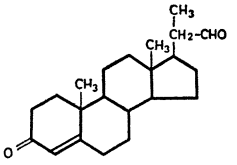
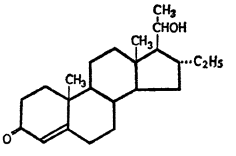
53 IM 556		6β,11α-Dihydroxy-4-pregnene-3,20-dione diacetate (U-0471) **P4 *II″$_{6\beta,11\alpha}$on$_{3,20}$**
54 IM 561		4-Pregnene-3,11,20-trione (B.R.L.) **P4on$_{3,11,20}$**
55 IM 555		4-Pregnene-3,6,11,20-tetrone (U-0460) **P4on$_{3,6,11,20}$**
56 IM 546		22-Hydroxy-23,24-bisnorchol-4-en-3-one (B.R.L.) **P4Iol$_{20}$on$_3$**
57 IM 548		3-Oxo-23,24-bisnor-4-cholen-22-oic acid (B.R.L.) **P4I″$_{20}$on$_3$**
58 IM 416		3,20-Dioxo-4-pregnene-16α-carboxylic acid methyl ester (U-35258) **P4I″$_{16\alpha}$/*$_{Iol}$on$_{3,20}$**
59 IM 547		3-Oxo-23,24-bisnor-4-cholen-22-al (B.R.L.) **P4Ial$_{20}$on$_3$**
60 IM 207		16α-Ethyl-20β-hydroxy-4-pregnen-3-one (Organon) **P4II$_{16\alpha}$ol$_{20}$on$_3$**

Table 135 A (continued)

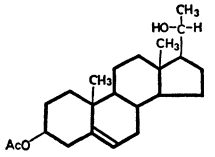
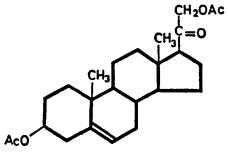
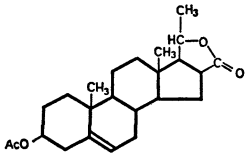
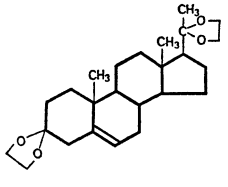
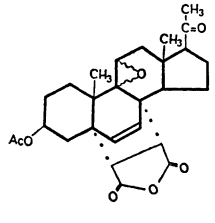
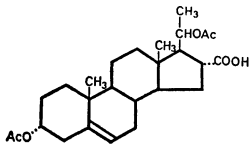
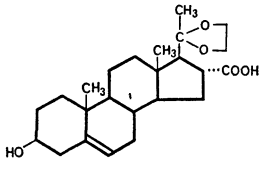
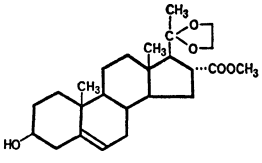
61 IM 524		5-Pregnen-3β-20β-diol 3-acetate (B.R.L.) **P**^{5}ol$_{20\beta}$ *II$''_{3\beta}$
62 IM 523		3β,21-Dihydroxy-5-pregnen-20-one diacetate (B.R.L.) **P**^{5} *II$''_{3\beta,21}$on$_{20}$
63 IM 467		16β-Carboxy-5-pregnene-3β,20β-diol γ-lactone 3-acetate (SC-5634) **P**^{5} I$''\gamma$lac$_{16\beta-20}$*II$''_{3\beta}$
64 IM 525		3,20-Dioxo-5-pregnene 3,3-20,20 bis-(ethylenedioxy) (B.R.L.) **P**$^{5}\vartheta\vartheta$II$_{3,20}$
65 IM 552		9ξ,11ξ-Epoxy-3β- hydroxy-5α-pregn-6-en-20-one- 5,8α-maleic anhydride adduct, acetate (U-0156) **P**$^{5}\alpha^{6}$ *II$''_{3\beta}$on$_{20}$ $\theta_{9\xi-11}$ maleic anhydride adduct$_{5\alpha-8}$
66 IM 465		16α-Carboxy-5-pregnene-3α,20-diol diacetate (SC-5934) **P**^{5}I$''_{16\alpha}$ *II$''_{3\alpha,20}$
67 IM 415		20,20-(Ethylenedioxy)-3β-hydroxy-5-pregnene- 16α-carboxylic acid (U-12872 E) **P**^{5}I$''_{16\alpha}$ol$_{3\beta}\theta\theta$II$_{20}$
68 IM 438		20,20-(Ethylenedioxy)-3β-hydroxy-5-pregnene- 16α-carboxylic acid methyl ester (U-36548) **P**^{5}I$''_{16\alpha}$/*$_{\text{I}o\text{l}3\beta}\theta\theta$II$_{20}$

Table 135 A (continued)

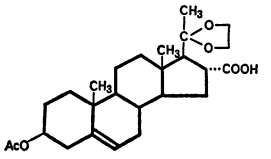
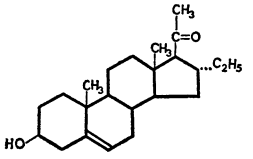
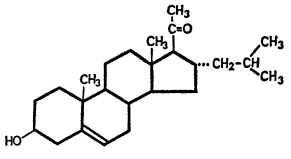
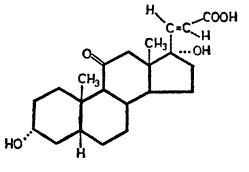
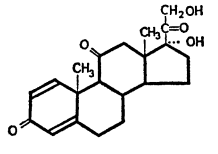
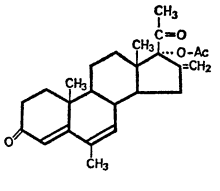
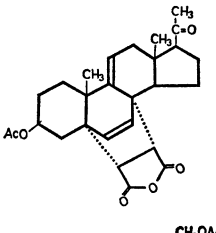
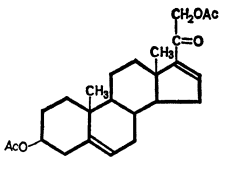
69 IM 437		20,20-(Ethylenedioxy)-3β-acetoxy-5-pregnene-16α-carboxylic acid (U-35939) $P^5I''_{16\alpha}$ *$II''_{3\beta\theta\theta}II_{20}$
70 IM 71		3β-Hydroxy-16α-ethyl-5-pregnen-20-one (Organon) $P^5II_{16\alpha}ol_{3\beta}on_{20}$
71 IM 210		3β-Hydroxy-16α-isobutyl-5-pregnen-20-one (Organon) $P^5III_{16\alpha/I//2}ol_{3\beta}on_{20}$
72 IM 499		3α,17α-Dihydroxy-11-oxo-5β-pregn-20-ene-21-carboxylic acid (Roussel) $P5\beta^{20}I''_{21}ol_{3\alpha,17}on_{11}$
73 IM 526		17,21-Dihydroxy-1,4-pregnadiene-3,11,20-trione *Prednisone* (B.R.L.) $P^{1,4}ol_{17,21}on_{3,11,20}$
74 IM 503		17-Hydroxy-6-methyl-16-methylene-4,6-pregnadiene-3,20-dione acetate (U-21240) $P^{4,6}I_6(CH_2)_{16}$ *$II''_{17}on_{3,20}$
75 IM 553		3β-Hydroxy-5α-pregna-6,9(11)-dien-20-one-5,8α-maleic anhydride adduct, acetate (U-0157) $P5\alpha^{6,9(11)}$ maleic anhydride $adduct_{5\alpha-8}$
76 IM 99		3β,21-Dihydroxy-5,16-pregnadien-20-one diacetate (Organon) $P^{5,16}$ *$II''_{3\beta,21}on_{20}$

Table 135 A (continued)

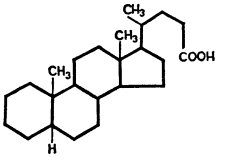
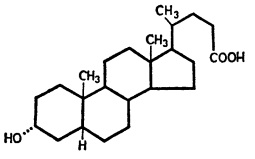
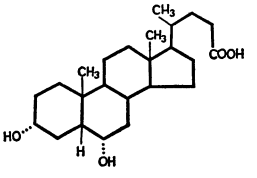
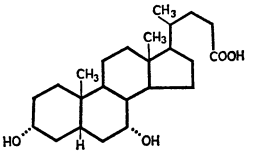
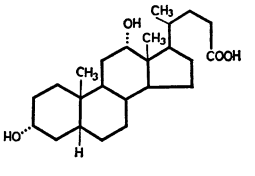
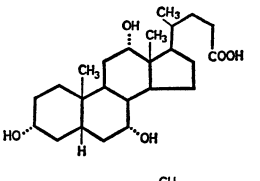
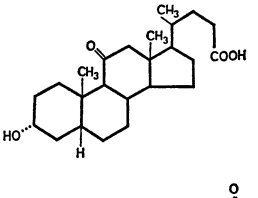
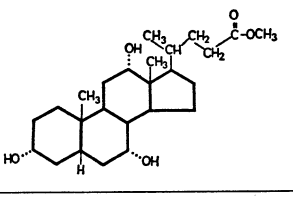
77 IM 296		5β-Cholan-24-oic acid (Roussel) **CH5β″$_{24}$**
78 IM 493		3α-Hydroxy-5β-cholan-24-oic acid *Lithocholic acid* (Roussel) **CH5β″$_{24}$ol$_{3\alpha}$**
79 IM 485		3α,6α-Dihydroxy-5β-cholan-24-oic acid. *Hyodesoxylic acid* (Roussel) **CH5β″$_{24}$ol$_{3\alpha,6\alpha}$**
80 IM 492		3α,7α-Dihydroxy-5β-cholan-24-oic acid. *Chenodesoxylic acid* (Roussel) **CH5β″$_{24}$ol$_{3\alpha,7\alpha}$**
81 IM 490		3α,12α-Dihydroxy-5β-cholan-24-oic acid. *Desoxycholic acid* (Roussel) **CH5β″$_{24}$ol$_{3\alpha,12\alpha}$**
82 IM 488		3α,7α,12α-Trihydroxy-5β-cholan-24-oic acid. *Cholic acid* (Roussel) **CH5β″$_{24}$ol$_{3\alpha,7\alpha,12\alpha}$**
83 IM 487		3α-Hydroxy-11-oxo-5β-cholan-24-oic acid (Roussel) **CH5β″$_{24}$ol$_{3\alpha}$on$_{11}$**
84 IM 550		3α,7α,12α-Trihydroxy-5β-cholan-24-oic acid methyl ester (U-0021) **CH5β″ ol$_{3\alpha,7\alpha,12\alpha}$*I$_{24}$**

Table 135 A (continued)

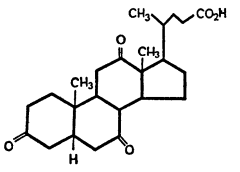
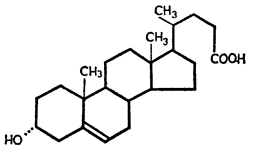
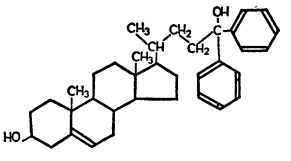
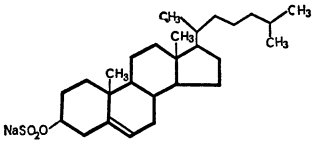
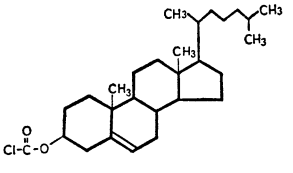
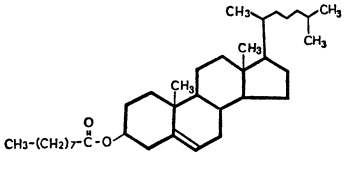
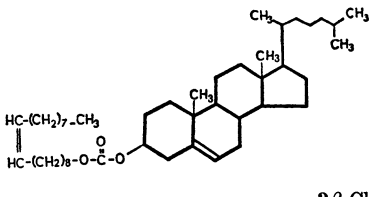
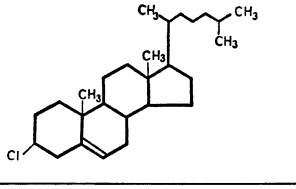
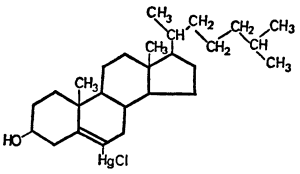
85 IM 541		3,7,12-Trioxo-5β-cholan-24-oic acid (B.R.L.) **CH 5 β″$_{24}$on$_{3,7,12}$**
86 IM 491		3α-Hydroxy-5-cholen-24-oic acid (Roussel) **CH^{5}″$_{24}$ol$_{3\alpha}$**
87 IM 554		24,24-Diphenyl-5-cholene-3β,24-diol (U-0359) **CH^{5}Ph$_{24,24}$ol$_{3\beta,24}$**
88 IM 198		3β-Hydroxy-5-cholestene 3-sodium sulphate (Organon) **CHT^{5} *S″Na$_{3\beta}$**
89 IM 532		5-Cholesten-3β-ol chloroformate (B.R.L.) **CHT^{5} *I″Cl$_{3\beta}$**
90 IM 530		5-Cholesten-3β-ol pelargonate (B.R.L.) **CHT^{5} *IX″$_{3\beta}$**
91 IM 531		5-Cholesten-3β-ol oleyl carbonate (B.R.L.) **CHT^{5} *XIX^{10}″$_{3\beta}$**
92 IM 529		3β-Chloro-5-cholestene (B.R.L.) **CHT^{5}Cl$_{3\beta}$**

Table 135 A (continued)

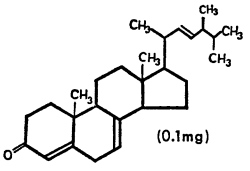
93
IM 558



6-(Chloromercuri)-5-cholesten-3β-ol
(U-0617)

CHT^{5}ol$_{3\beta}$HgCl$_{6}$

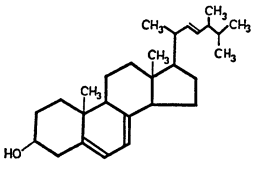
94
IM 536



4,7,22-Ergostatrien-3-one
(B.R.L.)

ER^{4,7,22}on$_{3}$

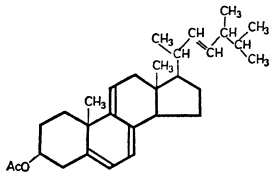
95
IM 535



5,7,22-Ergostatrien-3β-ol
Ergosterol
(B.R.L.)

ER^{5,7,22}ol$_{3\beta}$

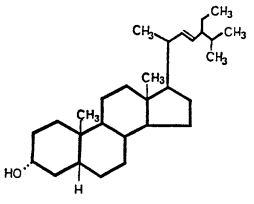
96
IM 551



5,7,9(11),22-Ergostatetraen-3β-ol acetate
(U-0025)

ER$^{5,7,9(11),22}$ *II$''_{3\beta}$

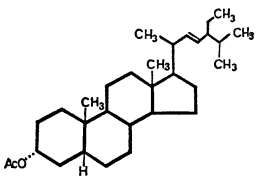
97
IM 537



3α-Hydroxy-5β-stigmast-22-ene
(B.R.L.)

ST5β^{22}ol$_{3\alpha}$

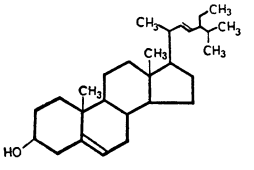
98
IM 538



3α-Hydroxy-5β-stigmast-22-ene acetate
(B.R.L.)

ST5β^{22} *II$''_{3\alpha}$

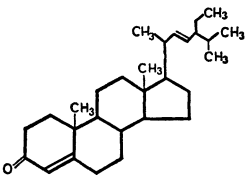
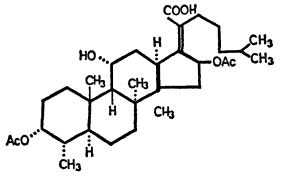
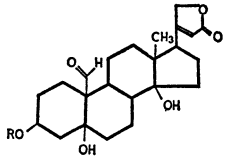
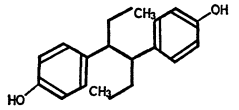
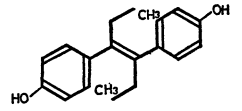
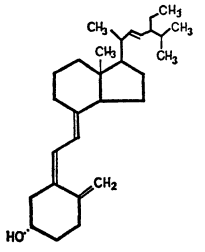
99
IM 533



5,22-Stigmastadien-3β-ol
Stigmasterol
(B.R.L.)

ST^{5,22}ol$_{3\beta}$

Table 135 A (continued)

100 IM 534		4,22-Stigmastadien-3-one (B.R.L.) $ST^{4,22}on_3$
101 IM 494		3α,16β-Diacetoxy-11α-hydroxy-31-nor-5α,8α,9β,13α,14β-dammaran-17(20)-en-21-oic acid (Roussel) **Dammaran $n31^{17(20)}5\alpha,8\alpha,9\beta,13\alpha,14\beta I''_{21}ol_{11\alpha}$ $*II_{3\alpha,16\beta}$**
102 IM 542		3β,5,14-Trihydroxy-19-oxo-5β,14β-card-20(22)-enolide mixture of 3-glycosides (β-glucose-β-glucose-cymarose-, β-glucose-cymarose-, cymarose) (B.R.L.) $CAR5\beta^{20(22)}Ial_{19\beta}ol_{5,14\beta}$ $*R_3$
103 IM 521		3,4-Bis(p-hydroxy-phenyl)-n-hexane **(Hexestrol)** (B.R.L.)
104 IM 520		3,4-Bis(p-hydroxy-phenyl)-n-hex-3-ene **(Diethylstilbestrol)** (B.R.L.)
105 IM 539		Calciferol (Vitamin D_2) (B.R.L.) Calciferol

eminently suitable for pharmaco-chemical correlation studies. It must be kept in mind, however, that these assessments of activity are subjective and hence only very high or essentially negative ratings deserve serious consideration as indexes of strong and negligible activity respectively. This is all the more true since, in many instances, the total number of rats had to be limited to five per group because the majority of the steroids tested were available only in small amounts. On the other hand, all compounds which are highly potent in increasing resistance to one or both of these toxicants were retested at lower dose levels.

Table 135B. *First step (ctd.): Synopsis of all 500 steroids tested for their ability to prevent digitoxin and indomethacin intoxication*

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
1					
IM 360	$G^4H_{13\alpha,17\alpha}ol_{17}on_3$ (d-Norbolethone)	10	3	3	**0.3**
2					
IM 343	$G^4H_{13,17\alpha}ol_{17}on_3$ (d,l-Norbolethone)	10	3	3	0.3
		0.5	0	0.5	**0.5**
		0.1	0	—	0
3					
IM 142	$E^4ol_{17\beta}$	10	0	3	**0.15**
4					
IM 147	E^4on_{17}	10	0	2	**0.1**
5					
IM 188	$E^4on_{3,17}$	10	1	2	**0.15**
6					
IM 146	$E^4ol_{17\beta}on_3$ (Nortestosterone)	10	2	0	**0.1**
7					
IM 522	$E^{4*}H''_{17\beta/Cl//2,2,2}on_3$	0.5	0	0	0
8					
IM 340	$E^{4*}III''Ph_{17\beta}on_3$ (Nandrolonephenyl pr.)	10	0	0	0
9					
IM 339	$E^{4*}X''_{17\beta}on_3$ (Nondrolone dec.)	10	0	0	0
10					
IM 186	$E^{1(10),5}ol_{3\beta}on_{17}$	10	0	3	**0.15**
11					
IM 33	$E^{1,3,5(10)}ol_{3,17\beta}$ (Estradiol)	10	0	—	0
		1	0	0.5	**0.25**
12					
IM 505	$E^{1,3,5(10)}ol_3{*}H''_{17\beta/Br//2,2,2}$	0.5	0	0	0
13					
IM 504	$E^{1,3,5(10)}ol_3{*}H''_{17\beta/Cl//2,2,2}$	0.5	0	0	0

[a] As in the previously reported data the figures indicate the means of the statistical difference grades of protection ranging from "0" (no protection) to "3" (perfect protection). For further data concerning the techniques *cf.* p. VIII.

[b] Based on the formula $\frac{D+I}{2} \times \frac{1}{mg}$, where D = reading for digitoxin, I = indomethacin, mg = the dose tested.

It is noteworthy that, among the 500 steroids listed in Table 135B, only 20 exhibited an activity index of 10 or more. It is hardly coincidental that all compounds of this select group are 16α-carbonitriles, except for TMACN (Cpd. 149) which is a 2α-carbonitrile, the 16α-17-methyl-oxazolyl (Cpd. 329), CS-1 (Cpd. 191), and dexamethasone acetate (Cpd. 360). It may be significant that within this small group of exceptions we find: one steroid (Cpd. 149) which also carries a carbonitrile substituent on the ring, although in the 2α-position, two 9α-fluoro-compounds (CS-1 and dexamethasone), one steroid (Cpd. 329) substituted at 16α, although not by a carbonitrile. It will be interesting to see whether other 16α-substituted or halogenated steroids possess considerable catatoxic activity and whether the latter can be further increased by the introduction of several apparently advantageous substituents into the same molecule.

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
14					
IM 506	$E^{1,3,5(10)}ol_3$*II″$_{17\beta/F//2,2,2}$	0.5	0	0	0
15					
IM 513	$E^{1,3,5(10)}ol_3$*III″$_{17\beta/ol//2}$	0.5	0	0	0
16					
IM 507	$E^{1,3,5(10)}ol_3$*III″$_{17\beta/I,ol//2}$	0.5	0	0	0
17					
IM 509	$E^{1,3,5(10)}ol_3$*V″$_{17\beta}$	0.5	0	0	0
18					
IM 510	$E^{1,3,5(10)}ol_3$*VII″$_{17\beta}$	0.5	0	0	0
19					
IM 511	$E^{1,3,5(10)}ol_3$*XVI″$_{17\beta}$	0.5	0	0	0
20					
IM 512	$E^{1,3,5(10)}ol_3$*Bz″$_{17\beta}$	0.5	0	0	0
21					
IM 508	$E^{1,3,5(10)}ol_3$*adamantoate$_{17\beta}$	0.5	0	0	0
22					
IM 518	$E^{1,3,5(10)}ol_{17\beta}$*II″$_{3/I//2,2,2}$	0.5	0	0	0
23					
IM 519	$E^{1,3,5(10)}ol_{17\beta}$*III″$_{3/I,Br//2}$	0.1	0	0	0
24					
IM 514	$E^{1,3,5(10)}$*II″$_{3/I//2,2,2}$ *II″$_{17\beta/Cl//2,2,2}$	0.5	0	0	0
25					
IM 516	$E^{1,3,5(10)}$*II″$_{3/I//2,2,2}$ *methyltartrate$_{17\beta}$	0.1	0	0	0
26					
IM 517	$E^{1,3,5(10)}$*furoate$_{3,17\beta}$	0.1	0	0	0
27					
IM 21	$E^{1,3,5(10)}ol_3on_{17}$ (Estrone)	10	0	2.5	**0.125**
28					
IM 515	$E^{1,3,5(10)}$*II″$_{17\beta/Cl//2,2,2}\theta III^2{}_3$	0.5	0	0	0
29					
IM 194	$E^{1,3,5(10)}on_{17}\theta I_3$	10	0	1.5	**0.075**
30					
IM 356	$E^{4,9,11}on_{3,17}$	10	0.5	0	**0.025**
31					
IM 355	$E^{4,9,11}on_3\theta III^2{}_{17\beta/I//2}$	10	1.5	3	**0.225**
		0.5	—	0	0
32					
IM 435	$E5\beta CN_5II^{1-1}{}_{17\alpha}ol_{17}on_3$	0.5	0	0	0
33					
IM 428	$E5\beta CN_5III^2{}_{17\alpha/I//2}ol_{17}on_3$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
34					
IM 430	$E5\xi CN_5III^2{}_{17\alpha/I//2}ol_{17}on_3$	0.5	0	0	0
35					
IM 192	$E^4I_{17\alpha}ol_{17}on_3$	10	3	3	**0.3**
		0.5	—	0	0
36					
IM 79	$E^4I_{17\alpha}ol_{4,17}on_3$	10	0	3	**0.15**
		0.5	—	0	0
37					
IM 59	$E^4II_{17\alpha}ol_{17}$ (Ethylestrenol)	10	3	3	**0.3**
		0.5	0	0	0
38					
IM 76	$E^4II_{17\alpha}ol_{17}{*}III''_{3\beta}$	10	0	3	**0.15**
39					
IM 57	$E^4II_{17\alpha}ol_{17}on_3$ (Norethandrolone)	10	3	0.5	**0.175**
40					
IM 193	$E^4II^1{}_{17\alpha}ol_{17}on_3$	10	1	2	**0.15**
41					
IM 315	$E^4III''ylac_{17\alpha}on_3$	10	3	2.5	**0.275**
42					
IM 101	$E^4III^2{}_{17\alpha}ol_{17}$ (Allylestrenol)	10	0	3	**0.15**
43					
IM 338	$E^{5(10)}II^{1-1}{}_{17\alpha}ol_{17}on_3$ (Norethynodrel)	10	0	0	0
44					
IM 392	$E^{1,3,5(10)}I_{16\alpha}CN_{16}{*}II''_{17\beta}\theta I_3$	0.5	0	0	0
45					
IM 395	$E^{1,3,5(10)}I_{16\xi}CN_{16}on_{17}\theta I_3$	0.5	0	0	0
46					
IM 35	$E^{1,3,5(10)}II^{1-1}{}_{17\alpha}ol_{3,17}$ (Ethynylestradiol)	10	0	0	0
47					
IM 304	$E^{1,3,5(10)}II^{1-1}{}_{17\alpha}ol_{17}\theta I_{3\beta}$	10	0	3	**0.15**
		0.5	—	0	0
48					
IM 168	$E^{1,3,5(10),7}furyl_{17\alpha/3}ol_{17}{*}II''_{3\beta}$	10	0	2.5	**0.125**
49					
IM 169	$E^{1,3,5(10),7}furyl_{17\alpha/3}ol_{17}\theta cycV/_{3\beta}$	10	0	3	**0.15**
50					
IM 380	$E^{1,3,5(10),16}CN_{17}{*}II''_3$	0.5	0	0	0
51					
IM 400	$E^{1,3,5(10),16}CN_{17}\theta I_3$	0.5	0	0	0
52					
IM 326	$E(O_2)^{4,9(10)}ol_{17\beta}on_3$	10	3	0	**0.15**
		0.5	1	—	2
53					
IM 328	$E(O_2)^{4,9,11}ol_{17\beta}on_3$	10	1.5	0.5	**0.1**

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
54					
IM 336	$Eab^{10(5\rightarrow4)}I_{7\alpha,17\alpha}ol_{17}on_{3,5}$	10	2	1.5	**0.175**
55					
IM 196	$A5\alpha ol_{11\beta,17\beta}$	10	3	0	**0.15**
56					
IM 183	$A5\alpha on_{3,17}$	10	3	0.5	**0.175**
57					
IM 100	$A5\alpha ol_{17\beta}on_3$ (Androstanolone)	10	2	0	**0.1**
58					
IM 191	$A5\alpha{*}S''Na_{3\alpha}on_{17}$	0.5	0	0	0
59					
IM 327	$A5\alpha on_{17}\theta_{2\beta-19}Cl_{3\alpha}$	10	0	2	**0.1**
60					
IM 252	$A5\alpha{*}II''_{3\beta}on_{17}Cl_{5,6\beta}$	10	0	0	0
61					
IM 229	$A5\alpha NOH_{17}$	10	3	3	**0.3**
62					
IM 213	$A5\alpha ol_{11\beta}NOH_{17}$	10	3	3	**0.3**
63					
IM 224	$A5\alpha ol_{11\beta}(NH_2)_{17\beta}$	10	1.5	0	**0.075**
64					
IM 246	$A5\alpha ol_{3\beta}$sp imidazolidyl$_{17/on//2,5}$	10	0	0	0
65					
IM 319	$A5\alpha ol_{3\alpha}on_{17}(N_3)_{2\beta}$	10	0	0	0
66					
IM 313	$A5\alpha ol_{2\beta}on_{17}NH_{3\alpha/I} \cdot HCl\,15H_2O$	0.5	0	0	0
67					
IM 140	$A5\alpha ol_{2\beta}on_{17}N_{3\alpha/I//1,1} \cdot HClH_2O$	0.5	0	0	0
68					
IM 284	$[A5\alpha{*}II''_{3\alpha}on_{17}N_{2\beta/I,II,II}]Br^-$	0.5	0	0	0
69					
IM 260	$[A5\alpha{*}II''_{3\alpha}on_{17}N_{2\beta/I,III,III}]Br^-$	0.5	0	0	0
70					
IM 253	$A5\alpha{*}II''_{3\beta}on_{17}Cl_5(N_3)_{6\beta}$	10	0	0	0
71					
IM 453	$A5\alpha{*}II''_{3\alpha,17\beta}$piperidino$_{2\beta,16\beta} \cdot \cdot 2\,HCl\,15\,H_2O$	0.5	0	0	0
72					
IM 361	$[A5\alpha{*}II''_{3\alpha,17\beta}$piperidino$_{2\beta,16\beta}]$ (Pancuronium Br) $\cdot$ 2 Br^-H_2O	10	0	1.5	**0.075**
73					
IM 185	$[A5\alpha ol_{3\alpha}on_{17}$piperidino$_{2\beta,16\beta/I/1,1}] \cdot 2\,Br$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
74					
IM 325	$A5\alpha{*}III''_{17\beta/cycV//3}S_{2\alpha-3}$	10	0	0	0
75					
IM 250	$A5\beta{*}II''_{3\beta}on_{17}\theta_{5\beta-6}$	10	0	0	0
76					
IM 141	$A^4on_{3,17}$ (Androstenedione)	10	1	0.5	**0.075**
77					
IM 8	$A^4on_{3,11,17}$ (Andrenosterone)	10	0	—	0
78					
IM 53	$A^4ol_{17\beta}on_3$ (Testosterone)	10	3	0.5	**0.175**
		0.5	0	0	0
79					
IM 131	$A^4{*}II''_{17\beta}on_3$ (Testosterone acetate)	10	0	0	0
80					
IM 58	$A^4{*}III''_{17\beta}on_3$ (Testosterone pr.)	10	0	0	0
81					
IM 199	$A^4{*}S''Na_{17\beta}on_3$	0.5	0	0	0
82					
IM 226	$A^4{*}II''_{11\alpha,17\beta}on_3$	10	0	2	**0.1**
83					
IM 563	$A^4ol_{11\alpha}on_{3,17}$	0.5	0	0	0
84					
IM 187	$A^4ol_{19}on_{3,17}$	10	0	0	0
85					
IM 358	$A^4{*}cycVI''_{17\beta}on_3F_{2\alpha}$	10	0	0	0
86					
IM 216	$A^4{*}III''_{17\beta}on_3F_{6\alpha}$	10	0	0	0
87					
IM 41	$A^4ol_{11\beta}on_{3,17}F_{9\alpha}$ (Fluorohydroxyandrostenedione)	10	0.5	1.5	**0.1**
88					
IM 251	$A^4ol_{3\beta}on_{17}(N_3)_{6\beta}$	10	0	3	**0.15**
89					
IM 17	$A^5ol_{3\beta,16\alpha}$ (Cetadiol)	10	0	0	0
90					
IM 139	$A^5{*}II''_{3\beta}{*}Bz''_{17\beta}$	10	0	2.5	**0.125**
91					
IM 31	$A^5ol_{3\beta}on_{17}$ (Dehydroepiandrosterone)	10	2	3	**0.25**
		0.5	0	0.5	0
92					
IM 27	$A^5{*}II''_{3\beta}on_{17}$ (Dehydroepiandrosterone acetate)	10	0	3	**0.15**
		0.5	—	0	0
93					
IM 362	$A^5{*}S''Na_{3\beta}on_{17}$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
94					
IM 184	$A^{5}ol_{3\beta,19}on_{17}$	10	0	0	0
95					
IM 245	$A^{5}ol_{3\beta}$sp imidazolidinyl$_{17/on//2,5}$	10	0	0	0
96					
IM 332	$A^{5}ol_{3\beta}$(2-Disopropylaminoethyl formamido)$_{17\beta}$	10	0	0	0
97					
IM 321	$A^{5}ol_{3\beta}$(3-Dimethylaminopropyl methylamino)$_{17\beta}$ · 2HCl	10	0	0	0
98					
IM 247	A^{5}*II″$_{3\beta}$sp oxazolyl$_{17/I//2}$	10	0	0	0
99					
IM 363	$A^{4,6}on_{3,17}\theta_{1\alpha-2}$	10	0	0	0
100					
IM 153	$A^{4,9(11)}ol_{17\beta}on_{3}$	10	0	1.5	**0.075**
101					
IM 280	**A5αI**$_{1\alpha}ol_{17\beta}on_{3}$	10	0.5	0	**0.025**
102					
IM 189	**A5αI**$_{4\alpha}ol_{17\beta}on_{3}$	10	3	0.5	**0.175**
103					
IM 51/d	**A5αI**$_{17\alpha}ol_{3\beta,11\beta,17}$ (Methyl-androstanetriol)	10	3	3	**0.3**
104					
IM 190	**A5αI**$_{17\alpha}ol_{17}on_{3}$	10	3	0	**0.15**
105					
IM 163	**A5αI**$_{17\alpha}ol_{11\beta,17}on_{3}$	10	3	3	**0.3**
106					
IM 320	**A5αI**$_{17\alpha}ol_{17}\theta I_{3\alpha}$	10 0.5	3 0	1.5 —	**0.225** 0
107					
IM 419	**A5αI**$_{17\alpha}ol_{3\alpha,17}SCN_{2\beta}$	0.5	0	0	0
108					
IM 248	**A5αI**$_{17\alpha}ol_{3\beta,5,17}(N_3)_{6\beta}$	10	0	0.5	**0.025**
109					
IM 454	**A5αI**$_{16\alpha,17\alpha}$*II″$_{3,17}$	10 0.5	3 0	— 0	**0.3** 0
110					
IM 451	**A5αCHOH**$_{2}ol_{17\beta}on_{3}$	0.5	0	0	0
111					
IM 427	**A5αCN**$_{1\alpha}ol_{17\beta}on_{3}$	0.5	0	0	0
112					
IM 433	**A5αCN**$_{1\alpha}$*II″$_{17\beta}on_{3}$	0.5	0	0	0
113					
IM 424	**A5αCN**$_{3\xi}ol_{17\beta}(NH_2)_3$	0.5	0	0	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
114					
IM 434	**A5αCN$_5$ol$_{17\beta}$on$_3$**	0.5	0	0	0
115					
IM 420	**A5βCN$_5$ol$_{17\beta}$on$_3$**	10	0	0	0
		0.03	0	0	0
		0.015	0	0	0
		0.005	0	0	0
		0.001	0	0	0
116					
IM 421	**A5αCN$_5$*III$''_{17\beta}$on$_3$**	0.5	0	0	0
117					
IM 422	**A5βCN$_5$*III$''_{17\beta}$on$_3$**	0.5	0	0	0
118					
IM 442	**A5αI$_{17\alpha}$Iol$_{2\alpha}$ol$_{3\beta,17}$**	0.5	0	0	0
119					
IM 98/a	**A5αI$_{17\alpha}$CHOH$_2$ol$_{17}$on$_3$** (Oxymetholone)	10	3	0	**0.15**
120					
IM 443	**A5αI$_{17\alpha}$(CHNH$_2$)$_2$ol$_{17}$on$_3$**	0.5	0	0	0
121					
IM 425	**A5αI$_{17\alpha}$CN$_5$ol$_{17}$on$_3$**	0.5	0	0	0
122					
IM 382	**A5αI$_{16\beta}$CN$_{17}$ol$_{3\beta,17\xi}$**	0.5	0	0	0
123					
IM 275	**A5αII$_{17\alpha/\text{on}//1}$ol$_{3\beta}\theta_{16\beta-17}$**	10	3	3	**0.3**
124					
IM 276	**A5αII$_{17\alpha/\text{on}//1}$on$_{3,11}\theta_{16\beta-17}$**	10	2	3	**0.25**
125					
IM 441	**A5αI$''_{16\beta}$II$_{17\alpha/\text{on}//1}$ol$_{3\beta}$**	0.5	0	0	0
126					
IM 432	**A5αII$^{1-1}{}_{17\alpha}$CN$_5$ol$_{3\alpha,17}$**	0.5	0	0	0
127					
IM 335	**A5αIII$_{17\alpha/\text{I}//2}$ol$_{3\beta,17}$**	10	3	3	**0.3**
		0.5	—	0	0
128					
IM 78	**A5α^{1}I$_1$*II$''_{17\beta}$on$_3$** (Methenolone acetate)	10	1.5	0	**0.075**
129					
IM 77	**A5α^{1}I$_1$*VII$''_{17\beta}$on$_3$** (Methenolone oenonthate)	10	2.5	0	**0.125**
130					
IM 318	**A5α^{2}I$_{17\alpha}$ol$_{17}$**	10	1.5	1	**0.125**
131					
IM 365	**A5α^{2}CN$_3$on$_{17}$**	10	2	2	**0.2**
		0.5	0	0	0
132					
IM 150	**A5α^2|$_{17\alpha}$isoxazolyl$_{2-3/3,4}$ol$_{17}$**	10	1.5	0.5	**0.1**
		0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
133					
IM 249	$A5\beta I_{17\alpha}ol_{17}{*}II''_{3\beta}\theta_{5\beta-6}$	10	0	1.5	**0.075**
134					
IM 165	$A5\beta I_{17\alpha}ol_{17}on_{3,11}F_{9\alpha}$	10	2	0.5	**0.125**
135					
IM 357	$A5\beta Ial_{17\beta}ol_{3\alpha,17}on_{11}$	10	0	0	0
136					
IM 386	$A5\beta CN_{17\beta}{*}II''_{3\alpha}$	0.5	0	0	0
137					
IM 377	$A5\beta CN_{17}ol_{17\xi}{*}II''_{3\alpha}$	0.5	0	0	0
138					
IM 426	$A5\beta I_{17\alpha}CN_5ol_{17}on_3$	0.5	0	0	0
139					
IM 489	$A5\beta I_{17\beta/I''//1,1}ol_{3\alpha}on_{11}$	0.5	0	1.5	**1.5**
		0.03	—	0	0
140					
IM 381	$A5\beta ICN_{17\beta}ol_{3\alpha}$	0.5	0	0	0
141					
IM 385	$A5\beta ICN_{17\beta}{*}II''_{3\alpha}on_{11}$	0.5	0	0	0
142					
IM 384	$A5\beta I_{17\beta/CN}$tetrahydropyranyl$_{3\beta/ol//2}$	0.5	0	0	0
143					
IM 369	$A5\beta I_{17\beta/CN//1,1}CN_{17}ol_{3\alpha}on_{11}$	0.5	0	0	0
144					
IM 495	A5β benzyloxy methyl-dimethyl malonate $_{17\beta}ol_{3\alpha}on_{11}$	0.5	0	0	0
145					
IM 368	$A5\beta^2CN_3ol_{17\beta}$	10	0.5	2	**0.125**
		0.5	0	0	0
146					
IM 254	$A5\beta^2II_{3\beta/on//1}ol_{17\beta}$	10	0	0	0
147					
IM 378	$A5\beta^{16}CN_{17}{*}II''_{3\alpha}$	0.5	0	0	0
148					
IM 393	$A5\beta^{17(20)}ICN_{17\beta}{*}II''_{3\alpha}on_{11}$	0.5	0	1.5	**1.5**
149					
IM 406	$A5\beta^{17(20)}I_{17\beta/CN//1,1}ol_{3\alpha}on_{11}$	0.5	0	0	0
150					
IM 120	$A^4I_{6\alpha}ol_{17\beta}on_3$ (Methyltestosterone)	10	2.5	3	**0.275**
151					
IM 208	$A^4I_{6\alpha}ol_{11\beta,17\beta}on_3$	10	3	3	**0.3**
		0.5	—	0	0
152					
IM 55	$A^4I_{17\alpha}ol_{17}on_3$ (Methyltestosterone)	10	3	1.5	**0.225**

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
153					
IM 90	$A^4I_{17\alpha}ol_{4,17}on_3$ (Oxymesterone)	10	0.5	3	**0.175**
154					
IM 223	$A^4I_{17\alpha}ol_{6\beta,17}on_3$	10	0	1	**0.05**
155					
IM 160	$A^4I_{17\alpha}ol_{11\alpha,17}on_3$	10	3	3	**0.3**
156					
IM 241	$A^4I_{17\alpha}ol_{11\beta,17}on_3$	10	3	3	0.3
		0.5	2	—	**4.0**
157					
IM 85	$A^4I_{17\alpha}ol_{17\beta}$*$SII''_{1\alpha,7\alpha}on_3$ (Emdabol)	10	2	1	0.15
		0.5	0.5	0	**0.5**
		0.1	0	—	0
158					
IM 81	$A^4I_{17\alpha}ol_{11\beta,17}on_3F_{9\alpha}$	10	3	2	0.25
	(Fluoxymesterone)	0.5	3	0	**3.0**
		0.1	0	—	0
159					
IM 162	$A^4I_{17\alpha}ol_{17}on_{3,11}F_{9\alpha}$	10	3	2	**0.25**
160					
IM 502	$A^4I''_{17\beta}on_3$	0.5	0	0	0
161					
IM 450	$A^4(CONH_2)_{7\beta}ol_{17\beta}on_3$	0.5	0	0	0
162					
IM 391	A^4CN_{17}*$II''_{17\xi}on_3$	0.5	0	0	0
163					
IM 374	$A^4CN_{17}ol_{17\xi}on_{3,11}$	0.5	0	0	0
164					
IM 375	A^4CN_{17}*$II''_{17\xi}on_{3,11}$	0.5	0	0	0
165					
IM 36	$A^4II^{1-1}_{17\alpha}ol_{17}on_3$ (Ethyltestosterone)	10	0	1	**0.05**
166					
IM 10	$A^4III''K_{17\alpha}ol_{11\beta,17}on_3F_{9\alpha}$ (CS-1)	10	3	3	0.3
		0.5	3	3	6.0
		0.1	1	1.5	**12.5**
		0.03	0	0	0
167					
IM 314	$A^4III''\gamma lac_{17\alpha}on_3$	10	3	3	**0.3**
		0.5	—	0	0
168					
IM 9	$A^4III''\gamma lac_{17\alpha}$*$SII''_{7\alpha}on_3$	10	3	3	0.3
	(Spironolactone)	0.5	0	0	**1.5**
		0.1	—	0	0
169					
IM 66	$A^4III''\gamma lac_{17\alpha}ol_{11\beta}on_3F_{9\alpha}$	10	3	2	0.25
		0.5	3	—	**6.0**

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
170					
IM 342	**A^{4}tetrahydrofuryl$_{17\alpha}$*SII''$_{7\alpha}$on$_3$** (Spiroxasone)	10	3	3	0.3
		0.5	0	1.5	1.5
		0.1	—	0.5	**5.0**
171					
IM 312	**A^{4}imidazolyl$_{17\beta/I/2}$on$_3$**	10	0	1	**0.05**
172					
IM 54	**A^{5}I$_{17\alpha}$ol$_{3\beta,17}$** (Methylandrostenediol)	10	0	0	0
173					
IM 159	**A^{5}I$_{4,4}$ol$_{17\beta}$on$_3$**	10	0	1.5	**0.075**
174					
IM 501	**A^{5}I''$_{17\beta}$ol$_{3\beta}$**	0.5	0	0	0
175					
IM 306	**A^{5}I''K$_{17\beta}$ol$_{3\beta}$(NH$_2$)$_{17}$**	10	0	0	0
176					
IM 255	**A^{5}I''$_{17\alpha/*I}$*II''$_{3\alpha}$NHII''$_{17}$**	10	3	1	**0.2**
177					
IM 390	**A^{5}CN$_{17}$ol$_{17\alpha}\theta\theta$II$_3$**	0.5	0	0	0
178					
IM 182	**A^{5}I$_{17\alpha}$I''$_{17/*I}$ol$_{3\beta}$**	10	0	1.5	**0.075**
179					
IM 568	**A^{5}I$_{4,4}$CN$_{2\alpha}$ol$_{17}$on$_3$**	0.5	2	0.5	**2.5**
		0.03		—	0
180					
IM 364	**A^{5}I$_{4,4,17\alpha}$CN$_{2\alpha}$ol$_{17}$on$_3$** (TMACN)	10	3	3	0.3
		0.5	2.5	3	5.5
		0.1	2	2	**20.0**
		0.03	0	0	0
181					
IM 308	**A^{5}II$_{17\alpha/on//1///ol////2}$*II''$_{3\beta}\theta_{16\beta-17}$**	10	0	0	0
182					
IM 448	**A^{5}I''$_{16\beta}$II$_{17\alpha/ol//1}$ol$_{3\beta}$**	0.5	1.5	3	**4.5**
		0.1	0.5	0	2.5
		0.03	0	0	0
183					
IM 440	**A^{5}I''$_{16\beta}$II$_{17\alpha/on//1}$ol$_{3\beta}$**	0.1	0	0	0
184					
IM 446	**A^{5}II$_{17\alpha/on//1}$(CONH$_2$)$_{16\beta}$ol$_{3\beta}$**	0.5	0	1.5	**1.5**
		0.1	0	0	0
185					
IM 417	**A^{5}CN$_{17}$I$_{17\beta/CN/1,1}$ol$_{3\beta}$**	0.5	0	0	0
186					
IM 118	**A^{5}II$^{1-1}{}_{17\alpha}$ol$_{3\beta,17}$**	10	0	0	0
187					
IM 350	**A^{5}III''ylac$_{17\alpha}$ol$_{3\beta}$**	10	0	3	**0.15**
		0.5	—	0	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
188					
IM 351	$A^{5}III^{1''}\gamma lac_{17\alpha}ol_{3\beta}$	10	3	3	0.3
		0.5	—	0.5	**1**
189					
IM 354	$A^{5}III^{1-1''}{}_{17\alpha}ol_{3\beta,17}$	10	0	0	0
190					
IM 500	$A^{5}III^{2-2''}{}_{17\alpha}ol_{3\beta,17}$	0.5	0	0	0
191					
IM 217	$A^{1,4}I_{6\alpha}on_{3,11,17}$	10	3	3	0.3
		0.5	—	1	**2**
192					
IM 281	$A^{1,4}I_{17\alpha}ol_{17}on_{3}$	10	0.5	1	**0.075**
193					
IM 164	$A^{1,4}I_{17\alpha}ol_{11\beta,17}on_{3}$	10	2	1.5	**0.175**
194					
IM 323	$A^{4,6}III''K_{17\alpha}ol_{17}on_{3}$	10	3	3	**0.3**
	(Aldadiene Kalium)	0.5	0	—	0
195					
IM 316	$A^{4,6}III''\gamma lac_{17\alpha}ol_{17}on_{3}$	10	3	3	**0.3**
	(Aldadiene)	0.5	0	—	0
196					
IM 157	$A^{4,9(11)}I_{17\alpha}ol_{17}on_{3}$	10	3	3	**0.3**
197					
IM 222	$A^{4,9(11)}I_{2\alpha,17\alpha}ol_{17}on_{3}$	10	3	1	**0.2**
198					
IM 389	$A^{4,16}CN_{17}on_{3}$	0.5	0	0	0
199					
IM 498	$A^{5,15}I''_{17\beta}{*}II''_{3\beta}$	0.5	0	0	0
200					
IM 396	$A^{5,16}CN_{17}{*}II''_{3\beta}$	0.5	0	0	0
201					
IM 322	$A(O_2)5\alpha I_{17\alpha}ol_{17}$	10	3	1.5	**0.225**
202					
IM 80	$A(O_2)^{4}I_{17\alpha}ol_{17}on_{3}$ (Oxandrolone)	10	3	3	**0.3**
		0.5	0	0	0
203					
IM 410	$A(N_4)II_{17\alpha}ol_{17}on_{3}$	10	1.5	3	**0.225**
		0.5	0	0	0
204					
IM 302	$AnA5\alpha on_{2,16}$	10	3	3	**0.3**
205					
IM 126	$AnB^{4}ol_{17\beta}on_{3}$ (B-Nortestosterone)	10	0.5	1	**0.075**
206					
IM 84	$AhoD(O_{17\alpha})^{1,4}on_{3,17}$	10	2	3	**0.25**
207					
IM 452	$An18,nC,hoD^{4}I_{17\alpha}ol_{17}on_{3}$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
208					
IM 143	$A^{4\,9}\beta,10\alpha ol_{17\beta}on_3$ (Retrotestosterone)	10	0	0.5	**0.025**
209					
IM 145	$A^{4\,9}\beta,10\alpha on_{3,17}$ (Retroandrostenedione)	10	3	0.5	**0.175**
210					
IM 243	$P5\alpha on_{3,20}$	10	0.5	0.5	0.05
211					
IM 72	$P5\beta on_{3,20}$(Pregnanedione)	10	0	2	0.1
		0.5	—	0	0
212					
IM 106	$P5\alpha on_{3,11,20}$	10	3	3	0.3
213					
IM 115	$P5\beta on_{3,11,20}$	10	2.5	3	**0.275**
		0.5	—	0	0
214					
IM 447	$P5\alpha ol_{3\beta}on_{20}$	0.5	0	0	0
215					
IM 261	$P5\alpha ol_{3\beta,16\alpha}on_{20}$	10	0	0.5	**0.025**
216					
IM 136	$P5\alpha ol_{3\beta}on_{11,20}$	10	1.5	3	**0.225**
217					
IM 301	$P5\beta ol_{3\beta}on_{11,20}$	10	3	3	**0.3**
		0.5	—	0	0
218					
IM 122	$P5\alpha ol_{3\beta,17}on_{11,20}$	10	3	3	**0.3**
219					
IM 277	$P5\alpha ol_{3\beta}on_{11,20}\theta_{16\alpha-17}$	10	3	3	**0.3**
220					
IM 272	$P5\alpha^{*}II''_{3\beta}on_{20}Cl_{5,6\beta}$	10	0	0.5	**0.025**
221					
IM 273	$P5\alpha ol_{3\beta,17}on_{20}Cl_{5,6\beta}$	10	0	0.5	**0.025**
222					
IM 215	$P5\alpha ol_5on_{3,20}F_{6\beta}$	10	0	0	0
223					
IM 290	$P5\alpha^{*}II''_{3\beta,20}\theta_{5\alpha-6}$methyloxazolyl$_{16\alpha-17}$	10	2	3	**0.25**
224					
IM 285	$P5\alpha^{*}II''_{3\beta}on_{11,20}$methyloxazolyl$_{16\alpha-17}$	10	0.5	1	**0.075**
225					
IM 286	$P5\alpha^{*}III''_{3\beta}on_{11,20}$ethyloxazolyl$_{16\alpha-17}$	10	1	0.5	**0.075**
226					
IM 331	$P5\alpha ol_{3\beta}$[N-(piperidinoethyl)-formamido]$_{20\beta}$	10	0.5	2.5	**0.15**
227					
IM 82	$[P5\alpha ol_{3\alpha}on_{20}$piperidino$_{2\beta/I//1}]Br^{-}$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
228					
IM 135	P5αol$_{3\alpha}$on$_{20}$NH$_{2\beta/II//Ph///2}$ · HCl	0.5	0	0	0
229					
IM 274	P5αol$_5$*II″$_{3\beta}$on$_{20}$(N$_3$)$_{6\beta}$	10	0	1.5	**0.075**
230					
IM 283	P5αol$_{3\beta,17}$on$_{11,20}$(N$_3$)$_{16\beta}$	10	0	0	0
231					
IM 282	P5αol$_{3\beta,16\alpha}$on$_{11,20}$(N$_3$)$_{17}$	10	2	1	**0.15**
232					
IM 460	P5αol$_{3\alpha}$on$_{20}$morpholino$_{2\beta}$ · HCl	0.5	0	0	0
233					
IM 269	P5α^{16}on$_{3,11,20}$	10	0	3	**0.15**
		0.5	—	0	0
234					
IM 264	P5α^{16}*II″$_{3\beta,11\alpha}$on$_{20}$	10	0	3	**0.075**
235					
IM 263	P5α^{16}*III″$_{3\beta}$on$_{11,20}$	10	0	0	0
236					
IM 270	P5α^{16}on$_{11,20}$θI$_{3,3}$	10	1.5	3	**0.225**
		0.5	—	0	0
237					
IM 265	P5α$^{17(20)}$*II″$_{3\beta,20}$	10	0	3	**0.15**
238					
IM 128	P5βol$_{3\alpha,12\alpha,20}$	10	3	3	**0.3**
		0.5	0	—	0
239					
IM 129	P5βol$_{3\alpha,20}$on$_{12}$	10	3	3	**0.3**
240					
IM 113	P5β*II″$_{3\alpha,20}$on$_{12}$	10	3	3	**0.3**
		0.5	0	0	0
241					
IM 112	P5βol$_{3\alpha}$on$_{20}$	10	0	3	**0.15**
242					
IM 110	P5βol$_{3\alpha}$*II″$_{12\alpha}$on$_{20}$	10	0	3	**0.15**
243					
IM 127	P5β*II″$_{3\alpha,12\alpha}$on$_{20}$	10	0.5	3	**0.175**
244					
IM 11	P5β*suc″Na$_{21}$on$_{3,20}$	10	2	0	**0.15**
	(Hydroxydione Sodium)	0.5	0	—	0
245					
IM 121	P5βol$_{3\alpha}$on$_{11,20}$	10	3	1.5	**0.225**
246					
IM 293	P5β*II″$_{3\alpha}$on$_{11,20}$	10	3	3	**0.3**
		0.5	—	0	0
247					
IM 294	P5βol$_{3\alpha,17}$on$_{11,20}$	10	2	3	**0.25**
		0.5	—	0	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
248					
IM 303	$P5\beta ol_{3\alpha,17}{*}II''_{21}on_{11,20}$	10	0	1.5	**0.075**
249					
IM 300	$P5\beta ol_{17}{*}II''_{3\alpha,21}on_{11,20}$	10	0	0	0
250					
IM 305	$P5\beta ol_{17}{*}II''_{21}on_{3,11,20}$	10	0	0.5	**0.025**
251					
IM 497	$P5\beta''_{21}ol_{3\alpha}on_{11}$	0.5	0	0	0
252					
IM 116	$P5\beta ol_{3\alpha,20}\theta_{11\beta-12}$	10	3	3	**0.3**
		0.5	—	0	0
253					
IM 74	$P^4on_{3,20}$ (Progesterone)	10	1.5	3	**0.225**
		0.5	0	0	0
254					
IM 561	$P^4on_{3,11,20}$	0.5	0	0	0
255					
IM 555	$P^4on_{3,6,11,20}$	0.5	0	0	0
256					
IM 133	$P^4ol_{3\beta,16\alpha,17}on_{20}$	10	0	0	0
257					
IM 119	$P^4ol_{11_\alpha}on_{3,20}$ (Hydroxyprogesterone)	10	1.5	3	**0.225**
		0.5	0	0	0
258					
IM 47	$P^4ol_{17}on_{3,20}$ (Hydroxyprogesterone)	10	3	3	**0.3**
259					
IM 73	$P^4{*}II''_{17}on_{3,20}$ (Acetoxyprogesterone)	10	3	3	0.3
		0.5	0	0.5	**0.5**
260					
IM 244	$P^4ol_{21}on_{3,20}$ (Desoxycorticosterone)	10	3	2	**0.25**
261					
IM 25	$P^4{*}II''_{21}on_{3,20}$	10	1	0	0.05
	(Desoxycorticosterone acetate)	0.5	—	0.5	**1.0**
262					
IM 53	$P^4{*}II''_{6\beta,11\alpha}on_{3,20}$	0.5	0	0	0
263					
IM 23	$P^4ol_{11\beta,21}on_{3,20}$	10	3	3	**0.3**
	(Corticosterone, Kendall "Cpd. B")	0.5	0	0	0
264					
IM 134	$P^4ol_{11\beta}{*}II''_{21}on_{3,20}$	10	2	1.5	**0.175**
	(Corticosterone acetate)				
265					
IM 70	$P^4ol_{17,21}on_{3,20}$	10	3	3	**0.3**
	(Hydroxydesoxy-corticosterone)				
266					
IM 235	$P^4ol_{11\alpha,17,21}on_{3,20}$	10	3	3	**0.3**

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
267					
IM 45/a	$P^4ol_{11\beta,17,21}on_{3,20}$ (Hydrocortisone, Cortisol)	10	3	1	**0.2**
268					
IM 45	$P^4ol_{11\beta,17}$*II″$_{21}on_{3,20}$ (Cortisol acetate)	10 0.5	3 0	0 0	**0.15** 0
269					
IM 298	$P^4ol_{11\beta,17}$*IV″$_{21/I//3,3}on_{3,20}$ (Cortisol tetrabutylacetate)	10	0	0	0
270					
IM 98	$P^4ol_{11\beta,17}$*suc″$_{21}on_{3,20}$ (Cortisol hemisuccinate)	10	0	0	0
271					
IM 337	$P^4ol_{11\beta,17}$*suc″$Na_{21}on_{3,20}$ (Cortisol sodium succinate)	10	2	0.5	**0.125**
272					
IM 256	$P^4ol_{14\alpha,17}$*II″$_{21}on_{3,20}$	10	1.5	2.5	**0.2**
273					
IM 237	$P^4ol_{6\beta}on_{3,11,20}$	10	0.5	1.5	**0.1**
274					
IM 557	$P^4ol_{21}on_{3,11,20}$	0.5	0	0	0
275					
IM 102	$P^4ol_{17,21}on_{3,11,20}$ (Cortisone)	10 0.5	3 0	0 —	**0.15** 0
276					
IM 22	P^4ol_{17}*II″$_{21}on_{3,11,20}$ (Cortisone acetate)	10 0.5	3 0	0 0	**0.15** 0
277					
IM 43	$P^4ol_{11\beta,21}on_{3,20}al_{18}$ (Aldosterone)	10	0	0	0
278					
IM 149	$P^4ol_{11\beta,21}on_{3,20}\theta\theta_{16\alpha-17I,I}$ (Cortisol acetonide)	10	3	0	**0.15**
279					
IM 130	P^4*II″$_{21}on_{3,20}\theta_{16\alpha-17}$ (Epoxy-desoxycorticosterone)	10	0	1.5	**0.075**
280					
IM 123	$P^4on_{3,20}\theta_{16\alpha-17}$ (Epoxyprogesterone)	10	0.5	3	**0.175**
281					
IM 16	$P^4on_{3,11,20}Br_{9\alpha}$	10 0.5	1 —	1 3	**0.2** 0
282					
IM 179	$P^4on_{3,20}Br_{17}$ (Bromoprogesterone)	10	0.5	3	**0.175**
283					
IM 18	P^4ol_{17}*II″$_{21}on_{3,11,20}Cl_4$ (Chlorocortisone acetate)	10	0.5	—	**0.05**
284					
IM 19	$P^4ol_{11\beta,17}$*II″$_{21}on_{3,20}Cl_{9\alpha}$ (Chlorocortisol acetate)	10	0	—	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
285					
IM 230	$P^4ol_{11\alpha}on_{3,20}F_{6\alpha}$	10	2	3	**0.25**
286					
IM 214	$P^4ol_{11\beta,17}{*}II''_{21}on_{3,20}F_{6\alpha}$ Fluorohydrocortisone acetate)	10	0	1.5	**0.075**
287					
IM 170	$P^4ol_{11\beta}{*}II''_{3\beta}on_{20}\theta\theta I_{16\alpha-17/I,I}F_{6\alpha}$	10	0	0	0
288					
IM 173	$P^4ol_{11\beta}on_{3,20}\theta\theta I_{16\alpha-17/I,I}F_{6\alpha}$	10 0.5	3 0	1.5 —	**0.225** 0
289					
IM 15	$P^4ol_{11\beta}on_{3,20}F_{9\alpha}$	10	3	3	**0.3**
290					
IM 39	$P^4ol_{11\beta,17}{*}II''_{21}on_{3,20}F_{9\alpha}$ (Fluorocortisol acetate)	10	1.5	2.5	**0.2**
291					
IM 174	$P^4ol_{11\beta}on_{3,20}\theta\theta I_{16\alpha-17/I,I}F_{6\alpha,9\alpha}$	10	0	0.5	**0.025**
292					
IM 524	$P^5ol_{20\beta}{*}II''_{3\beta}$	0.5	0	0	0
293					
IM 69	$P^5ol_{3\beta}on_{20}$ (Pregnenolone)	10	1	3	**0.2**
294					
IM 125	$P^5{*}II''_{3\beta}on_{20}$	10	0	1	**0.05**
295					
IM 195	$P^5ol_{3\beta,17}on_{20}$	10	0	0	0
296					
IM 132	$P^5ol_{17}{*}II''_{3\beta}on_{20}$	10	0	0	0
297					
IM 3	$P^5ol_{3\beta}{*}II''_{21}on_{20}$ (Acetoxypregnenolone)	10	0	1.5	**0.075**
298					
IM 523	$P^5{*}II''_{3\beta,21}on_{20}$	0.5	0	0	0
299					
IM 176	$[P^5{*}II''_{17}{*}S''_{3\beta}on_{20}]$pyridinium$^+$	10	1.5	3	**0.225**
300					
IM 108	$P^5ol_{17}{*}II''_{3\beta,21}on_{20}$	10	0	1	**0.05**
301					
IM 297	$P^5on_{20}\theta I_{3\beta}$	10 0.5	1 —	3 0	**0.2** 0
302					
IM 525	$P^5O,OII_{3,20}$	0.5	0	0	0
303					
IM 359	$P^5{*}I''_{11\alpha}O,OI_{17-20,20-21}O,OII_3$	10 0.5	0.5 —	3 0	**0.175** 0
304					
IM 177	$P^5ol_{3\beta}on_{20}\theta_{16\alpha-17}$	10	0	0.5	**0.025**

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
305					
IM 109	$P^{5}ol_{3\beta}{*}II''_{21}on_{20}\theta_{16\alpha-17}$	10	0	0.5	**0.025**
306					
IM 178	$P^{5}ol_{3\beta}on_{20}Br_{17}$ (Bromopregnenolone)	10	0	0	**0.15**
307					
IM 317	$P^{1,4}on_{3,15,20}$	10	3	3	**0.3**
308					
IM 7	$P^{1,4}ol_{11\beta,17,21}on_{3,20}$ (Prednisolone)	10	3	0.5	**0.175**
309					
IM 56	$P^{1,4}ol_{11\beta,17}{*}II''_{21}on_{3,20}$	10	2	3	**0.25**
	(Prednisolone acetate)	0.5	0	0	0
310					
IM 96	$P^{1,4}ol_{11\beta,17}{*}suc''_{21}on_{3,20}$	10	3	3	**0.3**
	(Prednisolone hemisuccinate)	0.5	0	—	0
311					
IM 97	$P^{1,4}ol_{11\beta,17}{*}Bz''_{21/S''Na//3}on_{3,20}$	10	3	1.5	**0.225**
	(Prednisolone m-sulfobenzoate sodium)				
312					
IM 526	$P^{1,4}ol_{17,21}on_{3,11,20}$	0.5	0	0	0
313					
IM 68/a	$P^{1,4}ol_{17}{*}II''_{21}on_{3,11,20}$	10	3	3	**0.3**
	(Prednisone acetate)	0.5	0	—	0
314					
IM 211	$P^{1,4}ol_{11\beta,17,21}on_{3,20}F_{6\alpha}$	10	3	0.5	**0.175**
	(Fluoroprednisolone)				
315					
IM 32	$P^{1,4}ol_{11\beta,16\alpha,17,21}on_{3,20}F_{9\alpha}$	10	0	0	0
	(Triamcinolone)	2	0	0	0
316					
IM 148	$P^{1,4}ol_{11\beta,21}on_{3,20}\theta\theta I_{16\alpha-17/I,I}F_{9\alpha}$	10	0	0	0
	(Triamcinolone acetonide)				
317					
IM 257	$P^{3,5}ol_{17}{*}II''_{3,21}on_{20}$	10	3	3	**0.3**
		0.5	—	0	0
318					
IM 288	$P^{3,5}{*}II''_{21}on_{20}\theta I_{3\beta}methyloxalolyl_{16\alpha-17}$	10	1.5	3	**0.225**
		0.5	—	0	0
319					
IM 171	$P^{4,6}{*}II''_{3\beta,17}on_{20}Cl_{6}$	10	2	1	**0.15**
320					
IM 172	$P^{4,6}{*}II''_{17}on_{3,20}Cl_{6}$	10	3	3	**0.3**
		0.5	0	0	0
321					
IM 206	$P^{4,16}on_{3,20}$	10	2	3	**0.25**
		0.5	—	0	0
322					
IM 154	$P^{4,17(20)}ol_{11\beta,21}on_{3}$	10	3	3	**0.3**
		0.5	0	0	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
323					
IM 240	$P^{4,17(20)}ol_{11\beta}{*}II''_{21}on_3$	10	1	0	**0.05**
324					
IM 239	$P^{4,17(20)''}{*}I_{21}on_{3,11}$	10	3	3	**0.3**
		0.5	—	0	0
325					
IM 156	$P^{4,17(20)}ol_{11\beta}on_3al_{21}$	10	0	0	0
326					
IM 180	$P^{5,16}ol_{3\beta}on_{20}$ (Dehydropregnenolone)	10	0	3	**0.15**
327					
IM 111	$P^{5,16}{*}II''_{3\beta}on_{20}$ (Dehydropregnenolone acetate)	10	0	1.5	**0.075**
328					
IM 99	$P^{5,16}{*}II''_{3\beta,21}on_{20}$	0.5	0	0	0
329					
IM 287	$P^{1,4,9(11)}{*}II''_{21}on_{3,20}$	10	3	3	0.3
	methyloxazolyl$_{16\alpha-17}$	0.5	3	3	6
		0.2	—	3	**10.0**
330					
IM 161	$P^{1,4,17(20)}ol_{11\beta,21}on_3$	10	3	3	**0.3**
		0.5	0	—	0
331					
IM 228	$P^{1,4,17(20)}ol_{11\beta}{*}II''_{21}on_3$	10	0	0.5	**0.025**
332					
IM 231	$P^{1,4,17(20)}ol_{11\beta,16\alpha}{*}II''_{21}on_3$	10	3	2	**0.25**
333					
IM 329	$P^{5,17(20),20}{*}II''_{3\beta,21}$	10	0	0	0
334					
IM 330	$P^{5,17(20),20}{*}II''_{3\beta}(SO_2CH_3)_{21}$	10	1	0	**0.05**
335					
IM 212	$P5\alpha I_{6\beta}ol_{5,11\alpha}on_{3,20}$	10	3	3	**0.3**
		0.5	0	0	0
336					
IM 233	$P5\alpha I_{6\beta}ol_{5,11\beta,17}{*}II''_{21}on_{3,20}$	10	0	1.5	**0.075**
337					
IM 262	$P5\alpha I_{16\alpha}ol_{17}{*}II''_{21}on_{3,20}$	10	0	0	0
338					
IM 266	$P5\alpha I_{16\beta}ol_{3\beta}on_{20}$	10	0	0	0
339					
IM 259	$P5\alpha I_{16\beta}ol_{11\alpha,17}{*}II''_{21}on_{3,20}$	10	3	3	0.3
		0.5	—	0.5	**1**
340					
IM 258	$P5\alpha I_{16\beta}ol_{17}{*}II''_{21}on_{3,11,20}$	10	2	3	**0.25**
		0.5	—	0	0
341					
IM 267	$P5\alpha I_{16\beta}ol_{3\beta,11\alpha}on_{20}\theta_{16\alpha-17}$	10	3	3	0.3
		0.5	0	0.5	**0.5**
342					
IM 431	$P5\alpha CN_5{*}II''_{17}on_{3,20}$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
343					
IM 449	P5αCN$_{6\beta}$ol$_{3\beta,5}$on$_{20}$	0.5	0	0	0
344					
IM 307	P5α(CH$_2$)$_{16}$ol$_{17}$*II″$_{3\beta}$on$_{11,20}$	10	0.5	0	**0.025**
345					
IM 496	P5αI″$_{21,21}$ol$_{3\beta}$	0.5	0	0	0
346					
IM 366	P5α^{2}CN$_{3\theta\theta}$II$_{20}$	10	0.5	2	0.125
		0.5	0	2	**2.0**
		0.03	—	0	0
347					
IM 552	P5α^{6}maleic anhydride adduct$_{5\alpha-8}$ *II″$_{3\beta}$on$_{20}\theta_{9\xi-11}$	0.5	0	0	0
348					
IM 402	P5α$^{20''}$CN$_{21}$ol$_{3\beta}$*II$_{21}$	0.5	0	0	0
349					
IM 553	P5α$^{6,9(11)}$maleic anhydride adduct$_{5\alpha-8}$*II″$_{3\beta}$on$_{20}$	0.5	0	0	0
350					
IM 28	P5α$^{6,9(11)}$maleic anhydrid adduct$_{5\alpha-8}$*II″$_{3\beta,21}$on$_{20}$	10	0	0	0
351					
IM 166	P5βI$_{11\alpha}$on$_{3,20}$	10	0	1	**0.05**
352					
IM 227	P5βI$_{11\alpha}$ol$_{11}$NOH$_{3,20}$ · H$_2$O	10	3	3	**0.3**
		0.5	0	—	0
353					
IM 545	P5βI″$_{20}$*II″$_{3\alpha}$	0.5	0	0	0
354					
IM 544	P5βIal$_{20}$*II$_{3\alpha}$	0.5	0	0	0
355					
IM 436	P5β(CONH$_2$)$_{16\alpha}$ol$_{3\alpha}$on$_{11,20}$	0.5	0	0	0
356					
IM 418	P5βCN$_{16\alpha}$*II″$_{3\beta}$on$_{11,20}$	0.5	2.5	2	4.5
		0.1	2.5	2	22.5
		0.03	0	1.5	25.0
		0.015	0	1.5	**50.0**
357					
IM 383	P5βCN$_{20}$ol$_{20\xi}$*II″$_{3\alpha}$	0.5	0	0	0
358					
IM 388	P5βCN$_{20}$ol$_{20\xi}$*II″$_{3\beta,21}$	0.5	0	0	0
359					
IM 403	P5βCN$_{20}$ol$_{20\xi}$*II″$_{3\alpha}$on$_{11}$	0.5	0	0	0
360					
IM 398	P5β″$_{21}$CN$_{20}$ol$_{3\alpha}$	0.5	0	0	0
361					
IM 399	P5βCN$_{21}$ICN$_{20}$*II″$_{3\alpha}$	0.5	0	0	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
362					
IM 367	$P5\beta^{2}CN_{3}\theta\theta II_{20}$	10	0	0	0
363					
IM 394	$P5\beta^{17(20)}CN_{20}{*}II''_{3\alpha}on_{11}$	0.5	0.5	1.5	**2.0**
364					
IM 371	$P5\beta^{17(20)''}CN_{20}{*}II''_{3\alpha}{*}IIol_{21}$	0.5	0	0	0
365					
IM 405	$P5\beta^{17(20)''}{}_{21}CN_{20}ol_{3\alpha}on_{11}$	0.5	0	0	0
366					
IM 408	$P5\beta^{17(20)''}CN_{20}{*}II''_{3\alpha}{*}II_{21}on_{11}$	0.5	0	0	0
367					
IM 499	$P5\beta^{20}I''_{21}ol_{3\alpha,17}ol_{3\alpha,17\alpha}on_{11}$	0.5	0	0	0
368					
IM 370	$P5\beta^{20''}CN_{21}ol_{3\alpha}{*}II_{21}$	0.5	0	0	0
369					
IM 158	$P^{4}I_{2\alpha}ol_{11\beta,17}{*}II''_{21}on_{3,20}$ (Methylcortisol acetate)	10	3	2.5	**0.275**
370					
IM 51/h	$P^{4}I_{2\alpha}ol_{17}{*}II''_{21}on_{3,20}\theta_{9\beta-11}$	10	3	3	0.3
		0.5	0	1.5	**1.5**
371					
IM 51/j	$P^{4}I_{2\alpha}ol_{11\beta,17}{*}II''_{21}on_{3,20}Cl_{9\alpha}$	10	0	1.5	**0.075**
372					
IM 51/f	$P^{4}I_{6\alpha}on_{3,11,20}$	10	3	3	**0.3**
		0.5	0	0	0
373					
IM 51/c	$P^{4}I_{6\alpha}ol_{11\beta}on_{3,20}$	10	3	3	**0.3**
		0.5	0	—	0
374					
IM 220	$P^{4}I_{6\alpha}ol_{17}on_{3,20}$	10	3	3	0.3
		0.5	0.5	—	**1**
375					
IM 219	$P^{4}I_{6\alpha}{*}II''_{17}on_{3,20}$	10	3	3	**0.3**
		0.5	0	—	0
376					
IM 51/i	$P^{4}I_{6\alpha}ol_{11\beta,17}on_{3,20}$	10	3	3	**0.3**
		0.5	0	—	0
377					
IM 242	$P^{4}I_{6\alpha}ol_{11\beta,17,21}on_{3,20}$ (Methylcortisol)	10	3	0	**0.15**
378					
IM 209	$P^{4}I_{6\alpha}ol_{11\beta,17}{*}II''_{21}on_{3,20}$ (Methylcortisol acetate)	10	2	0.5	**0.125**
379					
IM 546	$P^{4}Iol_{20}on_{3}$	0.5	0	0	0
380					
IM 416	$P^{4}I''_{16\alpha/*I}on_{3,20}$	0.5	0	0	0
381					
IM 155	$P^{4}Ial_{20\beta}on_{3}$	10	0.5	0	**0.025**

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
382					
IM 547	$P^4Ial_{20}on_3$	0.5	0	0	0
383					
IM 466	$P^4CN_{16\alpha}on_{3,20}$	0.1	3	0	**15.0**
		0.015	0	0	0
384					
IM 475	$P^4CN_{16\alpha}on_3\theta\theta III_{20/I//2,2}$	0.5	1.5	3	4.5
		0.03	0.5	0	**8.33**
385					
IM 225	$P^4I_{20\beta/NOH}NOH_3$	10	3	3	**0.3**
		0.5	0	—	0
386					
IM 207	$P^4II_{16\alpha}ol_{20}on_3$	0.5	0	0	0
387					
IM 472	$P^4ICN_{16\alpha/}on_{3,20}$	0.5	0	0	0
388					
IM 105	$P^5I_{16\alpha}*II''_{3\beta}on_{20}$	10	0	0.5	**0.025**
389					
IM 201	$P^5I_{16\alpha}ol_{3\beta,17}on_{20}$	10	0	0	0
390					
IM 200	$P^5I_{16\beta}ol_{3\beta}on_{20}\theta_{16\alpha-17}$	10	3	2	**0.25**
391					
IM 465	$P^5I''_{16\alpha}*II''_{3\alpha,20}$	0.5	0	0	0
392					
IM 415	$P^5I''_{16\alpha}ol_{3\beta}\theta\theta II_{20}$	0.5	0	0	0
393					
IM 437	$P^5I''_{16\alpha}*II''_{3\beta}\theta\theta II_{20}$	0.5	0	0	0
394					
IM 438	$P^5I''_{16\alpha/*I}ol_{3\beta}\theta\theta II_{20}$	0.5	0	0	0
395					
IM 352	$P^5(CH_2NH_2)_{16\alpha}ol_{3\beta}on_{20}$	2	0	0	0
396					
IM 444	$P^5CN_{16\alpha}ol_{3\beta,20}$	0.5	1.5	3	4.5
		0.1	1	1.5	**12.5**
		0.03	0	0	0
397					
IM 464	$P^5CN_{16\alpha}*II''_{3\beta,20\beta}$	0.1	0	2	**10.0**
		0.03	—	0	0
398					
IM 346	$P^5CN_{16a}ol_{3\beta}on_{20}$	10	3	3	0.3
	(Pregnenolone-16α-carbonitrile, PCN)	1	3	3	3.0
		0.5	3	3	6.0
		0.2	3	3	15.0
		0.1	3	3	30.0
		0.03	2	3	**83.0**
		0.015	0	0	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
399					
IM 413	$P^5CN_{16\alpha}$*II″$_{3\beta}$on$_{20}$	0.5	2.5	2	4.5
		0.1	0.5	2	12.5
		0.03	1	1.5	**41.67**
		0.015	0	0	0
400					
IM 479	$P^5CN_{16\alpha}$*VII″$_{3\beta}$on$_{20}$	0.5	3	3	6.0
		0.03	1	1.5	**41.67**
		0.015	0	0	0
401					
IM 476	$P^5CN_{16\alpha}$*suc″Na$_{3\beta}$on$_{20}$	0.5	3	3	6.0
	(PCN sodium hemisuccinate)	0.03	2.5	3	91.67
		0.015	2	1.5	116.67
		0.005	0	2	**200.0**
402					
IM 478	$P^5CN_{16\alpha}$*adamantoate$_{3\beta}$on$_{20}$	0.5	0.5	3	**3.5**
		0.03	—	0	0
403					
IM 483	$P^5CN_{16\alpha}$*S″(NH$_4$)$_{3\beta}$on$_{20}$	0.5	3	3	6.0
	(PCN ammonium sulfate)	0.03	3	3	**100.0**
		0.015	1	0.5	50.0
404					
IM 423	$P^5CN_{16\alpha}ol_{3\beta}on_{7,20}$	0.5	3	1.5	4.5
		0.03	0	0.5	**8.33**
405		0.5	3	2	5.0
IM 429	$P^5CN_{16\alpha}$*II″$_{3\beta}$on$_{7,20}$	0.03	0	1.5	**25.0**
406					
IM 411	$P^5CN_{16\alpha}on_{11,20}\theta\theta II_3$	0.5	2.5	2	4.5
		0.1	2.5	2	22.5
		0.03	0	1.5	25.0
		0.015	0	1 5	**50.0**
407					
IM 414	$P^5CN_{16\alpha}ol_{3\beta}\theta\theta II_{20}$	0.5	2.5	2	4.5
		0.1	2	2	20.0
		0.03	0.5	1.5	**33.3**
		0.015	0	0	0
408					
IM 412	$P^5CN_{16\alpha}on_{11}\theta\theta II_{3,20}$	0.5	2.5	2	4.5
		0.1	0	2	10.0
		0.03	0	1.5	**25.0**
		0.015	0	0	0
409					
IM 461	$P^5CN_{16\alpha}ol_{3\beta}\theta\theta III_{20/I//2,2}$	0.5	2	3	5.0
		0.03	2	0	**33.3**
		0.015	0	—	0
410					
IM 445	$P^5CN_{16\alpha}$*II″$_{3\beta}\theta\theta II_{20}$	0.5	1.5	3	4.5
		0.1	0	1.5	**7.5**
		0.03	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
411					
IM 482	$P^5CN_{16\alpha}ol_{3\beta}(NOCH_3)_{20}$	0.5	1	1.5	2.5
		0.03	1.5	0	**25.0**
		0.015	0	—	0
412					
IM 462	$P^5CN_{16\alpha}ol_{3\beta}NOH_{20}$	0.5	3	3	6.0
		0.03	2.5	3	**91.67**
		0.015	0.5	1	50.0
		0.010	—	0	0
413					
IM 404	$P^{5''}CN_{20}{*}II''_{3\beta}{*}II_{21}$	0.5	0	0	0
414					
IM 474	$P^5I_{4,4}CN_{16\alpha}on_{3,20}$	0.5	0.5	3	**3.5**
		0.03	—	0	0
415					
IM 481	$P^5I_{4,4}CN_{16\alpha}on_3\theta\theta III_{20/I//2,2}$	0.5	0	0	0
416					
IM 71	$P^5II_{16\alpha}ol_{3\beta}on_{20}$	0.5	0	0	0
417					
IM 469	$P^5ICN_{16\alpha}ol_{3\beta}on_{20}$	0.5	0	0	0
418					
IM 409	$P^5II''_{16\alpha/{*}II//CN///2}ol_{3\beta}on_{20}$	10	0	0	0
419					
IM 210	$P^5III_{16\alpha/I//2}ol_{3\beta}on_{20}$	0.5	0	0	0
420					
IM 467	$P^5I''\gamma lac_{16\beta-20}{*}II''_{3\beta}$	0.5	0	0	0
421					
IM 52	$P^{1,4}I_{6\alpha}ol_{11\beta,17,21}on_{3,20}$	10	3	3	**0.3**
	(Methylprednisolone)	0.5	0	0	0
422					
IM 52/a	$P^{1,4}I_{6\alpha}ol_{11\beta,17}{*}II''_{21}on_{3,20}$	10	3	2	0.25
	(Methylprednisolone acetate)	0.5	1	—	2.0
423					
IM 221	$P^{1,4}I_{6\alpha}ol_{11\beta,17}{*}suc''Na_{21}on_{3,20}$	10	3	1.5	0.225
		0·5	0	—	0
424					
IM 218	$P^{1,4}I_{6\alpha}ol_{11\beta,17}on_{3,20}F_{9\alpha}$	10	2	0	**0.1**
425					
IM 51/e	$P^{1,4}I_{6\alpha}ol_{11\beta,17}on_{3,20}F_{9\alpha,21}$	10	1.5	—	**0.15**
426					
IM 345	$P^{1,4}I_{16\alpha}ol_{11\beta,17}{*}II''_{21}on_{3,20}F_{6\alpha}$ (Methylprednisolone)	10	0.5	1.5	**0.1**
427					
IM 152	$P^{1,4}I_{16\alpha}ol_{11\beta,17}{*}II''_{21}on_{3,20}F_{9\alpha}$	10	3	0	0.15
	(Dexamethasone acetate)	1	3	1.5	2.25
		0.5	2.5	0.5	3.0
		0.1	3	1	20.0
		0.03	3	0	**50.0**
		0.015	0	—	0

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
428					
IM 527	$P^{1,4}I_{16\alpha}ol_{11\beta,17}$*$Bz''_{21/S''Na//3}on_{3,20}F_{9\alpha}$	0.5	0	1.5	**1.5**
		0.03	—	0	0
429					
IM 151	$P^{1,4}I_{16\beta}ol_{11\beta,17}$*$II''_{21}on_{3,20}F_{9\alpha}$	10	2	0	0.1
	(Betamethasone acetate)	2	3	3	1.5
		1	2	1.5	1.75
		0.5	1.5	1.5	3.0
		0.1	0	1	**5.0**
430					
IM 95	$P^{1,4}I_{21}ol_{11\beta,17}$*$II''_{21}on_{3,20}F_{9\alpha}$	10	3	2	**0.25**
	(Fluperolone acetate)	0.5	0	—	0
431					
IM 289	$P^{3,5}Ial_6$*$II''_{21}on_{20}\theta I_{3\beta}$methyl-oxazolyl$_{16\alpha-17}$	10	2	3	**0.25**
		0.5	—	0	0
432					
IM 459	$P^{3,5}CN_6$*$II''_{16\alpha}on_{20}\theta I_3$	0.5	0	0	0
433					
IM 458	$P^{3,5}CN_6ol_{11\beta,17\alpha}$*$II''_{21}on_{20}\theta I_3$	0.5	0	0	0
434					
IM 455	$P^{3,5}CN_6$*$II''_{21}on_{11,20}\theta II_{3\beta/Cl//2}$ $\theta\theta I_{16\alpha-17/I,I}F_{9\alpha}$	0.5	0	1	**1.0**
435					
IM 456	$P^{3,5}CN_6ol_{11\beta}$*$III''_{21/I//2,2}on_{20}\theta II_{3/Cl//2}$ $\theta\theta I_{16\alpha-17/I,I}F_{9\alpha}$	0.5	0	0	0
436					
IM 457	$P^{3,5}CN_6ol_{11\beta}$*$II''_{21}on_{20}\theta II_{3/Cl//2}$ $\theta\theta I_{16\alpha-17/I,I}F_{9\alpha}$	0.5	0	0	0
437					
IM 470	$P^{3,5}CN_{16\alpha}on_{7,20}$	0.1	0	0	0
438					
IM 471	$P^{3,5}CN_{16\alpha}ol_3on_{7,20}$	0.1	0	—	0
439					
IM 167	$P^{4,6}I_{17}$*$II''_{3\beta}on_{20}Cl_6$	10	3	3	**0.3**
		0.5	—	0	0
440					
IM 279	$P^{4,6}I_{1\alpha-2}$*$II''_{17}on_{3,20}Cl_6$	10	3	3	0.3
	(Cyproterone acetate)	1	2.5	3	2.75
		0.5	2	3	**5.0**
		0.1	0	0.5	2.5
441					
IM 175	$P^{4,6}I_{6,17}on_{3,20}$	10	3	3	0.3
		0.5	0	1.5	**1.5**
442					
IM 503	$P^{4,6}I_6(CH_2)_{16}$*$II''_{17}on_{3,20}$	0.5	0	0	0
443					
IM 295	$P^{5,16}I_6$*$II''_{3\beta}on_{20}$	10	0	3	**0.15**
444					
IM 268	$P^{5,16}I_{16}$*$I''_{3\beta}on_{20}$	10	3	1	**2**

Table 135 B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
445					
IM 373	$P^{5,17(20)''}CN_{20}{*}II''_{3\beta}{*}II_{21}$	0.5	0	0	0
446					
IM 278	$Pn19^{4}{*}VI''_{17}on_{3,20}$ (Depostat)	10	3	3	**0.3**
		0.5	—	0	0
447					
IM 407	$Pn19^{4,9,11}CN_{20\xi}ol_{20}on_{3}$	0.5	0	0	0
448					
IM 477	$P^{3\alpha(5)}5\alpha CN_{16\alpha}ol_{6\beta}on_{20}$	0.5	2	3	**5.0**
		0.03	0	0	0
449					
IM 480	$P^{3\alpha(5)}5\alpha CN_{16\alpha}ol_{6\beta}\theta\theta III_{20/I//2,2}$	0.5	1	3	**4.0**
		0.03	0	0	0
450					
IM 144	$P9\beta,10\alpha^{4}on_{3,20}$ (Retroprogesterone)	10	2	3	**0.25**
451					
IM 349	$CH5\beta ol_{3\alpha,12\alpha,24}$	10	0	1	**0.05**
452					
IM 296	$CH5\beta''_{24}$ (Cholanic acid)	0.5	0	0	0
453					
IM 493	$CH5\beta''_{24}ol_{3\alpha}$	0.5	0	0	0
454					
IM 485	$CH5\beta''_{24}ol_{3\alpha,6\alpha}$	0.5	0	0	0
455					
IM 492	$CH5\beta''_{24}ol_{3\alpha,7\alpha}$	0.5	0	0	0
456					
IM 490	$CH5\beta''_{24}ol_{3\alpha,12\alpha}$	0.5	0	0	0
457					
IM 488	$CH5\beta''_{24}ol_{3\alpha,7\alpha,12\alpha}$	0.5	0	0	0
458					
IM 311	$[CH5\beta''ol_{3\alpha,12\alpha}{*}II_{24/N//I,II,II}]Br^{-}$	10	0	0	0
459					
IM 550	$CH5\beta''ol_{3\alpha,7\alpha,12\alpha}{*}I_{24}$	0.5	0	0	0
460					
IM 310	$[CH5\beta''ol_{3\alpha,7\alpha,12\alpha}{*}II_{24/N//I,II,II}]Br^{-}$	10	0	0	0
461					
IM 541	$CH5\beta''_{24}on_{3,7,12}$	0.5	0	0	0
462					
IM 487	$CH5\beta''_{24}ol_{3\alpha}on_{11}$	0.5	0	0	0
463					
IM 117	$CH5\beta''{*}I_{24}{*}II''_{3\alpha,7\alpha}on_{12}$	10	0	0	0
464					
IM 309	$CH5\beta''{*}II_{24/N//II,II}on_{3,12}\cdot HCl$	10	0	0	0
465					
IM 491	$CH^{5''}{}_{24}ol_{3\alpha}$	0.5	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
466					
IM 282	$CH^{5}{''}ol_{3\beta}{*}I_{24}$	10	0	0	0
467					
IM 554	$CH^{5}Ph_{24,24}ol_{3\beta,24}$	0.5	0	0	0
468					
IM 124	$CHn\,24\,5\beta{''}ol_{3\alpha,12\alpha}{*}I_{23}$	10	3	3	**0.3**
	(Methyl nordeoxycholanate)	0.5	0	0	0
469					
IM 376	$CHn(24)\,5\beta\,CN_{20}ol_{3\alpha}on_{11}(CONH_2)_{20}$	0.5	0	0	0
470					
IM 401	$CHn(24)\,5\beta{''}CN_{20}{*}II{''}_{3\alpha}{*}II_{21}on_{11}$	0.5	0	0	0
471					
IM 107	$CHT\,5\alpha ol_{3\beta}$ (Dihydrocholesterol)	10	0	0	0
472					
IM 114	$CHT^{4}on_{3}$ (Cholestenone)	10	0	0	0
473					
IM 104	$CHT^{5}{*}II{''}_{3\beta}$ (Cholesteryl acetate)	10	0	0	0
474					
IM 530	$CHT^{5}{*}IX{''}_{3\beta}$	0.5	0	0	0
475					
IM 531	$CHT^{5}{*}$ carbonate$_{3\alpha/*XVIII}{}^{9}$	0.5	0	0	0
476					
IM 198	$CHT^{5}{*}S{''}Na_{3\beta}$	0.5	0	0	0
477					
IM 348	$CHT^{5}ol_{3\beta}on_{7}$	10	3	0	**0.15**
478					
IM 529	$CHT^{5}Cl_{3\beta}$	0.5	0	0	0
479					
IM 532	$CHT^{5}{*}I{''}Cl_{3\beta}$	0.5	0	0	0
480					
IM 558	$CHT^{5}ol_{3\beta}HgCl_{6}$	0.5	0	0	0
481					
IM 138	$CHT^{5,7}{*}Bz{''}_{3\beta}$ (Dehydrocholesteryl benzoate)	10	0	0	0
482					
IM 232	$CHT(NO_{25})^{5}ol_{3\beta}$	10	0	1.5	**0.075**
483					
IM 341	$ER\,5\alpha^{22}ol_{3\beta}on_{11}$	10	0	0	0
484					
IM 299	$ER\,5\alpha^{8,22}{*}II{''}_{3\beta}on_{11}$	10	0	0	0
485					
IM 536	$ER^{4,7,22}on_{3}$	0.1	0	0	0

Table 135B (continued)

Group	SSS Name	Dose (mg)	Digitoxin[a]	Indomethacin[a]	Activity index[b]
486					
IM 535	$ER^{5,7,22}ol_{3\beta}$	0.5	0	0	0
487					
IM 551	$ER^{5,7,9(11),22}*II''_{3\beta}$	0.5	0	0	0
488					
IM 537	$ST5\beta^{22}ol_{3\alpha}$	0.5	0	0	0
489					
IM 538	$ST5\beta^{22}*II''_{3\alpha}$	0.5	0	0	0
490					
IM 103	$ST^{5}ol_{3\beta}$ (β-Sitosterol)	10	0	0	0
491					
IM 534	$ST^{4,22}on_{3}$	0.5	0	0	0
492					
IM 533	$ST^{5,22}ol_{3\beta}$	0.5	0	0	0
493					
IM 204	$SP5\alpha*II''_{3\beta}$	10	0	0	0
494					
IM 203	$SP5\alpha*II''_{3\beta}on_{12}$	10	0	0	0
495					
IM 205	$SP5\beta ol_{3\beta}$	10	0	0	0
496					
IM 137	$SP^{5}*II''_{3\beta}$	10	0	0	0
497					
IM 202	$SP^{5}ol_{3\beta}on_{12}$	10	0	1.5	**0.075**
498					
IM 291	$SP5\alpha^{9(11)}*II''_{3\beta}$	10	0	0	0
499					
IM 542	$CAR5\beta^{20(22)}Ial_{19\beta}ol_{5,14\beta}*R_{3}$	0.5	0	0	0
500					
IM 494	**Dammaran-n31**$^{17(20)}$**5α,8α,9β, 13α,14β**$I''_{21}ol_{11\alpha}*II_{3\alpha,16\beta}$	0.5	0	0	0

The "Activity Index" is based on the formula $\frac{D+I}{2} \times \frac{1}{mg}$ where D = reading for digitoxin, I = indomethacin, and mg = the dose tested. Thus, the index reflects overall activity against both substrates at any one dose level. The highest Activity Index reading for each steroid is emphasized by boldface numerals, but direct intercomparisons are possible only at corresponding dose levels. Naturally, a compound tested only at the high level of 10 mg per dose could not reveal its full potency in this method of expression, but if it had little or no activity, lower doses were not tested.

On the other hand, steroids available only in very small amounts could be assayed only at dose levels too low for comparison with many other compounds.

For simplicity's sake the compounds are designated and sequentially enumerated according to the rules of the "SSS Nomenclature of Steroids" (*cf.* p. 774).

Second Step: Determination of "Protective Spectrum"

Having selected the most promising protective substances by first screening them for activity against digitoxin and indomethacin, we proceeded to appraise the "Protective Spectrums" of the most potent ones among them. These compounds were now tested against a heterogenous set of 10 pathogens, widely differing in their chemical structure and in the organ changes that they elicit.

The statistical significance of the results was computed (as outlined on p. 771) for the inhibition or aggravation of the changes produced by each of the 10 model toxicants. After this, the "Overall Protective Indexes" ("OPI") and the "Protective Spectrum Index" ("PSI") were calculated, as rough indications of the mean degree and the specificity of protection, that is of the quantitative and qualitative prophylactic potencies respectively. These data are summarized in Tables 136, p. 836 and 137.

For the 10 toxicants enumerated in the caption of Table 136 the techniques of administration and the manner in which protection is expressed have been described in an earlier paper (Selye G 70,420/71), as well as in the sections devoted to the effect of various steroids upon each toxicant in this monograph. Suffice it to recall that the highest possible degree of protection corresponds to grade "3", the "OPI" expresses the mean grade of protection, whereas the "PSI" gives the percentage of the toxicants tested against which significant protection is obtained (irrespective of the degree of significance). These two indexes — given in the last two vertical columns of Table 136 — do not run strictly parallel but the various compounds tested for protective potency are listed roughly in decreasing order of their OPIs. Whenever the material at our disposal permitted it, compounds active at a certain dose were retested at a lower dose level. Of course, the two indexes were not calculated for dose levels at which, because of toxicity or lack of material, not all protective compounds could be tested (marked with a dash); hence the OPI and PSI are listed only for the highest, but still well tolerated, dose of each protective compound. The last two horizontal lines in Table 136 list the indexes for the amenability of the toxicants to detoxication by the conditioning agents as explained on p. 772.

Perusal of Table 136 indicates that almost all **steroids** were active in offering protection, at least against some of the damaging agents; but this is so merely because most of the steroids tested here were included in this study precisely because they had shown some potency in preliminary tests.

It is noteworthy that among all steroids tested PCN (Cpd. 1), again exhibited the highest catatoxic activity as judged by both indexes. It was even more active than any of the other 16α-carbonitriles tested against all 10 substrates. CS-1, spironolactone, Cpds. 4, 5, cyproterone acetate, ethylestrenol, norbolethone, TMACN and Cpd. 10 were almost equally efficacious at the highest dose (10 mg), but at the 500 μg dose level, activity fell roughly in the order in which the compounds are mentioned here. Indomethacin and digitoxin are most readily detoxified by these steroids — in the case of PCN even at the dose level of 300 μg/kg. However, the general indexes of

activity are not very meaningful at the low dose levels at which efficacy against other toxicants has not been examined; hence they are listed only for the optimal protective dose. It will be noted that usually this is 10 mg (the highest dose tested) but in the case of such compounds as the strong glucocorticoids or estradiol, inherent toxicity of heavy overdosage counteracts the protective effect against drugs by causing severe mortality. In these instances, lower dose levels were selected for the computation of the OPI and PSI (last two vertical columns at right).

On the other hand, Cpd. 34 and the steroids listed after it in Table 136 exhibit only negligible if any activity, with the exception of occasional strong inhibitory effects (grade 2 or 3) against individual toxicants (e.g., pregnanedione against parathion, hexobarbital and indomethacin; progesterone against indomethacin; 11α-hydroxyprogesterone against indomethacin, hydroxydione against digitoxin, DOC against nicotine). This singular specificity of protection among compounds having a very low, if any, protective effect against other substrates may well depend upon specific so-called "physiologic antagonisms" (e.g., the anesthetic effect of hydroxydione or the mineralocorticoid action of DOC), but further experiments will be necessary to prove this.

A glance at the OPI and PSI of the toxicants (last two horizontal lines at the bottom of Table 136) shows that digitoxin, dioxathion, hexobarbital, progesterone, indomethacin and DHT were most readily detoxified by the largest number of conditioning agents, whereas parathion, nicotine, zoxazolamine and especially the infarctoid cardiopathy produced by fluorocortisol + Na_2HPO_4 + corn oil were most resistant.

Among the **nonsteroidal agents** of Table 137 rather specific antagonisms were quite common. For example, ACTH increased resistance to the neuromuscular blocking action of zoxazolamine, although it had little if any effect against other agents.

Vitamin E offered some protection against DHT; whereas acetylsalicylic acid protected against progesterone anesthesia, zoxazolamine paralysis and indomethacin ulcers, but these compounds offered no noteworthy protection against other toxicants.

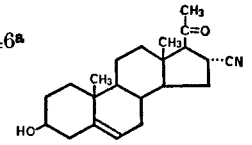
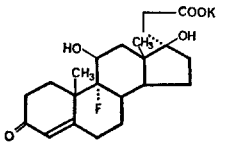
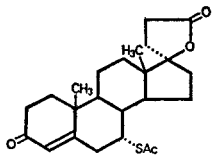
Bile duct ligature offered complete protection against DHT-induced calcinosis, the F-COL + Na_2HPO_4 + oil cardiopathy and indomethacin ulcers, but the mortality was not completely prevented and hence, the grade of protection — which reflects the mean inhibition of lesions plus mortality — is comparatively moderate. It is very likely that occlusion of the choledochus acts by preventing bile secretion, thereby interfering with lipid absorption and the enterohepatic circulation of drugs.

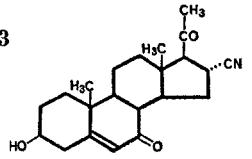
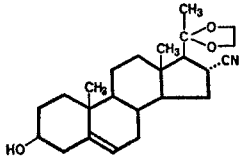
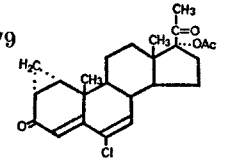
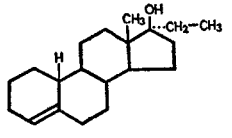
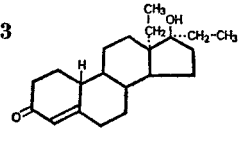
Digitoxin, indomethacin, vitamin A and vitamin D — all of which are readily detoxified under the influence of catatoxic steroids — do not act as inducers of protective enzymes against any of the substrates tested. Obviously, there is no relationship between amenability to detoxication by steroid-induced enzymes and the power to induce such enzymes.

As with the steroidal protective agents, indomethacin intoxication appears to be particularly easy to prevent, but digitoxin poisoning (which is likewise combated by virtually all catatoxic steroids) is singularly resistant to protection by nonsteroidal agents, with the exception of nicotine and phentolamine.

Among the nonsteroidal agents, the highest general protective indexes are exhibited by phetharbital, diphenylhydantion, phenobarbital and phenylbutazone, but at high dose levels, tolbutamide and compound W-1372 are also quite efficacious.

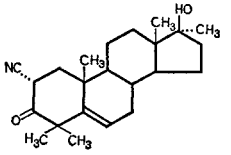
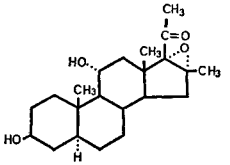
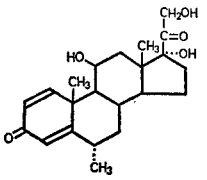
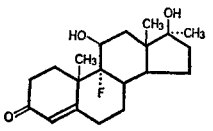
Table 136. *Second step: "Protective Spectrums" of some steroidal agents*

Group	Steroidal Conditioning Agent	Dose mg	Toxicant: Digitoxin	Dioxathion	Parathion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_2HPO_4 + Corn oil	Dihydrotachysterol (DHT)	OPI	PSI %
1 **IM 346**[a] 	3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile **PCN** (SC-4674)	10	3	3	2	2.5	3	3	3	3	1.5	2	**2.6**	**100**
		1	3	3	2.5	0	1	3	3	3	0.5	—		
		0.5	3	3	1.5	0	1	3	3	3	0	2		
		0.2	3	—	3	—	—	2	3	3	—	1.5		
		0.1	3	3	2	—	—	3	3	3	—	0		
	$P^5CN_{16\alpha}ol_{3\beta}on_{20}$	0.03	2	0.5	0	—	0	3	—	3	—	—		
		0.015	0	—	—	—	—	3	0	0	—	—		
		0.005	—	—	—	—	—	3	—	—	—	—		
		0.003	—	—	—	—	—	2	—	—	—	—		
		0.001	—	—	—	—	—	0	—	—	—	—		
2 **IM 10** 	9α-Fluoro-11β,17-dihydroxy-3-oxo-4-androstene-17α-propionic acid potassium salt **CS-1,** (SC-11927)	10	3	1.5	1.5	3	3	3	2	3	1.5	3	**2.5**	**100**
		0.5	3	3	0.5	0	0	3	1	3	0	1		
		0.1	1	1.5	—	—	—	2	0	1.5	—	0		
		0.03	0	0	—	—	—	0	—	0	—	—		
	$A^4III''K_{17\alpha}ol_{11\beta,17}on_3F_{9\alpha}$													
3 **IM 9** 	17-Hydroxy-7α-thioacetyl-3-oxo-4-androstene-17α-propionic acid γ lactone **Spironolactone** (SC-9420)	10	3	1.5	2	3	3	3	1	3	2	2	**2.4**	**100**
		0.5	0	0	0	0	0	2	0	1.5	0	0		
		0.1	—	—	—	—	—	0	—	0	—	—		
	$A^4III''\gamma lac_{17\alpha}on_3$ *$SII''_{7\alpha}$													

4 IM 423		16α-Cyano-3β-hydroxy-5-pregnene-7,20-dione (SC-6813) $P^5CN_{16\alpha}ol_{3\beta}on_{7,20}$	1 0.5 0.03	3 3 0	3 2 —	1.5 2 0	2.5 0 —	3 2 0	3 2 0	3 0 —	2 1.5 2.5	1 0 —	1.5 1.5 —	**2.4**	**100**
5 IM 414		3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile, cyclic (ethylene acetal) (U-19553) $P^5CN_{16\alpha}ol_{3\beta}\theta\theta II_{20}$	1 0.5 0.1 0.03 0.015	3 3 1 1.5 0	3 2.5 — 0 —	0.5 — — — —	2 0 — — —	2 3 — 0 —	3 3 — 3 —	2 3 — 0 —	2 3 1.5 1.5 0	0 — — — —	0.5 — — — —	**1.8**	**90**
6 IM 279		6-Chloro-17-hydroxy-1α,2α-dihydro-2'H-cyclopropa(1,2)-4,6-pregnadiene 3,20-dione acetate **Cyproterone acetate** (Schering) $P^{4,6}I_{1\alpha-2}\ *II''_{17\alpha}on_{3,20}Cl_6$	10 1 0.5 0.1 0.03	3 2.5 2 0 —	2 3 2 0 —	3 1.5 0.5 — —	3 0 — — —	3 1 0 — —	3 3 3 2 0	3 — 2 0 —	3 3 3 0.5 —	0 — — — —	1.5 — 0 — —	**2.5**	**90**
7 IM 59a		17α-Ethyl-4-estren-17-ol **Ethylestrenol** (Organon) $E^4II_{17\alpha}ol_{17}$	10 0.5 0.1 0.03	3 0 — —	1.5 0 — —	2 0 — —	3 0.5 0 —	3 1 0 —	3 2 3 0	2 0 — —	3 0 — —	0 — — —	3 0.5 — —	**2.4**	**90**
8 IM 343		13,17α-Diethyl-17-hydroxy-4-gonen-3-one **Norbolethone** (Wy-3475) $G^4II_{13,17\alpha}ol_{17}on_3$	10 0.5 0.1	3 0 0	1.5 2 —	1.5 0 —	3 0 —	3 0 —	3 2 0	0 — —	3 0.5 —	1 0 —	3 1 0	**2.2**	**90**

[a] Underneath the serial number of the compounds, the "IM" numbers (for Institut de Médecine et de Chirurgie expérimentales) are mentioned. These identify steroids in our collection and remain the same in all Tables as well as in other publications from this Institute.

Table 136 (continued)

Group	Steroidal Conditioning Agent	Dose mg	Toxicant: Digitoxin	Dioxathion	Parathion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_2HPO_4 + Corn oil	Dihydrotachysterol (DHT)	OPI	PSI %
9 IM 364 	17β-Hydroxy-4,4,17-trimethyl-3-oxo-5-androstene-2α-carbonitrile **TMACN** (Winthrop) $A^5I_{4,4,17\alpha}CN_{2\alpha}ol_{17}on_3$	10	3	2.5	2	0	3	3	3	3	2	2	**2.1**	**90**
		0.5	2.5	3	0.5	0	1	3	1	3	−0.5	1.5		
		0.1	2	1.5	—	—	0	3	0	2	—	0		
		0.03	0	0	—	—	—	3	—	0	—	—		
		0.015	—	—	—	—	—	0	—	—	—	—		
10 IM 267 	16β-Methyl-16,17-epoxy-3β,11α-dihydroxy-5α-pregnan-20-one (Lepetit) $P5\alpha I_{16\beta}ol_{3\beta,11\alpha}on_{20}\theta_{16\alpha-17}$	10	3	3	2	0	1	3	3	3	0.5	1.5	**2.0**	**90**
		0.5	0	0	1	—	0	1	0	0.5	0	0		
		0.1	—	—	0	—	—	0	—	—	—	—		
11 IM 52a 	6α-Methyl-11β,17,21-trihydroxy-1,4-pregnadiene-3,20-dione **6α-Methylprednisolone** "Medrol" (Upjohn) $P^{1,4}I_{6\alpha}ol_{11\beta,17,21}on_{3,20}$	10	3	3	0.5	0	3	3	2	3	0	1.5	**1.9**	**80**
		0.5	0	0	0	—	2	1	1	0	—	0.5		
		0.1	—	—	—	—	2	0	0	—	—	—		
		0.03	—	—	—	—	3	—	—	—	—	—		
		0.015	—	—	—	—	0	—	—	—	—	—		
12 IM 81 	9α-Fluoro-17α-methyl-11β,17-dihydroxy-4-androsten-3-one **Fluoxymesterone** (U-6040) $A^4I_{17\alpha}ol_{11\beta,17}on_3F_{9\alpha}$	10	3	3	0	1	2	3	3	2	0	2	**1.9**	**80**
		0.5	3	2	—	0	3	3	0	0	—	1		
		0.1	0	3	—	—	3	3	—	—	—	0		
		0.03	—	0.5	—	—	3	0	—	—	—	—		
		0.015	—	—	—	—	0	—	—	—	—	—		

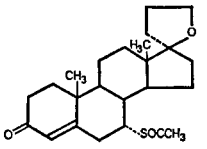
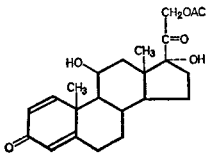
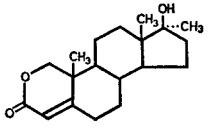
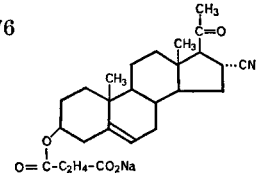
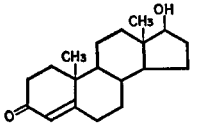
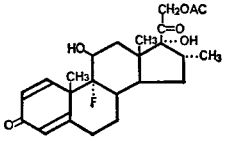
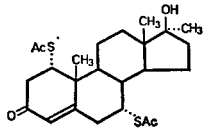
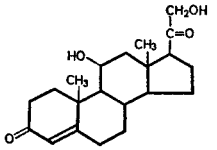
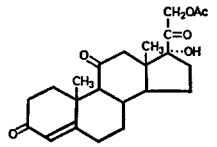
13 IM 342		7α-Thioacetyl-(17R)-spiro-[4-androsten-17,2'-(furan)]-3-one **Spiroxasone** (Merck Sharp & Dohme) **A^{4}tetrahydrofuryl$_{17\alpha}$*SII″$_{7\alpha}$on$_3$**	10 0.5 0.1	3 0 —	3 0 —	1.5 0 —	0 — —	2 1 0	3 1 0	0 — —	3 1.5 0.5	2 0.5 —	1.5 0.5 —	**1.9**	**80**
14 IM 56		11β,17,21-Trihydroxy-1,4-pregnadiene-3,20-dione 21-acetate **Prednisolone acetate** (Schering) **P^{1,4}ol$_{11\beta,17}$ *II″$_{21}$on$_{3,20}$**	10 0.5	2 0	1.5 0	0 —	0.5 0	3 0	3 0	0 —	3 0	0.5 0	1.5 0	**1.5**	**80**
15 IM 80		17α-Methyl-17-hydroxy-2-oxa-4-androsten-3-one **Oxandrolone** (Searle) **A(O$_2$)^{4}I$_{17\alpha}$ol$_{17}$on$_3$**	10 0.5 0.1	3 0 —	1.5 0 —	0 — —	3 0 —	3 0 —	2 2 0	0 — —	3 0 —	0 — —	3 0.5 —	**1.9**	**70**
16 IM 476		3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3-hemisuccinate sodium salt **PCN-sodium hemisuccinate** (U-36278A) **P^{5}CN$_{16\alpha}$*suc″Na$_{3\beta}$ on$_{20}$**	0.5 0.03 0.015 0.005	3 3 2 0	3 0.5 — —	3 0 — —	0 — — —	2 0 — —	2 2 — —	2 0 — —	3 3 1.5 2	0 — — —	0 — — —	**1.8**	**70**
17 IM 53		17β-Hydroxy-4-androsten-3-one **Testosterone** (Roussel, NBC) **A^{4}ol$_{17\beta}$on$_3$**	10 0.5	3 0	2.5 0	0 —	0 —	2 0	2 0	1 0	0.5 0	0 —	2.5 0	**1.4**	**70**

Table 136 (continued)

Group	Steroidal Conditioning Agent	Dose mg	Toxicant: Digitoxin	Dioxathion	Parathion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_2HPO_4 + Corn oil	Dihydrotachysterol (DHT)	OPI	PSI %
18	9α-Fluoro-16α-methyl-11β,17,21-	10	3	—	—	—	—	—	—	0	—	—		
IM 152 	trihydroxy-1,4-pregnadiene-3,20-	1	3	3	—0.5	0	2	3	0	1.5	0	1	1.3	60
	dione 21-acetate	0.5	2.5	3	0	—	3	3	—	0.5	—	0		
	Dexamethasone acetate	0.1	3	0	0	—	1	0	—	1	—	0		
	(Schering)	0.03	3	—	—	—	2	—	—	0	—	0		
	$P^{1,4}I_{16\alpha}ol_{11\beta,17}$ *$II''_{21}on_{3,20}F_{9\alpha}$	0.015	0	—	—	—	2	—	—	—	—	—		
		0.005	—	—	—	—	2	—	—	—	—	—		
		0.003	—	—	—	—	0	—	—	—	—	—		
19	17α-Methyl-17-hydroxy-1α,7α-	10	2	2	0	0	3	3	0	1	0	2	1.3	60
IM 85 	dithio-4-androsten-3-one 1,7-di-	0.5	0.5	0	—	—	0	0	—	0	—	0		
	acetate	0.1	0	—	—	—	—	—	—	—	—	—		
	Emdabol													
	(Merck, Sharp & Dohme)													
	$A^4I_{17\alpha}ol_{17}$*$SII''_{1\alpha,7\alpha}on_3$													
20	11β,21-Dihydroxy-4-pregnene-3,20-	10	3	0.5	0.5	0	0	3	0	3	0	2	1.2	60
IM 23 	dione	0.5	0	—	—	—	—	1	—	0	—	0		
	Corticosterone, Kendall "Cpd. B"	0.1	—	—	—	—	—	0	—	—	—	—		
	(Merck, Sharp & Dohme)													
	$P^4ol_{11\beta,21}on_{3,20}$													
21	17,21-Dihydroxy-4-pregnene-	10	3	0.5	0	0	3	2	3	0	—1	1	1.2	60
IM 22 	3,11,20-trione 21-acetate	0.5	0	0	0	—0.5	0	0	0	0	0	0		
	Cortisone acetate													
	(Upjohn)													
	P^4ol_{17} *$II''_{21}on_{3,11,20}$													

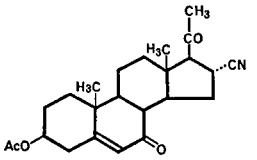
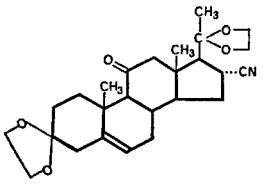
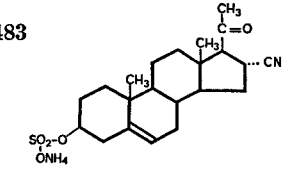
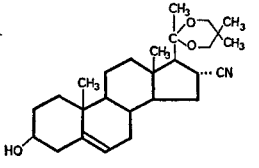
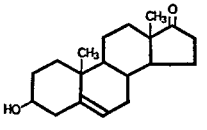
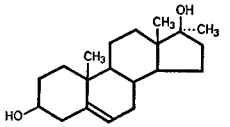
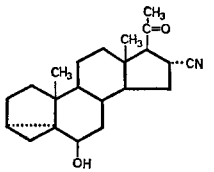
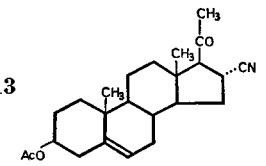
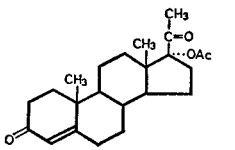
22 **IM 429**		16α-Cyano-3β-hydroxy-5-pregnene-7,20-dione acetate (SC-6703) $P^5CN_{16\alpha}{*}II''_{3\beta}on_{7,20}$	1 0.5 0.03	3 3 0	2	0 — —	0.5	0 —	2	3 3	1.5 2 1.5	0 —	0 —	**1.2**	**60**
23 **IM 412**		3,11,20-Trioxo-5-pregnene-16α-carbonitrile, cyclic 3,20-bis (ethylene acetal) (U-35910) $P^5CN_{16\alpha}on_{11}\theta\theta II_{3,20}$	1 0.1 0.03 0.015	3 0 — —	3	3	0 — —	0 —	1	3	2.5 1.5 1.5 0	0 —	0 —	**1.6**	**60**
24 **IM 483**		3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3-sulfate ammonium salt **PCN ammonium sulfate** (U-37863C) $P^5CN_{16\alpha}{*}S''(NH_4)_{3\beta}\,on_{20}$	0.5 0.03 0.015	3 3 1	3 1.5 2	0 — —	0 — —	3	2 0 —	0 —	3 3 0.5	0 — —	0.5 — —	**1.5**	**60**
25 **IM 461**		3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile, cyclic(2,2-dimethyltrimethylene acetal) (U-36961) $P^5CN_{16\alpha}ol_{3\beta}\theta\theta III_{20/I//2,2}$	0.5 0.03 0.015	2 2 0	3 0	0.5 —	0 —	1	3 0	0 —	3 0	0 —	0 —	**1.3**	**60**
26 **IM 31**		3β-Hydroxy-5-androsten-17-one **Dehydroepiandrosterone** (Ayerst) $A^5ol_{3\beta}on_{17}$	10 0.5 0.1	2 0 —	0.5 — —	1 0 —	0 — —	2 2 0	2 0 —	0 —	3 0.5 —	0 —	0 —	**1.1**	**60**

Table 136 (continued)

Group	Steroidal Conditioning Agent	Dose mg	Toxicant: Digitoxin	Dioxathion	Parathion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_2HPO_4 + Corn oil	Dihydrotachysterol (DHT)	OPI	PSI %
27 **IM 54** 	17α-Methyl-5-androstene-3β,17-diol **Methylandrostenediol "MAD"** (Organon)	10 0.5	0 —	3 0	0 —	0 —	3 0	3 0	3 0	0 —	0 —	2 0	**1.4**	**50**
	$A^5I_{17\alpha}ol_{3\beta,17}$													
28 **IM 477** 	6β-Hydroxy-20-oxo-3α,5α-cyclo-pregnane-16α-carbonitrile (U-36710)	0.5 0.03 0.015	2 0 	2 0 	0 — 	0 — 	0 — 	3 1 0	3 0 	3 0 	0 — 	0 — 	**1.3**	**50**
	$P^{3(5)}5\alpha CN_{16\alpha}ol_{6\beta}on_{20}$													
29 **IM 413** 	3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile acetate **PCN acetate** (U-34889, Syntex)	0.5 0.1 0.03 0.015	1.5 0.5 0 —	1 	0 — — —	0 — — —	0 — — —	3 — 0 —	3 	2 1.5 1.5 0	0 — — —	0 — — —	**1.1**	**50**
	$P^5CN_{16\alpha}$ *$II''_{3\beta}on_{20}$													
30 **IM 73** 	17α-Hydroxy-4-pregnene-3,20-dione acetate **17-Acetoxyprogesterone** (U-5533)	10 0.5	3 0	1 0.5	0 —	0 —	0 —	2 0	0 —	3 0.5	0 —	2 0	**1.1**	**50**
	P^4 *$II''_{17}on_{3,20}$													

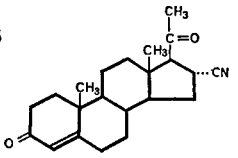
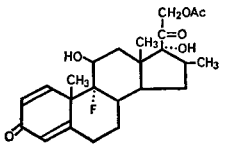
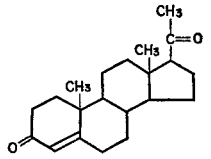
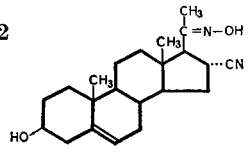
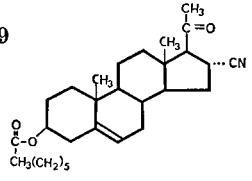
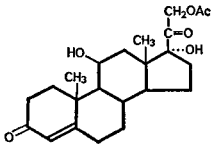
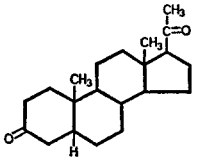
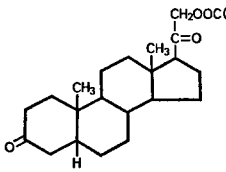
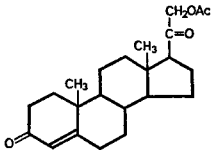
31 IM 466		16α-Cyano-4-pregnene-3,20-dione **Cyanoprogesterone** (SC-4688) **P⁴CN$_{16\alpha}$on$_{3,20}$**	0.5 0.1 0.03 0.015	— 3 0	1	0 — — —	0 — — —	1	3 0 —	2 2 	— 0 — 0	0 — — —	0 — — —	**0.9**	**50**
32 IM 151		9α-Fluoro-16β-methyl-11β,17,21-trihydroxy-1,4-pregnadiene-3,20-dione 21-acetate **Betamethasone acetate** (Schering) **P^{1,4}I$_{16\beta}$ ol$_{11\beta,17}$*II″$_{21}$on$_{3,20}$F$_{9\alpha}$**	10 2 1 0.5 0.1 0.03	2 3 2 1.5 0 —	— — 1 0.5 0 —	— — —1.5 0 0 —	— — 0 — — —	— — 0 — 1 0	— — 3 3 1 0	— — 0 — — —	0 3 1.5 1.5 —1 —	— — 0 — — —	— — 1.5 0.5 0 —	**0.8**	**50**
33 IM 74		4-Pregnene-3,20-dione **Progesterone** (Roussel, Organon) **P^{4}on$_{3,20}$**	10 0.5	1.5 0	0.5 0.5	0 —	1 0	0 —	0 —	0 —	3 0	0 —	1.5 0	**0.8**	**50**
34 IM 462		3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile, 20-oxime (U-37722) **P^{5}CN$_{16\alpha}$ol$_{3\beta}$NOH$_{20}$**	0.5 0.03 0.015 0.01	3 2.5 0.5 0	3 0 	0 — —	0 — —	0 0 —	3 0 —	0 — —	3 3 1	0 — —	0 — —	**1.2**	**40**
35 IM 479		3β-Hydroxy-20-oxo-5-pregnene-16α-carbonitrile 3-heptanoate **PCN heptanoate** (U-37001) **P^{5}CN$_{16\alpha}$ *VII″$_{3\beta}$on$_{20}$**	0.5 0.03 0.015	3 1 	3 0 	0 — —	0 — —	0 0 	2 0 	0 — —	3 1.5 0	0 — —	0 — —	**1.1**	**40**

Table 136 (continued)

Group		Steroidal Conditioning Agent	Dose mg	Toxicant: Digitoxin	Dioxathion	Parathion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_2HPO_4 + Corn oil	Dihydrotachysterol (DHT)	IPO	PSI %
36 **IM 45**		**11β,17,21-Trihydroxy-4-pregnene-3,20-dione 21-acetate** **Cortisol acetate** (Roussel) $P^4ol_{11\beta,17}$ *$II''_{21}on_{3,20}$	10 0.5 0.1 0.03	**3** **0** — —	**0** **0** — —	**—0.5** **0** — —	**—0.5** **0** — —	**3** **0** — —	**0** **3** **2** **0**	**3** **2** **1** **0**	**0** **0** — —	**—0.5** **0.5** — —	**1.5** **1** **0** —	**0.9**	**40**
37 **IM 72**		5β-Pregnane-3,20-dione **Pregnanedione** (Searle) **P5β** $on_{3,20}$	10 0.5	**0** —	**1** **0**	**2.5** **0**	**0** —	**2** **0**	**0** —	**0** —	**2** **0**	**0** —	**0** —	**0.8**	**40**
38 **IM 11**		**21-Hydroxy-5β-pregnane-3,20-dione hemisuccinate sodium salt** **Hydroxydione sodium** (Schering, Pfizer) **P5β** *suc''Na_{21} $on_{3,20}$	10 0.5	**3** **0**	**1.5** **0**	**0.5** **0**	**0.5** **0**	**0** —	**0** —	**0** —	**0** —	**0** —	**0** —	**0.6**	**40**
39 **IM 25**		**21-Hydroxy-4-pregnene-3,20-dione acetate** **Desoxycorticosterone acetate** **"DOC-Ac"** (Schering) P^4 *$II''_{21}on_{3,20}$	10 0.5	**0** —	**1.5** **0**	**0** —	**2** **0**	**0** —	**0** —	**0** —	**1** **0.5**	**0** —	**0.5** —	**0.5**	**40**

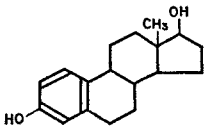
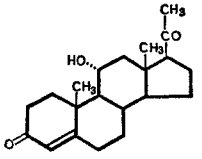
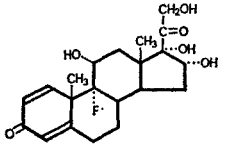
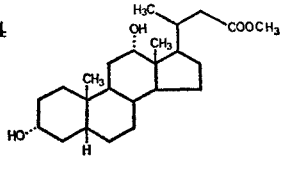
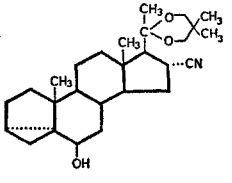
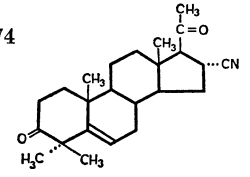
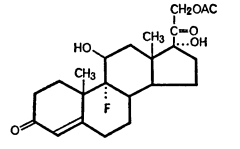
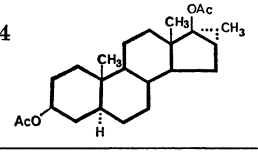
40 **IM 33**		1,3,5(10)-Estratriene-3,17β-diol	10	0	—	—	0	—	—	0	—	0.5	0		
		Estradiol	1	0	1.5	—1.5	0	1	1	0	0.5	0	0	**0.3**	**40**
		(Roussel)	0.5	—	0.5	0	—	0	3	—	—	—	—		
			0.1	—	—	—	—	0	2	—	—	—	—		
		$E^{1,3,5(10)}ol_{3,17\beta}$	0.03	—	—	—	—	—	0	—	—	—	—		
41 **IM 119**		11α-Hydroxy-4-pregnene-3,20-dione	10	1.5	0	0	0	0	2	0	3	0	0	**0.7**	**30**
		11α-Hydroxyprogesterone	0.5	0	—	—	—	—	0	—	0	—	—		
		(SKF, Ayerst)													
		$P^{4}ol_{11\alpha}on_{3,20}$													
42 **IM 32**		9α-Fluoro-11β,16α,17,21-tetra-	10	—	—	—	0	—	—	0	—	0.5	1.5		
		hydroxy-1,4-pregnadiene-3,20-dione	2	0	—1.5	—0.5	0	1	0	—	0	1	1.5	**0.2**	**30**
		Triamcinolone	0.5	—	0	0	—	0	—	—	—	1.5	0		
		(Lederle)	0.1	—	—	—	—	—	—	—	—	0	—		
		$P^{1,4}ol_{11\beta,16\alpha,17,21}on_{3,20}F_{9\alpha}$													
43 **IM 124**		3α,12α-Dihydroxy-24-nor-5β-cholan	10	3	0	0	—0.5	0	3	0	3	0	0	**0.9**	**30**
		23-oic acid methyl ester	0.5	0	—	—	—	—	0	—	0	—	—		
		Methyl nordeoxycholanate													
		(SKF)													
		CHn24,5β''$ol_{3\alpha,12\alpha}$ *I_{23}													
44 **IM 480**		6β-Hydroxy-20-oxo-3α,5α-cyclo-	0.5	1	0	0	—0.5	0	2	0	3	0	0	**0.6**	**30**
		pregnane-16α-carbonitrile,	0.03	0	—	—	—	—	0	—	0	—	—		
		cyclic(2'2'-dimethyltrimethylene													
		acetal)													
		(U-37483)													
		$P^{3(5)}5\alpha CN_{16\alpha}ol_{6\beta}\theta\theta III_{20/1//2,2}$													

Table 136 (continued)

Group	Steroidal Conditioning Agent	Dose mg	Toxicant: Digitoxin	Dioxathion	Parthion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_3HPO_4 + Corn oil	Dihydrotachysterol (DHT)	OPI	PSI %
45 IM 474 	4,4-Dimethyl-3,20-dioxo-5-prenene-16α-carbonitrile (U-35641) $P^5I_{4,4}CN_{16\alpha}on_{3,20}$	0.5 0.03	0.5 —	0 —	0 —	0 —	1	0 —	0 —	3 0	0 —	0 —	**0.5**	**30**
46 IM 39 	9α-Fluoro-11β,17,21-trihydroxy-4-pregnene-3,20-dione 21-acetate **9α-Fluorocortisol (F-COL) acetate** (U-4845) $P^4ol_{11\beta,17}\ *II''_{21}on_{3,20}F_{9\alpha}$	2 0.5 0.1	0 — —	0 — —	0 — —	—0.5 — —	2 0 0	0 — —	0 — —	0.5 0 —	— — —	0 — —	**0.2**	**20**
47 IM 454 	16α,17-Dihydro-3′H-cyclopropa(16,17)-5α-androstane-3β,17β-diol diacetate (SC-21940) $A5\alpha I_{16\alpha-17}\ *II''_{3\beta,17\beta}$	0.5	0	0	0	0	0	0	0	0	0	0	**0**	**0**
	OPI		**2.2**	**1.6**	**0.6**	**0.6**	**1.5**	**2.1**	**1.2**	**2.1**	**0.3**	**1.0**		
	SPI%		**80**	**80**	**40**	**30**	**70**	**80**	**50**	**90**	**20**	**60**		

The infarctoid cardiopathy produced by fluorocortisol + Na_2HPO_4 + corn oil, which is inhibited by several steroids (particularly spironolactone and spiroxasone, among those listed in Table 136), was consistently resistant to prophylaxis by any of the nonsteroidal drugs in Table 137. Of course, potassium salts (e.g., KCl) or potassium-sparing agents (e.g., amiloride, triamterene) offer excellent protection against this cardiopathy, as shown by our previous investigations, and since spironolactone and spiroxasone likewise retain potassium, it is probable that here they also act primarily through this mechanism.

The relative amenability of the other toxicants to protection by nonsteroidal conditioning agents can be most readily appraised on the basis of the indexes listed in the last two horizontal lines of Table 137, p. 848. It will be seen that indomethacin and dioxathion have the highest PSI, but in general, the overall protective effect of these nonsteroidal agents falls far short of that of the steroids listed in Table 136. Indeed, whatever overall protective values can be ascribed to the set of nonsteroidal agents are mainly due to the comparatively high efficacy of phenobarbital, phetharbital, diphenylhydantoin, phenylbutazone, and to a lesser extent, of tolbutamide and W-1372; the other agents in this list are either inactive or offer protection only against very few toxicants.

Third Step: Identification of Damaging Agents Amenable to Prophylaxis

As previously stated, this third step of the screening procedure was designed primarily to identify the types of compounds that can be detoxified by steroids. However, for comparative purposes, we have also tested thyroxine and phenobarbital under identical conditions, as examples of nonsteroidal agents previously shown to influence resistance against many toxicants. The steroids included in this battery of tests were purposely selected to comprise proven syntoxic or catatoxic substances, as well as compounds which had never been shown to protect against any toxic agent.

The prophylactic steroids were administered, as outlined on p. VIII, in 1 ml water by a stomach tube, twice daily, from the first day until termination of the experiment, unless otherwise stated in the footnotes. Thyroxine was administered at the dose of 0.2 mg in 0.2 ml water s.c., once daily, and phenobarbital at the dose of 6 mg in 1 ml water p.o., twice daily, as described in Table 137. The treatment with the toxicants and the assessment of the lesions they produce was again expressed as outlined on p. 834. The results are summarized in Table 138, p. 850.

In this series of experiments, the "OPI" and "PSI" refer to the amenability of the individual damaging agents to the protective effect of the compounds listed in the caption of Table 138. In other words, in the two last vertical columns of Tables 135, 136, 137, these indexes were computed to express the protective action of many agents against a standard set of toxicants, whereas in Table 138 (as in the two last horizontal lines of Tables 136, 137) they are meant to reflect the amenability of diverse toxicants to inactivation by a standard set of potential prophylactic agents.

In Table 137, the damaging agents are listed merely in alphabetic order but a glance at the OPI column reveals that the **toxicants most amenable to prophylaxis** by diverse agents are: cocaine, cyclobarbital, cycloheximide, digitoxin, EPN, ethion, ethylmorphine, glutethimide, hexobarbital, indomethacin, methyprylon, nicotine,

Table 137. *Second step (Ctd.): "Protective Spectrums" of some nonsteroidal agents*

Nonsteroidal Conditioning Agent	Dose[a]	Toxicant: Digitoxin	Dioxathion	Parathion	Nicotine	Hexobarbital	Progesterone	Zoxazolamine	Indomethacin	Fluorocortisol + Na_2HPO_4 + Corn oil	Dihydrotachysterol	OPI	PSI%
Phetharbital (Burroughs Wellcome) in 1 ml water, p.o., twice daily	10	2	3	2	2.5	3	3	3	3	0	2.5	**2.4**	**90**
	0.5	0	1.5	1	0	0	0	0	0	—	0		
	0.1	—	0	0	—	—	—	—	—	—	—		
Diphenylhydantoin (Eastman) in 1 ml water, p.o., twice daily	25	1.5	1.5	2	3	3	3	3	3	0	0.5	**2.1**	**90**
	6	0	2.5	3	0.5	3	2	2	3	—	0.5		
	1	0	0	1.5	0	0		0	0	—	0.5		
Phenobarbital sodium (BDH) in 1 ml water, p.o., twice daily	6	0	3	2.5	3	3	3	1	3	0	2	**2.1**	**80**
	0.5	0	2	1	0	3	3	0	3	—	0		
	0.1	0	0	0.5	—	1	1	0	0	—	—		
	0.03	—	—	—	—	0	—	—	—	—	—		
Phenylbutazone (Geigy) in 1 ml water, p.o., twice daily	10	0	3	1	3	3	3	0	3	0	0	**1.6**	**60**
	0.5	—	0	0	0	0	1	—	0	—	—		
	0.1	—	—	—	—	—	0	—	—	—	—		
Tolbutamide (Hoechst Pharmaceutical) in 1 ml water, p.o., twice daily	50	0	3	1	0	3	3	0	2	0	0.5	**1.3**	**60**
	10	—	0.5	0	—	0	2	—	0	—	—		
	5						0						
W-1372 (Wallace) in 1 ml corn oil, p.o., twice daily	10	0	3	0	0	3	3	3	3	0	0	**1.5**	**50**
	5	0	3	1	0	0	2	3	0	0	—		
	1	0	1.5	0.5	0	0	0	0	0	0	—		
	0.5	—	0.5	—	—	—	—	0	—	—	—		
Vitamin E (Distillation Products Ind.) in 1 ml corn oil, p.o., twice daily	50	0	1	0	0	1	1	0	0	0	1.5	**0.5**	**40**
	10	—	—	—	—	0	—	—	—	—	0		
Bile duct ligature	—	0	0.5	0	0	—3	—3	—3	1.5	1.5[b]	1.5	**—0.4**	**40**
Acetylsalicylic acid (Merck) in 2 ml water, p.o., twice daily	10	0	0	0	0	0	3	1	3	0	0	**0.7**	**30**
	0.5	—	—	—	—	—	0	0	0	—	—		

Sodium salicylate (Fisher) in 1 ml water, p.o., twice daily	10	0	0	1	0	0	1	0	3	0	0	0.5	30
	0.5	—	—	0	—	—	0	—	0	—	—		
Nicotine (Eastman Organic Chemical) in 1 ml water, p.o., twice daily	3	1	0.5	0	0	0	0	0	1.5	0	0	0.3	30
	0.15	0	—	—	—	—	—	—	0	—	—		
ACTH (Nordic Biochemical Ltd.) in 0.2 ml water, s.c., twice daily	50 I.U.	0	0	0	0	0	0	2	0	0	1	0.3	20
	2.5 I.U.	—	—	—	—	—	—	1	—	—	0		
Vitamin D_2 (Wander) in 0.5 ml corn oil, p.o., twice daily	0.025	0.5	0	0.5	0	0	0	0	0	0	0	0.1	20
Vitamin A (Hoffmann-La Roche) in 0.5 ml corn oil, p.o., twice daily	2500 I.U.	0	0	0	0	0	0	0	0	0	0.5	0.1	10
Indomethacin (Merck Sharp & Dohme) in 0.2 ml water, s.c., twice daily	0.15	0	0	0	0	0	0	0	0	0	0	0	0
STH (C.H.Li) in 0.2 ml water, s.c., twice daily	1	0	0	0	0	0	0	0	0	0	0	0	0
	0.05	—	—	—	—	—	2	0	—	—	—		
	0.01	—	—	—	—	—	0	—	—	—	—		
Vitamin C (Fisher) in 1 ml water, p.o., twice daily	50	0	0	0	0	0	0	—1	0	0	0	—0.1	0
Digitoxin (Roussel) in 1 ml water, p.o., twice daily	0.15	0	0	0	0	0	0	0	0	0	—0.5	—0.1	0
L-Thyroxine (BDH) in 0.2 ml water, s.c., daily	0.2	0	—1.5	—2	0	0	0	0	0	0	0	—0.4	0
	0.01	—	0	0	—	—	—	—	—	—	—		
Phentolamine (Ciba) in 0.2 ml water, s.c., twice daily	3	2	1	—	—	0	—	—	0.5	—	—		
	0.5	0	0	—	—	—	—	—	—	—	—		
OPI		0.4	0.9	0.4	0.6	0.8	1.1	0.5	1.3	0.1	0.5		
PSI %		25	50	36	21	35	47	31	55	5	42		

[a] Doses in international units (I.U.) are so indicated: all other dosages are expressed in milligrams.

[b] This inhibition may be spurious since all rats were moribund or dead by the end of the experiment, although they showed no cardiac necrosis.

Table 138. *Third step: Identification of damaging agents amenable to prophylaxis*

Toxicant	Conditioning Agent: Phenobarbital	PCN	Ethylestrenol	CS-1	Spironolactone	Norbolethone	Oxandrolone	Prednisolone-Ac	Progesterone	Triamcinolone	DOC-Ac	Hydroxydione	Estradiol	Thyroxine	MPI[1]	OPI[2]	PSI[3]
Acetanilide	1.5	1.5	1.5	1.5	1.5	1.5	0	1.5	0	0	0	0	—1	1.5	**0.8**	**12**	**60**
Acrylamide	3	3	0	1.5	0.5	0	0	0	0	0	0	0	0	0	**0.6**	**8**	**30**
Aminoacetonitrile	0	0	0	0	0	0	0	3	1	3[c]	1	0	1[b]	3	**0.9**	**12**	**40**
o-Aminophenol HCl	0	—	0	0	0	0	0	0	0	0	0	—0.5	0	—1.5	**—0.2**	**0**	**0**
Aminopyrine	1	2.5[b]	3	2.5	1	2.5	0	2.5	2.5	2.5	0	0	0	0	**1.4**	**20**	**60**
DL-Amphetamine	0	0	0.5	0	0.5	0	0	—0.5	0	—1.5	0	0	0	—1.5	**—0.2**	**1**	**10**
Arsenic pentoxide	1	0	0	0	1	0	0	—1.5	0	—0.5	0	0	—3	0	**—0.2**	**2**	**10**
Barbital	—2	0	0	—1	0	—1	0	2	0	0[a]	—3	—1	—3[b]	0	**—0.6**	**2**	**10**
Bile duct ligature	—	—	0	—	0	0	0	—1	0	—1[a]	0	0	0[b]	0	**—0.2**	**0**	**0**
Bishydroxycoumarin	0	0	1	1	2	1	2	0	0	0[a]	0	0	3	0	**0.7**	**10**	**40**
Bromobenzene	—	—	—0.5	0	0	0	0	—1	—0.5	—1	0	0	0	0	**—0.3**	**0**	**0**
Brompheniramine maleate	—	—	0	0	0	0	0	0	0	0[a]	0	0	0[b]	0	**0**	**0**	**0**
Cadmium chloride	0	1.5	1	0	1	0.5	0	2.5	0	1.5	0	0	2.5	1	**0.8**	**12**	**60**
Caramiphen HCl	0.5	1.5	1	0.5	0	0	0	1	0	0	0	0	0	—1	**0.3**	**5**	**40**
Carisoprodol	0	1	1	3	2	0	0	2	—1	0	0	0	0	—1	**0.5**	**9**	**40**
Chlordiazepoxide	0	3	3	3	1	1	0	0	0	0	0	0	0	0	**0.8**	**11**	**40**
Cinchophen	3	3	2	0	3	2	0	3	0	0[a]	0	0	0[b]	0	**1.1**	**16**	**40**
Cocaine	3	3	3	3	3	2.5	2	3	1.5	0	0	1	3	0	**2.0**	**28**	**80**
Colchicine	2	3	1.5	3	3	0	2	0	0.5	0[a]	0.5	0	0[b]	0	**1.1**	**16**	**60**
DL-Coniine	0	2	0	0	0	0	0	1.5	0	2	—0.5	0	2	0	**0.5**	**8**	**30**
Croton oil	0	2	0	0	0	0	0	2	0	2[a]	0	0	0[b]	0	**0.4**	**6**	**20**
Cyclobarbital	3	3	3	3	3	3	3	3	0	3[a]	0	0	0[b]	0	**1.9**	**27**	**60**
Cycloheximide (100 μg)	3	3	3	3	3	3	3	0	3	0[a]	1	1	0	0	**1.9**	**26**	**70**

Cyclophosphamide	0	3	1	3	3	1.5	0	—1.5	1.5	—1.5	0	0	—1.5	—0.5	**0.6**	**13**	**40**
DDT	1.5	0	0	0	0	0	0	0	0	—0.5[a]	0	0	0[b]	—0.5	**0.04**	**2**	**10**
DHT	3	2	3	3	2	3	3	1.5	1.5	1.5[a]	0.5	0	0	0	**1.7**	**24**	**80**
Digitoxin	0	3	3	3	3	3	3	2	1.5	0[a]	0	3	0	0	**1.8**	**25**	**60**
Digitoxin + Na_2HPO_4 + Oil	0.5	1.5	2	2.5	2	2.5	1.5	0	2	0	0	0	0	0	**1.0**	**15**	**60**
Diisopropylfluorophosphate "DFP"	0	0	0.5	0.5	0.5	0	0	0.5	0	0[a]	0	0	0[b]	0	**0.1**	**2**	**30**
Dimercaprol	—	0	0	0	0	0	0	0	0	0	0	—	0	—0.5	**—0.04**	**0**	**0**
Dinitrophenol	—	0	0	0	0	0	0	0	0	0	0	—	0	—2	**—0.2**	**0**	**0**
Dioxathion	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	0.5	—1.5[a]	1.5	0	1.5[b]	—1.5	**0.9**	**16**	**80**
Diphenylhydantoin	1	0	0	1	1	0	0	—3	0	—3[a]	0	0	0[b]	—1	**—0.3**	**3**	**20**
Dipicrylamine	0	2	0	2	0	0	0	0	0	—1.5	0	0	0	—1.5	**0.1**	**4**	**10**
DOC-Ac + NaH_2PO_4	0.5	0	0	0	0	0	0	—3	0	—3[a]	0	0	0[b]	1.5	**—0.3**	**2**	**10**
Doxepin	0	0	1	0	0	0	0	0	0	0	0	0	0	0	**0.1**	**1**	**10**
E-coli endotoxin No. 08	0	0	0	—0.5	0	—0.5	0	1.5	0	3[a]	0	0	0[b]	—1	**0.2**	**5**	**10**
Edrophonium chloride	—	—	0	0	0	0	0	0	0	0	0	—	0	0	**0**	**0**	**0**
Elementary yellow phosphorus	—	—	0	0	0	0	0	0	0	0[a]	0	—	0[b]	0	**0**	**0**	**0**
Emetine HCl	0	1.5	0	1.5	0.5	0	—0.5	—0.5	—0.5	0	—0.5	0	0	—0.5	**0.1**	**4**	**20**
Ephedrine sulfate	0.5	0	0	0	0	0	0	—0.5	0	0	0	0	0	—1	**—0.1**	**1**	**10**
Epinephrine	—0.5	0	0	0	0	0	0	—0.5	0	—1.5	0	—0.5	—1	—3	**—0.5**	**0**	**0**
EPN	3	3	3	3	3	3	3	0.5	0.5	0[a]	1.5	2	2[b]	—1	**1.9**	**28**	**90**
Estradiol + NaH_2PO_4	0	—0.5	0	0	0	—0.5	0	—1.5	0	—1.5[a]	0	0	0[b]	0.5	**—0.3**	**1**	**10**
Ethion	3	3	3	3	3	3	3	2.5	3	0[a]	0	0	1[b]	0	**2**	**28**	**70**
Ethyl alcohol	0	0	0	0	0	0	0	—1.5	0	—0.5[a]	0	0	0[b]	0	**—0.1**	**0**	**0**
Ethylene chlorohydrin	—	0	0	0	0	0	0	0	0	0.5[a]	0	—	0[b]	0	**0.04**	**1**	**10**
Ethylene glycol	0	0	0	0	—0.5	0	0	1.5	0	2[a]	0	0	—2	2.5	**0.3**	**6**	**20**
Ethylmorphine	3	3	3	3	3	2	2	1	2	0	1.5	0	3	0	**1.9**	**27**	**80**
Fasting	—	—	0	—	0	0	0	0	0	0	0	0	0	—	**0**	**0**	**0**
Flufenamic acid	2	1.5	2	1.5	1.5	0.5	0	—1	0	—1	0	0	0.5	—0.5	**0.5**	**10**	**50**
Fluorocortisol + $NaClO_4$ + Oil	1	1.5	0	2	2	0.5	0	—0.5	0	—1[a]	0	0	0	0	**0.4**	**7**	**40**

[a–c] Administered at the dose level of: [a] 2 mg, [b] 1 mg, [c] 0.5 mg.

[1] Algebraic sum of all grades divided by numbers of toxicants tested.

[2] Arithmetic sum of all grades.

[3] Percentage of all positive grades taking grade "3" protection by all conditioning agents as 100%.

Table 138 (continued)

Toxicant	Conditioning Agent: Pheno-barbital	PCN	Ethyl-estrenol	CS-1	Spirono-lactone	Nor-bolethone	Oxan-drolone	Predni-solone-Ac	Proges-terone	Triamci-nolone	DOC-Ac	Hydroxy-dione	Estradiol	Thyroxine	MPI[1]	OPI[2]	PSI[3]
Fluorocortisol + Na_2HPO_4 + Oil	0	1.5	0	1.5	2	1	0	0.5	0	0.5	0	0	0.5	0	**0.5**	**8**	**50**
Fluorocortisol + Na_2HPO_4 + Restraint	1	2	0	1.5	2	0	0	—0.5	0	—0.5	0	1	0	0	**0.5**	**8**	**40**
Fluphenazine di HCl	0	3	1	3	0	0	0	1	0	0	1	0	0	0	**0.6**	**9**	**40**
Glutethimide	3	3	3	3	3	3	1	3	0	3	0	0	2	0	**1.9**	**27**	**70**
Glycerol	0.5	0	1.5	1	0	1	0.5	1.5	1	—2	0	0	0	1.5	**0.5**	**9**	**60**
Griseofulvin	0	0	0	0	0	0	0	0.5	0	0[a]	0	—0.5	0[b]	0	**0**	**1**	**10**
Guthion	3	3	3	3	3	3	3	1	0	0[a]	0	0	0[b]	0	**1.6**	**22**	**60**
Haloperidol	0	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1	—1.5	0	0	0	—1.5	**0.6**	**12**	**60**
Heptachlor	0	0	0	0	0	—0.5	0	0.5	—0.5	0	0	0	—0.5	—0.5	**—0.1**	**1**	**10**
Hexamethonium Cl	0	0	0	0	0	0	0	3	0	3	0	0	0	0	**0.4**	**6**	**10**
Hexobarbital	3	3	3	3	3	3	3	3	0	1[a]	0	0	1[b]	0	**1.9**	**26**	**70**
Homatropine HBr	—	2	0	0	0	0	0	0	0	0	0	0	0	0	**0.2**	**2**	**20**
Hydrazine	0.5	0	0	0	0	0	0	2	0	1.5	0	0	0	0.5	**0.3**	**5**	**30**
Hydroquinone	2	2	3	1	0	0	0	—1	0	—3	0	0	0	0	**0.3**	**8**	**30**
Imipramine	2.5	2	2	1.5	2	0	0	0	0	0	0	0	0.5	0	**0.8**	**11**	**40**
Indium trichloride	—	0	0	0	0	0	0	0	0	—0.5	0	0	0	—0.5	**—0.1**	**0**	**0**
Indomethacin	3	3	3	3	3	3	3	3	3	0[a]	1	0	0.5[b]	0	**2.0**	**29**	**80**
LSD	1	3	3	3	0	3	0	3	1	3	0	0	3	0	**1.6**	**23**	**60**
Mechlorethamine HCl	0	0	0	0	0	0	—1	—3	0	—3	0	—3	0	—3	**—0.9**	**0**	**0**
Mephenesin	0	3	3	3	3	3	0	1	0	0[a]	1	0	0	0	**1.2**	**17**	**50**
Meprobamate	3	3	3	3	3	3	3	3	0	0[a]	0	0	0[b]	0	**1.7**	**24**	**60**
Mercuric chloride (400 μg)	0	0	0.5	0	3	0	0	0.5	2	0	0	0	1	—0.5	**0.5**	**7**	**40**
Mersalyl (4 mg)	0	0	2	0	3	1.5	0.5	3	0.5	1	0	0	0	0	**0.8**	**12**	**50**

Methadone	0	0	1.5	0	0	0	0	0	—1	0.5	0	0	0	—0.5	**0.04**	**2**	**10**
n-Methylaniline	2	2[b]	1	0.5	0	1	0	—0.5	—2	—2	0	0	0.5	—2	**0.3**	**7**	**40**
Methylphenidate	0	0	0	0	0	0	0	0	0	0	0	0	0	—1.5	**—0.1**	**0**	**0**
Methylsalicylate	0.5	1.5	1.5	1.5	1.5	1.5	0	—0.5	0	—0.5	0.5	0	0	—0.5	**0.5**	**9**	**50**
Methyprylon	3	3	3	3	3	3	3	3	3	3	0	1	2	0	**2.4**	**33**	**90**
Morphine sulfate	0	0	0	0	0	0	0	0	0	0	0	0	0	—0.5	**—0.04**	**0**	**0**
α-Naphthylisothiocyanate	3	3	3	2.5	0	0.5	0	0	0	0	0	0	0	0	**0.9**	**12**	**40**
Neostigmine Br	1.5	1.5	0.5	0.5	0	0	0	0.5	—0.5	—1.5	0	0	—1	—1.5	**0**	**5**	**40**
Nephrectomy	—	—	0	—	0	0	0	0	0	0	0	0	0	0	**0**	**0**	**0**
Nicotine	3	2.5	3	3	3	3	3	0.5	1	0	2	0.5	0	0	**1.8**	**25**	**80**
Nikethamide	1.5	1	1.5	1.5	0.5	1.5	0	0	1.5	—1.5	1.5	1	1.5	—0.5	**0.8**	**13**	**70**
p-Nitroanisole	0	2	0.5	0	0	0.5	0	0	0	2	0	0	0.5	0	**0.4**	**6**	**40**
OMPA (1 mg)	0	0	0	0	0	0	0	0	0	0	0	0	—3	0	**—0.2**	**0**	**0**
Pancuronium Br	0	1.5	1	3	3	0	0	2.5	0	1.5	0	0	0	0.5	**0.9**	**13**	**50**
Parathion	2.5	2	2	1.5	2	1.5	0	0	0	—0.5[a]	0	0.5	—1.5[b]	—2	**0.6**	**12**	**50**
Pentylenetetrazol	1	3	0	0	0	2.5	0	0	2.5	0	0	0	0	0	**0.6**	**9**	**30**
Phenindione	3	1.5	3	3	3	3	3	0	1	0[a]	0.5	1	0.5[b]	0	**1.6**	**23**	**80**
Phenyl isothiocyanate	3	3	2.5	0.5	3	1	0.5	0	0	0	0	0	0	0	**1.0**	**14**	**50**
Phenyramidol	3	3	3	3	3	3	2	2	0	0	0	0	3	0	**1.8**	**25**	**60**
Physostigmine sulfate	0	0.5	1	1	0.5	0	0	0.5	0	0[a]	0	0	—1[b]	—2	**0.04**	**4**	**40**
Picrotoxin	3	3	3	3	3	3	3	3	3	0.5[a]	2	1	0[b]	0	**2.2**	**31**	**90**
Piperidine	3	0	3	3	0	3	3	0	3	3	1	3	0	1	**1.9**	**26**	**70**
Pipradol	—	1.5	0	0	0	0	0	0	0	0	0	0	0	—0.5	**0.1**	**2**	**10**
Pralidoxime Cl	0.5	0	1	0	0	0	0	2.5	0	2	0.5	0	1	0	**0.5**	**8**	**40**
Progesterone	3	3	3	3	3	3	2	3	0	0[a]	0	0	1[b]	0	**1.7**	**24**	**60**
Propionitrile	0	—2.5	0	0	0	0	0	—2.5	0	—3	0	0.5	0.5	—3	**—0.7**	**1**	**10**
Propylthiouracil	2	2	1.5	2	1.5	1.5	2	0	0.5	—0.5	0	0	0	—0.5	**0.9**	**13**	**60**
Pyrilamine	—	—	0	0	0	0	0	0	0	0	0	0	0	0	**0**	**0**	**0**
SKF 525-A	1	2	1	3	3	1	3	3	3	3[a]	0	1	0[b]	0	**1.7**	**24**	**80**
Sodium perchlorate	0	0	0	—1.5	0	0	0	—1	0	0[a]	0	0	0[b]	1.5	**—0.1**	**2**	**10**
Strychnine	3	0.5	0.5	0	0.5	0	0	3	0	3	0	0	3	—0.5	**0.9**	**14**	**50**
Tetraethyl ammonium Cl	1.5	0	0	0	0	0	0	2.5	0.5	3	0	0	1	0.5	**0.6**	**9**	**40**
Thallium chloride	—	0	0	0	0	0	0	0	0.5	0.5	0	0	0.5	0	**0.1**	**2**	**20**

Table 138 (continued)

Toxicant	Conditioning Agent: Phenobarbital	PCN	Ethylestrenol	CS-1	Spironolactone	Norbolethone	Oxandrolone	Prednisolone-Ac	Progesterone	Triamcinolone	DOC-Ac	Hydroxydione	Estradiol	Thyroxine	MPI[1]	OPI[2]	PSI[3]
Theobromine	3	3	0	3	0	0	0	0	2.5	0	0	0	0	0	**0.8**	**12**	**30**
Theophylline	3	3	3	3	0.5	2	2	3	3	0	0.5	0.5	0	0	**1.7**	**24**	**80**
Thimerosal	0	1.5	1.5	1	1	2	0.5	1	0	0	0	0.5	2	0	**0.8**	**11**	**60**
Thioacetamide	—0.5	—2	0	—2	0	—0.5	—2	—2	—2	—1	—2	0	0	—2	**—1.1**	**0**	**0**
Thiopental	3	3	3	3	3	0	2	3	0	0[a]	0	0	0[b]	0	**1.2**	**20**	**40**
Tremorine	0	—	0	0.5	0	0	0	0.5	0	0	0	0	0	0	**0.1**	**1**	**20**
Triamcinolone	—1	3	0	3	2	2	0	—3	0	—3[a]	—1	0	—3[b]	0	**—0.1**	**10**	**30**
Tribromoethanol	0	3	3	3	3	3	0	3	—1	0[a]	—1	0	0[b]	0	**1.1**	**18**	**60**
Trichloroethanol	3	3	3	2	3	2	0	3	0	0	0	0	—2	1	**1.3**	**20**	**60**
3,3,5-Triiodo-L-thyronine	0	0	0.5	0	0	1	0	—1.5	0	—1	0	0	—0.5	0	**—0.1**	**2**	**10**
Tri-o-cresyl phosphate	—1	—1	0	0	0	—0.5	0	—0.5	0	—1	0	0	0	1	**—0.2**	**1**	**10**
d-Tubocurarine	0	3	3	3	3	2	0	3	2	3	0	0	0	0	**1.6**	**22**	**60**
Tyramine HCl	0	0	0.5	0	1	1	0	1	0	2	1	0	0	0	**0.5**	**7**	**40**
L-Tyrosine	3	3	3	1.5	2	1	0	1.5	1	1.5[a]	0	0	0.5	—1	**0.6**	**18**	**60**
W 1372 (40 mg)	2	3	1.5	3	3	1.5	1	0	1.5	0	1	0.5	2	0	**1.4**	**20**	**80**
Warfarin	0	0	0	1	0	0	0	0	0	0	0	0	0	0	**0.1**	**1**	**10**
Zoxazolamine	1	3	2	2	1	0	0	0	0	0	0	0	0	0	**0.6**	**9**	**40**

phenyramidol, picrotoxin, and piperidine. The PSI of these toxicants runs roughly parallel to their OPI, that is to say the agents whose toxicity is most significantly impeded by various prophylactics are, in general, also detoxified by the largest number of potentially prophylactic substances.

Several substrates in Table 138 are very amenable to detoxication but only by few compounds; hence, despite their great activity in one or two respects, they have extremely low general protective indexes. For example, mercuric chloride is almost completely detoxified by spironolactone, yet its overall amenability to protection is very low, because its detoxication depends upon a steroid-borne thioacetyl group. In this series of conditioners, such a substituent occurs only in this particular steroid. Similarly, the intoxications amenable to protection by glucocorticoids only (lathyrogens, ganglioplegics, croton oil, E. coli, pralidoxime, strychnine, ethylene glycol), give comparatively low overall protective indexes, because only two of the prophylactic substances tested possess strong glucocorticoid potency.

Reading the columns in Table 138 vertically, we confirmed that the best **protection against the largest number of toxicants** is offered by the typical catatoxic steroids: PCN, ethylestrenol, CS-1, spironolactone, norbolethone, and oxandrolone.

Furthermore, some systemic toxicants (e.g., cyclobarbital, methyprylon, SKF 525-A, d-tubocurarine) are combated both by catatoxic steroids and by the two glucocorticoids included in this series. Several other drugs (e.g., barbital, cinchophen, cocaine, ethion, meprobamate, picrotoxin, thiopental, tribromoethanol, trichloroethanol) are well detoxified by prednisolone but not by triamcinolone, although the latter is the more potent glucocorticoid. Presumably, prednisolone possesses both catatoxic and syntoxic properties. Yet, here again we must remember that such in vivo tests can only determine whether a compound is or is not amenable to detoxication by steroids that have syntoxic or catatoxic actions with respect to other substrates. Further investigations will be required to identify the underlying mechanism.

DOC, hydroxydione, estradiol and thyroxine are comparatively **ineffective** both as regards the intensity and the spectrum of protection. In fact, in many cases, pretreatment with thyroxine results in toxication rather than detoxication. In the computation of the OPI and PSI, the results of toxication are not deducted from those of detoxication, but merely considered as "O"; hence the aggravating effect of thyroxine emerges only from the MPI.

In general, intoxications that can be inhibited by catatoxic steroids are also amenable to **prophylaxis by phenobarbital.** Yet, here again, there are exceptions: chlordiazepoxide, cyclophosphamide, digitoxin, digitoxin + Na_2HPO_4 + corn oil, haloperidol, mephenesin, triamcinolone, tribromoethanol, and tubocurarine, though readily detoxified by most catatoxic steroids, are virtually resistant to phenobarbital. In several other instances, phenobarbital is effective, but much less so than the most potent catatoxic steroids. The converse is rarely if ever the case; among the intoxications tested, only strychnine and piperidine poisoning responded much better to phenobarbital than to catatoxic steroids, but perhaps here, the hypnotic effect of the drug played a special role.

It must be emphasized that we cannot draw far-reaching conclusions as to the **specificity of catatoxic and syntoxic steroid actions** from this experimental series, since in some groups, the number of experimental animals may have been too small to compensate for individual variations in susceptibility. Furthermore, the conditioners

were administered in fixed amounts; dose effect curves would be required in each case to make more meaningful comparisons. Still even these data suffice to show that there are qualitative differences in the prophylactic actions of the various conditioning agents.

In surveying Table 138, it is striking how few conditioning agents induced a considerable **decrease in resistance** (— 2.5 or more) to the toxicants tested. This is all the more noteworthy since overdosage with two drugs (the toxicant and the conditioning agent) might be expected to be more difficult to resist than treatment with the toxicant alone. Most of the apparent decreases in resistance were seen after conditioning with prednisolone and triamcinolone, but of course here the toxic effect of heavy glucocorticoid overdosage (loss of body weight with a predisposition for spontaneous infections) was probably the decisive factor. In addition, when given in combination with DOC-Ac + NaH_2PO_4, the glucocorticoids notoriously sensitize to myocardial necrosis. Furthermore, when triamcinolone was used as a toxicant, it was obvious a priori that additional treatment with the same or another glucocorticoid would aggravate the overdosage syndrome. It is perhaps more remarkable that intoxication with hydroquinone was greatly accentuated by triamcinolone.

Among the other steroids, let us point out that PCN appears to sensitize to the toxic effects of propionitrile, whereas estradiol considerably aggravates poisoning with OMPA and triamcinolone. Thyroxine markedly diminishes resistance to epinephrine and propionitrile, but to a lesser extent, it also accentuates the toxicity of several other agents and rarely offers protection.

Synoptic "Diagram Table"

The "Diagram Table" (Table 139) graphically summarizes the highlights of Table 138 except that:

1. The results are registered only for inhibitions (not for aggravations) of intoxications.

2. Intermediates between the four basic grades (0, 1, 2 and 3) are disregarded in that:

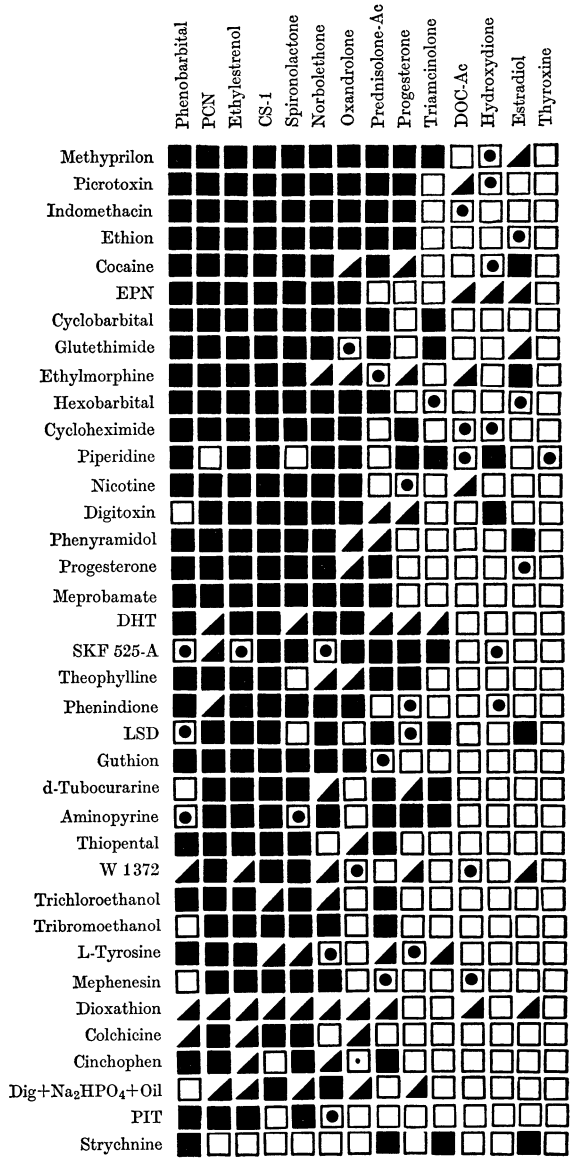
□ = 0.5 or less
◉ = 1
◢ = 1.5 to 2
■ = 2.5 to 3

The absence of any sign indicates that the corresponding experiment has not been done.

3. Here, the toxicants are listed according to decreasing "Total OPI" values.

When thus arranged, it becomes particularly obvious that among the hormonal conditioning agents, PCN is effective against the largest number of toxicants and its protective potency roughly parallels that of phenobarbital at the dose levels employed here. However — as mentioned in discussing Table 138 — the parallelism in the protective value of these two compounds is not absolute. Tables 138 and 139 clearly show furthermore that (in addition to PCN) ethylestrenol, CS-1, spironolactone and oxandrolone have high protective potencies, prednisolone and progesterone are much less active, whereas triamcinolone, DOC, hydroxydione, estradiol and thyroxine protect only against very few toxicants.

Table 139. "*Diagram Table*" [a]



[a] In this Table the compounds are enumerated in the same order as in Table 138. However, in the latter, grades of 0.5 or less were also considered whereas here they were taken as 0 (□). Furthermore, no distinction is made here between 1.5 and 2 or 2.5 and 3. This explains the minor discrepancies between the grading judged by the pictorial symbols and the sequence in which the compounds are listed.

Table 139 (continued)

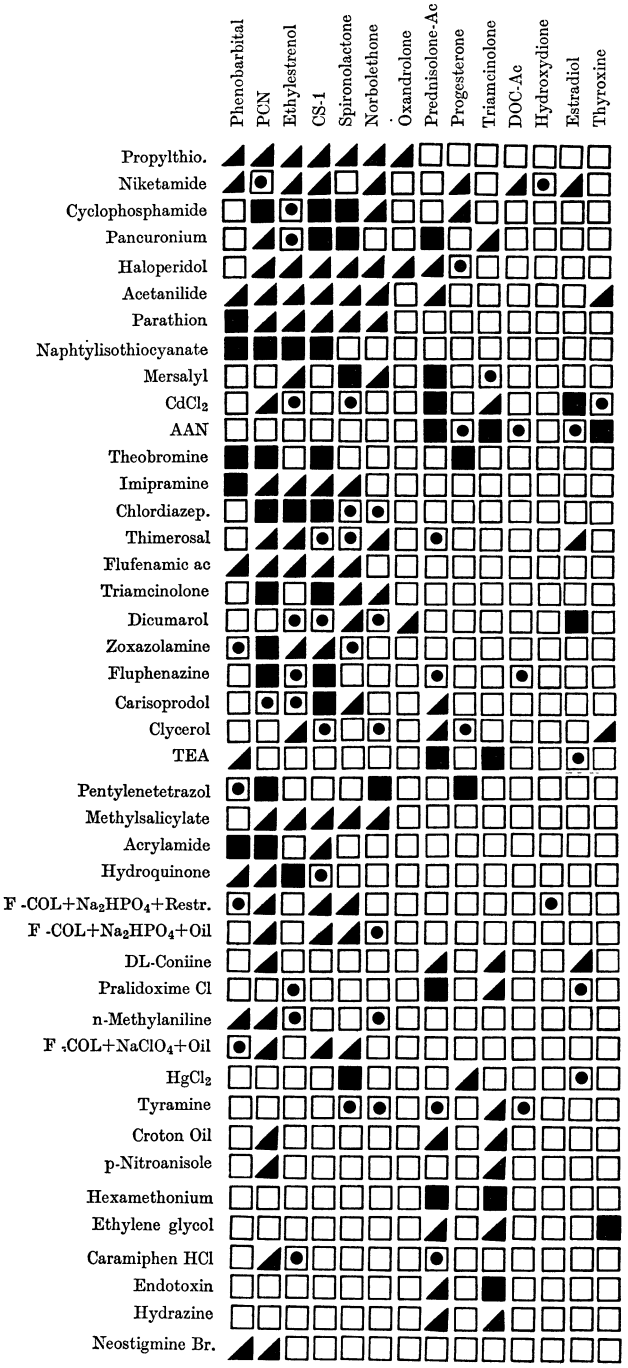
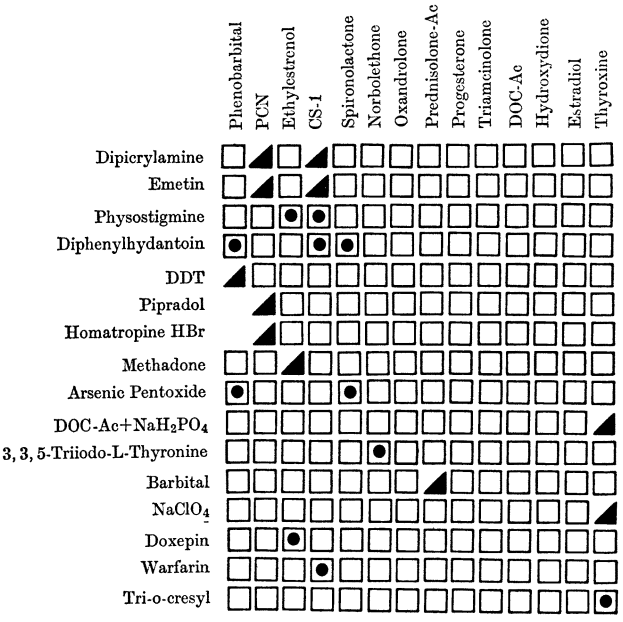


Table 139 (continued)



No significant protection was obtained by any of the conditioners against:

DFP	Bromobenzene
TlCl	Brompheniramine
Propionitrile	Dimercaprol
Griseofulvin	Dinitrophenol
DL-Amphetamine	Endrophonium
Tremorine	Yellow phosphorus
Estradiol + NaH_2PO_4	Ephedrine
Heptachlor	Ethyl alcohol
OMPA	Ethylene chlorohydrin
Epinephrine	Nephrectomy
Mechlorethamine	Methylphenidate
o-Aminophenol	$InCl_3$
Fasting	Morphine
Bile duct ligature	Pyrilamine

Varia

Conney F88,649/67: A review of the literature on more than 200 drugs leads to the conclusion that "there is no apparent relationship between either their actions or structure and their ability to induce enzymes. It is of interest that most of the inducers are soluble in lipid at a physiological pH."

Moscona & Piddington F90,487/67: In retinal explants of 12-day chick embryos, glutamine synthetase activity can be induced by the addition of various corticoids to the culture medium. Cortisol, corticosterone and aldosterone are particularly active in this respect, whereas pregnenolone, progesterone, DOC, 11-desoxycortisol, 17α-hydroxyprogesterone, and 11α-hydroxyprogesterone had little activity. 11β-Hydroxyprogesterone and 11β,17α-dihydroxyprogesterone exhibited intermediate degrees of activity. Apparently, "the 11β-position is of primary significance in the activity of these molecules in inducing retinal glutamine synthetase in this system.

Table 140. *Effect of the standard conditioners upon body and organ weights*

Treatment[a]	Final body weight (g)	Liver weight (g)	Kidney weight (mg)	Preputial glands weight (mg)
None	131 ± 2	5.0 ± 0.1	533.1 ± 8.9	48.8 ± 1.9
PCN	130 ± 2 NS	7.4 ± 0.2 ***	518.4 ± 8.0 NS	51.4 ± 2.3 NS
CS-1	140 ± 3 *	6.4 ± 0.2 ***	528.7 ± 10.6 NS	68.9 ± 3.3 ***
Ethylestrenol	142 ± 3 **	5.8 ± 0.1 ***	512.8 ± 6.9 NS	106.5 ± 5.4 ***
Spironolactone	138 ± 2 **	5.9 ± 0.1 ***	484.9 ± 6.4 ***	47.1 ± 2.9 NS
Norbolethone	144 ± 3 ***	5.6 ± 0.2 **	529.0 ± 10.7 NS	99.7 ± 4.8 ***
Oxandrolone	135 ± 2 NS	5.1 ± 0.1 NS	520.1 ± 8.4 NS	88.0 ± 3.1 ***
Prednisolone-Ac	82 ± 1 ***	7.1 ± 0.2 ***	726.6 ± 12.2 ***	43.3 ± 2.9 NS
Triamcinolone (2 mg)	90 ± 2 ***	5.9 ± 0.2 ***	609.6 ± 10.4 ***	42.1 ± 2.8 NS
Progesterone	130 ± 3 NS	5.3 ± 0.1 NS	531.1 ± 6.3 NS	66.3 ± 2.6 ***
Estradiol	90 ± 3 ***	6.1 ± 0.3 ***	666.3 ± 17.0 ***	41.9 ± 4.6 NS
DOC-Ac	125 ± 2 NS	4.9 ± 0.1 NS	546.7 ± 8.3 NS	54.1 ± 2.5 NS
Hydroxydione	124 ± 2 *	4.9 ± 0.1 NS	546.5 ± 13.7 NS	56.9 ± 2.6 *
Thyroxine	127 ± 2 NS	5.7 ± 0.1 ***	618.7 ± 8.3 ***	54.8 ± 4.1 NS
Phenobarbital	129 ± 2 NS	6.8 ± 0.2 ***	554.7 ± 11.8 NS	62.0 ± 3.2 ***

[a] 25 ♀ 100 g rats per group. All treatments were given at the standard dose levels (*cf.* p. VIII) from the 1st to the 8th day. Final body weights were registered on the 9th day at which time the organs were fixed in formalin saturated with picric acid. All organ weights are listed as a percentage of the final body weight. (Student's t-test comparing all weights with the corresponding values of the untreated controls.)

For further details on technique of tabulation, *cf.* p. VIII.

This conclusion is further supported by the fact that cortisone, which has a ketone group in the 11-position, had no effect under these conditions."

Wattenberg et al. G71,805/68: In rats, the relationship between chemical structure and benzpyrene hydroxylase activity was studied on a large series of 2-phenylbenzothiazoles. "Introduction of an appropriate halogen into the 4′-phenyl position approximately doubles inducing activity compared to unsubstituted 2-phenylbenzothiazole. Other substitutions and modifications of the molecule result in either lesser increases in inducing activity or, in some instances, reduction or total loss of inducing activity. Most compounds show similar inducing effects on both lung and liver."

Selye G70,421/70: 304 Steroid were tested for their ability to protect rats against usually fatal intoxication with indomethacin or digitoxin. Using a "Simplified Activity Grading" system, 65 among these compounds were found to be active against indomethacin, 54 against digitoxin, and 23 against both substrates at a 10 mg dose level. But only 4 of these steroids were still capable of inhibiting one or both of these substrates at the 0.5 mg dose level. The only steroid still active against both substrates at the dose of 0.2 mg was pregnenolone carbonitrile "PCN" (3β-hydroxy-20-oxo-5-pregnene-16α-carbonitrile).

Selye G70,480/71: A review (28 pp., 111 refs.) on "Hormones and Resistance." The historic developments and present status of our concept of syntoxic and catatoxic steroids is summarized and the standard techniques for the in vivo bioassay of protective hormones are described at length.

Solymoss et al. G60,084/71: In rats, ethylestrenol, norbolethone, progesterone, triamcinolone, prednisolone and hydroxydione exhibited no antimineralocorticoid properties (Kagawa's test). Thus, the catatoxic effect of steroids appears to be unrelated to antimineralocorticoid potency.

Selye PROT. 43477: In otherwise untreated 100 g ♀ rats, the influence of the most commonly used conditioners upon the final body and organ weights (the latter expressed as a percentage of the body weight) has been determined for comparison with the experiments in which such conditioning agents were given conjointly with toxicants. After eight days of treatment, the final *body weight* was diminished

Table 140 (continued)

Uterus weight (mg)	Ovary weight (mg)	Thymus weight (mg)	Thyroid weight (mg)	Adrenal weight (mg)
149.6 ± 31.0	11.3 ± 0.8	244.5 ± 14.3	6.8 ± 0.2	14.2 ± 0.3
164.0 ± 27.1 NS	7.9 ± 0.8 **	267.2 ± 8.7 NS	9.0 ± 0.4 ***	15.9 ± 0.4 **
133.8 ± 13.0 NS	10.3 ± 0.7 NS	234.7 ± 19.4 NS	8.5 ± 0.4 **	12.4 ± 0.3 ***
183.4 ± 12.5 NS	8.5 ± 0.6 **	137.6 ± 9.2 ***	7.4 ± 0.6 NS	10.4 ± 0.3 ***
147.0 ± 26.3 NS	9.0 ± 0.7 *	253.2 ± 12.1 NS	7.3 ± 0.6 NS	12.2 ± 0.3 ***
168.6 ± 13.2 NS	7.0 ± 0.7 ***	157.5 ± 11.6 ***	8.2 ± 0.4 **	10.8 ± 0.4 ***
331.1 ± 29.9 ***	9.5 ± 0.5 NS	174.2 ± 8.9 ***	7.4 ± 0.4 NS	12.5 ± 0.3 ***
144.3 ± 8.4 NS	10.6 ± 0.6 NS	19.3 ± 1.5 ***	10.0 ± 0.6 ***	10.1 ± 0.5 ***
105.8 ± 12.7 NS	13.1 ± 0.9 NS	25.0 ± 2.0 ***	8.2 ± 0.4 *	10.6 ± 0.8 ***
144.5 ± 37.1 NS	7.8 ± 0.7 **	249.4 ± 16.8 NS	7.2 ± 0.5 NS	12.4 ± 0.3 ***
535.2 ± 98.4 **	14.7 ± 1.6 NS	63.7 ± 8.4 ***	8.3 ± 0.3 **	19.6 ± 0.8 ***
144.9 ± 37.1 NS	8.7 ± 0.7 *	217.9 ± 16.0 NS	8.0 ± 0.4 *	12.8 ± 0.4 *
155.3 ± 46.3 NS	8.4 ± 0.8 *	279.3 ± 23.3 NS	8.2 ± 0.5 *	15.0 ± 0.7 NS
74.5 ± 9.8 *	6.7 ± 0.7 ***	243.5 ± 17.5 NS	6.2 ± 0.4 NS	15.0 ± 0.6 NS
137.7 ± 31.3 NS	10.4 ± 0.8 NS	290.9 ± 14.9 *	10.0 ± 0.4 ***	15.5 ± 0.5 *

by prednisolone, triamcinolone and estradiol. To a lesser extent, hydroxydione also caused a decrease in body weight gain, but this may have been due to the prolonged daily anesthetic effect of this steroid. The rats treated with anabolics, particularly ethylestrenol and norbolethone, showed a body weight increase but this was not pronounced during this short time experiment. Although oxandrolone likewise proved to be anabolic in earlier experiments (in that it combated the catabolism produced by DHT or glucocorticoids) it did not cause an absolute increase in body weight. When organ weights were expressed as percentages of the final body weight, significant *hepatic* enlargement was obtained by PCN, CS-1, spironolactone, norbolethone, prednisolone, triamcinolone, estradiol and phenobarbital. This is of interest since triamcinolone and estradiol are virtually devoid of catatoxic potency. Among all steroids, PCN caused the greatest hepatic enlargement, exceeding even that elicited by phenobarbital. The percental increase in *renal* weight was especially conspicuous following treatment with prednisolone, triamcinolone, estradiol and thyroxine, although the glucocorticoids and estradiol markedly diminished the absolute final body weight of these same animals. Phenobarbital and PCN caused no renal enlargement. The *preputial glands* are only imperfect indicators of testoid activity. Our earlier experiments showed that ordinary pregnenolone (3β-hydroxy-5-pregnen-20-one) possesses a pronounced preputial gland stimulating effect, although it is virtually devoid of typical testoid actions upon the capon's comb or the castrate rat's seminal vesicles. The insignificant increase in preputial gland weight, observed under our experimental conditions following treatment with PCN, suggests that the attachment of the carbonitrile group to this molecule decreases rather than increases its preputial gland stimulating action. In any event, the compound is much less "virilizing" under these conditions than is progesterone. The preputial glands were most markedly enlarged following treatment with ethylestrenol, norbolethone and oxandrolone, but to a lesser extent also by CS-1, progesterone and phenobarbital, perhaps owing to the endogenous production of testoid metabolites. As expected, *uterine* enlargement was greatest after treatment with estradiol, but some increase in uterine weight was noted also under the influence of oxandrolone, whereas thyroxine had an inverse effect. None of the conditioners tested caused *ovarian* enlargement, but PCN, ethylestrenol, norbolethone, progesterone and thyroxine induced significant ovarian atrophy. The *thymus* weight was most markedly diminished by prednisolone, triamcinolone and estradiol, but to a lesser extent also by ethylestrenol, norbolethone and oxandrolone. Curiously, phenobarbital appears to have caused a barely significant and PCN a nonsignificant increase in thymus weight. Phenobarbital, prednisolone,

PCN, CS-1, norbolethone and estradiol also increased the weight of the *thyroid* approximately in decreasing order as enumerated. The most pronounced *adrenal* hypertrophy was elicited by estradiol, but minor degrees were also obtained by PCN and phenobarbital. Possibly, detoxication of endogenous corticoids might have led to a compensatory hypertrophy of this magnitude. On the other hand, CS-1, ethylestrenol, spironolactone, norbolethone, oxandrolone, prednisolone, triamcinolone, progesterone and DOC caused a diminution of the percental adrenal weight, *cf.* Table 140, p. 860.

Selye PROT. 22393: There appears to be no obligatory relationship between glucocorticoid and catatoxic activity. Thus, large doses of prednisolone offer excellent protection against the ulcerogenic effect of indomethacin, whereas triamcinolone, a much more potent glucocorticoid, is devoid of this protective effect over a broad dose range *cf.* Table 141.

Solymoss PROT. 42234: As judged by the Kagawa test, PCN is practically devoid of antimineralocorticoid activity. In male adrenalectomized adult rats, 6 μg of DOC s.c. produced a pronounced decrease in the urinary Na/K excretion, which could not be counteracted by 1 mg of PCN.

Table 141. *Glucocorticoid potency not responsible for inactivation of indomethacin by prednisolone*

Treatment (mg)[a]	Intestinal ulcers (%)[b]	Mortality (Dead/Total)[b]
None	100	5/5
Triamcinolone 0.1	100	5/5
Triamcinolone 0.5	100	5/5
Triamcinolone 10	100	5/5
Prednisolone 0.1	100	5/5
Prednisolone 0.5	100	4/5
Prednisolone 10	0	0/5

[a] In addition to the treatments listed in this column, the rats (100 g ♀) of all groups received indomethacin 1 mg in 0.2 ml water/day, s.c., on 4th day ff. Triamcinolone and prednisolone were given at the doses indicated in 1 ml water × 2/day, p.o., 1st day ff.

[b] Intestinal ulcers (% positive) and mortality on 10th day.

SUMMARY AND OUTLOOK

This treatise attempts to outline the history and present status of research on the regulation of resistance by hormones. It would obviously be impossible to give a meaningful résumé of this vast field here, but it may be helpful to summarize the highlights of the research with which our group has had personal experience, namely: the effect of natural and artificial steroids upon comparatively nonspecific resistance phenomena.

The protective agents are classified, according to their mechanism of action, into two main groups: 1. "syntoxic" compounds which improve tissue tolerance by permitting a symbiotic type of coexistence with the pathogen (e.g., by suppressing inflammatory reactions); 2. "catatoxic" substances which actually destroy the aggressor (e.g., through the induction of hepatic microsomal enzymes).

The syntoxic effects are virtually limited to glucocorticoids, and since these have received sufficient attention in the past, this monograph deals primarily with recent studies on catatoxic steroids.

We have tested more than 500 steroids, under comparable conditions, for their possible protective effect against numerous toxicants. The results of these studies are tabulated and their evaluation revealed the following principal facts:

1. **The catatoxic effect can manifest itself independently of all classic hormonal actions,** although it is frequently associated with anabolic, antimineralocorticoid or glucocorticoid properties.

2. Some of the most potent catatoxic steroids are **carbonitriles**; these also have an unusually broad "spectrum of activity", in that they protect against many toxicants.

3. The **16α-position of the —CN group** appears to be particularly advantageous for catatoxic activity. Its introduction into a virtually ineffective steroid, e.g., 5-pregnenolone, endows the latter with sufficient catatoxic potency to protect a rat, at dose levels as low as 300 μg/kg, against fatal digitoxin or indomethacin intoxication.

4. Steroids may serve as especially favorable **carriers** of pharmacologically active groups, for example of thioacetyl (for the detoxication of mercury), quaternary ammonium bases (for the induction of a neuromuscular block), oncolytic agents, etc.

5. Certain catatoxic steroids possess **abortifacient** properties and interfere with **lactation.**

6. It is not yet proven that effective amounts of catatoxic steroids are normally **secreted in response to a need** (as glucocorticoids are during stress). However, they certainly represent basic **"soil-factors"** determining normal resistance; for example testosterone in amounts secreted by the testis raises the resistance of male rats far above that of females or gonadectomized animals of either sex. Furthermore, corticosterone — the natural life-maintaining steroid of the rat — possesses catatoxic activity against several substrates and is undoubtedly secreted in response to a need during stress.

7. Certain substrates, which are not subject to inactivation by steroidal or non-steroidal catatoxic compounds, can be **"opsonized"** (that is made amenable to this type of detoxication) by the addition of a radical. Thus, morphine is resistant, whereas ethylmorphine is highly sensitive, to inactivation by various compounds (catatoxic steroids, phenobarbital). This may well be due to a decrease in the polarity of the compound which may facilitate its penetration through membranes to the sites where enzymic detoxication can occur. It is interesting to speculate about the multitude of toxicants that might be made amenable to steroid-induced degradation if we learned how to stimulate their fat solubilization in vivo.

8. Many observations suggest that both syntoxic and catatoxic steroids may have important **clinical applications** in a variety of diseases caused by exogenous or endogenous toxicants. This is particularly true of maladies due to pathogens amenable to biotransformation by hepatic microsomal enzymes. In principle, here, enzyme induction would have to precede contact with the pathogen, and, hence, catatoxic steroids would be expected to have only prophylactic potency. Yet, if the pathogens act over a prolonged period (e.g., chronic indomethacin or digitoxin intoxication), curative effects have been obtained even when treatment was begun only after clinical manifestations of intoxication had become evident. Finally, if catatoxic steroids are important, "soil-factors" determining normal resistance, we should search for the possible existence of diseases caused by inadequate production of these compounds, or of disturbances in the responsiveness of the enzyme-inducing mechanisms which they regulate. Such maladies might be expected to result not only in deficient detoxication of exogenous pathogens but also in deficient or excessive degradation of endogenous chemical constituents of the body, such as hormones and metabolites.

As we have said in the Introduction to this monograph, animals are endowed with a complex hormonal defense system comparable in its scope to those based upon nervous, or immunologic reactions. Through conscious planning of defense, conditioned reflexes, or autonomic "emergency reactions," the body can adapt standard responses of its nervous system to defense against a multitude of specific injuries. Appropriate

immunologic reactions can adjust the basic phenomenon of antibody formation to cope with a great variety of potential pathogens which possess antigenic properties.

The main purpose of this monograph was to show that there exists a third general adaptive system in which a group of hormones and hormone derivatives (particularly steroids) offer resistance to agents not easily combated through the first-mentioned two defensive mechanisms; here, syntoxic hormones help to tolerate a pathogen, whereas catatoxic substances destroy it.

The 35 years of research — from the first description of the defensive role of corticoids in combating stress, to the latest observations on the extraordinary catatoxic potency of steroid carbonitriles — represent only a rough outline of the introductory phase in the elucidation of the hormonal defense system. The many references cited in the preceding pages clearly show that even in this first sketch, our own observations provided only a small percentage of the established facts. We have raised more questions than we have answered; but perhaps, by providing an inventory of pertinent facts, this monograph will help others to clarify this new field, which appears to have implications in virtually all phases of homeostasis under physiologic and pathologic conditions.

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