*Original Research Article*

Chemical Evaluation of the Effects of Analgesics on the Body's Chemical Cells

Martin Alphin

Department of Research and Development, UOP, Vian, Austria

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ABSTRACT

Pain is a sensory state and indicates a disorder in the body. It can be affected in two ways. First, the sensitivity of the relevant receptors can be reduced to such an extent that their excitability is reduced or eliminated by pain waves, which results in a reduction or disappearance of the sensation of pain. It is also possible to induce analgesia by attenuating or eliminating pain waves which enter the thalamus and affect sensitive cortices in the brain. With the synthesis and discovery of new analgesics, the classification of analgesics into non-narcotic compounds and other narcotic compounds is no longer common, and today they are divided into two main groups in terms of potency: weak analgesics and strong analgesics. The first group consists of compounds which are suitable for the treatment of simple and moderate pain and most of them have antipyretic, anti-inflammatory (anti-inflammatory), and anti-rheumatic effects. Very severe pain such as pain from surgery, cancer pain, renal colic, and biliary cannot be relieved with the first group of compounds, and for this, strong analgesics are used which are mostly drugs.

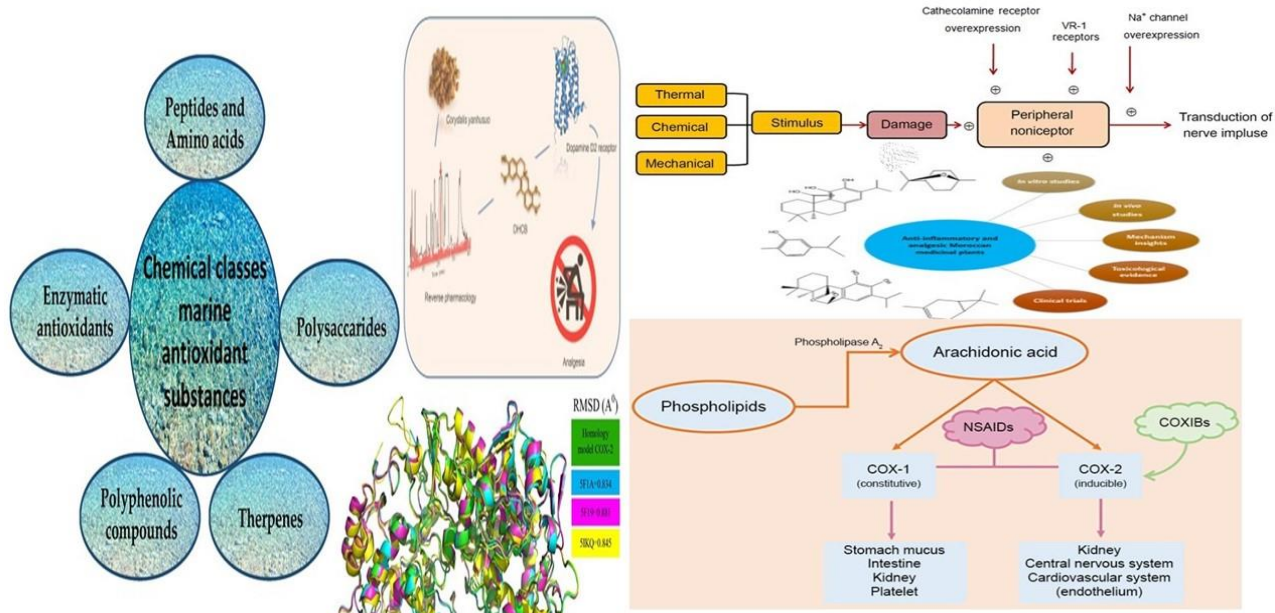
* Corresponding author: Martin Alphin

✉ E-mail: mrtnalphin@gmail.com

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GRAPHICAL ABSTRACT



INTRODUCTION

Salicylic acid derivatives, parahydroxy aniline derivatives (paramorphines), pyrazole derivatives, anthranilic acid derivatives, phenylalkanoic acid derivatives, and the related compounds form the main groups of mild to moderate analgesics [1]. The treatment spectrum of these drugs is wider than that of morphine derivatives, unlike narcotic analgesics; they do not cause addiction even in long-term use. Analgesics in this group, which are involved in the synthesis and release of prostaglandins and are able to enter the central nervous system, also have a hypothermic effect which is increased by the local release of prostaglandins by microorganisms' pyrogens [2-4]. These compounds, known as antipyretic drugs, have no effect on the body's normal heat odor [5].

1- Salicylic acid derivatives: Salicylic acid itself cannot be used as an analgesic due to its inflammatory effect on gastric mucosa, but its derivatives such as acetylsalicylic acid (aspirin), salicylamide, autoxy bisamidogenetics, good analgesic drugs are prominently utilized [6].

A) Acetylsalicylic acid: In 1898, a scientist named Hoffmann, who was trying to obtain a salicylate derivative with better tolerability and less toxicity than salicylic acid, succeeded in synthesizing acetylsalicylic acid at the Bayer plant in Germany [7-9].

This compound is one of the most important drugs today, which is less prepared and consumed in the world than other drugs. The antipyretic and analgesic effects of aspirin are similar to those of salicylic acid, but it is more tolerable and can be mixed with magnesium carbonate or aluminum hydroxide in order to prevent inflammation of the gastric mucosa caused by salicylic acid derivatives. To produce a therapeutic effect, salicylic acid, which is produced by the hydrolysis of aspirin, is not solely responsible and has a greater effect than all other aspirin molecules. Aspirin can cause allergic reactions in some people, such as asthma and urticaria [10-12]. Although the toxicity of this drug is lower than that of sodium salicylate and salicylic acid, and also aspirin

poisoning has been reported. Doses of 30-40 grams of aspirin are lethal [13-15].

A1) Specifications: Aspirin is composed of white crystalline powder or almost colorless crystals with almost no odor and sour taste, which are very good in ethanol, good in ether and chloroform, and difficult to dissolve in water, as well. Its melting point is 136-140 degrees. Aspirin is stable in dry air, but is slowly hydrolyzed by moisture and smells of acetic acid. It is also hydrolyzed by boiling water, hydroxides, and alkaline carbonates [16-18].

A2) Synthesis: It is obtained by acetylation of salicylic acid by heat with Stanidrid in the presence of a small amount of sulfuric acid as an aspirin catalyst. In addition to physical and physicochemical methods such as IR, GC, HPLC, and TLC, the following chemical methods can also be used [19-21].

- Aspirin is hydrolyzed by boiling water and salicylic acid formed with Fe^{3+} ions produce a purple color.
- Due to its heating with 6 N sodium and then, acidification of the solution cooled by 6 N sulfuric acid, a white crystalline precipitate of salicylic acid is formed, which after washing and drying, its melting point is measured.
- If a little ethanol and concentrated sulfuric acid are added to the smooth hydrolysis product, the smell of ethyl acetate is created.

A3) Determination of amount: Aspirin can be titrated with a one-tenth of a normal soda solution in the presence of phenolphthalein reagent. According to the American Pharmacopoeia, aspirin can be first heated with a certain amount of semi-normal caustic soda solution, and after an additional period of time, the caustic soda can be titrated with semi-normal sulfuric acid and the amount of caustic soda can be determined from the number of aspirin [22-25].

A4) Impurities: As mentioned, aspirin is easily hydrolyzed to form salicylic acid. Salicylic acid is an important impurity and can be determined by

the presence or absence of trivalent iron ions in aspirin. The maximum allowable amount of salicylic acid in aspirin is five per ten thousand. By comparing the color of the ethanolic solution, the concentration of salicylic acid to which ammonium ferrous sulfate solution was added with the color of the ethanolic solution of aspirin containing ammonium ferrous sulfate indicated a greater amount of this impurity. Acetylsalicylic acid is mostly used to relieve pain and fever, as well as in acute and chronic rheumatic complications. For successful utilization in the treatment of rheumatic complications, large initial doses should be taken which can increase the amount of blood aspirin to 30 mg per 100 ml of plasma. In this case, side effects such as dizziness, headache, and ringing in the ears are not uncommon. In long-term use of aspirin, there is a possibility of interfering with the synthesis of prothrombin in the liver and greater readiness for bleeding, and also due to the effect on the gastric mucosa, limited bleeding (at the micro level) occurs. In recent years, low concentrations of aspirin have been used to prevent and treat thrombosis. The effect of aspirin is probably due to its contrast with bradykinin or the interruption in the synthesis and release of prostaglandins. It is possible that its analgesic effect is related to the environmental effect on the origin and location of pain and vasodilation. Likewise, its antipyretic effect is most likely related to vasodilation and increased percutaneous heat loss. There is also empirical evidence that aspirin exerts its antipyretic effect by affecting the heat-regulating nerve center in the hypothalamus [26]. Depending on the particle size and formulation, aspirin is rapidly absorbed in the stomach or upper small intestine and also, peaks in the bloodstream two hours after ingestion. Initially, unchanged aspirin in the blood is superior to salicylic acid, but after a short time, most of it in the hydrolyzed blood loses the acetyl group. Aspirin is excreted mainly by the kidneys and

the rate of excretion depends on the pH of the urine, so that with alkaline urine about eighty-five percent of one unit is excreted as free salicylate and the rest as conjugated forms in a short time. In the case of acidic urine, due to the reabsorption of free salicylic acid, the action is longer and only about ten percent of a dose is excreted freely and most of it is excreted in conjugated forms (glucuronide, sulfate, etc.). A small amount of salicylate is oxidized to genetic acid in the liver and then, excreted in conjugate [27].

B) Salicylamide: Salicylamide was discovered in 1834 and in 1890, it was recommended to be prescribed as a well-tolerated salicylic acid derivative, however at first it could not find a place in medicine until 1946 in the United States. It was considered again and entered the pharmaceutical market. It has a good analgesic and antipyretic effect, but its anti-rheumatic effect is low and the cause can probably be traced to the salicylamide inability to form a chelate complex [28].

B1) Etz amide: The analgesic effect of this compound, which is ethyl ether of salicylamide, is twice that of salicylamide, and the fever effect on it is better than salicylamide. Excretion of this compound is slow and the consumed unit is completely eliminated from the body after 48 hours [29].

C) Genetic acid: This compound is a powerful antioxidant with many applications. Its sodium salt has toxicity as well as analgesic and fever effect on less than sodium salicylate and it is sometimes used in the treatment of rheumatic complications. Its effect is probably to inhibit the action of the enzyme hyaluronidase [30].

Para-hydroxy aniline derivatives (para-aminophenols): The analgesic effect of aniline derivatives was discovered in 1886 by Hep and Kahan. Knowing the toxic effect of aniline, which is the production of methemoglobin and inhibition of oxygen transport, these two

scientists, to eliminate its toxicity, conducted research on the other compounds of this group and in 1887 succeeded in discovering the antipyretic and anti-inflammatory effect. The pain was stanilized. Stanilide was initially widely used, however because it is also hydrolyzed in the body to form toxic derivatives such as aniline, it is rarely used anymore. In general, any substitution on the amine group which reduces the base power of this group will also lead to a decrease in the physiological activity of the compound [31].

A) Phenastine: This compound is composed of colorless glossy crystals or bitter white crystalline powder that is dissolved in ethanol and chloroform and is insoluble in water and ether. The melting point of Fenastin is 134-137 degrees. Fanastin is decomposed by dilution with dilute mineral acids of the amide group and by heat with concentrated mineral acids by the amide and autoxy groups [32].

A1) Synthesis: Para-nitrophenol is converted into para-nitrofenidine in an acidic medium with ethanol or alkali-ethanol sulfate, and then the nitro group is reduced by iron filings in an acidic medium. Fanastin is obtained by heating 4 ethoxy anilines obtained with stannidride or glacial acetic acid [33].

Pyrazole derivatives (5-pyrazolones and 3 and 5-pyrazolidine d)

Although it has been a long time since pyrazolone derivatives entered the pharmaceutical market, some of these compounds are still among the most important analgesic, antipyretic, and anti-inflammatory drugs. Pyrazolones do not exist in nature and are all synthesized. The first compound of this group is a drug called phenazone, which was synthesized in 1883 by a scientist named Connor (Figures 1 and 2).

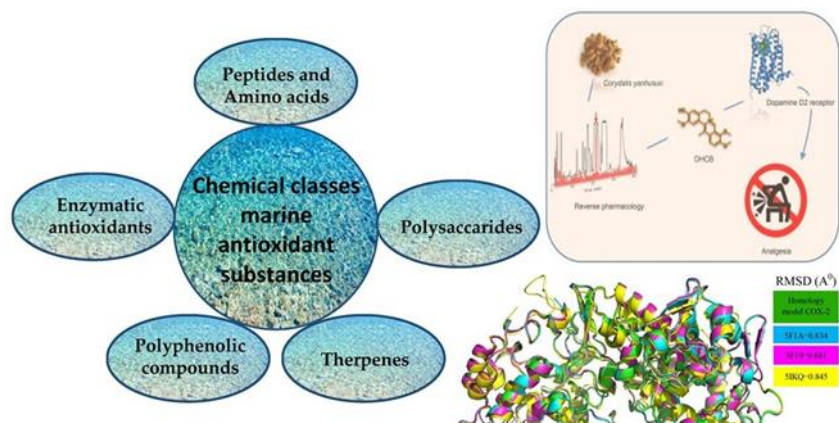


Figure 1. Drug with new mechanisms of action

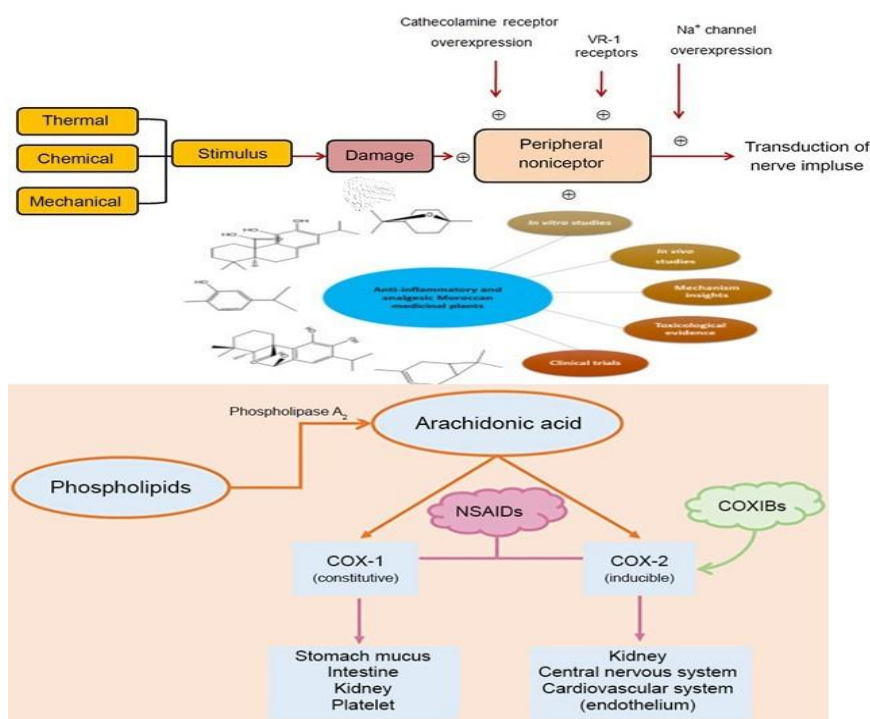


Figure 2. Antinociceptive effect of essential oils

The scientist wanted to synthesize compounds of quinoline that were similar to quinine, but the substance he obtained from the acetyl acetate of streptophenyl hydrazine differed from the imagined formula, but this substance (phenazone) and similar compounds in Pharmacological tests indicated good analgesic, antipyretic, and anti-inflammatory effects. The most important analgesics and antifungals in

this group, which are mostly among the official drugs, are 5 pyrazolones such as phenazone, amino phenazone dipyrone, propyphenazone, isopropyl amino phenazone, isopropyl aminase phenazone, and isopropyl aminase and 3 ones such as phenylbutazone, oxyphenobutazone, pheniprazine, mofobazone, and sulfine pyrazone [34].

Sedatives, hypnotics

The excitability of the central nervous system can be changed to different amounts and intensities by drugs. While a slight decrease in irritability creates a state of relaxation, it can be induced by a stronger drug or by a larger amount of the same drug. Temporary elimination or strong reduction of nervous system irritability can also be caused by anesthetics. To cause these three conditions, drugs with different chemical structures can be used or sometimes a drug can be taken with different drugs. For instance, phenobarbital has a sedative effect in small amounts (15 mg), but in large quantities (200 mg) is a good hypnotic [35]. Oral administration of 250 mg of hexobarbital also induces sleep, while intravenous injection of 500 mg of sodium hexobarbital induces anesthesia. Thus, hypnotics can be described as drugs which, in appropriate doses, reduce the central nervous system excitability to a state that resembles physiological sleep with reduced body functions such as respiration, pulse rate, blood pressure, muscle tension, and reflexes. Contrary to popular belief, this sleep is not a state of the brain inactivation, however is characterized by constructive respiratory action in all organs. The difference between sleep and anesthesia is that in anesthesia, unlike sleep, the central nervous system is almost and temporarily paralyzed, with the exception of vital centers in the spinal cord [36]. Hypnotics and sedatives have very different chemical structures and their effect is probably due to their binding to specific receptors, but is more related to their physical and chemical properties. The good results that Hansch et al. obtained about the relationship between the effect and hydrophobicity of several drugs do not apply to most hypnotics. Likewise, the theories and results of specific experiments which perform actions such as inhibition of oxidative interactions (i.e., oxidative phosphorylation), enzymatic (specific inhibition of respiratory enzymes), and bioelectrical

(inhibition of cytogenesis) are responsible for cell transmission. They knew, they generally do not seem to be enough. Kyogoko and Yu have illustrated that barbitol binds specifically to the adenine moiety of molecules such as FAD and NAD. These results and increasing symptoms suggest that barbiturates, due to their structural similarity to thymine, form hydrogen bonds with adenine macromolecules such as FAD and NAD, thereby interfering with significant biochemical processes [37].

Sedative-sedative classification

Plant extracts such as valerian root extract were among the first valuable drugs in this group. The active ingredients of this plant, after conducting numerous scientific studies, were finally extracted and identified by Tis et al. These materials are extracted in weakly acidic medium or lipophilic solvents and also, purified on aluminum oxide, including Valtratum, Didrovaltratum, and Acevaltratum. These substances, as well as the homolones and lupulins found in barley extract, are still used as sedatives. The use of mineral bromides as sedative compounds in 1850 led to the discovery in a short time of other effective compounds such as chloral, paraldehyde, sulfonal, and urethane. The sedative-hypnotic compounds used today are divided into the following seven groups [38].

A) Alcohols: The sedative-healing effect of alcohols has been known for centuries. However, the first member of this group, methanol, did not use drugs due to blindness, and the therapeutic use of ethanol, which is an instance of an alcohol with a sedative-hypnotic effect, has become obsolete due to alcoholism and is often stimulant for It is used to create vitality. The power of the hypnotic effect as well as the dissolution of normal alcohols in fat increase by maximizing the number of carbon atoms to octanol. Substitution of hydrogen atoms by halogens creates a similar situation, especially for low molecular weight alcohols. The potency of type 3

alcohols is probably higher than that of primary alcohols due to their greater resistance to metabolism to inactive compounds than secondary alcohols and strong secondary alcohols. Bifurcation in alkyl chains also increases the effect strength. In unsaturated alcohols, compared to similar saturated alcohols, in addition to increasing potency, the toxicity effect also increases. Alcohols such as ethchlorvynol and methyl pentinol, which are still used, are consistent with these results [39].

B) Aldehydes and their derivatives: Compounds of this group are among the first organic hypnotics. In 1869, chloroform was introduced as an intelligence drug, assuming that it was converted into chloroform in the body by haloform reaction, however in contrast to the reaction of haloform, which can be performed in vitro. Chloral is regenerated in the body and converted into trichloroethanol, which is an active metabolite (halogenated alcohol) and is probably responsible for the chloral effect. Chlorinated latent forms like petrochloride as petrochloride and chloral betaine, etc. are used to reduce or eliminate chloral damage [40].

C) Chloral Hydrate: Unlike liquid and oily chloral, this compound is composed of colorless or white crystals which have a pungent odor, a bitter burning taste and a zero-degree melting point. Chloral hydrate, like chloral, is broken down by alkalis to form chloroform and formate. Chlorine is prepared by chlorination of ethanol or acetaldehyde. Chlorination of ethanol produces a semi-acetal which is decomposed into chloral and ethanol by concentrated sulfuric acid.

D) Carbamates: With the exception of meprobamate, t-bamate and chlorazepate, which are esters of carbamic acid esters and are considered as central nervous system depressants with muscle relaxant properties. Etinamate is the most prominent carbamate with a sedative-like effect similar to a weak barbiturate. This combination is used to induce a

pleasant sleep quickly and without complications after sleep. The effect of etinamate begins after twenty minutes and lasts about three to five hours. It also has a weak anticonvulsant effect and local anesthesia.

E) Non-cyclic ureas: Unlike cyclic ureas (such as barbiturates), which are derivatives of carboxylic acid urea, these substances are composed of urea derivatives to monocarboxylic acids. Scarbermal, carbural, and bromine isovaleramide are mild hypnotics which are rapidly absorbed in the gastrointestinal tract and last for about three to four hours. These compounds were used as a daily sedative to relieve simple states of anxiety and nervousness and also to relieve sleep disorders. To prepare non-cyclic ureides, first the corresponding alkyl or dialkyl malonic acid is decarboxylated and after halogenation with red phosphorus and bromine, the obtained halide acid is condensed with urea [41].

F) Cyclic amides and imides: With the exception of methaqualone, the other compounds in this group can be divided into two groups: piperidine-2,4-d, such as pyridyls d-on, and meth-prilone, and piperidine-2, -6 d, such as pyridyls d-on, and meth-prilon, and piperidine-2,6. They were divided like glutathione.

G) Benzodiazepines: Among the group of benzodiazepines, several drugs such as chlorazepate, diazepam, flurazepam, lorazepam, nitrazepam, nimetazepam, oxazepam, and temazepam have been synthesized and entered the market. These drugs have generally a sedative-hypnotic effect, however they are often considered anxiolytic drugs and are prescribed to relieve excitement and anxiety. Benzodiazepines or anxiolytics have a long onset time and a long inactivation time. This means that most of them in the body are first converted into another active metabolite and then, inactivated and excreted by conjugation. Examples of these drugs are diazepam, chlorazepate, or oxazepam, which is itself the active

metabolite of the previous two compounds. This type of benzodiazepines is also utilized to reduce the acute side effects of quitting alcoholics and drug addicts. Some benzodiazepines are also used to reduce the acute side effects of quitting alcoholism in drug addicts. Some benzodiazepines, such as clonase pam, which are rapidly excreted from the body, are prescribed to treat epileptic seizures. Derivatives of benzodiazepines, which have a short onset of action and rapid excretion, are also utilized to relieve sleep disturbances caused by excitement, fear, and arousal. The most important of these drugs are nitrazepam and flurazepam, which are widely used as hypnotics [42].

G1) Nitrazepam: The effect of this drug appears after taking a unit of 2.5-10 mg after about 30-60 minutes and is followed by a quiet sleep of 6-8 hours. Nitrazepam is absorbed through the gastrointestinal tract and 5% of it is excreted unchanged in the urine in the first 48 hours along with about 20% of amino and acetyl amino metabolites. This drug is preferred over barbiturate and non-barbiturated hypnotics due to its low toxicity (even in high doses). Long-term use of nitrazepam can be addictive.

G2) Flurazepam: Menu and hydrochloride reminders of this drug like nitrazamide are prescribed as a hypnotic. The hypnotic effect appears about 20-45 minutes after taking a dose of 30 mg and guarantees a 7-8-hour sleep. Flurazepam is easily absorbed by the gastrointestinal tract and is rapidly metabolized, and also its metabolites are excreted in the urine with very little unchanged form.

H) Barbiturates: Barbiturates are various derivatives of barbituric acid. This acid was developed in 1836 by a scientist named Bayer from condensing manolic acid with urea [43].

Topical disinfectants

These compounds are divided into antiseptics and disinfectants according to their uses for humans or contaminated materials and objects such as drinking water, clothing, surgical

supplies, etc. The term antiseptic includes bacteriostatic compounds used to disinfect living tissue. Antiseptics kill most of the germs on the treated area but do not necessarily sterilize the area. It is not common to take these compounds orally or by injection to kill internal infections, and they are only in direct contact with germs outside the body. Iodine disinfectants have a bactericidal effect and are used to disinfect and kill germs from non-living objects and contaminants such as surgical instruments, drinking water, toilets, contaminated feces, as well as vaccines and blood products. Although topical disinfectants have lost their former importance with the discovery of sulfonamides and antibiotics, they are still in many cases used to kill pathogens on the external surfaces of the human and animal bodies, especially objects and materials contaminated with these compounds are used.

Basically, a good disinfectant compound should have the following properties:

- Strong effect (bactericidal or bacteriostatic)
- Suitable physical and chemical properties (durability, solubility, rate of effect, etc.)
- These compounds should be effective against a variety of bacteria and their spores, as well as fungi, viruses, and protozoa, and are not specific to chemotropic and antibiotics.
- The potency of these compounds should not be diminished as much as possible by inactive substances.
- The toxicity of these compounds should be negligible so that they do not cause poisoning if absorbed.
- Antiseptic and disinfectant compounds have different chemical structures and are divided into the following groups:
 - Alcohols such as ethanol, propanol, isopropanol, propylene glycol, and triethylene glycol.
 - Aldehydes such as formaldehyde, paraformed aldehyde, and glutaraldehyde.

- Organic acids such as caprylic acid, eunsilic acid, mandelic acid, salicylic acid, and benzoic acid.
- Phenols and their halogenated derivatives such as phenol, cresol, cresol chlorine, thymol, parachlorophenol, chlorhexidine, hexachlorophene, and resorcinol.
- Halogens and halogenophores such as chloroamine, halazone, sodium hypochlorite, and povidone iodide [44].
- Oxidizing compounds such as permanganate, oxygenated water, carbamide peroxide, and benzoyl peroxide.
- Nitrofurantoin derivatives such as nitrofurantoin and nitrofurazone).
- Mercury derivatives such as merbromine, thiomersal, phenylmercury nitrate, nitromerzol, etc.
- Dyes such as Janesian Violet, Amaranth Methylene Blue, Foxin Acroflavin, and Ethacridine are the last two compounds of acridine derivatives.
- Active disinfectants on the surface of the anionic group include soaps and detergents containing sodium lauryl sulfate, sodium atasulfate, and cationic groups such as benzalkonium chloride, acetyl trimethylpyridinem bromide, acetyl trimethylpyrimidyl benzyl amide have become active [45].

Conclusion

Stanilide, as the best instance of reducing the amino group base strength by acetylation, is toxic in high and low doses without significant analgesic effect. Another example is fermentilide, which is easily hydrolyzed in addition to strong side effects. Larger homologs than stabilization also have a weak effect due to poor solubility. If this substitution is done by aromatic acids, the obtained compounds cannot be used as analgesics for various reasons. For instance, benz anilide has no analgesic effect, salicylic anilide has the antifungal effect of

vaxalgin and is highly toxic. Hydroxylated anilines at the ortho-metaphoric sites, known as aminophenols, are receiving more attention due to their lower toxicity. Para-aminophenol is a metabolite of aniline which is less toxic than other isomers. Although this compound has an analgesic and antipyretic effect, it is not allowed to be taken as a medicine due to its toxicity. By acetylating the amine para-aminophenol group, a body called anastyl-para-aminophenol (acetaminophen) is formed, which is a relatively good analgesic and antipyretic. Another way to reduce para-aminophenol toxicity is to ether the phenolic hydroxyl group. The most important compounds obtained by this method are anisidine and phenytoin, which are ethyl and methyl ether of para-aminophenol. By acetylation of the amine group and ethering of the hygosylphenyl group, one of the most significant para-aminophenol derivatives called phenestin, of which ethyl ether is para-aminophenol acetyl ether was synthesized and introduced. Methyl and propyl ether-acetyl-para-aminophenol homologues are not used due to the severe side effects such as nausea, diuresis, etc.

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