HUMAN PHARMACOLOGY pdf

1: Human pharmacology of MDMA: pharmacokinetics, metabolism, and disposition.

We are happy to announce that ACCP will launch a new journal, Journal of the American College of Clinical Pharmacy (JACCP). The journal will publish papers across the spectrum of clinical pharmacy practice.

Toxicology[edit] Toxicology is the study of the adverse effects , molecular targets, and characterization of drugs or any chemical substance in excess including those beneficial in lower doses. Theoretical pharmacology[edit] Theoretical pharmacology is a relatively new and rapidly expanding field of research activity in which many of the techniques of computational chemistry, in particular computational quantum chemistry and the method of molecular mechanics, are proving to be of great value. Theoretical pharmacologists aim at rationalizing the relation between the activity of a particular drug, as observed experimentally, and its structural features as derived from computer experiments. They aim to find structureâ€"activity relations. Furthermore, on the basis of the structure of a given organic molecule, the theoretical pharmacologist aims at predicting the biological activity of new drugs that are of the same general type as existing drugs. More ambitiously, it aims to predict entirely new classes of drugs, tailor-made for specific purposes. Posology[edit] Posology is the study of how medicines are dosed. This depends upon various factors including age, climate, weight, sex, elimination rate of drug, genetic polymorphism and time of administration. There is a close collaboration between environmental science and medicine in addressing these issues, as healthcare itself can be a cause of environmental damage or remediation. Human health and ecology are intimately related. Demand for more pharmaceutical products may place the public at risk through the destruction of species. The entry of chemicals and drugs into the aquatic ecosystem is a more serious concern today. In addition, the production of some illegal drugs pollutes drinking water supply by releasing carcinogens. Experimental pharmacology[edit] Experimental pharmacology involves the study of pharmacology through bioassay , to test the efficacy and potency of a drug. Dental pharmacology[edit] Dental pharmacology relates to the study of drugs commonly used in the treatment of dental disease. With the knowledge of cell biology and biochemistry increasing, the field of pharmacology has also changed substantially. It has become possible, through molecular analysis of receptors , to design chemicals that act on specific cellular signaling or metabolic pathways by affecting sites directly on cell-surface receptors which modulate and mediate cellular signaling pathways controlling cellular function. A chemical has, from the pharmacological point-of-view, various properties. Pharmacokinetics describes the effect of the body on the chemical e. When describing the pharmacokinetic properties of the chemical that is the active ingredient or active pharmaceutical ingredient API , pharmacologists are often interested in L-ADME: Liberation â€" How is the API disintegrated for solid oral forms breaking down into smaller particles , dispersed, or dissolved from the medication? Absorption â€" How is the API absorbed through the skin , the intestine , the oral mucosa? Distribution â€" How does the API spread through the organism? Metabolism â€" Is the API converted chemically inside the body, and into which substances. Are these active as well? Could they be toxic? Excretion â€" How is the API excreted through the bile, urine, breath, skin? Medication is said to have a narrow or wide therapeutic index or therapeutic window. This describes the ratio of desired effect to toxic effect. A compound with a narrow therapeutic index close to one exerts its desired effect at a dose close to its toxic dose. A compound with a wide therapeutic index greater than five exerts its desired effect at a dose substantially below its toxic dose. Those with a narrow margin are more difficult to dose and administer, and may require therapeutic drug monitoring examples are warfarin , some antiepileptics , aminoglycoside antibiotics. Most anti- cancer drugs have a narrow therapeutic margin: Medicine development and safety testing[edit] Development of medication is a vital concern to medicine , but also has strong economical and political implications. To protect the consumer and prevent abuse, many governments regulate the manufacture, sale, and administration of medication. In the United States , the main body that regulates pharmaceuticals is the Food and Drug Administration and they enforce standards set by the United States Pharmacopoeia. In the European Union , the main body that regulates pharmaceuticals is the EMA and they enforce standards set by the European Pharmacopoeia. The metabolic stability and the reactivity of a library of

candidate drug compounds have to be assessed for drug metabolism and toxicological studies. Many methods have been proposed for quantitative predictions in drug metabolism; one example of a recent computational method is SPORCalc. This means that when a useful activity has been identified, chemists will make many similar compounds called analogues, in an attempt to maximize the desired medicinal effect s of the compound. This development phase can take anywhere from a few years to a decade or more and is very expensive. It needs to be determined how safe the medicine is for human consumption, its stability in the human body and the best form for delivery to the desired organ system, like tablet or aerosol. After extensive testing, which can take up to 6 years, the new medicine is ready for marketing and selling. To recoup this outlay pharmaceutical companies may do a number of things: The FDA requires that all approved drugs fulfill two requirements: The drug must meet safety criteria by being subject to animal and controlled human testing. Gaining FDA approval usually takes several years. Testing done on animals must be extensive and must include several species to help in the evaluation of both the effectiveness and toxicity of the drug. The dosage of any drug approved for use is intended to fall within a range in which the drug produces a therapeutic effect or desired outcome.

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2: Human Pharmacology Research & Studies | Bioavailability & Bioequivalence

Unique among pharmacology textbooks, HUMAN PHARMACOLOGY: MOLECULAR TO CLINICAL organizes drugs of a class according to the characteristics of the class, with individual drugs discussed in relation to other drugs of the class.

The other common feature is their similar structure. The amino end of the protein remains extracellular whereas the carboxylic end, which is intracellular, interacts with the G-protein, which is bound to the intracellular surface of the cell membrane. When the appropriate drug interacts with the ligand-binding domain of the receptor protein, which is on the extracellular aspect of one of the transmembrane helices, a conformation change in the receptor protein takes place such that the intracellular end stimulates the G-protein. In this way the drug is able to induce a change inside the target cell without itself having to enter the cell. Examples of such proteins are the enzymes adenylyl cyclase and phospholipase C PLC and certain potassium and calcium ion channels. The released cAMP is then able to act within the cell in which it is produced, but because of its hydrophilic nature it is unable to cross the lipid cell membrane and leave that cell. Cyclic AMP is an example of a second messenger. These second enzymes alter the activity of key intracellular regulatory proteins, such as enzymes, by their phosphorylation of serineâ€”threonine residues; cAMP-dependent protein kinases may also phosphorylate serineâ€”threonine residues of certain ion channels. Depending upon the regulatory protein concerned, the phosphorylation may result in either activation or inhibition. In this way, the stimulation of a G-protein coupled receptor may result in either an increase or a decrease in the activity of the target cell or tissue. The action of cAMP is terminated by its metabolism by the intracellular enzyme phosphodiesterase, of which there are several subtypes. The consequence of the stimulation of PLC by the G-protein coupled receptor is the catalysis of the hydrolysis of phosphoinositides, which are phospholipids normally found within the cell membrane. This results in the production of inositol triphosphate IP3 and diacylglycerol DAG , both of which act as second messengers. This in turn activates calcium- and calmodulin-dependent protein kinases with results similar to those described for cAMP. Unlike cAMP and IP3, DAG is lipid soluble, thus it remains within the cell membrane where it stimulates a subtype of protein kinase, called protein kinase C, which, like the other protein kinases, catalyses the phosphorylation of regulatory proteins such as enzymes. It can therefore be seen how the interaction of a ligand with a G-protein coupled receptor results in a change in cellular activity. Several other features of G-protein coupled receptors remain to be described. First, it is not unusual for several different types of receptor, in a single cell, all to be associated with a single G-protein, association of two receptors of the same type with a single G-protein would explain the phenomenon of spare receptors see Section 1. Conversely, a single receptor may interact with more than one G-protein, and hence utilise two different second messengers. Because the G-protein may remain stimulated for several seconds, it is able to catalyse many reactions, thus a single ligand or drug â€”receptor interaction may result in the generation of over molecules of second messenger. Similarly, activation of a steroid receptor may either increase or, more commonly, decrease the expression of that or another receptor. In the case of the membrane-bound receptors, it has been shown that these receptors are able to migrate across the cell surface but that they are held within the plane of the cell membrane, and may congregate in clusters of receptors of a similar type. It has also been observed that sections of cell membrane containing drug receptors may invaginate to produce vesicles. This may occur following binding of the drug to the receptor, where it is a mechanism by which the drug is rapidly dissociated from the receptor. The drug may then be broken down by intracellular enzymes while the receptor is recirculated to the membrane, ready for restimulation. However, this may also occur to unbound receptors. Invagination may occur following repeated stimulation of that receptor by an agonist, or it may occur following stimulation of another receptor. While in the vesicles, the receptors may be susceptible to breakdown by enzymes, thus there may be a reduction in the total number of receptors available within that cell. This process is sometimes called receptor downregulation, and is a common response of a receptor population to continued, excessive stimulation, this is one basis of drug tolerance see later. Another form of drug tolerance involves a reduction in the number of receptors available following a reduction of receptor synthesis, this takes somewhat longer to occur than the

invagination desribed above. Again, this form of down-regulation may occur following repeated stimulation of the receptor by its agonist, or as a result of stimulation of another receptor, in which case it is called heterologous down-regulation. An increase in receptor synthesis may produce up-regulation. This is a rapid response and is called receptor desensitisation. In these cases the cause of the disorder lies at the receptor level rather than at the messenger level. The effect of any drug is dependent upon the dose used, thus an increase in dose induces an increase in response in the case of graded responses or, in the case of quantal responses, an increase in the proportion of the population exhibiting the response. The doseâ€" response relationship is not linear, the effect of the drug reaches a maximum at higher doses, after which an increase in dose does not cause an increase in response. It is common to portray the relationship between dose and response using a graph of the response versus the logarithm of the dose or drug concentration , this not only allows a wider range of doses to be plotted, but also gives a curve, the central portion of which is approximately linear. An increase in drug concentration results in a greater number of drugâ€"receptor interactions and thus a greater response; occupation of all available receptors results in the maximal response being achieved. These drugs, antagonists, interact with the receptors but do not produce an effect, other than to prevent access to the receptor by full agonists. Thus antagonists reduce the effects of agonists. Antagonists may be either competitive, in which case they bind to the same sites on the receptor as the agonists, or they may be non-competitive, in which case they bind to separate, but closely associated sites. The molecular nature of receptors is now well understood. Because of the mechanism of action, drugs acting on these receptors n.

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