



Factors Affecting Ratio of Poisons: Article

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Abstract

Pharmacology the best characteristics options of poison their modes of response their modes of response once to bear with the organism and the clinical and biochemical uses of drugs the pharmacology and toxicology intersect with each in their responses to produce adverse effect.

Keywords: Therapeutic index, toxicology, pharmacology, synergic effects, cumulative action.

INTRODUCTION

Toxicology is the branch of bio forensics that deals with the symptoms, adverse effect of poison, drugs to living organisms with the out viewing the postmortem response of the habitual poison or drug habitual body their treatment their exposure their detection whereas toxicants and toxins each have major distributive variations toxins is principally made by the living organism in response to substance whereas toxicants if the foreign material extract from the manmade or human semi artificial in nature that ends up in unwell organic process of the person and together this toxicants and toxin leads to become poison at a poison or poisoning substances at a point poisoning inherently lead to damage the psychological method of the human anatomy's the functioning of the correct bio concentration of the chemicals produces unwell effects briefly interval of time. Whereas the pharmacopeia is a patent poison or a term through which gene related factors through which is a factor which decides for any poison to its adverse potential for creating any substance a poison is set by the analysis of the dose response that is in graded dose response in a private or the measure dose response ranked response is given to a population which ends up in bigger magnitude of response as dose increased and in the quantal dose response relationship the percentage of the population affected uplift as the dose is raised this dose relationship offers method|how|some way|the way|the simplest way} to the broad way that is dose LD_{50} that is outlined because the live of the quantity of the substance needed to kill 50% of the tested population it live the acute toxicity of the substances soul chiefly perform experiments on rats to check the animals primarily the formula lies that that quantity of chemical administered/100gm for tiny animal and in kgs for big animals they are entered through several routes whereas dermal injections are most popular (LD_{50})lethal concentration stands for the concentration of chemical in air which might kill 50% of tested animals. These studies contribute to therapeutic dose that determines any substance to be a therapist or the hepatotoxic poison the therapeutic indefinite quantity is that the quantitative relation that compares because the blood concentration at which drug become toxic and the concentration at which it become effective the larger the therapeutic the safer is that the medication if the TI is smaller than that is the excellence between the two concentration is little the drug toxicity appears and produces sign and symptoms on the body. Therapeutic ratio is the amount of drug that causes the therapeutic effect to the amount that causes toxicity if TI is < 10 it is poison. The primary concern of rhetorical medical scientist isn't the legal outcome of the poison or hepatotoxic investigation or technology wont to encounter it but lies on the ways of obtainment and interpretation of results for example hexachlorohexahydro dimethanonaphthalene an insecticide which belongs to organochlorine pesticides metabolized into dieldrin which needs an in depth investigation of necessary factors sources analysis and chemical purity concentration to verify the identification of aldrin poison. In the cases of poison analysis, a blood sample of approximately 05 ml requires to screen and confirm toxic analytes that elaborate the profile of substances of the toxicologist poison.

ACTIONS OF POISON/ CAUSES MODIFYING THE ACTION OF POISON

Poisons mainly act into three ways local which is the chemical destruction by corrosives second is the remote which is NUX vomica producing action on spinal cord combined action where carbolic acid was thought to have action on both local and remote control actions.

- Quantity increases the toxicity increases example is copper sulphate fatal dose is 20g if ingested more than this than it cannot kill but emesis occur suddenly their mechanism of action suddenly vary because large dose of arsenic can kill person without producing irritant symptoms moderate dose produce shocking irritant symptoms and small dose produce therapeutic action.
- Forms of physical state poison found to be active in gaseous forms less in liquid form and least active in solid form.
- Solubility in the case of solid solubility increases toxicity also increases where insoluble salt may become soluble in the acid secretion of stomach and can become poisonous.
- Mechanical combination if combined automatically inert substances become harmless example alkaloid is poisons nous however once it's combined with charcoal it become harmless.
- Chemical combination two strong poison when taken together sulphuric acid and sodium hydroxide are both strong in their nature but when taken together it do not cause harm whereas phosphorous and CuSO_4 both form in combined effect cupric phosphide silver nitrate and hydrogen chloride when taken together form silver chloride. Dilution when strong poison diluted with water other substances become harmless .Mode of administration if we talk about fastest to slowest they lie in a line of intravascular-inhalation-sublingual-intranasal-intramuscular-rectal-oral-subcutaneous-topical- transdermal rate of absorption from the gastrointestinal is faster on empty stomach rate is faster if associated substances dissolve poison material gastrectomy intestinal absorption of poison are faster because it transfer from intestine faster basic drugs include morphine, quinine, they get ionized in stomach faster salicylates and barbiturates remain unionized in stomach Condition of the body children are more susceptible to drugs and poisons as they have low rate of metabolism. Gray baby syndrome develop to a child who are unable to glucuronidase chloramphenicol .Young ruke for child doses given by Thomas young because of increase susceptibility children need less dosage of drug the proportionate dose for (1-12y) Doses of child: $\text{age in y}/\text{age in y} + 12^*$ (adult dose) thus a child of 6y need 1/3 of ordinary dose of adult.
- IDOSYNCRASY adverse drug reaction inherent personal hypersensitivity to a specific drugs or food items it causes fever, vomit, rigors, git dysfunction. Drugs showing specialness area unit acetylsalicylic acid, belladonna, bromine, cocaine, iodine, mercury. Tolerance: habit is the ability of an organism to show num response to a fixed dose due to long repeating of doses that leads to habitual drugs because it decrease the reaction between toxin and reactors induction of enzymes increases metabolism example arsenic can produce tolerance among the person if continues uptake in small doses produces tolerance among patient, Cumulative action poison which have a slow rate of excretion from the body they get accumulate in the body if poison gets accumulated from a so long time example arsenic and lead. Synergism: when two poison taken together it produce synergic effect such as produce allergy or hypersensitivity alcohol and barbiturates

CONCLUSIONS

It is found from this article that quantity and dose is the key factor on which the modification or action of poison depend which hit the person at a small amount to large amount and the combined effect of poison which is responsible to produce synergic effects.

REFERENCES

1. Ahmad N, Harsas W, Marolt RS, Morton M, Pollack KJ (1988) Total DDT and dieldrin content of human adipose tissue. Bull Environ Contam Toxicol 41(6): 802-808.
2. Bandyopadhyay, S. K., Tiwari, R. K., Bhattacharyya, A., & Chatterjee, G. C. (1982). Effect of dieldrin on rat liver plasma membrane enzymes. Toxicology letters, 11(1-2), 131-134.
3. Bano, Y. (1982). Effects of aldrin on serum and liver constituents of freshwater catfish *Clarias batrachus* L. Proceedings: Animal Sciences, 91(1), 27-32.
4. Campagna, A. F., Eler, M. N., Fracácio, R., Rodrigues, B. K., & Verani, N. F. (2007). The toxic potential of aldrin and heptachlor on *Danio rerio* juveniles (Cypriniformes, Cyprinidae). Ecotoxicology, 16(3), 289-298.
5. Cicchetti, R., & Argentin, G. (2003). The role of oxidative stress in the in vitro induction of micronuclei by pesticides in mouse lung fibroblasts. Mutagenesis, 18(2), 127-132.
6. luKerr SR, Vass WP. (2013). Pesticides residue in aquatic invertebrates. Environmental Science Research 3: 134-180.

7. Treon, J. F., Cleveland, F. P., Stemmer, K. L., Cappel, J., Boller, R. A., Shaffer, F. E., ... & Coomer, J. (1955). The toxicity of aldrin when fed to suckling dogs, and the toxicity of aldrin, dieldrin, DDT and lindane when incorporated in the diets of older dogs over a period of more than fifteen months. Cincinnati, OH. The Kettering Laboratory in the Department of Preventive Medicine and Industrial Health, College of Medicine, University of Cincinnati.
8. Treon, J. F., Dutra, F. R., Shaffer, F. E., Cleveland, F. P., Wagner, W., & Gahegan, T. (1951). The toxicity of aldrin, dieldrin, and DDT when fed to rats over the period of six months. Cincinnati, OH. The Kettering Laboratory in the Department of Preventive Medicine and Industrial Health, College of Medicine, University of Cincinnati.
9. Thorpe, E., & Walker, A. I. T. (1973). The toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, β -BHC and γ -BHC. *Food and cosmetics toxicology*, 11(3), 433-442.
10. Lamai, S. L., Warner, G. F., & Walker, C. H. (1999). Effects of Dieldrin on Life Stages of the African Catfish, *Clarias gariepinus* (Burchell). *Ecotoxicology and environmental safety*, 42(1), 22-29.
11. Rocha, E. (1999). Histology and cytology of fish liver: a review. *Ichthyology: Recent research advances*, 321-344.
12. Spiotta, E.J. (1951). Aldrin poisoning in man; report of a case. *AMA Arch Ind Hyg Occup Med* 4(6): 560-566.