Handbook of teratology pdf

The six principles of James Graves Wilson's teratology, published in 1959, direct research on teratogenic agents and their effects on developing organisms. Wilson's six principles were inspired by the five principles of Gabriel Madeleine's experimental teratology, published in 1877. Teratology is the study of birth defects, and teratogen is something that either causes or enhances abnormal embryonic or fetal development and causes birth defects. Detailed in his 1973 monographEnvironment and Birth Defects, Wilson's principles helped scientists experimentally investigate teratogenes. Between the 1860s and 1900s, Gabriel Madeleine Camille Darreste, who studied embryology and zoology in Paris, France, relied on the early work of the father and son of the duo Etienne Jeffroy and Isidore Jeffroy Saint-Iler. In the early nineteenth century in Paris, France, Etienne experimented on chicken eggs, subjecting them to various physical manipulations such as tingling, inversion, abnormally high and low temperatures, etc., and he studied as a result of malformations. He argued that some abuses can lead to specific deformities. While the deformities did materialize, Saint-Iler did not identify their exact causes. His son Isidore then went on to report on Etienne's results between 1832 and 1837 in Histoire g'an'rale et particuli're des anomalies de l'organization chez l'homme et les animaux (Common History and Specific Anomalies of the Organization for Humans and Animals). Relying on the work of the Ilers, Dareste manipulated temperatures, shaking and chemically processing the embryos of chicks. He found that the most serious anomalies occurred when he manipulated embryos in the early stages of development. Dareste claimed that his experiments had been delayed or completely arrested. Dareste developed a set of five principles of teratology, which he described in detail in his 1891 Recherches sur la production artificielle des monstruosit's: ou, Essais de t'ratog'nie exp'rementale Research on the artificial production of monsters, or Experimental testing of teratogenicity. James G. Wilson, who studied embryology at the University of Rochester School of Medicine in Rochester, New York, expanded five of Dareste's principles in his 1959 article, Experimental Research on Congenital Malformations. Wilson further revised these five principles and added a sixth generalization about dosage effects in his 1973 monograph of the environment and birth defects. The first principle described in Wilson's 1959 work, labeled as susceptibility to teratogenesis, depends on the genotype of the Concept and the manner in which it interacts with adverse environmental factors, has four sections. In Section A, View Differences, Wilson says that some species to specific teratogenes where others don't, or at least don't Just the same degree. For example, humans and other primates are extremely vulnerable to thalidomide, a sedative used in the 1950s to treat morning sickness. When exposed to thalidomide during embryonic development, the fetus develops malformations of the limbs and face. Other mammals, including rats and mice, however, are resistant to thalidomide. In section B, Tension and Intralitter Differences, Wilson notes that animals of the same species with different genetic backgrounds may differ in the frequency and intensity of anomalies caused by teratogenes because some lines are more resistant to teratogenes than others. Section C, The interaction of the genome and the environment, emphasizes the interaction between the environment and genetics, leading to different anomalies between organisms with the same genome raised in different environments and between organisms with different genomes raised in the same environment. Maternal characteristics, such as the ability of a pregnant woman to assimilate teratogenes, partly determine whether the fetus will develop abnormalities. In section D, a multifactorial cause-and-effect relationship, Wilson argues that interactions between genes and the environment involving more than one gene and/or more than one environmental factor can affect the severity of birth defects caused by teratogen. Principle two, susceptibility to teratogenesis varies depending on the stage of development during exposure to adverse effects, has in six sections. Beginning with the introductory section of A., Span Development Division, principle two chronologically illustrates the stages of development from the early fireproof period to birth and after. Wilson goes on to describe the susceptibility of embryos to teratogenes at every stage of development. Titled Highly Receptive Period Organogenesis, Section C is the result of Wilson's studies of teratogenes, and it depicts the process of organogenesis, the development of organs in the body, along with an increase in the incidence of malformations caused by teratogenes due to the sharp differentiation of the tissue concept. Last section, F. What about germ cells? Wilson says factors such as the environment, drugs and dietary deficiency can damage the germ cells and germ layers from which they originate; however, when Wilson published his principles, the researchers did not determine whether teratogenes could affect certain stages of gametogenesis, or the formation of gametes. Principle Three, Teratogenic Agents Act in specific ways (mechanisms) to develop cells and tissues to initiate sequences of abnormal development events (pathogenesis), has two sections: A. Mechanisms of teratogenesis, and B. Pathogenesis defect. In these sections, Wilson argues that specific teratogenic agents produce distinctive patterns of malformations. Thanks to this third principle, Wilson that people can take supplements to protect against specific teratogenic agents. Principle 4, Access to adverse influences to developing tissues depends on the nature of influence (agent) divides teratogenic agents into A. Physical agents and B. Chemical agents. The pregnant woman's body protects the developing tissues of germ cells, embryos and fetuses, and Wilson argues that in placental mammals, many physical agents such as low-energy radiation do not greatly affect developing fetuses. However, Wilson viewed ionizing radiation as an exception among physical agents because of his ability to reach developing tissues. Thus, one of the first environmental agents classified as teratogen was x-radiation. Unlike physical agents, however, chemical agents almost always reach developing tissues, usually through the mother's blood. Due to the factors of maternal metabolization, absorption and elimination, the concentration of the chemical agent varies depending on the time the agent reaches the fetus, a change that affects the degree of abnormal development. A chemical that can be teratogenic in vitro, or an embryo in a test tube, does not necessarily mean that the same chemical will be a teratogenic agent in vivo, an embryo within its normal biological environment, such as in the womb. Wilson's fifth principle, Four manifestations of deviant development - death, malformations, backwardness of growth and functional deficit, has sections on each of these manifestations in the title of the principle. Wilson argues that encounters with teratogenic agents at any time during development have the ability to produce one or more of these manifestations, and that some manifestations are likely to occur at certain stages of development. After implantation of the embryo into the uterine wall, but before its cells are differentiated, the most common manifestation of deviant development is the death of the embryo. In addition, organisms sensitive to teratogenes are more susceptible to the death of their embryos than to the development of these abnormalities; reverse is the case for lines of organisms somewhat resistant to teratogenes. The sixth and final principle, listed by Wilson, manifestations of deviant development increase in frequency and degree as the dosage increases, from non-effect to completely lethal level, has five sections. In Section A of Thresholds in Teratogenesis, Wilson describes the concept of teratogenic thresholds and explains that while studies may indicate that organisms may have a threshold for teratogenes, or that pregnant women may interact with specific teratogenes and offspring that show no evidence of any defect, a large sample of test animals still needs to definitively establish the existence of Level. Sections B to D on embryotoxicity and maternal toxicity and Curve. The dose reaction curve depicts how quickly the developmental effect can change depending on the dosage of the teratogenic agent and the stage of embryonic development. Section E. Dosage level and response level, describes a range of toxicity from no effect to completely lethal. It also describes accessory effects such as elongated pregnancies during developmental period in which the defect forms. In addition, increasing the dose of teratogen can lead to certain malformations occur at several stages of development. Nearly three per cent of infants exhibit significant morphological anomalies, and malformations account for about twenty-five per cent of neonatal deaths, making them the leading cause of infant mortality. Scientists are investigating the mechanisms by which teratogens work, six wilson principles will ground their work. Bateson Sources, William. Mendel's principles: Defense. Cambridge: University Press, 1902. (access to May 16, 2014). Brent, Robert L. In Memory: James G. Wilson (1915-1987). Teratology 39 (1989): 317-19. Dreste, Camilla. Recherches sur la production artificielle de monstruosit's: ou, Essais de t'ratog'nie exp'rementale Research on artificial production of monsters, or experimental tests of teratogenicity. Paris: K. Reinwald, 1877. 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