gest that, following treatment of carpet, only a very small fraction of the applied pyrethroid can indeed be regarded as potentially bioavailable by inhalation (max. 0.2% of the active ingredient applied to the carpet). Therefore, assessment of health hazards in the indoor environment based solely on "vacuum cleaner" sampling (as used by analytical chemists to search for indoor contaminants) rather than examination of the actual airborne concentration, including other relevant airborne materials, is prone to tremendous errors and misjudgments.

These examples demonstrate that new regulatory requirements, *e.g.,* the EU biocide directive, and public perception and concern may trigger complex and demanding toxicological examinations and standardization of such complex studies with regard to the generation of test atmospheres, mode and duration of exposure, and selection of adequate toxicological endpoints, as a basic prerequisite for state-of-the-art hazard identification and risk assessment.

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Adverse effects of environmental exposures may occur not only in the individuals directly exposed, but also in their progeny

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Individual differences in the risk of cancer are ultimately determined by the interaction between the environment, that is the complex of factors that are not part of the genetic patrimony at the time of conception, and the genotype. Beginning prenatally, humans are exposed throughout life to a variety of environmental agents, among them many carcinogens and mutagens. Adverse effects of such exposure may occur not only in the individuals directly exposed, but also in their progeny.

Prenatal events may contribute to increase the cancer burden following either: i) exposure to a noxious agent during pregnancy with consequent direct exposure of fetal cells *in utero* (transplacental carcinogenesis), or ii) exposure of one or both parents to a carcinogen/mutagen before conception, resulting in a possible alteration of the germ cells (transgenerational carcinogenesis).

There is experimental evidence of a transplacental carcinogenic effect for a large number and variety of chemical carcinogens. Evidence in humans is instead limited to exposure during pregnancy to diethyisfilbestrol (DES) and X-rays. Studies in rodents, however, cover the entire lifespan of the progeny and the increase in cancer frequency is generally observed in adult or late life. Observations in humans are almost exclusively confined to the occurrence of Cancer in childhood.

Even if many experimental studies remain open to criticism, particularly due to the relatively small number of animals used and the consequent lack of statistical power of the data, there is convincing evidence for a transgenerational effect in laboratory animals of a few chemical Carcinogens and of internal and external exposure to radiation. The epidemiological data in humans are mostly derived from the investigation of paternal occupational exposures to chemicals, chemical mixtures or radiation and, although suggestive of a transgenerational effect, they remain Controversial. Observations in humans, again, are limited to the occurrence of childhood cancer.

The hypothesis has been proposed that certain environmental exposures of parents before conception may cause alterations of the germ cells which affect the susceptibility of the progeny to cancer. Most likely, different mechanisms underlie the high incidence of tumors appearing at an early age in certain familial syndromes, and the surfacing of an increased predisposition to cancer revealed by a relatively modest increase in tumor incidence late in life. A better understanding of pre- and postnatal gene-environment interactions in determining cancer risks would have important implications for public health, as it could considerably improve the efficacy of primary prevention.

Experimental adenocarcinoma of the esophagus: Implications in the sequence Barrett's esophagus-adenocarcinoma

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No suitable models of esophageal carcinogenesis were available until the discovery by Duckrey *et al.* that N-methyl-N-nitrosoaniline given orally to rats resulted in a high incidence of squamous cell carcinomas of the esophagus. Since then, most of the experimentally induced squamous cell carcinomas of the esophagus have been due to the exposure of rats to several nitrosamines by various routes of administration.

In recent years, the incidence rates for adenocarcinoma of the esophagus have risen rapidly in Western countries, especially among white males. Most of these tumors arise from areas of columnar-lined esophagus (Barrett's esophagus), a condition clearly associated with a chronic gastroesophageal reflux of acid and duodenal-content secretions. These epidemiological observations prompted us