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# BEITRÄGE ZUR TABAKFORSCHUNG INTERNATIONAL

# CONTRIBUTIONS TO

## TOBACCO RESEARCH

### **CONTENTS**

1.	Editors' Note	297
2.	Implications of Evolving Medical Science for Proof of Lung Cancer Causation	
	Larry C. Frarey and Javier Martinez	298
3.	The Pore Size Distribution of Naturally Porous Cigarette Paper and its Relation to Permeability and Diffusion Capacity	
	Bernhard Eitzinger, Maria Gleinser, Stefan Bachmann, and Dietmar Volgger	312
4.	Prediction Model for Cigarette Yields Derived from Data Obtained under Two Different Machine Smoking Regimes	
	Stéphane Colard	320
5.	Evaluation of the Content of Free Amino Acids in Tobacco by a New Liquid Chromatography-Tandem Mass Spectrometry Technique	
	Serban C. Moldoveanu, Jeff Zhu, and Wayne A. Scott	334

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### **EDITORS' NOTE**

This issue of *Beiträge zur Tabakforschung International* (BTFI) includes a paper by Frarey and Martinez, titled "Implications of Evolving Medical Science for Proof of Lung Cancer Causation" (pp 298–311). Advisory Board and Editors of BTFI have been reasoning for some time before deciding to publish this article in the Journal. One point considered was the fact that the topic may be beyond the scope of *Beiträge*. A second issue was that the authors are affiliated with a law firm and the article is written from a legal perspective. There are, however, good reasons in favor of a publication in BTFI. First, the paper is written clearly and understandably, and certainly of considerable interest for the majority of our readers. Second, it has been positively reviewed by biomedical experts in the field. Third (and crucial for our decision), it deals with an old and formidable challenge for judicature, public health, industry and science: causality in multi risk factor diseases, which need decades for development and outbreak.

Concurrently with the release of the first U.S. Surgeon General Report ('Terry Report') in 1964 (1), the British statistician and epidemiologist, Sir Austin Bradford Hill, published his famous criteria for causation in epidemiology (2). These include strength (of the association between risk factor and diseases), consistency (reproducibility in different studies), specificity (no other likely explanation), temporality (effect must occur after exposure), biological gradient (dose-response relationship), plausibility (biological mechanism), coherence (with animal or *in vitro* studies), experiment (e.g., the effect of smoking cessation on disease risk), analogy (effect of similar factors). It is well accepted that the association between smoking and lung cancer fulfills the Hill criteria for causality, which, of course, is based on population data. Individual cases, on the other hand, are in the focus of the Frarey and Martinez paper. Since the mid 1960s epidemiology and medical science have achieved major progress in the direction of 'individualization'. 'Molecular epidemiology' includes biomarkers (of exposure and effect) as well as genetic factors (e.g., genetic polymorphisms), which result in the more objective assessment of exposure and more detailed description of the risk of a population. The inter-disciplinary science of 'molecular pathology and epidemiology' tries to elucidate disease etiology at the molecular, individual and population level by making use particularly of the modern 'omics' methodologies. At the end of this development towards individualization emerge the models of 'precision medicine' and 'personalized medicine'. Although they are primarily focused on custom-tailored therapies of diseases, it is conceivable that etiology also becomes part of these disciplines. It should be kept in mind however, that even at the individual level the element of probability retains its importance in the interplay of genetic and other biological or environmental factors. Only very rarely can one causal factor be correctly assigned to a disease. Generally, statistical probability and not a distinct cause-effect relationship will form the basis for scientific judgment.

In our view, the article of Frarey and Martinez contributes sensibly to the considerations discussed above. We would like to encourage interested readers to communicate their views in form of a letter to the editor.

Wolf-Dieter Heller Gerhard Scherer

### REFERENCES

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