CURRICULUM VITAE

April 2018

Title and name
Dr. Gabriele Aquilina

Nationality
Italian

Panel / Scientific Committee
Panel on Food Additives and Flavourings (FAF)

Education

Specialisation in Microbiology, 1985, Università degli Studi di Roma "La Sapienza", Rome, Italy
Degree in Biology, 1981, Università degli Studi di Roma "La Sapienza", Rome, Italy

Work Experience

2007 – present	Istituto Superiore di Sanità (ISS)	Senior scientist. Research activity: Analysis of genotoxicic activity of chemicals. Study of the molecular mechanisms of mutagenesis. Institutional and regulatory tasks and responsibilities: Inspector for Good laboratory practice, as expert for mutagenesis and carcinogenesis. Italian National Coordinator of the OECD Test Guideline Programme. Head of the Italian Delegation to the Joint Meeting of the Chemicals Committee and the Working Party on Chemicals, Pesticides and Biotechnology. Risk assessment activity (as expert for genotoxicity and carcinogenicity): Member of the Committee for Risk Assessment (RAC) of the European Chemicals Agency (ECHA). Chair of the Working Group "Evaluation of intrinsic properties and classification of chemicals" established by the ISS following a request of the Italian Competent Authority for REACH Regulation. Member of the "Working Group on Biocides" of the ISS.
1986 – 2006	Istituto Superiore di Sanità (ISS)	Researcher. Research activity: Analysis of the genotoxic activity of chemicals in bacterial systems and

		cultured mammalian cells. Study of the molecular mechanisms of mutagenesis. Analysis of the role of the mismatch repair system in the modulation of the genotoxicity of chemicals. Regulatory and risk assessment tasks: Member of the Advisory Committee for Biocides the Italian Ministry of Health. Member of the ISS Expert Panel on Biocides for the evaluation of biocides as expert for genotoxicity and carcinogenicity.
1981 – 1986	Istituto Superiore di Sanità (ISS)	Holder of post-graduate scholarships and grants, Analysis of the genotoxic activity of chemicals in bacterial systems and cultured mammalian cells.

Scientific expertise

Genetic Toxicology
In vitro and *in vivo* genotoxicity testing methods
Mechanism of genotoxicity and carcinogenicity

Most relevant scientific publications within the fields of EFSA

ORCID code: https://orcid.org/0000-0002-3280-7883

Narciso L, Catone T, **Aquilina G**, Attias L, De Angelis I, Iuliano MG, Tassinari R, Mantovani A, Maranghi F, 2017. The juvenile toxicity study as a tool for a science-based risk assessment in the children population group. Reprod Toxicol;72:136-141. doi: 10.1016/j.reprotox.2017.06.188. DOI: 10.1016/j.reprotox.2017.06.188

Blasi MF, Ventura I, **Aquilina G**, Degan P, Bertario L, Bassi C, Radice P, Bignami M, 2006. A human cell-based assay to evaluate the effects of alterations in the MLH1 mismatch repair gene. Cancer Res;66(18):9036-44. DOI: 10.1158/0008-5472.CAN-06-1896

Russo MT, Blasi MF, Chiera F, Fortini P, Degan P, Macpherson P, Furuichi M, Nakabeppu Y, Karran P, **Aquilina G**, Bignami M, 2004. The oxidized deoxynucleoside triphosphate pool is a significant contributor to genetic instability in mismatch repair-deficient cells. Mol Cell Biol;24(1):465-74.

Aquilina G, Bignami M, 2001. Mismatch repair in correction of replication errors and processing of DNA damage. J Cell Physiol;187(2):145-54. DOI: 10.1002/jcp.1067

Aquilina G, Ceccotti S, Martinelli S, Soddu S, Crescenzi M, Branch P, Karran P, Bignami M, 2000. Mismatch repair and p53 independently affect sensitivity to N-(2-chloroethyl)-N'-cyclohexyl-N-nitrosourea. Clin Cancer Res;6(2):671-80.

Aquilina G, Crescenzi M, Bignami, 1999. M.Mismatch repair, G(2)/M cell cycle arrest and lethality after DNA damage. Carcinogenesis;20(12):2317-26.

Aquilina G, Ceccotti S, Martinelli S, Hampson R, Bignami M, 1998. N-(2-chloroethyl)-N'-cyclohexyl-N-nitrosourea sensitivity in mismatch repair-defective human cells. Cancer Res;58(1):135-41.

Aquilina G, Fiumicino S, Zijno A, Martinelli S, Overkamp WJ, Zdzienicka MZ, Oshimura M, Wild CP, Bignami M, 1997. Reversal of methylation tolerance by transfer of human chromosome 2. Mutat Res;385(2):115-26.

Aquilina G, Hess P, Fiumicino S, Ceccotti S, Bignami M, 1995. A mutator phenotype characterizes one of two complementation groups in human cells tolerant to methylation damage. Cancer Res;55(12):2569-75

Aquilina G, Hess P, Branch P, MacGeoch C, Casciano I, Karran P, Bignami M, 1994. A mismatch recognition defect in colon carcinoma confers DNA microsatellite instability and a mutator phenotype. Proc Natl Acad Sci U S A;91(19):8905-9.

Branch P, **Aquilina G**, Bignami M, Karran P, 1993. Defective mismatch binding and a mutator phenotype in cells tolerant to DNA damage. Nature;362(6421):652-4.

Aquilina G, Biondo R, Dogliotti E, Meuth M, Bignami M, 1992. Expression of the endogenous O6-methylguanine-DNA-methyltransferase protects Chinese hamster ovary cells from spontaneous G:C to A:T transitions. Cancer Res;52(23):6471-5.

Aquilina G, Giammarioli AM, Zijno A, Di Muccio A, Dogliotti E, Bignami M, 1990. Tolerance to O6-methylguanine and 6-thioguanine cytotoxic effects: a cross-resistant phenotype in N-methylnitrosourea-resistant Chinese hamster ovary cells. Cancer Res;50(14):4248-53.

Aquilina G, Zijno A, Moscufo N, Dogliotti E, Bignami M. Tolerance to methylnitrosourea-induced DNA damage is associated with 6-thioguanine resistance in CHO cells. Carcinogenesis. 1989 Jul;10(7):1219-23.

Aquilina G, Frosina G, Zijno A, Di Muccio A, Dogliotti E, Abbondandolo A, Bignami M, 1988. Isolation of clones displaying enhanced resistance to methylating agents in O6-methylguanine-DNA methyltransferase-proficient CHO cells. Carcinogenesis. 9(7):1217-22.

Moreover, co-author of over 300 Scientific Opinions published in the EFSA Journal, including 69 opinions as responsible for the assessment of the genotoxic potential and 13 opinions as rapporteur.