

Justo Lorenzo Bermejo

1997 Engineer, Madrid Polytechnic University (UPM) Spain
1998 PhD Quant. Genetics, Kiel University (CAU) Germany
2002 Mathematician, Spanish Open University (UNED)
2003-8 Scientist, German Cancer Research Center (dkfz)
2009- Group leader, University Hospital Heidelberg (IMBI)



Current Research

My research has focused on the quantification of the familial aggregation of cancer based on the Swedish Family-Cancer Database, the identification of cancer susceptibility genes and the combination of data from familial and genetic association studies.

-I have adapted statistical techniques that permit to impute unobserved genotypes based on public data repositories. I have applied these techniques to explore the relationship between *TGF β* variants and acute lymphoblastic leukemia, and the effect of variants in the *UBC9* gene on breast cancer risk.

- I have applied both traditional (logistic regression) and novel (multidimensionality reduction) statistical methods to investigate the relationship between combinations of genetic variants and cancer.

- The design of preventive strategies, the identification of prognostic biomarkers and the translation into improved therapies may benefit from the characterization of (sub)phenotypes. I have used gene expression data to characterize heterogeneous nevi and to investigate the relationship with *BRAF* mutational status.

- I have developed and applied tailored statistical methods to optimize the identification of genetic variants involved in cancer and other complex diseases (for example, in rheumatoid arthritis).

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Future Projects and Goals

I will use data on familial aggregation to characterize cancer phenotypes and to prioritize genotyping. Genetic models will be designed in order to integrate data from familial and association studies.

Current statistical methods for identification of cancer related genes behave quite poorly under slight violations of the model assumptions. I will develop robust methods to identify the models best fitting to the majority of the data, and to detect outlying observations.

Selected Publications

Gene-environment interactions and familial relative risks **Hum Hered**; 66: 170-9.

Gene-environment studies: any advantage over environmental studies? **Carcinogenesis**; 28: 1526-32.

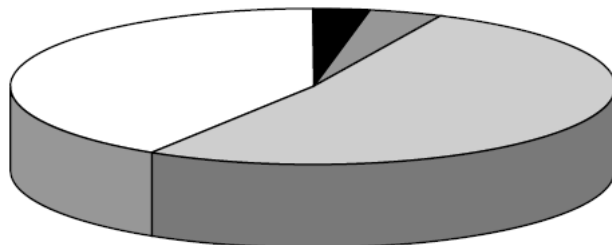
The balance between heritable and environmental aetiology of human disease. **Nat Rev Genet**; 7: 958-65.

Folate metabolic gene polymorphisms and childhood acute lymphoblastic leukaemia. **Leukemia**; 21: 320-5.

SNPs in DNA repair genes and basal cell carcinoma of skin. **Carcinogenesis**; 27: 1655-60.

Familial risk of cancer shortly after diagnosis of the first familial tumour. **J Natl Cancer Inst**; 97:1575-9.

- High penetrance (HNPCC, APC, MUTYH, LKB1, SMAD4, BMPR 1A, PTEN, ENG), PAF=3%
- ▣ Moderate penetrance (CHEK2 1157T, APC missense), PAF= 4%
- Low penetrance (six loci), PAF=52%



Genomic landscape of colorectal cancer.
Published in the
Am J Gastroenterology; 104: 789-90.