

BIOGRAPHICAL SKETCH

NAME: Cinzia Borgogna, PhD

POSITION TITLE: Associate Professor of Medical Microbiology

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
University of Turin, Turin, Italy	BSc	07/2005	Medical Biotechnology
University of Piemonte Orientale, Novara, Italy	PhD	12/2009	Virology
National Institute for Medical Research, London, UK	Postdoctoral	12/2011	Tumor Virology

A. Personal Statement

I am an Associate Professor of Medical Microbiology with 15 years of experience in the field of oncogenic viruses and emerging infectious diseases, particularly Human Papillomaviruses, Polyomaviruses, and SARS-CoV-2. I have a broad background in molecular virology, with specific expertise in virus-host interactions, innate immune responses, and the molecular mechanisms underlying viral persistence, oncogenesis, and immune evasion. Since 2013, I have served as Principal Investigator or co-Investigator on several competitively funded research projects supported by various funding agencies. I am currently leading a national PRIN PNRR grant focusing on host restriction factors against positive-sense single-stranded RNA viruses. My recent work has centered on characterizing the antiviral activity of IFI16, including its role as an RNA-binding protein during coronavirus infection.

In parallel with my research, I am the director of the residency school in Microbiology and Virology at the University of Piemonte Orientale, where I am actively involved in the advanced training of medical and biological specialists in clinical microbiology, virology, and infectious diseases. I also serve as a faculty member in the PhD program in Medical Sciences and Biotechnology and I regularly lecture in several degree programs, including Biology, Nursing, and Medicine, providing both theoretical and practical training.

In addition, I am a member of several institutional scientific committees, including the animal welfare Board (OPBA), the Biobank scientific committee, and spin-off and patent evaluations committee. These roles have provided me with significant experience in research leadership, project coordination, teaching, ethical oversight, and the translational application of biomedical research.

B. Positions, Scientific Appointments, and Honors

Positions and Appointments:

2021-to date: **Associate Professor of Medical Microbiology**, University of Piemonte

Orientale (UPO)

2011- 2021: **Assistant Professor of Medical Microbiology**, UPO

2024- to date **Director of the residency school in Microbiology and Virology**, UPO

2024- to date **Member of the board for the animal welfare (OPBA)**, UPO

2024- to date **Member of the spin off and patent evaluations committee**, UPO

2021-to date: **Member of the UPO Biobank scientific committee**, UPO

2020-to date: **Faculty member of Ph.D. Program in Medical Sciences and Biotechnology (XXXVI cycle)**, UPO

2019: **Faculty member of Ph.D. Program in Food, Health and Longevity (XXXV cycle)**, UPO

2014-2018: **Faculty member of Ph.D. Program in Medical Sciences and Biotechnology (XXX-XXXIV cycle)**, UPO

Honors:

2018: **In the Issue - Journal of Immunology** (doi: 10.4049/jimmunol.1890002). "HPV18 Persistence Impairs both Basal and DNA Ligand-Mediated IFN- β and IFN- λ_1 Production Through Transcriptional Repression of Multiple Downstream Effectors of Pattern Recognition Receptor Signaling" (Albertini et al., J Immunol 2018).

2011: **Winner of a travel award** for participating to the "27th International Papillomavirus Conference", Berlin, Germany.

2010: **Winner of the "Young Scientist Travel Award"** for participating to the "Fourth European Congress of Virology" Cernobbio, Italy.

2010: **FEMS Advanced Fellowship 2010**, awarded by the Federation of European Microbiological Societies (FEMS) to support a one-year research period at the National Institute for Medical Research, London, UK.

2006: **Winner of the "Biotech" award for the "Start-Cup Piemonte 2006" competition.**

C. Contributions to Science

Over the years, my research has covered multiple areas within virology and infectious diseases. The main research directions and scientific contributions I have made are detailed as follows:

1. The role of β HPV in skin carcinogenesis

I contributed to advancing the understanding of the role of β HPV in cutaneous carcinogenesis by developing innovative techniques to detect β HPV infection in human and murine skin lesions, including E4 immunostaining as a marker of productive infection. By applying this methodology to HPV8 transgenic mice, I established a novel model of β HPV-induced skin carcinogenesis characterized by the aberrant expansion of Lrig1-positive keratinocyte stem cells in the upper hair follicle (Lanfredini et al., *J Invest Dermatol*, 2017; Olivero et al., *Front Microbiol*, 2018). I further demonstrated the

progression of β HPV-positive actinic keratosis to squamous cell carcinoma through xenografting of human skin tumors in immunodeficient mice (Borgogna et al., *Front Microbiol*, 2018). More recently, I generated immunodeficient HPV8-transgenic mice, providing proof of concept of the synergistic role of immunosuppression, UV exposure, and β HPV infection in cutaneous cancer development (Borgogna et al., *J Invest Dermatol*, 2023).

2. Polyomavirus infection, reactivation, nephropathy, and tumorigenesis

In collaboration with clinical nephrologists and pathologists, my work has addressed the role of human polyomaviruses in kidney transplant recipients. I demonstrated that BK polyomavirus (BKPyV) undergoes in vivo mutagenesis during viral nephropathy, acquiring mutations that confer a replicative advantage (Peretti et al., *Cell Host Microbe*, 2018). I also uncovered that HPyV reactivation occurs in the urinary tract but not within renal tumors in a large cohort of kidney transplant recipients (Borgogna et al., *Viruses*, 2021), contributing to the understanding of viral latency and pathogenesis in immunosuppressed patients.

3. Virus-induced skin carcinogenesis in the elderly

Within the five-year “Department of Excellence in Aging Science” program at UPO, I expanded my research interests to Merkel Cell Carcinoma (MCC), a neuroendocrine skin carcinoma driven by Merkel cell polyomavirus (MCPyV). In collaboration with Dr. Nicole Fischer’s laboratory, a leading center for MCPyV research, I investigated the contribution of cancer-associated fibroblasts (CAFs) to the tumorigenesis of MCC in elderly patients. Through these studies, we characterized how MCC-derived CAFs enhance tumor growth and immune evasion mechanisms (Albertini et al., *J Invest Dermatol*, 2023).

4. Development of molecular and serological tools for SARS-CoV-2 infection management

During the COVID-19 pandemic, I led efforts to develop advanced diagnostic and monitoring tools for SARS-CoV-2 infection, including VSV-based pseudovirus neutralization assays and droplet digital PCR (ddPCR) protocols for viral load quantification in various biological matrices such as saliva. These tools have been employed in collaborative clinical studies to monitor humoral immune responses and vaccine efficacy in different patient cohorts, particularly immunocompromised individuals (Borgogna et al., *Blood Cancer J*, 2022; Borgogna et al., *Br J Haematol*, 2022; Monzani et al., *Viruses*, 2022; Caneparo et al., *Microbiol Spectr*, 2022).

5. The role of IFI16 in regulating coronavirus replication

My current research focuses on the role of IFI16, a well-known interferon-inducible DNA sensor, in regulating RNA virus replication, specifically investigating human coronaviruses (HCoVs). I demonstrated that IFI16 restricts SARS-CoV-2 and OC43 replication in an interferon-independent manner, while supporting NL63 replication. Upon SARS-CoV-2 infection, IFI16 translocates to the cytoplasm, where it binds viral RNA and the Nucleocapsid (N) protein, disrupting viral RNA-induced liquid-liquid phase separation (LLPS), a process crucial for the formation of viral condensates and efficient viral replication. Unlike SARS-CoV-2 and OC43, which possess an N-terminal intrinsically disordered region (IDR) essential for LLPS, NL63 lacks this region and thus evades IFI16-mediated restriction. These findings provide novel mechanistic insights into IFI16’s role as a modulator of coronavirus replication by targeting LLPS-dependent viral processes. (Cislaghi, I. et al. *Commun. Biol.* [Second round of revision]).

Complete list of publications:

<https://www.scopus.com/authid/detail.uri?authorId=23093248700>