University of Birmingham

# Dr Yun Fan PhD

Birmingham Fellow
Principal Investigator

**School of Biosciences (/schools/biosciences/index.aspx)**

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## About

Dr Yun Fan's research interest centers on understanding how cell death, cell proliferation and cell differentiation are coordinated to maintain tissue homeostasis. This has important implications for cancer development and tissue regeneration. As one of our Birmingham Fellows recruited worldwide, he joined us and established his laboratory at Birmingham in November 2012.

## Qualifications

2005 PhD - Biozentrum, University of Basel, Basel, Switzerland
2001 MS - Sun Yat-sen (Zhongshan) University, Guangzhou, China
1998 BE - Huazhong University of Science and Technology, Wuhan, China

## Biography

Dr Fan was initially trained as a molecular biologist in China. He then completed his PhD in the field of Neurobiology in Switzerland. He subsequently worked at the University of Texas MD Anderson Cancer Center and the University of Massachusetts Medical School in the United States during which time his research focused on regulation of program cell death (apoptosis) and its related compensatory cell proliferation.

## Teaching

BIO152 - Cell Biology and Physiology

BIO268 - Cell and Developmental Biology

BIOM03

Dr Fan is an Associate Fellow recognized by the Higher Education Academy (HEA)

## Postgraduate supervision

Competition funded PhD studentships are available. Applicants are encouraged to contact Dr Fan directly.

Please find details of the PhD research project at **www.findaphd.com/search/ProjectDetails.aspx?PJID=37217&LID=124 (http://www.findaphd.com/search/ProjectDetails.aspx?PJID=37217&LID=124)**

## Research

**Regulation of apoptosis and compensatory cell proliferation in tissue homeostasis**

Tissue homeostasis is the maintenance of normal tissue morphology and function under physiological or pathological conditions. In multi-cellular organisms, this requires coordinated cell death (e.g. apoptosis), cell proliferation and cell differentiation. On the one hand, in response to stresses such as radiation and accidental injury, cells can get damaged and are removed by a self-destruct mechanism called apoptosis. On the other hand, surprisingly, apoptotic cells can actively induce proliferation of their neighboring cells to compensate for the cell loss. This phenomenon is termed apoptosis-induced compensatory cell proliferation (apoptosis-induced proliferation). Therefore, both apoptosis and apoptosis-induced proliferation are critical for tissue recovery and organismal survival. Under pathological conditions, mis-regulated apoptosis or apoptosis-induced proliferation can lead to many human diseases including degenerative disorders and cancer. Our research is to investigate the cellular control of apoptosis and apoptosis-induced proliferation, especially how these cellular processes are coordinated and regulated.

Work by us and others has revealed that stress-induced apoptotic cells can send growth signals to trigger compensatory cell proliferation through a non-apoptotic function of caspases, a family of cysteine-proteases that normally execute apoptosis. Intriguingly, we also discovered that apoptosis can induce cell proliferation through distinct mechanisms in different developmental contexts, e.g. in proliferating versus differentiating tissues. However, it is not yet clear how apoptosis-induced proliferation is regulated at the molecular level following activation of caspases, and why distinct mechanisms are employed in a cell context-dependent manner. By taking advantages of Drosophila as a genetically tractable model organism, we have developed unique assays to systematically identify and characterize novel regulators of apoptosis-induced proliferation. Dissecting its underlying regulatory mechanisms will make substantial contributions to our understanding of the cellular strategies and genetic pathways used to maintain tissue homeostasis in response to apoptosis. Our long-term research goal is to elucidate the relevance of apoptosis-induced proliferation in tissue regeneration and tumorigenesis.

MARIE CURIE ACTIONS (http://ec.europa.eu/research/mariecurieactions/about-mca/actions/cig/index_en.htm)

BBSRC bioscience for the future (http://www.bbsrc.ac.uk/home/home.aspx)

Dr Fan's research is supported by the EU FP7 Marie Curie Actions (CIG) and the Biotechnology and Biological Sciences Research Council in the UK.

## Other activities

Members of the British Society for Developmental Biology and the Genetics Society

2011-2012, Research Assistant Professor, University of Massachusetts Medical School, Worcester, USA

2009-2011, Instructor, University of Texas MD Anderson Cancer Center, Houston, USA

2006-2009, Postdoctoral Fellow, University of Texas MD Anderson Cancer Center, Houston, USA

## Publications


- **Fan Y*** and Bergmann A* (2014). Multiple mechanisms modulate distinct cellular susceptibilities towards apoptosis in the developing *Drosophila* eye. ***Dev Cell*** 30(1):48-60.
  (*corresponding authors) **http://www.ncbi.nlm.nih.gov/pubmed/24981611 (http://www.ncbi.nlm.nih.gov/pubmed/24981611)**
- **Fan Y***, Wang S, Hernandez J, Yenigun VB, Hertlein G, Fogarty CE, Lindblad JL and Bergmann A* (2014). Genetic models of apoptosis-induced proliferation decipher activation of JNK and identify a requirement of EGFR signaling for tissue regenerative responses in Drosophila. ***PLoS Genetics*** 10(1): e1004131.
  (*corresponding authors) **http://www.ncbi.nlm.nih.gov/pubmed/24497843 (http://www.ncbi.nlm.nih.gov/pubmed/24497843)**
- Huang Q, Tang X, Wang G, **Fan Y**, Ray L, Bergmann A, Belenkaya TY, Ling X, Yan D, Lin Y, Ye X, Shi W, Zhou X, Lu F, Qu J and Lin X (2014). Ubr3 E3 ligase regulates apoptosis by controlling the activity of DIAP1 in *Drosophila*. ***Cell Death Differ*** 21(12):1961-70.
  **http://www.ncbi.nlm.nih.gov/pubmed/25146930 (http://www.ncbi.nlm.nih.gov/pubmed/25146930)**
- Christiansen AE, Ding T, **Fan Y**, Graves HK, Herz HM, Lindblad JL and Bergmann A (2012). Non-cell autonomous control of apoptosis by ligand-dependent Hedgehog signaling in *Drosophila*. ***Cell Death Differ***20(2):302-11.
  **http://www.ncbi.nlm.nih.gov/pubmed/23018595 (http://www.ncbi.nlm.nih.gov/pubmed/23018595)**
- Lee TV*, **Fan Y***, Wang S, Srivastava M, Broemer M, Meier P and Bergmann A (2011). *Drosophila* IAP1-mediated ubiquitylation controls processing, but not protein stability, of the initiator caspase DRONC. ***PLoS Genetics***7 (9): e1002261.
  (*co-first authors) **http://www.ncbi.nlm.nih.gov/pubmed/21909282 (http://www.ncbi.nlm.nih.gov/pubmed/21909282)**
- **Fan Y**, Lee T, Xu D, Chen Z, Lamblin AF, Steller H and Bergmann A (2010). Dual roles of *Drosophila* p53 in cell death and cell differentiation. ***Cell Death Differ***17 (6): 912-921.
  **http://www.ncbi.nlm.nih.gov/pubmed/19960025 (http://www.ncbi.nlm.nih.gov/pubmed/19960025)**
- **Fan Y** and Bergmann A (2010). The cleaved-Caspase-3 antibody is a marker of Caspase-9-like DRONC activity in *Drosophila*. ***Cell Death Differ***17 (3), 534-539.
  **http://www.ncbi.nlm.nih.gov/pubmed/19960024 (http://www.ncbi.nlm.nih.gov/pubmed/19960024)**
- Xu D, Woodfield SE, Lee TV, **Fan Y**, Antonio C and Bergmann A (2009). **Genetic control of programmed cell death (apoptosis) in *Drosophila*. (http://www.ncbi.nlm.nih.gov/pubmed/19182545?ordinalpos=3&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_DefaultReportPanel.Pubmed_RVDocSum)** ***Fly*** *(Austin)* 3(1):78-90.
  **http://www.ncbi.nlm.nih.gov/pubmed/19182545 (http://www.ncbi.nlm.nih.gov/pubmed/19182545)**
- **Fan Y** and Bergmann A (2008). Apoptosis-induced compensatory proliferation. The Cell is dead. Long live the Cell! ***Trends Cell Biol***18 (10), 467-473.
  **http://www.ncbi.nlm.nih.gov/pubmed/18774295 (http://www.ncbi.nlm.nih.gov/pubmed/18774295)**
- **Fan Y** and Bergmann A (2008). Distinct mechanisms of apoptosis-induced compensatory proliferation in proliferating and differentiating tissues in the *Drosophila* eye. ***Dev Cell***14 (3), 339-410. (evaluated by Faculty of 1000 Biology as “Must Read”)
  **http://www.ncbi.nlm.nih.gov/pubmed/18331718 (http://www.ncbi.nlm.nih.gov/pubmed/18331718)**