



# HKUST Institutional Repository (IR)

<http://repository.ust.hk/ir/>

Becky Lee  
October 2014

# Agenda

- What is actually HKUST IR doing?
- Why does the University need IR?
- How could IR work with the scholars?
- How could IR benefit scholars and the University?



# HKUST Institutional Repository (IR)

## Features

- It is a database using **DSpace for storage**, a software originally developed at MIT
- It complies with the **Open Archives Initiative (OAI)** which allows the content to be **easily discovered by web search engines**, services, and indexing tools, such as Google and [OAster](#)
- Its content can be **searched by keyword(s) in all fields** or particularly in article title, journal title, author, subject or ISSN/ISBN field.

# HKUST Institutional Repository (IR)

## 1. Indexes HKUST community's scholarly output

Collect different types of output

Over 61,000 records

Home Scholar Profiles

Search Publications:  All Fields  Advanced

Showing 1 - 20 of 61,176 (0.07 seconds) Sort by Relevance

Export 0 items to:  Email  CSV  Refworks

1 2 3 4 5 6 7 8 9 10 11 Next » [3059]

**Narrow Search**

FORMAT	Count
Article	32,835
Conference paper	20,220
Thesis	4,670
Book chapter	1,706
Book	650
Working paper	347
Patent	307
Technical report	199
Book review	100
Presentation	99
Preprint	22
Composition	8
Other	7
Research report	4
Dataset	2
ISS ...	

Effects of Ar- and Ar/O2-plasma-treated amorphous and crystalline polymer surfaces revealed by ToF-SIMS and principal component analysis  
Author(s): Ren, Xianwen ; Weng, Lu-Tao ; Ng, Kai-Mo ; Chan, Chi-Ming  
Source: Surface and interface analysis, v. 45, (7), 2013, Jul, p. 1158-1165  
Article, 2013

An overview of data replication on the internet  
Author(s): Loukopoulos, T ; Ahamd, I ; Papadias, D  
Source: I-SPAN'02: INTERNATIONAL SYMPOSIUM ON PARALLEL ARCHITECTURES, ALGORITHMS AND NETWORKS, PROCEEDINGS, 2002, p. 31-36  
Conference paper, 2002

Assessment of Non-uniform Temperature Effect on BGA De-component Process  
Author(s): Tang, Kun ; Song, Fubin ; Lee, S. W. Ricky ; Lo, Jeffery C. C.  
Source: 2012 IEEE 62ND ELECTRONIC COMPONENTS AND TECHNOLOGY CONFERENCE (ECTC), 2012, p. 977-980  
Conference paper, 2012

# HKUST Institutional Repository (IR)


2. Provides **Scholar Profile** to showcase HKUST faculty members' publications, bibliometrics, research interests and projects

HKUST Institutional Repository

Home ▾ Scholar Profiles

Search Publications:  All Fields ▾ Q Find Advanced

Search Profiles:  All Fields ▾ Q Find

 **Ip, Nancy Y Y (葉玉如)**

- » Dean, Office of the Dean of Science
- » Director, State Key Laboratory of Molecular Neuroscience, Division of Life Science
- » Co-Director, Molecular Neuroscience Center, Division of Life Science
- » The Morningside Professor of Life Science, Division of Life Science
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Homepage: <http://life-sci.ust.hk/faculty/Prof.N.Ip/index.html>  
Scopus: 7005756760, 35899235100  
[Co-authorship graph \(Beta Version\)](#)

**Publications** Bibliometrics Research Interests Projects

**Summary**

	Total	Article	Conference paper	Book chapter	Patent	Book
All publications	468	235	221	7	4	1
HKUST affiliated	439	210	218	6	4	1

Provide detailed information for each individual scholar

# HKUST Institutional Repository (IR)

3. Preserves scholarly output and allow them to be openly accessible

Provide real time citation counts in WoS and Scopus

Allow OA version of the article in full-text for public to download

HKUST Institutional Repository

Home Scholar Profiles

Search Publications: | All Fields Q Find Advanced

Cyclin-Dependent Kinase 5 Supp...

Please use this identifier to cite or link to this item: <http://hdl.handle.net/1783.1/3530>

### Cyclin-Dependent Kinase 5 Supports Neuronal Survival through Phosphorylation of Bcl-2

**Authors** Cheung, Zelda H. Gong, Ke Ip, Nancy Y.

**Issue Date** 2008

**Source** JOURNAL OF NEUROSCIENCE, v. 28, (19), 2008, MAY 7, p. 4872-4877

**Summary** Accumulating evidence indicates that deregulation of cyclin-dependent kinase 5 (Cdk5) activity is associated with apoptosis in various neurodegenerative disease models. Interestingly, recent studies suggest that Cdk5 may also favor neuronal survival. Nonetheless, whether Cdk5 is directly required for neuronal survival during development remains enigmatic. In the current study, we established the pivotal role of Cdk5 in neuronal survival during development by demonstrating that reduction or absence of Cdk5 activity markedly exacerbated neuronal death in cultures and in vivo. Interestingly, the antiapoptotic protein Bcl-2 (B-cell lymphoma protein 2) was identified as a novel substrate of Cdk5. We found that Cdk5-mediated phosphorylation of Bcl-2 at Ser70 was required for the neuroprotective effect of Bcl-2. Intriguingly, inhibition of this phosphorylation conferred proapoptotic property to Bcl-2. Furthermore, overexpression of a Bcl-2 mutant lacking the Cdk5 phosphorylation site abolished the protective effect of Cdk5 re-expression in Cdk5(-/-) neurons, suggesting that Ser70 phosphorylation of Bcl-2 contributed to Cdk5-mediated neuronal survival. Our observations revealed that Cdk5-mediated Bcl-2 phosphorylation is pivotal for the antiapoptotic effect of Bcl-2 and contributes to the maintenance of neuronal survival by Cdk5. Our study has also identified Cdk5 as a regulator of Bcl-2 function in neuronal apoptosis.

**Subjects** Apoptosis P35 Bcl-2 Cdk5 Neuronal survival Retinal ganglion cells p35

**ISSN** 0270-6474

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**Language** English

**Format** Article

**Access** [View full-text via DOI](#)  
[View full-text via Web of Science](#)  
[View full-text via Scopus](#)  
[Find](#)

**Files in this item:**

File	Description	Size	Format
4872.full.pdf	Published version	365504 B	Adobe PDF

# Why does the University need IR?

Yes, this is not a must!

## Functional needs

- The University needs an online archive for preserving its scholarly output
- The University needs a centralized platform for showcasing its research experts and output

## Mandates

1. **Institution's mandate** : Faculty member needs to grant the institution the non-exclusive permission to make his or her research output be openly accessible.

The examples of **institutions** implemented OA mandate:

- Massachusetts Institute of Technology (MIT)
- University College London (UCL)
- Nanyang Technological University (NTU)
- The Hong Kong Polytechnic University

# Why does the University need IR?

## Mandates

2. **Funder's mandate** : The funding agency requires its researchers to deposit a copy of the research publication in a OA repository immediately upon publication or with a few months of embargo period.

The examples of **funders** implemented OA mandate:

- National Institutes of Health (NIH) in US
- European Research Council (ERC)
- Hong Kong Research Grants Council (RGC)

The  
responsibility of  
Principal  
Investigator (PI)

\* An extract from the section 12 of Application Form (GRF1)

.... I undertake that upon acceptance of a paper for publication,

- I will check whether the publisher already allows (A) **full open access** to the **publisher's version**, or (B) my depositing a copy of the paper (either the publisher's version or **the final accepted manuscript after peer-review**) in the **institutional repository for open access**;
- if both (i) (A) and (B) are not allowed, I will request the publisher to allow me to place either version in my institutional repository **for restricted access immediately upon publication** or **after an embargo period** of up to twelve months if required by the publisher; and
- subject to the **publisher's agreement** on (i) or (ii) above, I will deposit a copy of the publication **in my institutional repository as early as possible** but no later than six months after publication or the embargo period, if any.





# How could IR work with the scholars?

## HKUST Scholars

1. Keep the pre-published versions of the research publication
  - ❑ **Pre-print**: the final manuscript/draft submitted to the publisher, AND/OR
  - ❑ **Post-print**: the final accepted version after peer-review and ready to be published
2. Submit the above version(s) to IR after the publication is published
  - ❑ by email via [lbir@ust.hk](mailto:lbir@ust.hk)
  - ❑ by online submission via [http://library.ust.hk/ir\\_submit](http://library.ust.hk/ir_submit)

## IR team

1. **Clear copyright issue** with the publishers/copyright owners
  - Confirm which version(s) can be deposited in IR
  - Clarify on the license requirement/notice of acknowledge/copyright statement
  - Note the embargo periods if any
2. Post the record with **correct version** of the article into IR for open access

**Cited By Counts**

10  
Scopus

- Similar Items**
- Coordinated relay beamforming for amplify-and-forward two-hop interference networks  
Author(s): Shi, Y.; Zhang, Jun; Letaief, K. B. 2012
  - Interference alignment in MIMO interference relay channels  
Author(s): Chen, X.; Song, S.H.; Ben Letaief, K. 2012
  - Distributed Power Allocation in Two-Hop Interference Channels: An Implicit-Based Approach  
Author(s): Shi, Y.; Ben Letaief, Khaled; Mallik, Ranjan K. ... 2012
  - Randomized power control for two-hop interference channels  
Author(s): Shi, Y.; Mallik, R.K.; Letaief, K.B. 2010
  - Performance analysis of multiple-relay decode-and-forward cooperation system  
Author(s): Zhang, Jun; Lok, T. M. 2005

Please use this identifier to cite or link to this item: <http://hdl.handle.net/1783.1/8165>

**Interference management with relay cooperation in two-hop interference channels**

**Authors** Zhang, Jun Letaief, K. B.

**Issue Date** 2012-02

**Source** IEEE Wireless Communications Letters , v.1, (3), June 2012, p.165-168

**Summary** Relaying has been proposed as an efficient technique to extend coverage and improve throughput in future wireless networks. However, its performance gain is degraded in the presence of co-channel interference, which is intensified as the network size increases. In this paper, we propose a relay selection strategy to reduce the interference between the network and the relay. The proposed strategy is based on the relay selection strategy between the network and the relay. The proposed strategy is based on the relay selection strategy between the network and the relay.

**Note** This work was supported by the National Natural Science Foundation of China (Grant No. 610311).

**Subjects** Wireless networks  
Interference management  
Two-hop interference channel  
Decode-and-forward relaying

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**Language** English

**Format** Article

**Access** View full-text via DOI  
View full-text via Scopus

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File	Description	Size	Format
RelayCoop_WCL_Revision.pdf	Pre-published version	168282 B	Adobe PDF

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Allow "Pre-published version" for OA

**Cited By Counts**

42  
WoS

43  
Scopus

- Similar Items**
- The role of cyclin-dependent kinase 5 in retinal ganglion cell survival  
Author(s): Cheung, Zelda H.; Ip, Nancy Y. 2004
  - Distinct cellular compartment of cyclin-dependent kinase 5 (Cdk5) and neuron-specific Cdk5 activator protein (p35(nck5a)) in the developing rat cerebellum  
Author(s): Matsushita, M; Tomizawa, K; Lu, YF ... 1998
  - Cyclin-dependent kinase 5 – an emerging player in Parkinson's disease pathophysiology, in Mechanisms in Parkinson's Disease - Models and Treatments  
Author(s): Cheung, Zelda H.; Ip, Nancy Y. 2012
  - Cdk5 activity is required for BDNF-stimulated neuronal survival and synaptic plasticity  
Author(s): Xu, Pei 2008
  - Involvement of Cdk5/p35 in EphB2-dependent dendritic spine development  
Author(s): Wu, Qian 2008

Please use this identifier to cite or link to this item: <http://hdl.handle.net/1783.1/3530>

**Cyclin-Dependent Kinase 5 Supports Neuronal Survival through Phosphorylation of Bcl-2**

**Authors** Cheung, Zelda H. Gong, Ke Ip, Nancy Y.

**Issue Date** 2008

**Source** JOURNAL OF NEUROSCIENCE , v. 28, (19), 2008, MAY 7, p. 4872-4877

**Summary** Accumulating evidence indicates that deregulation of cyclin-dependent kinase 5 (Cdk5) activity is associated with apoptosis in various neurodegenerative disease models. Interestingly, recent studies suggest that Cdk5 may also favor neuronal survival. Nonetheless, whether Cdk5 is directly required for neuronal survival during development remains enigmatic. In the current study, we established the pivotal role of Cdk5 in neuronal survival during development by demonstrating that reduction or absence of Cdk5 activity markedly exacerbated neuronal death in cultures and in vivo. Interestingly, the antiapoptotic protein Bcl-2 (B-cell lymphoma protein 2) was identified as a novel substrate of Cdk5. We found that Cdk5-mediated phosphorylation of Bcl-2 at Ser70 was required for the neuroprotective effect of Bcl-2. Intriguingly, inhibition of this phosphorylation conferred proapoptotic property to Bcl-2. Furthermore, overexpression of a Bcl-2 mutant lacking the Cdk5 phosphorylation site abolished the protective effect of Cdk5 re-expression in Cdk5(-/-) neurons, suggesting that Ser70 phosphorylation of Bcl-2 contributed to Cdk5-mediated neuronal survival. Our observations revealed that Cdk5-mediated Bcl-2 phosphorylation is pivotal for the antiapoptotic effect of Bcl-2 and contributes to the maintenance of neuronal survival by Cdk5. Our study has also identified Cdk5 as a regulator of Bcl-2 function in neuronal apoptosis.

**Subjects** Apoptosis  
P35  
Bcl-2  
Cdk5  
Neuronal survival  
Retinal ganglion cells  
p35

**ISSN** 0270-6474

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**Language** English

**Format** Article

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4872.full.pdf	Published version	385504 B	Adobe PDF

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# How could IR benefit scholars and the University?

## Scholar can

- ✓ own and maintain your **centralized profile** showcasing publications, bibliometrics, research interests and projects all at once
- ✓ manage and **store digital content (including data)** connected with your research
- ✓ **increase the visibility** of your research output after making them openly accessible
- ✓ **increase the research impact** as OA research output can be cited more easily
- ✓ fulfill the requirement of **OA mandates** if any
- ✓ be the **pioneer in those fast moving subjects** such as Electronics because you can make preprints open access via IR to establish that they were the first and to get feedback quickly

# How could IR benefit scholars and the University?

## The University can

- ✓ **store and preserve institutional digital assets**, not only scholarly research output but also grey literature such as theses or technical report
- ✓ **provide persistent access** to all content
- ✓ **increase the worldwide visibility** of the researchers and their output, groups and centres, research topics and interests
- ✓ **increase the ranking position** by a greater web presence
- ✓ **contribute to social and economic development** in HK and beyond by providing free access to the research output to other smaller research institutions and SMEs
- ✓ **stimulate new research partnerships** after getting greater visibility of research results through more channels
- ✓ **demonstrate the support and commitment** to Open Access, Open Innovation and Open Science

# Further details about .....

*Thank you!*

- ? Which **OA option** suits me best
- ? How to consider the **licensing requirement**
- ? What if the publisher **does not have OA policy** or option
- ? How to **evaluate** the OA journals
- ? How do I know which journal is with “**Green OA**” and which is in “**Gold**”
- ? When and how to **keep/transfer** my copyright
- ? .....

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department in details

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~ 23<sup>rd</sup> October 2014 ~

***Q & A session***