Dataset for the proteomic inventory and quantitative analysis of the breast cancer hypoxic secretome associated with osteotropism

The cancer secretome includes all of the macromolecules secreted by cells into their microenvironment. Cancer cell secretomes are significantly different to that of normal cells reflecting the changes that normal cells have undergone during their transition to malignancy. More importantly, cancer secretomes are known to be active mediators of both local and distant host cells and play an important role in the progression and dissemination of cancer. Here we have quantitatively profiled both the composition of breast cancer secretomes associated with osteotropism, and their modulation under normoxic and hypoxic conditions. We detect and quantify 162 secretome proteins across all conditions which show differential hypoxic induction and association with osteotropism. Mass Spectrometry proteomics data have been deposited to the ProteomeXchange Consortium with the dataset identifier PXD000397 and the complete proteomic, bioinformatic and biological analyses are reported in Cox et al. (2015) [1].
Integrative analysis of kinase networks in TRAIL-induced apoptosis provides a source of potential targets for combination therapy

Tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) is an endogenous secreted peptide and, in preclinical studies, preferentially induces apoptosis in tumor cells rather than in normal cells. The acquisition of resistance in cells exposed to TRAIL or its mimics limits their clinical efficacy. Because kinases are intimately involved in the regulation of apoptosis, we systematically characterized kinases involved in TRAIL signaling. Using RNA interference (RNAi) loss-of-function and cDNA overexpression screens, we identified 169 protein kinases that influenced the dynamics of TRAIL-induced apoptosis in the colon adenocarcinoma cell line DLD-1. We classified the kinases as sensitizers or resistors or modulators, depending on the effect that knockdown and overexpression had on TRAIL-induced apoptosis. Two of these kinases that were classified as resistors were PX domain-containing serine/threonine kinase (PXK) and AP2-associated kinase 1 (AAK1), which promote receptor endocytosis and may enable cells to resist TRAIL-induced apoptosis by enhancing endocytosis of the TRAIL receptors. We assembled protein interaction maps using mass spectrometry-based protein interaction analysis and quantitative phosphoproteomics. With these protein interaction maps, we modeled information flow through the networks and identified apoptosis-modifying kinases that are highly connected to regulated substrates downstream of TRAIL. The results of this analysis provide a resource of potential targets for the development of TRAIL combination therapies to selectively kill cancer cells.
Kinome-wide Decoding of Network-Attacking Mutations Rewiring Cancer Signaling

Cancer cells acquire pathological phenotypes through accumulation of mutations that perturb signaling networks. However, global analysis of these events is currently limited. Here, we identify six types of network-attacking mutations (NAMs), including changes in kinase and SH2 modulation, network rewiring, and the genesis and extinction of phosphorylation sites. We developed a computational platform (ReKINect) to identify NAMs and systematically interpreted the exomes and quantitative (phospho-)proteomes of five ovarian cancer cell lines and the global cancer genome repository. We identified and experimentally validated several NAMs, including PKCy M501I and PKD1 D665N, which encode specificity switches analogous to the appearance of kinases de novo within the kinome. We discover mutant molecular logic gates, a drift toward phospho-threonine signaling, weakening of phosphorylation motifs, and kinase-inactivating hotspots in cancer. Our method pinpoints functional NAMs, scales with the complexity of cancer genomes and cell signaling, and may enhance our capability to therapeutically target tumor-specific networks.

General information

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Organisations: Department of Systems Biology, Cellular Signal Integration, University of Copenhagen, Yale School of Medicine, University of Zurich, University of Rome Tor Vergata, Tottori University
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CoreFlow: A computational platform for integration, analysis and modeling of complex biological data

A major challenge in mass spectrometry and other large-scale applications is how to handle, integrate, and model the data that is produced. Given the speed at which technology advances and the need to keep pace with biological experiments, we designed a computational platform, CoreFlow, which provides programmers with a framework to manage data in real-time. It allows users to upload data into a relational database (MySQL), and to create custom scripts in high-level languages such as R, Python, or Perl for processing, correcting and modeling this data. CoreFlow organizes these scripts into project-specific pipelines, tracks interdependencies between related tasks, and enables the generation of summary reports as well as publication-quality images. As a result, the gap between experimental and computational components of a typical large-scale biology project is reduced, decreasing the time between data generation, analysis and manuscript writing. CoreFlow is being released to the scientific community as an open-sourced software package complete with proteomics-specific examples, which include corrections for incomplete isotopic labeling of peptides (SILAC) or arginine-to-proline conversion, and modeling of multiple/selected reaction monitoring (MRM/SRM) results. Biological significanceCoreFlow was purposely designed as an environment for programmers to rapidly perform data analysis. These analyses are assembled into project-specific workflows that are readily shared with biologists to guide the next stages of experimentation. Its simple yet powerful interface provides a structure where scripts can be written and tested virtually simultaneously to shorten the life cycle of code development for a particular task. The scripts are exposed at every step so that a user can quickly see the relationships between the data, the assumptions that have been made, and the manipulations that have been performed. Since the scripts use commonly available programming languages, they can easily be transferred to and from other computational environments for debugging or faster processing. This focus on ‘on the fly’ analysis sets CoreFlow apart from other workflow applications that require wrapping of scripts into particular formats and development of specific user interfaces. Importantly, current and future releases of data analysis scripts in CoreFlow format will be of widespread benefit to the proteomics community, not only for uptake and use in individual labs, but to enable full scrutiny of all analysis steps, thus increasing experimental reproducibility and decreasing errors.

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BFI (2018): BFI-level 1
Web of Science (2018): Indexed yes
BFI (2017): BFI-level 1
Scopus rating (2017): CiteScore 3.85 SJR 1.43 SNIP 0.982
Web of Science (2017): Impact factor 3.722
Web of Science (2017): Indexed yes
BFI (2016): BFI-level 1
Scopus rating (2016): CiteScore 4.05 SJR 1.421 SNIP 1.049
Web of Science (2016): Impact factor 3.914
Web of Science (2016): Indexed yes
BFI (2015): BFI-level 1
Scopus rating (2015): CiteScore 4.09 SJR 1.506 SNIP 1.132
Web of Science (2015): Impact factor 3.867
Web of Science (2015): Indexed yes
BFI (2014): BFI-level 1
Scopus rating (2014): CiteScore 4.02 SJR 1.367 SNIP 1.115
Web of Science (2014): Impact factor 3.888
Web of Science (2014): Indexed yes
BFI (2013): BFI-level 1
Scopus rating (2013): CiteScore 4.23 SJR 1.311 SNIP 1.018
Web of Science (2013): Impact factor 3.929
ISI indexed (2013): ISI indexed yes
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BFI (2012): BFI-level 1
Scopus rating (2012): CiteScore 4.29 SJR 1.231 SNIP 1.166
Web of Science (2012): Impact factor 4.088
ISI indexed (2012): ISI indexed yes
Web of Science (2012): Indexed yes
BFI (2011): BFI-level 1
Scopus rating (2011): CiteScore 4.81 SJR 1.23 SNIP 1.191
Web of Science (2011): Impact factor 4.878
ISI indexed (2011): ISI indexed no
BFI (2010): BFI-level 1
Scopus rating (2010): SJR 1.03 SNIP 1.051
Web of Science (2010): Impact factor 5.074
BFI (2009): BFI-level 1
Scopus rating (2009): SJR 0.785 SNIP 0.949
BFI (2008): BFI-level 1
Scopus rating (2008): SJR 0.739 SNIP 0.856
Scopus rating (2007): SJR 0.51 SNIP 0.755
Scopus rating (2006): SJR 0.618 SNIP 0.679
Scopus rating (2005): SJR 0.525 SNIP 0.766
Scopus rating (2004): SJR 0.697 SNIP 1.001
Scopus rating (2003): SJR 0.615 SNIP 0.762
Scopus rating (2002): SJR 0.801 SNIP 0.511
Scopus rating (2001): SJR 0.469 SNIP 0.521
Scopus rating (2000): SJR 0.392 SNIP 0.39
Scopus rating (1999): SJR 0.379 SNIP 0.605

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Identification of Hypoxia-Regulated Proteins Using MALDI-Mass Spectrometry Imaging Combined with Quantitative Proteomics

Hypoxia is present in most solid tumors and is clinically correlated with increased metastasis and poor patient survival. While studies have demonstrated the role of hypoxia and hypoxia-regulated proteins in cancer progression, no attempts have been made to identify hypoxia-regulated proteins using quantitative proteomics combined with MALDI-mass spectrometry imaging (MALDI-MSI). Here we present a comprehensive hypoxic proteome study and are the first to investigate changes in situ using tumor samples. In vitro quantitative mass spectrometry analysis of the hypoxic proteome was performed on breast cancer cells using stable isotope labeling with amino acids in cell culture (SILAC). MS analyses were performed on laser-capture microdissected samples isolated from normoxic and hypoxic regions from tumors derived from the same cells used in vitro. MALDI-MSI was used in combination to investigate hypoxia-regulated protein localization within tumor sections. Here we identified more than 100 proteins, both novel and previously reported, that were associated with hypoxia. Several proteins were localized in hypoxic regions, as identified by MALDI-MSI. Visualization and data extrapolation methods for the in vitro SILAC data were also developed, and computational mapping of MALDI-MSI data to IHC results was applied for data validation. The results and limitations of the methodologies described are discussed.

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Web of Science (2019): Indexed yes
BFI (2018): BFI-level 2
Web of Science (2018): Indexed yes
BFI (2017): BFI-level 2
Scopus rating (2017): CiteScore 4.16 SJR 1.818 SNIP 0.982
Web of Science (2017): Impact factor 3.95
Web of Science (2017): Indexed yes
BFI (2016): BFI-level 2
Scopus rating (2016): CiteScore 4.34 SJR 1.76 SNIP 1.018
Web of Science (2016): Impact factor 4.268
Web of Science (2016): Indexed yes
BFI (2015): BFI-level 2
Scopus rating (2015): CiteScore 4.45 SJR 1.933 SNIP 1.08
Web of Science (2015): Indexed yes
BFI (2014): BFI-level 2
Scopus rating (2014): CiteScore 4.64 SJR 1.959 SNIP 1.174
Web of Science (2014): Impact factor 4.245
Web of Science (2014): Indexed yes
BFI (2013): BFI-level 2
Scopus rating (2013): CiteScore 5.16 SJR 2.012 SNIP 1.248
Web of Science (2013): Impact factor 5.001
ISI indexed (2013): ISI indexed yes
KinomeXplorer: an integrated platform for kinome biology studies

A letter to the editor is presented related to the KinomeXplorer, an integrated platform providing workflows to efficiently analyze phosphorylation dependent interaction networks or kinase signaling networks.

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Organisations: Department of Systems Biology, Center for Biological Sequence Analysis, Cellular Signal Integration, Systems Biotechnology, Memorial Sloan-Kettering Cancer Center, European Molecular Biology Laboratory, University of Rome Tor Vergata, University of Copenhagen
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BFI (2019): BFI-level 2
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Modeling Cancer Metastasis using Global, Quantitative and Integrative Network Biology

In order to respond to alterations in its environment, a cell has to integrate multiple input-cues and modulate its signaling networks accordingly, in order to elicit a specific response such as proliferation or apoptosis. This process becomes
significantly altered during cancer development, with genomic modifications giving rise to differential protein dynamics, ultimately resulting in disease. The exact molecular signaling networks underlying specific disease phenotypes remain elusive, as the definition thereof requires extensive analysis of not only the genomic and proteomic landscapes within a particular tumor, but also the phenotypic response to perturbations. Thus, there is a critical need for an integrative global approach, which assesses a biological system such as cancer from several molecular aspects in an un-biased fashion. This thesis summarizes the efforts that were undertaken as part of my PhD in an attempt to positively contribute to this fundamental challenge. The thesis is divided into four parts. In Chapter I, we introduce the complexity of cancer, and describe some underlying causes and ways to study the disease from different molecular perspectives. There is a nearly infinite number of biological aspects that would need to be understood to enable comprehensive treatment regimens specific to each patient (i.e. personalized medicine). However, in the approaches outlined in this thesis, we chose metastasis as a key process for interrogating the clinical potential of targeting cancer networks using Network Biology. Technologies key to this, such as Mass Spectrometry (MS), Next-Generation Sequencing (NGS) and High-Content Screening (HCS) are briefly described. In Chapter II, we cover how signaling networks and mutational data can be modeled in order to gain a better understanding of molecular processes which are fundamental to tumorigenesis. In Article 1, we propose a novel framework for how cancer mutations can be studied by taking into account their effect at the protein network level. In Article 2, we demonstrate how global, quantitative data on phosphorylation dynamics can be generated using MS, and how this can be modeled using a computational framework for deciphering kinase-substrate dynamics. This framework is described in depth in Article 3, and covers the design of KinomeXplorer, which allows the prediction of kinases responsible for modulating observed phosphorylation dynamics in a given biological sample. In Chapter III, we move into Integrative Network Biology, where, by combining two fundamental technologies (MS & NGS), we can obtain more in-depth insights into the links between cellular phenotype and genotype. Article 4 describes the proof-of-principle concept of how one can look at DNA mutations and protein dynamics in an integrative fashion. This has, for example, allowed us to investigate how mutations at the DNA level are propagated at the proteome level. Article 5 demonstrates how by taking a global, multi-platform approach, combined with extensive computational analysis, it is possible to gain a better understanding of colorectal cancer metastasis, and obtain potential clinical benefits. Chapter IV briefly summarizes the findings of the thesis and closes by proposing some future directions based on the work that was presented. Overall, the thesis aims to demonstrate the value of deploying several experimental platforms, each studying a different biological aspect, combined with in-depth computational analysis, in order to shed light on the fundamental molecular processes which underlie a complex disease like cancer and provide possible avenues for therapeutic intervention.

**General information**

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**Organisations:** Department of Systems Biology

**Contributors:** Schoof, E., Brunak, S., Linding, R., Erler, J.

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**Modulation of the Chromatin Phosphoproteome by the Haspin Protein Kinase**

Recent discoveries have highlighted the importance of Haspin kinase activity for the correct positioning of the kinase Aurora B at the centromere. Haspin phosphorylates Thr3 of the histone H3 (H3), which provides a signal for Aurora B to localize to the centromere of mitotic chromosomes. To date, histone H3 is the only confirmed Haspin substrate. We used a combination of biochemical, pharmacological, and mass spectrometric approaches to study the consequences of Haspin inhibition in mitotic cells. We quantified 3964 phosphorylation sites on chromatin-associated proteins and identified a Haspin protein-protein interaction network. We determined the Haspin consensus motif and the co-crystal structure of the kinase with the histone H3 tail. The structure revealed a unique bent substrate binding mode positioning the histone H3 residues Arg2 and Lys4 adjacent to the Haspin phosphorylated threonine into acidic binding pockets. This unique conformation of the kinase-substrate complex explains the reported modulation of Haspin activity by methylation of Lys4 of the histone H3. In addition, the identification of the structural basis of substrate recognition and the amino acid sequence preferences of Haspin aided the identification of novel candidate Haspin substrates. In particular, we validated the phosphorylation of Ser137 of the histone variant macroH2A as a target of Haspin kinase activity. MacroH2A Ser137 resides in a basic stretch of about 40 amino acids that is required to stabilize extranucleosomal DNA, suggesting that phosphorylation of Ser137 might regulate the interactions of macroH2A and DNA. Overall, our data suggest that Haspin activity affects the phosphorylation state of proteins involved in gene expression regulation and splicing.

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**Organisations:** Department of Systems Biology, Center for Biological Sequence Analysis, Cellular Signal Integration

Identification of a novel immunoregulatory signaling pathway exploited by M. tuberculosis in dendritic cells

The causative agent of tuberculosis, Mycobacterium tuberculosis, has infected over a third of the world's population and poses a massive burden to health care systems and human well-being. Most M. tuberculosis infections are latent and are not cleared fully by the host immune system due to the highly sophisticated infectious machinery employed by the bacterium. The dendritic cell (DC) plays a crucial role in shaping the nature of the immune response after exposure to pathogens, and the interaction between M. tuberculosis and the dendritic cell is of profound importance for the course of infection. During their interaction, the DC is exposed to multiple M. tuberculosis-derived ligands recognized by a range of pattern recognition receptors, but the result is typically an immune response that is not very effective at clearing the bacteria from the host. The reason why the induced immune response is ineffective at clearing the bacteria is not fully understood, but clues may be given in the signaling pathways induced in DCs upon M. tuberculosis-exposure.
Identification of A Novel Immunoregulatory Signaling Pathway Exploited by Mycobacterium tuberculosis in Dendritic Cells

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Web of Science (2017): Impact factor 2.314
Web of Science (2017): Indexed yes
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Scopus rating (2016): CiteScore 2.03 SJR 0.979 SNIP 0.644
Web of Science (2016): Impact factor 2.256
BFI (2015): BFI-level 1
Scopus rating (2015): CiteScore 1.97 SJR 0.933 SNIP 0.679
Web of Science (2015): Impact factor 2.27
Web of Science (2015): Indexed yes
BFI (2014): BFI-level 1
Scopus rating (2014): CiteScore 1.91 SJR 0.901 SNIP 0.665
Web of Science (2014): Impact factor 1.739
Web of Science (2014): Indexed yes
BFI (2013): BFI-level 1
Scopus rating (2013): CiteScore 2.05 SJR 0.875 SNIP 0.709
In Vivo SILAC-Based Proteomics Reveals Phosphoproteome Changes during Mouse Skin Carcinogenesis

SILAC technology in combination with high-resolution mass spectrometry (MS) can be successfully used to measure phosphoproteomes in vivo. Here, Zanivan, Mann, and colleagues have applied SILAC-based MS to investigate phosphoproteomic changes during skin carcinogenesis, using the DMBA/TPA two-stage mouse model. Using this approach, the authors have revealed the phosphoproteomic dynamics that accompany skin cancer progression and predict specific kinase activities associated with tumor malignancy.

General information
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Organisations: Department of Systems Biology, Center for Biological Sequence Analysis, Beatson Institute for Cancer Research, Mayo College of Medicine, Max Planck Institute, National Institute of Environmental Health Sciences Research, University of Copenhagen
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Mutational properties of amino acid residues: implications for evolvability of phosphorylatable residues

As François Jacob pointed out over 30 years ago, evolution is a tinkering process, and, as such, relies on the genetic diversity produced by mutation subsequently shaped by Darwinian selection. However, there is one implicit assumption that is made when studying this tinkering process; it is typically assumed that all amino acid residues are equally likely to mutate or to result from a mutation. Here, by reconstructing ancestral sequences and computing mutational probabilities for all the amino acid residues, we refute this assumption and show extensive inequalities between different residues in terms of their mutational activity. Moreover, we highlight the importance of the genetic code and physico-chemical properties of the amino acid residues as likely causes of these inequalities and uncover serine as a mutational hot spot. Finally, we explore the consequences that these different mutational properties have on phosphorylation site evolution, showing that a higher degree of evolvability exists for phosphorylated threonine and, to a lesser extent, serine in comparison with tyrosine residues. As exemplified by the suppression of serine's mutational activity in phosphorylation sites, our results suggest that the cell can fine-tune the mutational activities of amino acid residues when they reside in functional protein regions.
Navigating cancer network attractors for tumor-specific therapy.

Cells employ highly dynamic signaling networks to drive biological decision processes. Perturbations to these signaling networks may attract cells to new malignant signaling and phenotypic states, termed cancer network attractors, that result in cancer development. As different cancer cells reach these malignant states by accumulating different molecular alterations, uncovering these mechanisms represents a grand challenge in cancer biology. Addressing this challenge will require new systems-based strategies that capture the intrinsic properties of cancer signaling networks and provide deeper understanding of the processes by which genetic lesions perturb these networks and lead to disease phenotypes. Network biology will help circumvent fundamental obstacles in cancer treatment, such as drug resistance and metastasis, empowering personalized and tumor-specific cancer therapies.

General information
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Organisations: Department of Systems Biology, Center for Biological Sequence Analysis, University of Copenhagen
Contributors: Creixell, P., Schoof, E., Erler, J. T., Linding, R.
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Scopus rating (2017): CiteScore 12.94 SJR 18.252 SNIP 6.062
Web of Science (2017): Impact factor 35.724
Web of Science (2017): Indexed yes
BFI (2016): BFI-level 2
Scopus rating (2016): CiteScore 13.16 SJR 20.666 SNIP 6.42
Web of Science (2016): Impact factor 41.667
Web of Science (2016): Indexed yes
BFI (2015): BFI-level 2
Scopus rating (2015): CiteScore 11.88 SJR 18.263 SNIP 5.553
Web of Science (2015): Impact factor 43.113
Web of Science (2015): Indexed yes
BFI (2014): BFI-level 2
Scopus rating (2014): CiteScore 11.4 SJR 16.609 SNIP 5.37
Web of Science (2014): Impact factor 41.514
Web of Science (2014): Indexed yes
BFI (2013): BFI-level 2
Response to Comment on "Positive Selection of Tyrosine Loss in Metazoan Evolution"

Su et al. claim guanine-cytosine (GC) content variation can largely explain the observed tyrosine frequency variation, independent of adaptive evolution of cell-signaling complexity. We found that GC content variation, in the absence of selection for amino acid changes, can only maximally account for 38% of the observed tyrosine frequency variation. We also uncovered other mechanisms acting to reduce tyrosine phosphorylation that further support our previous proposal.

General information
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Organisations: Center for Biological Sequence Analysis, Department of Systems Biology, Mount Sinai Hospital, University of California
Contributors: Tan, C. S. H., Schoof, E., Creixell, P., Pasculescu, A., Lim, W. A., Pawson, T., Bader, G. D., Linding, R.
Pages: 917
Publication date: 2011
Web of Science (2002): Indexed yes
Scopus rating (2001): SJR 10.987 SNIP 6.94
Web of Science (2001): Indexed yes
Scopus rating (2000): SJR 15.245 SNIP 7.042
Web of Science (2000): Indexed yes
Scopus rating (1999): SJR 16.615 SNIP 7.018
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http://www.sciencemag.org/
Source: orbit
Source-ID: 277591
Research output: Research - peer-review › Journal article – Annual report year: 2011

Projects:

**Immune stealing of bio-therapeutics**
Lie-Andersen, O., PhD Student, Department of Biotechnology and Biomedicine
auf dem Keller, U., Main Supervisor, Department of Biotechnology and Biomedicine
Hansen, M., Supervisor, Department of Micro- and Nanotechnology
Justesen, S. F. L., Supervisor, Department of Systems Biology
Schoof, E., Supervisor, Department of Biotechnology and Biomedicine
Thorgrimsen, S. P., Supervisor
01/01/2019 → 31/12/2021
Project: PhD

**Characterising Phosphorylation Network Dynamics in Colon Cancer Metastasis**
Schoof, E., PhD Student, Department of Biotechnology and Biomedicine
Linding, R., Main Supervisor, Department of Systems Biology
Brunak, S., Supervisor, Department of Biotechnology and Biomedicine
Erler, J. T., Supervisor
Molin, S., Examiner, Department of Biotechnology and Biomedicine
Abersold, R., Examiner
Sansom, O., Examiner
Abersold, R., Examiner
Sansom, O., Examiner
Institut stipendie (DTU)
01/02/2011 → 04/06/2014
Award relations: Characterising Phosphorylation Network Dynamics in Colon Cancer Metastasis
Project: PhD