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Punit Kaur, Ph.D.

Assistant Professor
Department of Microbiology, Biochemistry & Immunology

Department of Pathology and Anatomy

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Dr Punit Kaur Biography

Dr Kaur obtained a Bachelor of Science (B.Sc.) in 1999 from Government College for Girls, Chandigarh, India majoring Chemistry, Zoology and Botany. Dr Kaur was awarded a M.Sc. degree in Microbiology in 2001 from Guru Nanak Dev University majoring Microbiology, Genetics and Biochemistry. Dr Kaur's studies demonstrated that the soil samples from various sites in Amritsar, India showed the presence of actinomyctes. Dr Kaur was awarded a Ph.D. degree from Post Graduate Institute of Medical Education and Research (PGIMER), the largest institute for patient care and medical research in Chandigarh, India. During this time, Dr Kaur identified and characterized acid-shock induced outer membrane proteins (ASP) in Enteroaggregative Escherichia coli (EAEC), and elucidated the mechanisms by which these bacteria adapt and grow in the acid shock environment of stomach. Dr Kaur demonstrated for the first time the stress proteome in EAEC and found that these acid-shock proteins show cross reactivity with Heat Shock Proteins (HSP). The putative ASP with cell surface associated with an acid-tolerant phenotype provides new information regarding EAEC pathogenesis in humans.Dr Kaur has published 25 scientific articles, book chapters and reviews. Dr Kaur's studies have been selected for poster presentations at 30 national and international conferences. More recently, Dr Kaur has been selected as an invited speaker to 6 international and national conferences. Dr Kaur has played major role in the advisory responsibilities in undergraduate (15), international undergraduate (53), graduate (2) postdoctoral (3) and medical residents (1) training, and in teaching at Texas A&M Health Science Center College of Medicine, Scott and White Hospital and Clinic, Temple College, Temple, TX and Morehouse School of Medicine, Atlanta, GA.

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Dr Punit Kaur Research Interest

- Cancer Research
- Hyperthermia
- Nanotechnology
- Neuroscience
- Infectious Diseases
- Medicinal Plants
- Proteomics

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Recent Publications Authored by Dr Punit Kaur

Asea A, Kaur P, Panossian A, Wikman G. Evaluation of molecular chaperones Hsp72 and neuropeptide Y as characteristic markers of adaptogenic activity of plant extracts. Phytomedicine 2013; 3: S0944-7113(13)00245-6. PMID: 23920279.

Kaur P, Nagaraja GM, Zheng H, Gizachew D, Galukande M, Krishnan S, Asea A. A mouse model for triple-negative breast cancer tumor-initiating cells (TNBC-TICs) exhibits similar aggressive phenotype to the human disease. BMC Cancer 2012; 12:120-128. PMID: 22452810.

Panossian A, Wikman G, Kaur P, Asea A. Adaptogens stimulate neuropeptide Y and Hsp72 expression and release in neuroglia cells. Front Neurosci 2012; 6: 1-12. PMID: 22347152.

Nagaraja GM, Kaur P, Neumann W, Asea EE, Bausero MA, Multhoff G, Asea A. Silencing Hsp25/Hsp27 gene expression augments proteasome activity and increases CD8+ T cell-mediated tumor killing and memory responses. Cancer Prev Res (Phila) 2012; 5 (1):122-137 (Figure featured on journal cover). PMID: 22185976.

Kaur P, Hurwitz MD, Krishnan S, Asea A. Combined hyperthermia and radiotherapy for the eradication of cancer. *Cancers* 2011; 3: 3799-3823. PMID: 24213112.

Kaur P, Asea A. Quantitation methods for heat shock proteins in clinical samples using mass spectrometry. Methods Mol Biol 2011; 787:165-188. PMID: 21898236.

Kaur P, Nagaraja GM, Asea A. Combined lentiviral and RNAi technologies for the delivery and permanent silencing of the hsp25 gene. Methods Mol Biol 2011; 787:121-136. PMID: 21898232.

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A Mouse Model for Triple-Negative Breast Cancer Stem Cells (TNBC-CSC) Exhibits an Aggressive Phenotype

Editorial Board Member
Journal of Proteomics and Bioinformatics

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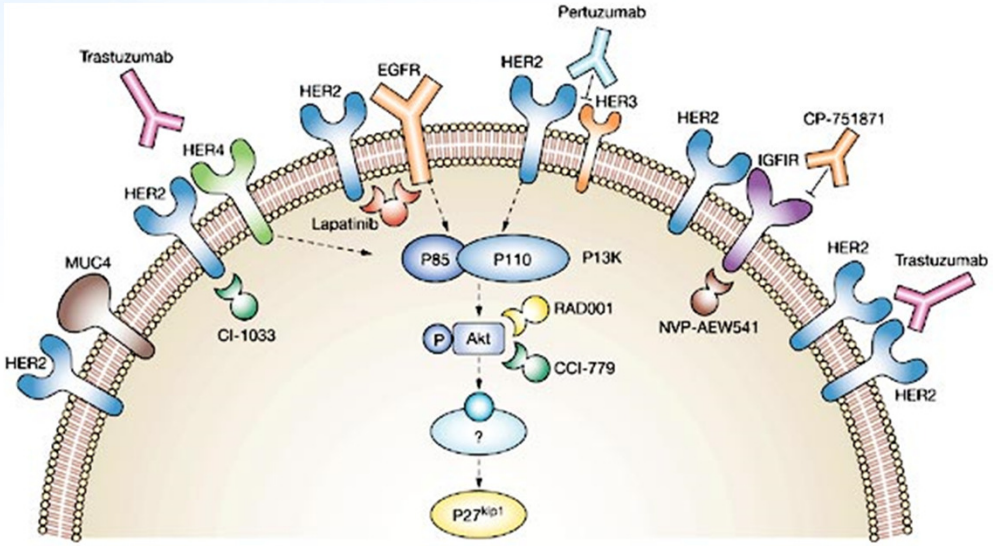
Triple-Negative Breast Cancer (TNBC)

Recent studies on surface receptors and gene expression of breast tumors have come up with a term triple-negative breast cancer (TNBC).

The disease gets its name because of testing negative for:

Estrogen Receptor (ER)
Progesterone Receptor (PgR)
Human Epidermal Growth Factor Receptor 2 (HER2) gene



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Triple-Negative Breast Cancer (TNBC)

Despite lower incidence and the steady improvement in screening, African-American and Hispanic women are more likely to die of triple-negative breast cancer than Caucasian women.

DOI: 10.1093/jnci/djq215
Advance Access publication on June 23, 2010.

ARTICLE

Race and Ethnicity and Breast Cancer Outcomes in an Underinsured Population

Ian K. Komenaka, Maria Elena Martinez, Robert E. Pennington Jr, Chiu-Hsieh Hsu, Susan E. Clare, Patricia A. Thompson, Colleen Murphy, Noelia M. Zork, Robert J. Goulet Jr

Manuscript received August 28, 2009; revised April 26, 2010; accepted M

Correspondence to: Ian K. Komenaka, MD, Department of Surgery, Wishard Memor
(e-mail: komenska@hotmail.com).

American Journal of Epidemiology

Vol. 162, No. 8
DOI: 10.1093/aje/kwi278
Advance Access publication August 24, 2005

Original Contribution

Risk Factors for Fatal Breast Cancer in African-American Women and White Women in a Large US Prospective Cohort

Marjorie L. McCullough, Heather Spencer Feigelson, W. Ryan Diver, Alpa V. Patel, Michael J. Thun, and Eugenia E. Calle

From the Department of Epidemiology and Surveillance Research, American Cancer Society, Atlanta, GA.

Received for publication March 28, 2005; accepted for publication June 1, 2005.

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Triple-Negative Breast Cancer (TNBC)

TN-BC ($HER2^-/ER^-/PgR^-$) - Creates a disease quite distinct from that seen in Caucasian women ($HER2^+/ER^+/PgR^+$), and is a much more aggressive disease without tumor-specific treatment options and accounts for 15% of all types of breast cancer with higher percentages in premenopausal African American and Hispanic women.

Although slightly responsive to chemotherapy, TNBC is more difficult to treat and generally insensitive to most available hormonal or targeted therapeutic agents.

Depending on its stage of diagnosis, TNBC can be extremely aggressive - recurring and metastasizing more often than other subtypes of breast cancer.

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Research Plan

Elevated heat shock protein (HSP) levels have been found in various tumors including breast, prostate, pancreatic, gastric, uterine, ovarian, head and neck cancer and also cancers arising from the nervous system and urinary system.

High expression of Hsp27 and Hsp72 is one of the reasons for TNBC aggressiveness.

This area of research has direct implications for clinical and patient care because understanding fundamental mechanisms by which combined HT and RT induces the killing of TNBC can be directly implemented for the treatment of patients with TNBC.

My goal is to understand the molecular mechanisms by which combined hyperthermia (HT) and radiotherapy (RT) induces the killing of TNBC cells.

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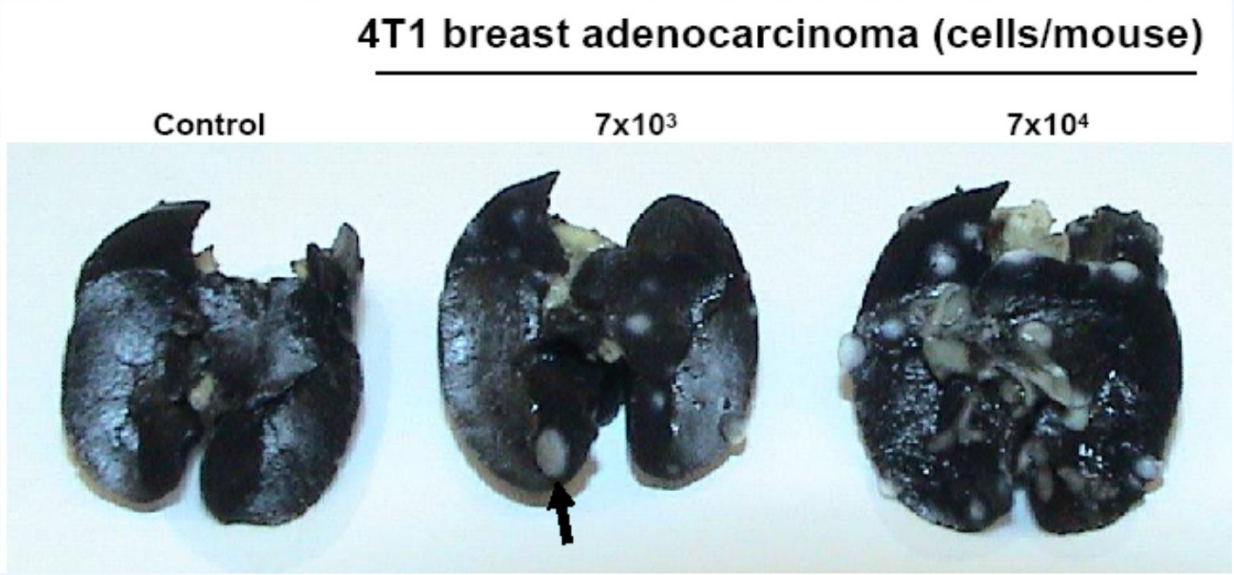
In vivo TNBC Tumor Model



Murine breast carcinoma 4T1 cells are a 6-thioguanine-resistant cell line selected from 410.4 tumor without mutagen treatment. When injected into the abdominal breast gland of female BALB/c mice (8–12 weeks old), 4T1 spontaneously produce highly metastatic tumors that can metastasize to the lung, liver, lymph nodes and brain while the primary tumor is growing in situ. The primary tumor does not have to be removed to induce metastatic growth. The tumor growth and metastatic spread of 4T1 cells in BALB/c mice very closely mimic human Stage IV breast cancer.

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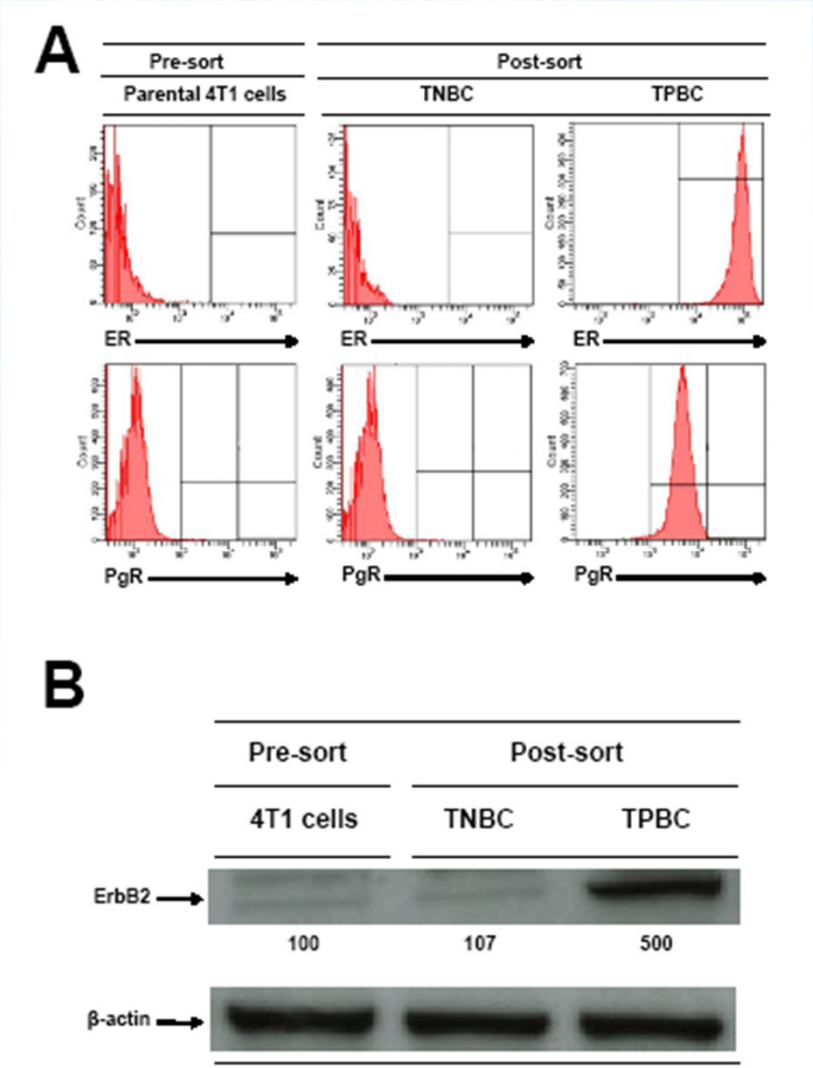
Transfection of Rat HER2, ER and PgR Genes to Produce TNBC Population



Kaur *et al.*, BMC Cancer, 2012

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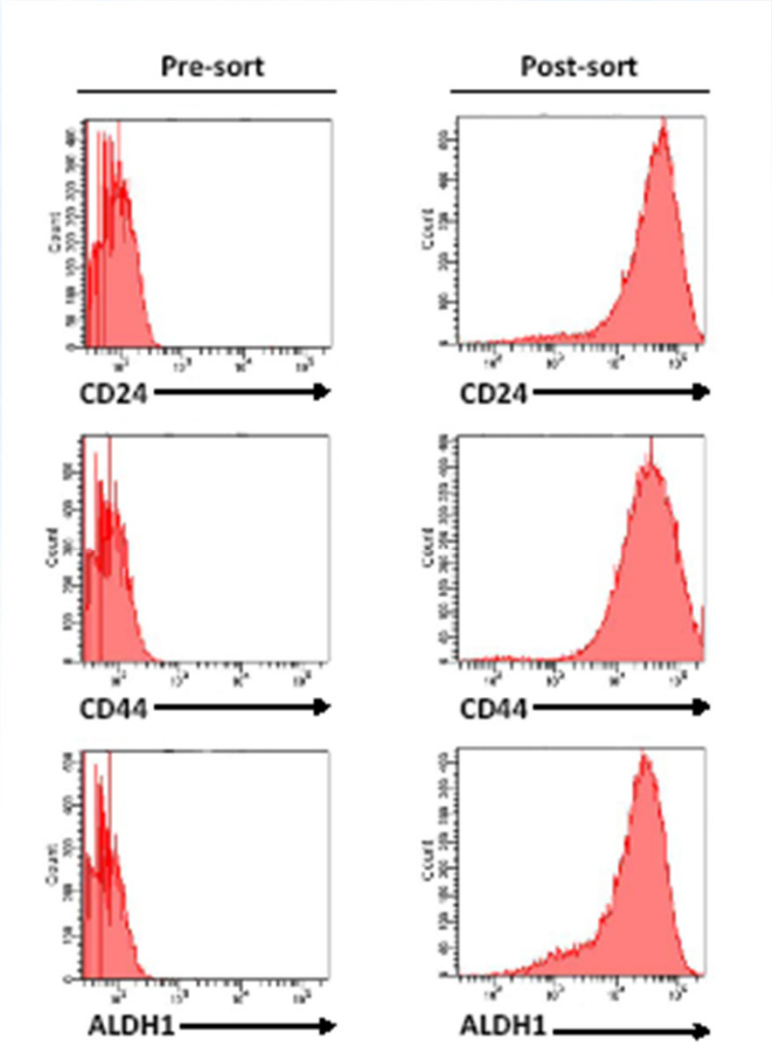
Selection of TNBC-Tumor Initiating Cells (TNBC-TICs)



Kaur *et al.*, BMC Cancer, 2012

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Higher Tumor Growth Potential in TNBC-TICs

Table 1. TNBC-TICs exhibit a greater clonogenic growth potential as compared to TNBC, TPBC-TICs, TPBC or parental 4T1 cells.

Cell type[1]	Number of cells transplanted	Fraction of mice with tumors[2]
TPBC-derived:		
$CD24^{+}/ALDH1^{+}/CD44^{high}$	500	5/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	100	5/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	50	4/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	25	2/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	10	3/5
TPBC-derived:		
$CD24^{+}/ALDH1^{+}/CD44^{low}$	500	1/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	100	0/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	50	0/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	25	0/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	10	0/5
TNBC-derived:		
$CD24^{+}/ALDH1^{+}/CD44^{high}$	500	5/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	100	5/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	50	5/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	25	5/5
$CD24^{+}/ALDH1^{+}/CD44^{high}$	10	4/5
TNBC-derived:		
$CD24^{+}/ALDH1^{+}/CD44^{low}$	500	1/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	100	0/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	50	0/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	25	0/5
$CD24^{+}/ALDH1^{+}/CD44^{low}$	10	0/5

[1]Tumor cells derived from TPBC or TNBC cells were stably transfected with eGFP using the lentivirus transduction technique as described in detail in the Methods section.

[2]Twenty-one days post tumor cell transplantation into the mammary pad of naïve female BALB/c mice, animals were scarified and eGFP signal from the tumors were measured using the Maestro™ *in vivo* imaging system (CRI), and spectral unmixing was performed to segregate skin and hair auto fluorescence and to measure the true eGFP signal, as described in detail in the Methods section. Data is the fraction of mice with tumors (n=5).

Kaur *et al.*, BMC Cancer, 2012

Confidence Interval (95%) for Re-populating Mammary Cell Frequency

Table 2. Confidence interval (95%) for repopulating mammary cell frequency.

Cell type	Confidence intervals for 1/(TICs frequency)[1]		
	Lower	Estimate	Upper
TPBC-derived:			
$CD24^{+}/ALDH1^{+}/CD44^{high}$	51.0	27.02	14.4
$CD24^{+}/ALDH1^{-}/CD44^{low}$	22124.7	3168.93	454.3
TNBC-derived:			
$CD24^{+}/ALDH1^{+}/CD44^{high}$	14.9	6.22	2.8
$CD24^{+}/ALDH1^{-}/CD44^{low}$	22124.7	3168.93	454.3

[1]Confidence interval (95%) for repopulating mammary cell frequency for the data in Table 1. Data were generated using ELDA, a software application for limiting dilution analysis (LDA) as previously described [34].

Kaur *et al.*, BMC Cancer, 2012

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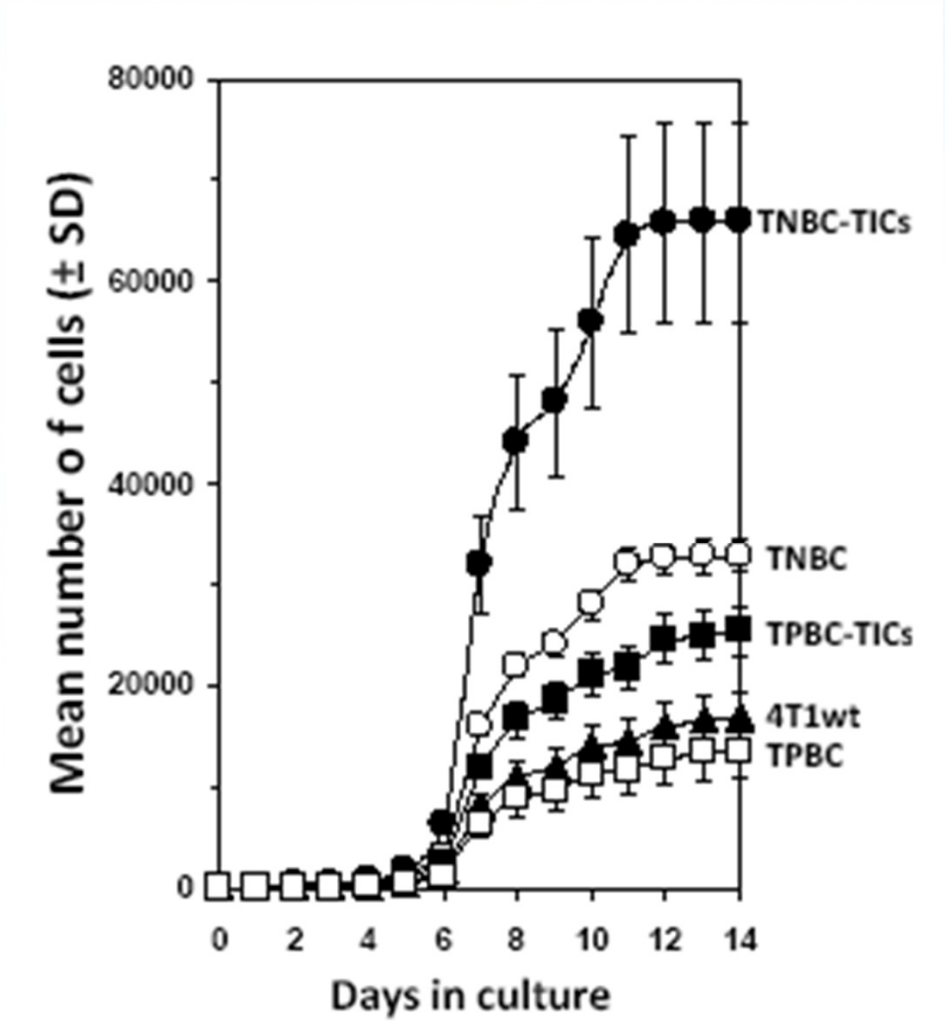
Fast Cell Proliferation in TNBC-TICs



Kaur *et al.*, BMC Cancer, 2012

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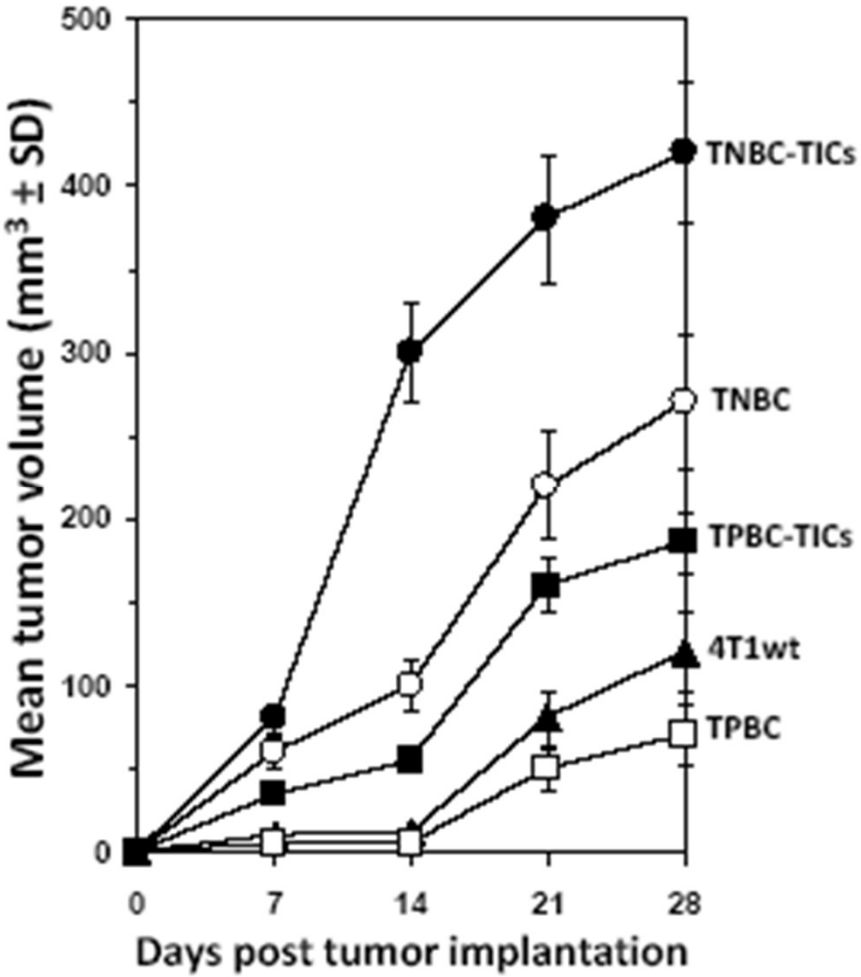
Larger Tumor Growth in TNBC-TICs



Kaur *et al.*, BMC Cancer, 2012

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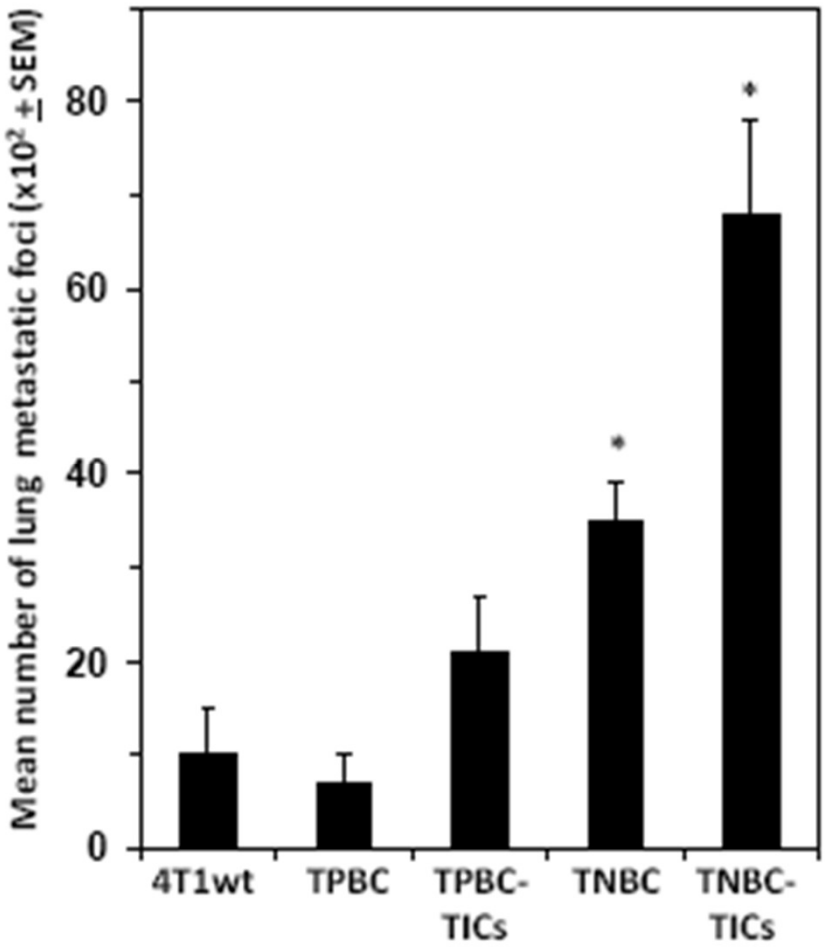
Production of More Metastatic Foci in TNBC-TICs



Kaur *et al.*, BMC Cancer, 2012

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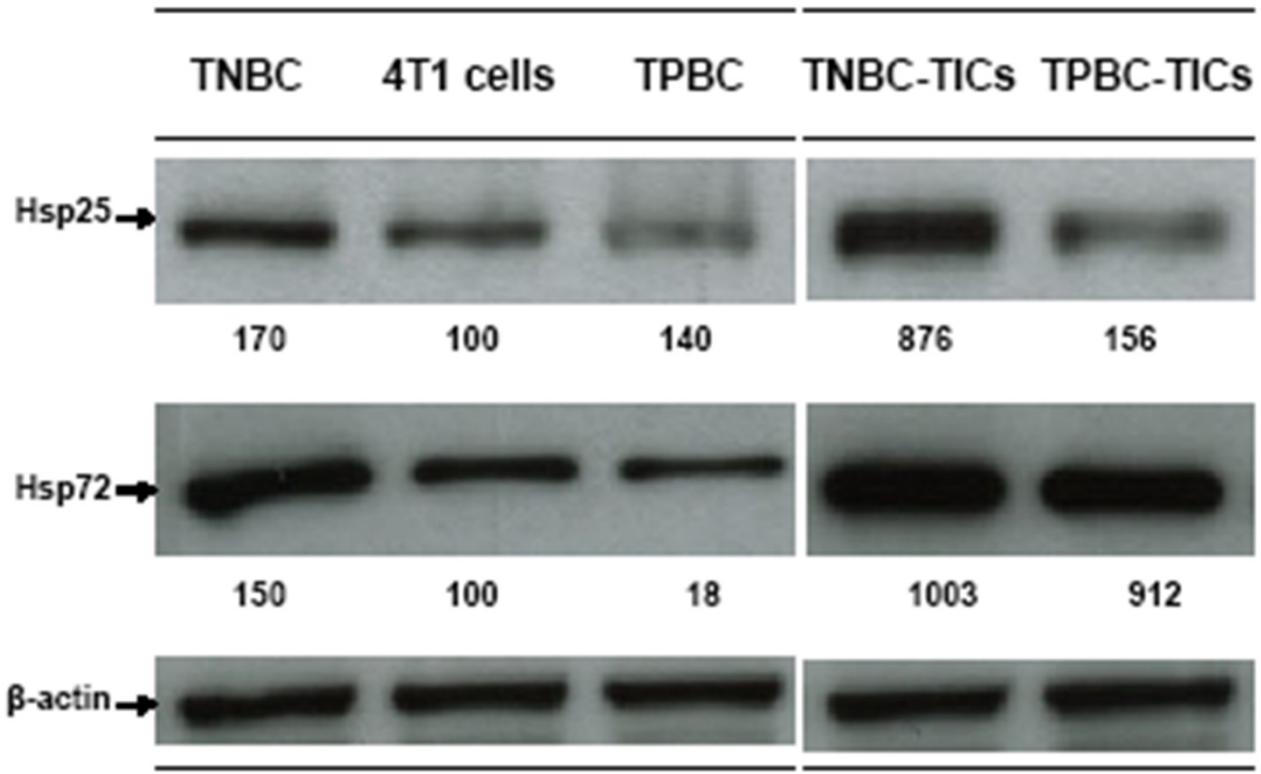
Higher Expression of Hsp25 and Hsp72/HspA1A in TNBC-TICs



Kaur *et al.*, BMC Cancer, 2012

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Summary

We have produced a TNBC model that can be used to study why human TNBC is so aggressive and will help in design of new targets to treat TNBC.

TNBC-TICs have:

- **$CD24^{+}/ALDH1^{+}/CD44^{high}$**
- **High proliferation rate**
- **High tumor growth rate**
- **High metastatic potential**
- **Higher expression of Hsp25**
- **Higher expression of Hsp72**

Kaur *et al.*, BMC Cancer, 2012

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Proteomics in Biomarker Discovery

Proteome is a complete complement of proteins that make up a cell or tissue:

- **Study of protein expression, regulation, modification and function in living systems for understanding how living systems use proteins**

- **Using a variety of techniques, proteomics can be used to study how proteins interact within a system or how proteins change due to applied stresses**

- **Proteomics requires the use of advanced measurement techniques with an emphasis on separations and mass spectrometry**

Mass spectrometry measures the mass of molecules by determining the mass to charge ratios (m/z)

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Obesity and Diabetes

Obesity is a complex disease and is a known risk factor for a variety of chronic diseases including:

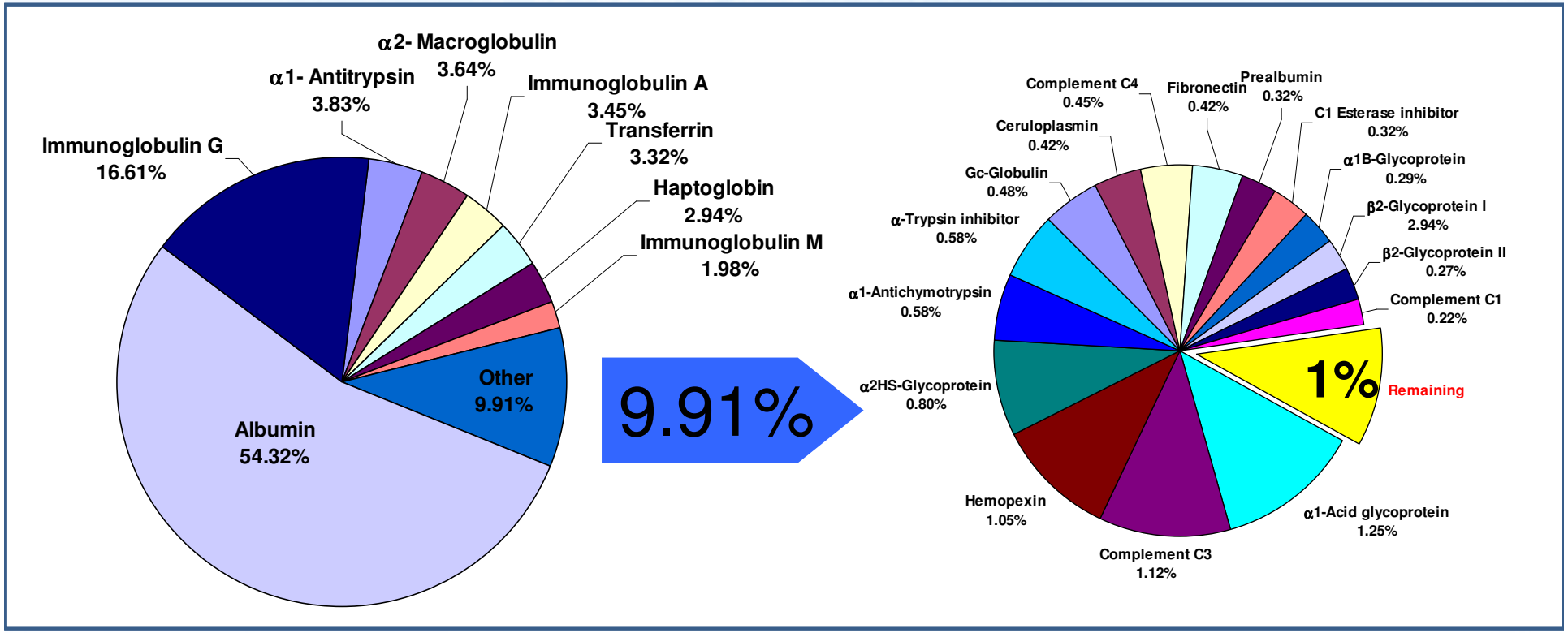
- **Hypertension**
- **Coronary Heart Disease**
- **Type 2 Diabetes**

Over the past decade obesity has become a major public health problem in most industrialized nations and the costs associated with the management of obesity and obesity-related diseases account for about 5% of total healthcare expenditures in most industrialized nations.

Body mass index (BMI) has been demonstrated to correlate well with fat mass, morbidity and mortality, and effects obesity-related disease risk.

Obese individuals have a greater than 10-fold increased risk of developing type 2 diabetes as compared to normal weight individuals.

Removal of High Abundant Proteins from Blood



Concentration range of proteins: 1 to 10^{12}

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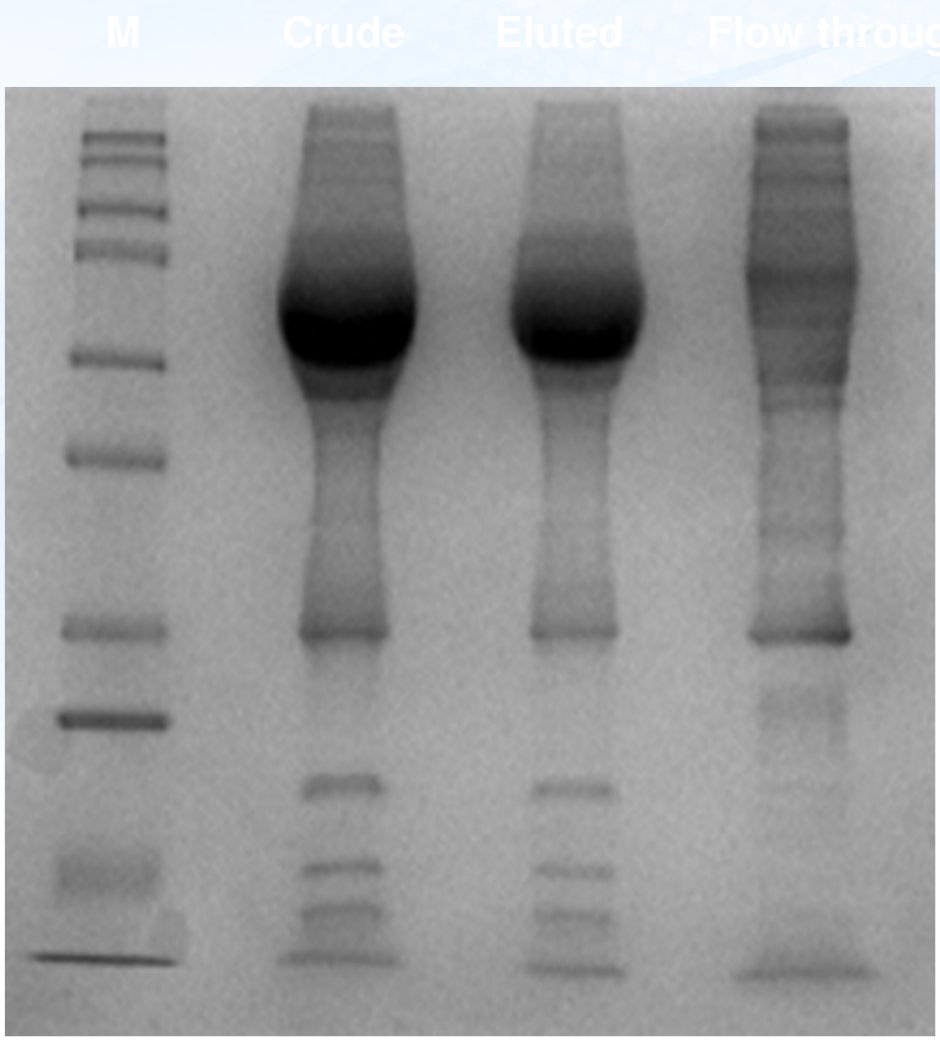
Depletion of High Abundant Proteins



Immunodepletion of plasma sample with affinity binding to column

Kaur *et al.*, J Prot Bioinform, 2010

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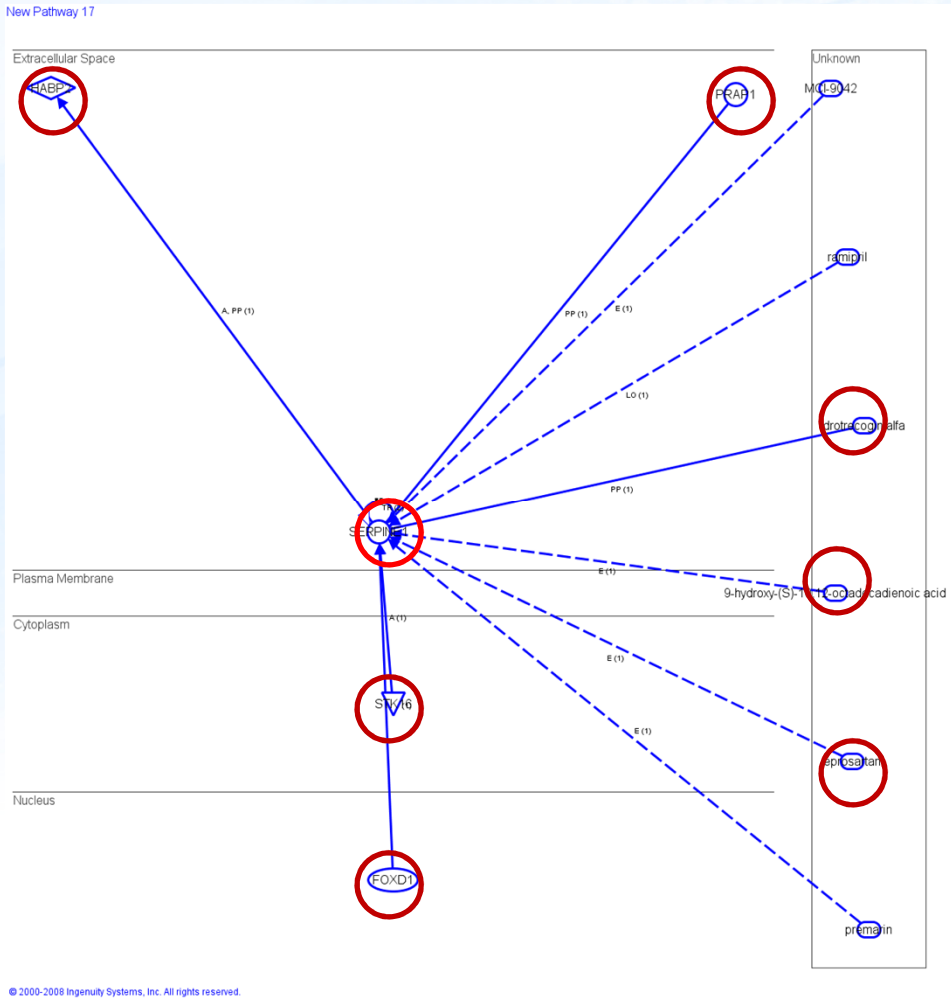
Canonical Pathways for Obesity



•**FOXD1, a transcription regulator, found in the cell nucleus directly acts on SERPINE1, which then activates the peptidase HABP2 and translocate to the kinase STK16.**

•**Drugs used for obesity including drotrecogin alfa was shown to directly bind to SERPINE1, whereas 9-hydroxy-(S)-10, 12-octadecadiencic acid indirectly acts on SERPINE1. Eprosartan drug used in both obesity and hypertension, were shown to indirectly act on SERPINE1.**

•**PRPA1, a protein known to play an important role in cancer was also shown to directly act on SERPINE1.**

Kaur *et al.*, J Prot Bioinform, 2010

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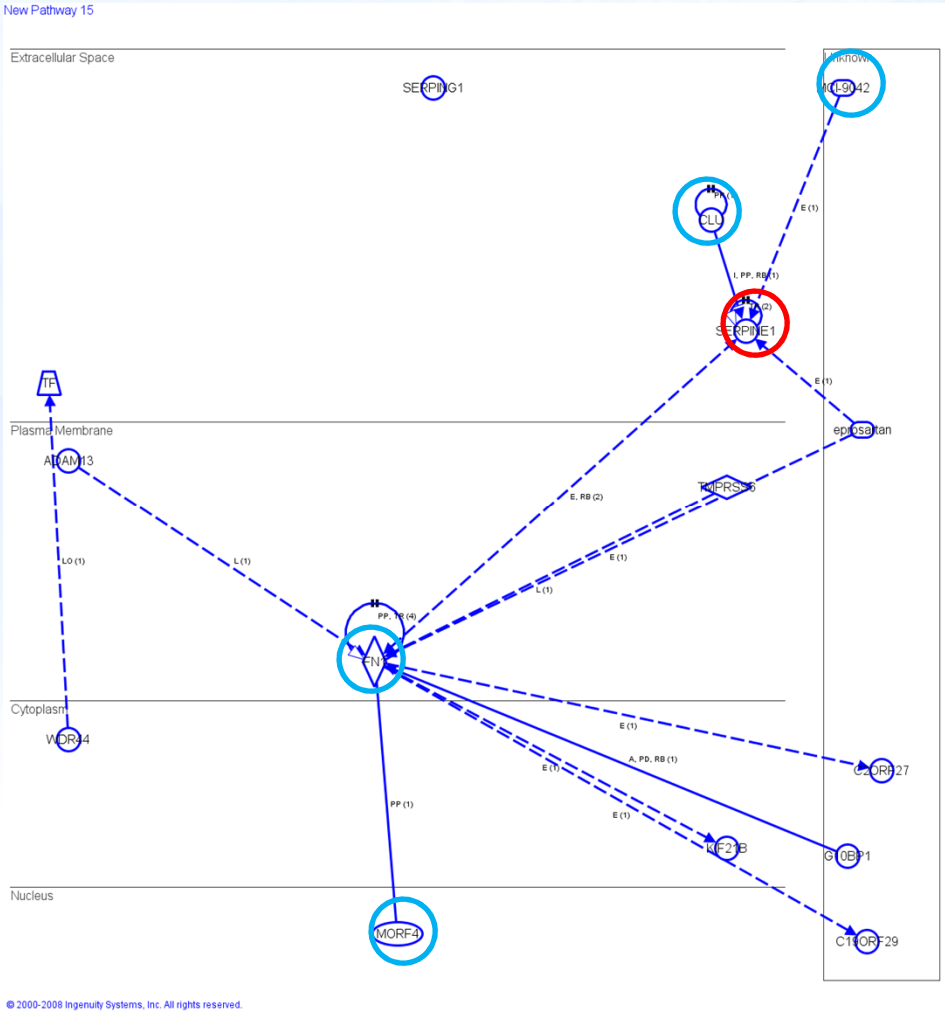
Canonical Pathways for Diabetes



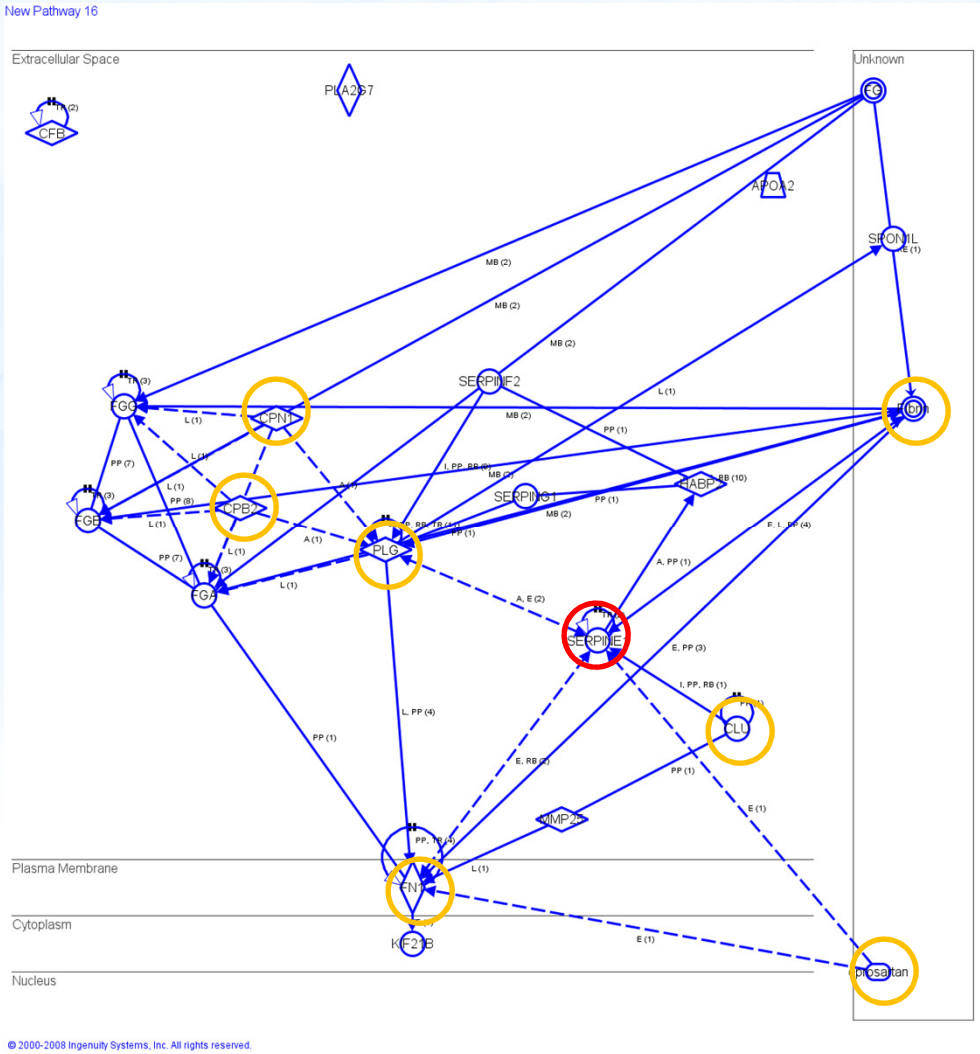
- **Protein CLL found in the extracellular space directly acts on SERPINE1.**

- **SERPINE1 in turn indirectly acts on the enzyme FN1, found in the plasma membrane, which is binds to the transcription regulators MORF4.**

- **The diabetic drugs MCL-9042 and eprostartan indirectly act on SERPINE1.**

Kaur *et al.*, J Prot Bioinform, 2010

Canonical Pathways for Cardiovascular Diseases



- The enzyme, FN1, and the peptidase, PLG, both known to play an important role in cardiovascular disease indirectly act on SERPINE1.
- Fibrin was found to directly act on SERPINE1.
- Epipsartan, was also shown to indirectly act on SERPINE1 in the cardiovascular disease pathway.
- CLL inhibits and acts on SERPINE1.
- Peptidases CPB2 and CPN1 were shown to indirectly act on SERPINE1 through PLG.

Kaur *et al.*, J Prot Bioinform, 2010

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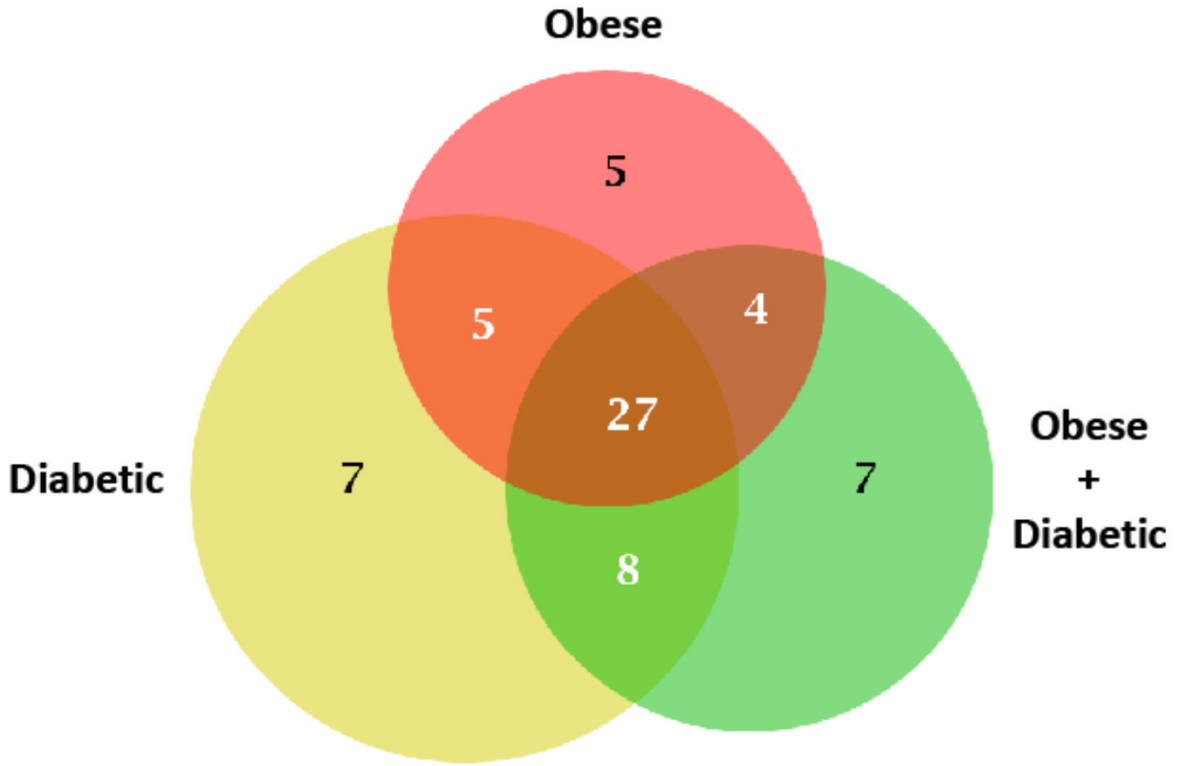
Venn Diagram for Protein Expression in Obesity and Diabetics



Kaur *et al.*, J Prot Bioinform, 2010

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Summary

Our data demonstrates a first step in understanding a link between diabetes and obesity.

Our ultimate goal of using mass spectrometry was to perform rapid screening of a large number of clinical samples, to identify the proteins that change in their relative abundance due to a particular disease for early detection.

This study is a proof-of-concept of protein profiling to investigate the usefulness of the immunodepletion approach for the clinical proteomics.

This study with small sample set is not statistically significant it is a step ahead to explore the some candidate protein profiles associated with significant changes in blood plasma proteins of obese and diabetic patients.

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