營養基因體學之介紹與應用

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研究領域

罕見疾病 / 敗血症 / 營養生化指標分析 / 保健食品專利

本研究室已陸續建立不同生物技術平台，以支援及進行不同研究計畫：**第一爲人類罕見疾病分子檢測平台**，可提供肝醣貯存症 Glycogen storage disease，GSD）第四型，神經纖維瘤（Neurofibromatosis）第 I 及 II 型 ，馮希伯-林島氏症 von Hippel-Lindau 症突變基因篩選；**第二爲蛋白質體學平台**，目前以二維膠體電泳及質譜法（Two-dimensional gel electrophoresis、MALDI-TOF）比較檢體差異展現蛋白質群；**第三爲 HPLC-高效能液相層析儀平台**，可快速分析患者血漿或尿液樣品，維生素 B2、B6、A、D、E 及 Beta-胡蘿蔔素含量。**第四爲細胞培養平台**，可培養不同型式初代及融合細胞，目前涵蓋主題包括「GSD-肝醣貯存症」、「Sepsis-敗血症」、「國人營養生化指標分析計畫」、「專利地圖分析」、「代謝症候群」等。

A. 罕見疾病基因及蛋白質體學研究

本研究室已建立患者皮膚纖維母細胞（ Fibroblast ）培養及 DNA 萃取、白血球（WBC）細胞 DNA 萃取流程，並針對標靶基因設計專一性引子進行 Genomic PCR、PCR 產物回收及核酸定序比對。肝醣貯存症（GSD）之分子生物學研究方面，檢體由杜克醫學大學醫學中心提供 GSD 患者 Fibroblast 細胞，結合北醫大、中研院生醫所陳垣崇所長實驗室已建立之分子檢測平台，進行 GSD 患者突變基因檢測。神經纖維瘤之分子遺傳檢測方面，由國泰醫院醫師提供 NF 病人血液檢體，進行 family base 之 NF1 及 NF2 突變基因篩選及蛋白質體學研究，以找尋國人 NF 基因突變點處及患者血漿是否有其它蛋白質之病理標記。

B. 維生素與敗血症機制探討

行政院衛生署民國九十五年的死亡原因統計報告中指出，敗血症已躍升至國人主要死因的第十二位，通常是加護病房患者的死亡主要原因。敗血症（sepsis）主要是由於細菌感染所引起的症狀，釋放至血液中的毒素會引起全身性嚴重的發炎反應，會導致體內細胞激素、腫瘤壞死因子等的產生，一連串激活了補體、白血球和血管內皮細胞等，最後常導致患者血液凝結、多重器官衰竭及休克死亡。本研究室 2006-2007 年藉由注射內毒素誘發小鼠產生敗血症後，敗血症小鼠注射高濃度水溶性維生素可提高敗血症小鼠的存活率，暗示了某些水溶性維生素可能有助於敗血症患者的輔助醫療。2007-2008 年與北醫附醫感染室合作的研究發現，加護病房（ICU）敗血症存活者及死亡者與血漿維生素濃度高低有關，因此以 HPLC 法進行患者血漿維生素的分析，將有助於敗血症病因之瞭解、病程之監控與營養輔助醫療。蛋白質體學 (Proteomics)是近年來被廣泛應用於生命科學的一種研究方法，由於敗血症成因複雜，又是 ICU 病房患者死亡的最主要原因，因此本研究室利用此一利器，分析患者血漿找到的特定蛋白質群，能否成爲診斷 sepsis 生物標記的潛力。

C. 國人營養生化指標分析

「國人營養生化指標分析計畫」，其研究目標爲以客觀具效力的生化指標評估國人營養狀況，以達到有效掌握國人飲食、營養與健康狀況，及瞭解國人營養攝取情形。營養生化指標是最客觀的評估國人營養狀況方法之一，可直接了解整體國人之健康狀況，亦可評估特殊族群如婦女、青少年、老人、弱勢族群及偏遠地區與其他族群、都會區之差異性。本研究進行水溶性及脂溶性維生素生化指標分析，並且透過比較新舊分析方法之差異性，並制定國民營養狀況標準生化分析方法。

D. 食品專利地圖分析

食品專利地圖（Food Patent Map）係指透過專利檢索技巧，檢索出與保健食品相關之專利資料，並以統計分析之方法，加以縝密及精細之剖析整理製成各種可分析、解讀、以圖表格式呈現之加值化專利資訊。目前本研究室所使用的分析軟體爲凌網科技公司團隊所研發的 PatentGuider【 專利領航員 】，使用者可以檢所保健食品關鍵字句，如同閱讀地圖般，用簡單與清晰的圖表即可獲取包含在其內的豐富專利資訊內涵以進行專利地圖分析，產出一目了然的專利情報，有助於擴展保健食品專利訊息，促進產業技術升級。

E. 素食者大腸結直腸癌的功效評估

衛生署 96 年國人十大死因統計，大腸癌爲所有癌症死因中之第三位，利用飲食頻率問卷(FFQ)能夠瞭解大腸結直腸癌人群營養狀況的數據，以進行營養基因體學 (Nutrigenomics) 的研究，研究團隊結合慈濟醫院陳建華醫師、中研院生醫所李玲慧博士、實踐大學陳巧明博士及本系周仁施純光博士，以提供大腸直腸癌患者營養照護更多學理依據。

- 罕見疾病基因研究
- 蛋白質體學研究
- 維生素與敗血症機制探討
- 國人營養生化指標分析
- 專利地圖分析
- 素食者大腸結直腸癌評估
- 代謝症候群保健食材評估

Glycogen storage disease type IV - Wikipedia, the free encyclopedia - Microsoft Internet Explorer

檔案(F) 編輯(E) 檢視(V) 我的最愛(A) 工具(T) 說明(H)

上一頁 搜尋 我的最愛

網址(D) http://en.wikipedia.org/wiki/Glycogen_storage_disease_type_IV 移至

Google glycogen storage disease typ 開始 書籤 724 已攔截 拼字檢查 傳送到 glycogen storage disease type IV 設定

Glycogen storage disease type IV

From Wikipedia, the free encyclopedia

Glycogen storage disease type IV is a very rare hereditary metabolic disorder. It is also known as:-

- **Glycogenosis type IV**,
- **Andersen's disease**,
- **Glycogen Branching Enzyme Deficiency (GBED)**,
- **polyglucosan body disease**.
- **Amylopectinosis**

Human pathology [edit]

It is a result of the absence of the glycogen branching enzyme amylo-1,4-1,6 transglucosidase, which is critical in the production of glycogen. This leads to very long unbranched glucose chains being stored in glycogen. The long unbranched molecules (known as amylopectin) have a low solubility which leads to glycogen precipitation in the liver. These deposits subsequently build up in the body tissue, especially the heart and liver. The end result is liver failure and eventual death occurring in the first year of life.

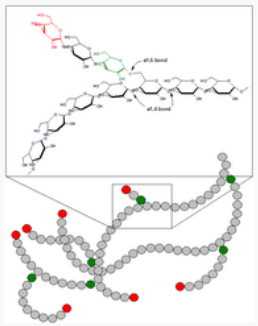
Horse pathology [edit]

See main article: Glycogen branching enzyme deficiency

External links [edit]

- *synd/78* at Who Named It
- *697958448* at GPnotebook

Glycogen storage disease type IV
Classification & external resources



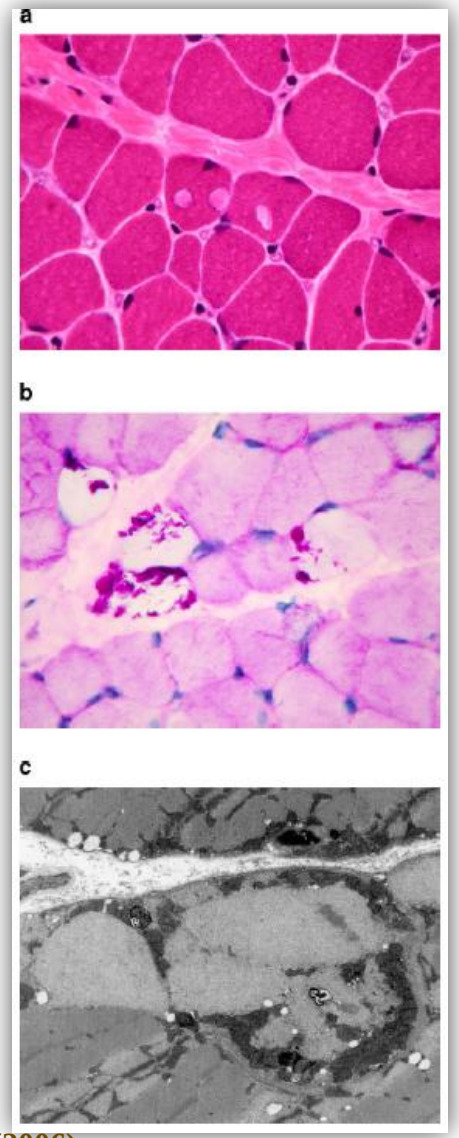
Glycogen

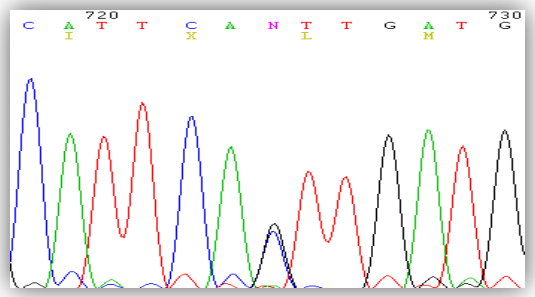
ICD-10	E74.0
ICD-9	271.0
OMIM	232500
DiseasesDB	5303
eMedicine	med/910 ped/97
MeSH	*C16.320.565.202.449.540*

v · d · e	Metabolic pathology / Inborn error of metabolism (E70-90, 270-279) [hide]
Amino acid	*Aromatic* (Phenylketonuria, Alkaptonuria, Ochronosis, Tyrosinemia, Albinism, Histidinemia) - *Branched chain* (Maple syrup urine disease, Propionic acidemia, Methylmalonic acidemia, Isovaleric acidemia, 3-Methylcrotonyl-CoA carboxylase deficiency) - *Transport* (Cystinuria, Cystinosis, Hartnup disease, Fanconi syndrome, Oculocerebrorenal syndrome) - *Sulfur* (Homocystinuria, Cystathioninuria) - *Urea cycle disorder* (N-Acetylglutamate synthase deficiency, Carbamoyl phosphate synthetase I deficiency, Ornithine transcarbamylase deficiency, Citrullinemia, Argininosuccinic aciduria, Hyperammonemia) - Glutaric acidemia type 1 - Sarcosinemia
	Lactose intolerance - Glycogen storage disease (type I, type II, type III, **type IV**, type V, type VI, type VII) - *fructose metabolism* (Fructose intolerance, Fructose bisphosphatase deficiency,

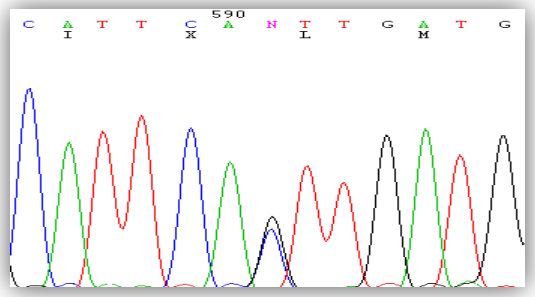
罕見疾病基因研究-GSD IV

FIG. 1. A photograph of the patient at 2 years of age.

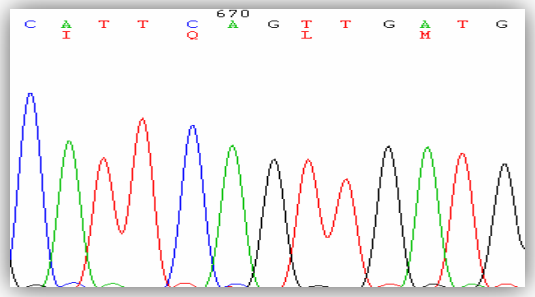




Proband



Father



Mother

American Journal of Medical Genetics 140A:878–882 (2006)

罕見疾病基因研究

-GSD IV

J Inherit Metab Dis
DOI 10.1007/s10545-009-9026-5

J Inherit Metab Dis. 2010 Jan 8

RESEARCH REPORT

Glycogen storage disease type IV: novel mutations and molecular characterization of a heterogeneous disorder

Sing-Chung Li · Chiao-Ming Chen · Jennifer L. Goldstein · Jer-Yuarn Wu · Emmanuelle Lemyre · Thomas Andrew Burrow · Peter B. Kang · Yuan-Tsong Chen · Deeksha S. Bali

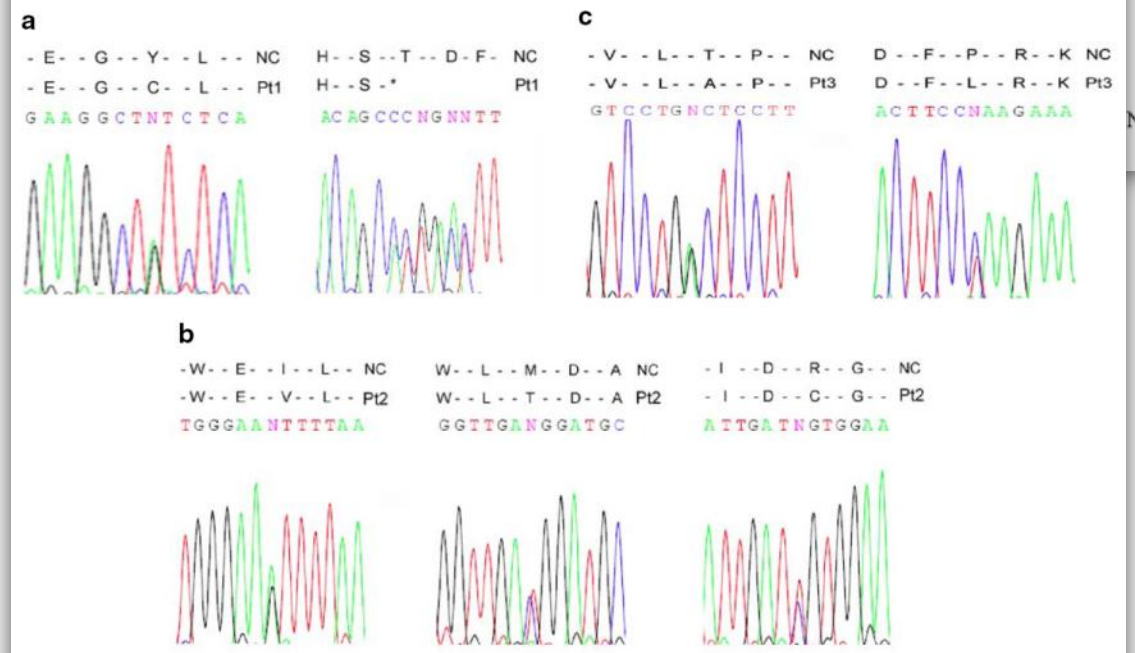


Fig. 2 The sequence analysis of glycogen-branching enzyme (*GBE1*) gene variation in patient 1(**a**), patient 2(**b**), and patient 3 (**c**)

November 2009

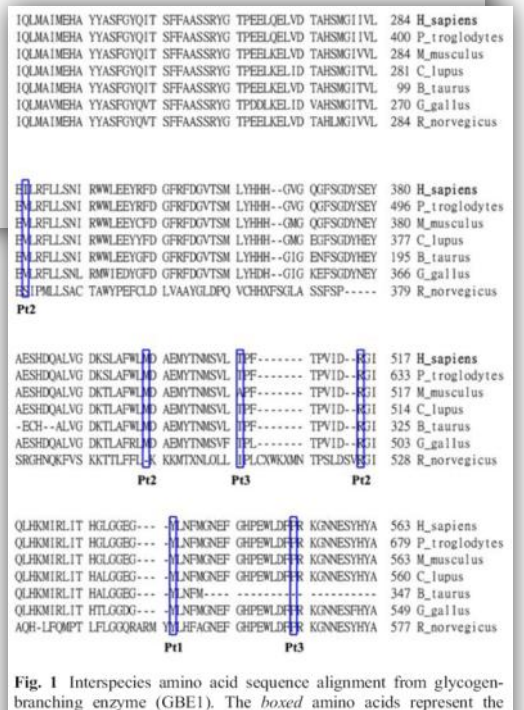


Fig. 1 Interspecies amino acid sequence alignment from glycogen-branching enzyme (GBE1). The *boxed* amino acids represent the amino acids that were altered in GBE1 in patients 1, 2, and 3, as compared with GBE1 sequences in other species

蛋白質體學研究

Methylprednisolone

19 wk chicken

12 wk chicken

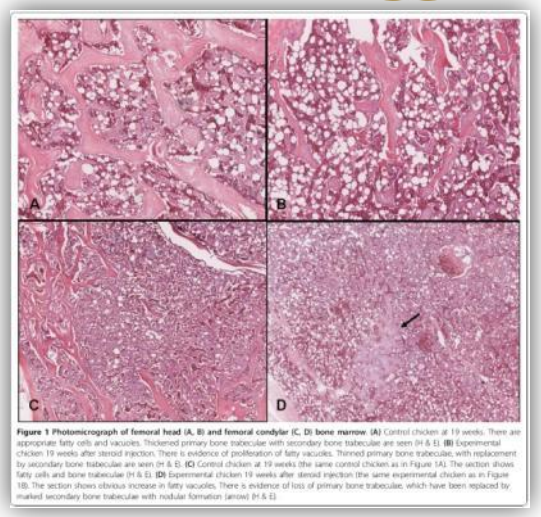
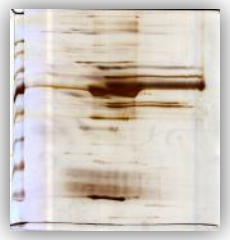


Figure 1 Photomicrograph of femoral head (A, B) and femoral condylar (C, D) bone marrow. (A) Control chicken at 19 weeks. There are appropriate fatty cells and vacuoles. Thickened primary bone trabeculae with secondary bone trabeculae are seen (H & E). (B) Experimental chicken 19 weeks after steroid injection. There is evidence of proliferation of fatty vacuoles. Thinned primary bone trabeculae, with replacement by secondary bone trabeculae are seen (H & E). (C) Control chicken at 19 weeks (the same control chicken as in Figure 1A). The section shows fatty cells and bone trabeculae (H & E). (D) Experimental chicken 19 weeks after steroid injection (the same experimental chicken as in Figure 1B). The section shows obvious increase in fatty vacuoles. There is evidence of loss of primary bone trabeculae, which have been replaced by marked secondary bone trabeculae with nodular formation (arrow) (H & E).



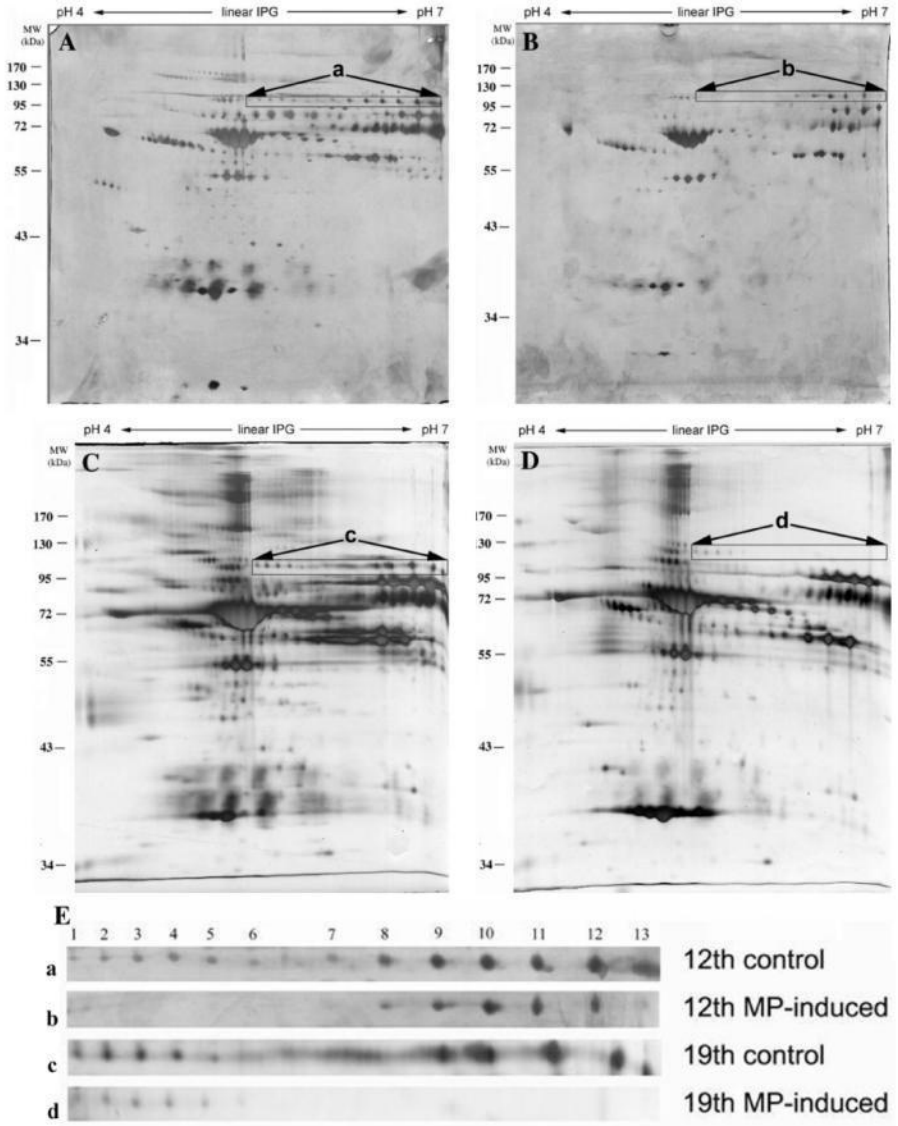


Figure 2 Representative proteome maps after separation by 2-dimensional gels and silver staining. **(A)** Control chicken bone marrow at 12 weeks (pooled sample of 3 chickens). **(B)** Steroid-induced chicken bone marrow at 12 weeks (pooled sample of 4 chickens). **(C)** Control chicken bone marrow at 19 weeks (pooled sample of the same 3 control chickens as in Figure 2A). **(D)** Steroid-induced chicken bone marrow at 19 weeks (pooled sample of the same 4 steroid-induced chickens as in Figure 2B). **(E)** Magnification of the proteome maps of (A), (B), (C) and (D). The 13 protein spots shown in the rectangle were further identified by MALDI-TOF MS.

Table 1 Differentially expressed proteins in femoral marrow of chickens within 12 weeks and 19 weeks after methylprednisolone (MP) induction (pooled sample of 4 chickens), compared with control subjects (pooled sample of 3 chickens)

Spot no.	Protein name	% coverage (score)	Swiss Prot. Accession no.	Matched species	Function	LF in 12th weeks	LF in 19th weeks
1	Coiled-coil domain-containing protein 43	41% (66)	Q5ZK95	Chicken	Evidence at transcript level.	0.9	1.4
2	Cell death activator CIDE-B	33% (69)	O70303	Mouse	Activates apoptosis [31]	1.1	2.1
3	Uncharacterized protein C3orf59	17% (61)	Q8IYB1	Human	Unknown	1.1	2.0
4	Haptoglobin precursor	31% (64)	P00738	Human	Combines with free plasma hemoglobin, preventing loss of iron	1.4	1.7
5	Serum amyloid P-component precursor	26% (79)	P02743	Human	May be involved in transcriptional regulation [12].	1.3	3.1
6	Zinc finger protein 28	23% (61)	P17035	Human	May be involved in transcriptional regulation.	1.2	4.1
7	Endothelial zinc finger protein 71	28% (57)	Q9NQZ8	Human	May be involved in transcriptional regulation.	1.2	5.6
8	T-box transcription factor 3 TBX3	12% (59)	Q7TST9	Chicken	Transcriptional repressor involved in developmental processes. Probably plays a role in limb pattern formation [9].	1.0	6.2
9	Cyclin-dependent kinase inhibitor 1	28% (62)	P39689	Mouse	May be the important intermediate by which p53 mediates its role as an inhibitor of cellular proliferation in response to DNA damage. Binds to and inhibits cyclin-dependent kinase activity, preventing phosphorylation of critical cyclin-dependent kinase substrates and blocking cell cycle progression [32].	0.9	7.4
0	Uncharacterized protein UNQ1940PRO4423 precursor	27% (60)	Q6UWF9	Human	Evidence at transcript level.	0.8	7.5
11	Uncharacterized protein C12orf52 homolog	39% (66)	Q2HJ75	Bovine	Evidence at transcript level.	0.9	6.6
12	Myosin	23% (85)	Q17R14	Bovine	Evidence at transcript level.	1.5	7.4
13	Dimethylaniline monooxygenase	19% (75)	Q8K4B7	Rat	This protein is involved in the oxidative metabolism of a variety of xenobiotics such as drugs and pesticides [29,34]	2.6	7.6

The lowing factors (LF) were established as the densitometric volume ratio of control over MP-induced group.

Li *et al. Proteome Science* 2010, **8**:47
http://www.proteomesci.com/content/8/1/47

PROTEOME SCIENCE

RESEARCH Open Access

Chicken model of steroid-induced bone marrow adipogenesis using proteome analysis: a preliminary study

Sing Chung Li[1], Ching Yu Lin[2], Tzong Fu Kuo[3], Yun Ho Lin[4], Chia Chun Chen[5], Way Neng Lin[1], Wing P Chan[5,6*]

Abstract

Background: Steroid-induced adipogenesis increases fat-cell volume and pressure in bone marrow. This may be a contributing factor in some forms of osteonecrosis. In this observational study, we aimed to determine the protein expression relating to steroid-induced adipogenesis of femoral bone marrow with use of a chicken model. We compared the histologic features of the femoral marrow of eight methylprednisolone (MP)-treated chickens with those of three control chickens and assessed differential proteins with 2-dimensional gel electrophoresis and differential proteins were identified by MALDI-TOF MS.

Results: One MP-induced chicken died of overdose anesthesia. Methylprednisolone-induced proliferation of adipose tissue and new bone formation were found on histologic examination. In our study, 13 proteins in the control and MP-induced groups were differently expressed and nine protein spots showed marked threefold downregulation after 19 weeks of MP treatment. These were serum amyloid P-component precursor, zinc finger protein 28, endothelial zinc finger protein 71, T-box transcription factor 3, cyclin-dependent kinase inhibitor 1, myosin 1D, dimethylaniline monooxygenase, and two uncharacterized proteins.

Conclusions: Proteomic profiling can be a useful dynamic approach for detecting protein expression in MP-induced adipogenesis of the femur in chickens.

J Exp Clin Med 2010;2(5):231–238

ELSEVIER

ORIGINAL ARTICLE

Screening of Ethylnitrosourea Mice With Fatty Acid Oxidation Disorders by a Candidate Gene Approach After Proteome Analysis

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KEY WORDS:
ENU mice;
proteomic;
short chain fatty acid

Background/Purpose: Ethylnitrosourea (ENU) is an alkylating agent and primarily induces point mutations such as AT to TA transversions and AT to GC transitions. Due to its high mutagenicity, ENU mouse mutagenesis enables the generation and identification of mouse mutants with aberrance in various phenotypes and to identify novel genes relevant for the expression of the phenotype. The purpose of this study was to investigate the candidate genes involved in fatty acid oxidation disorders by the proteomic approach.
Methods: We screened ENU mice from 39 families from previously published data and identified two mutant mice that had a striking elevation in blood C4-OH short chain fatty acids compared with ENU controls. Total mitochondrial proteins were extracted from the gastrocnemius for two-dimensional electrophoresis, and two downregulated proteins, adenylate kinase isoenzyme 1 (AK1) and adenosine-5′-triphosphate (ATP) synthase D chain (ATP5H), were identified in the mutant mice through matrix-assisted laser desorption/ionization time-of-flight mass spectrometry.
Results: After genomic polymerase chain reaction and direct sequencing of *Ak1* and *Atp5h*, no variation was found in both gene sequence analyses.
Conclusion: Proteomic profiling can be a useful approach for detecting dynamic protein expression in ENU-induced mice. It is important to further clarify mechanisms of the mutant C4-OH disorder responsible for this expression.

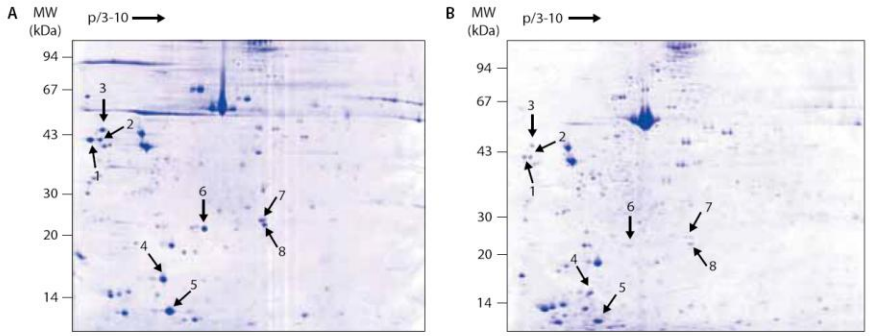


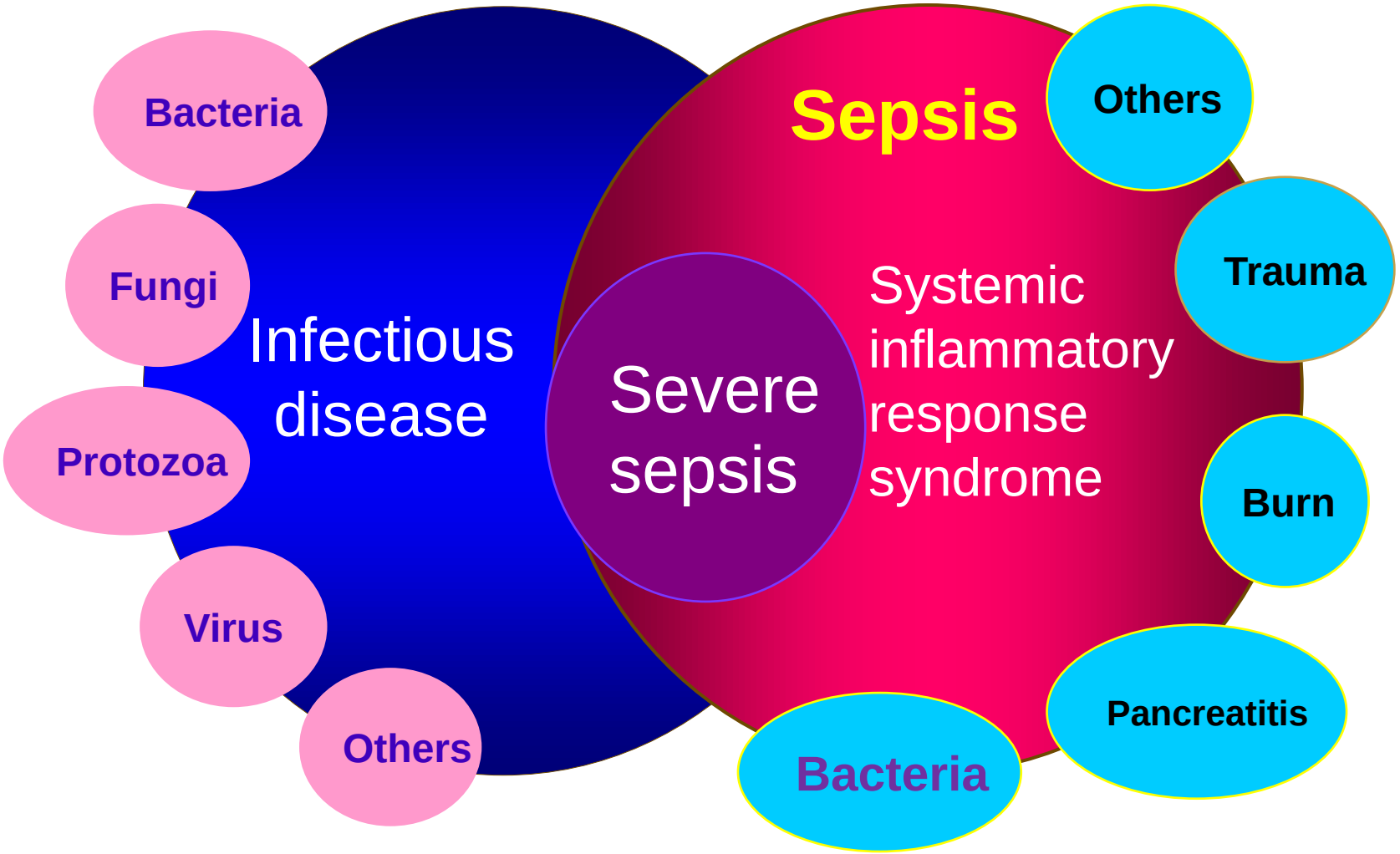
Figure 1 Two-dimensional analysis of mitochondrial proteins from normal and ethylnitrosourea mice. The mitochondria were extracted from gastrocnemius muscle by a commercial kit: (A) ethylnitrosourea control; (B) affected. Experiments were carried out in triplicate.

Table 2 Mitochondrial protein identification and quantification of ethylnitrosourea (ENU) control and C4-OH mice

Spot no.	Gene name	Description	Accession no.	Experimental *pI*/Mw (kD)	Fold change
1	Calsequestrin-1	Calcium-binding protein	O09165 (CASQ1_MOUSE)	3.9/45.6	5.2
2	Calsequestrin-1	Calcium-binding protein	O09165 (CASQ1_MOUSE)	3.9/45.6	2.4
3	Calsequestrin-1	Calcium-binding protein	O09165 (CASQ1_MOUSE)	3.9/45.6	2.7
4	Myosin light chain 2	This chain binds calcium	P97457 (MLRS_MOUSE)	4.8/18.9	2.2
5	Myosin light chain 3	Regulatory light chain of myosin	P05978 (MLE3_MOUSE)	4.6/16.6	1.5
6	Myosin light chain 6B	Regulatory light chain of myosin	Q8CI43 (MYL6B_MOUSE)	5.4/22.5	3.2
7	Adenylate kinase isoenzyme 1	Catalyzes the reversible transfer of the terminal phosphate group between ATP and AMP	Q9R0Y5 (KAD1_MOUSE)	5.7/21.5	4
8	ATP synthase D chain	Mitochondrial membrane ATP synthase	Q9DCX2 (ATP5H_MOUSE)	5.6/18.6	2.1

Differential spots were identified through matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and data were evaluated in the MASCOT and NCBI databases. ATP = adenosine triphosphate; AMP = adenosine monophosphate.

維生素與敗血症機制探討



Bone RC, et al. Chest 1992; 101: 1644-55

維生素與敗血症機制探討

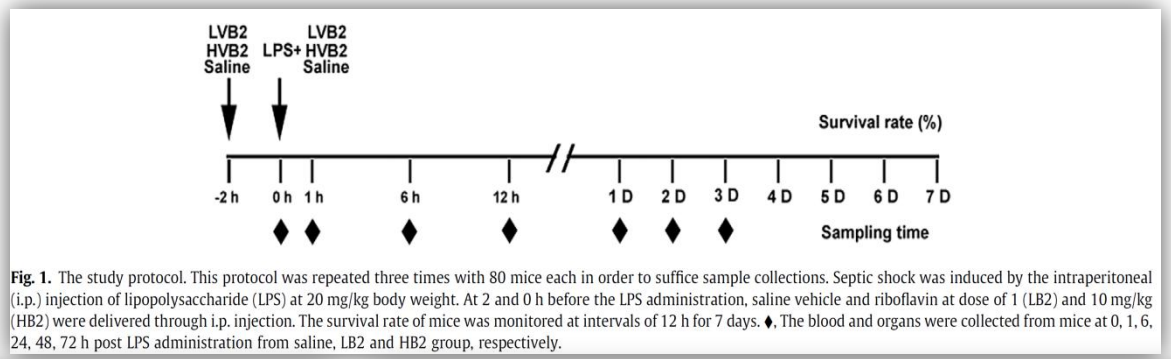


Fig. 1. The study protocol. This protocol was repeated three times with 80 mice each in order to suffice sample collections. Septic shock was induced by the intraperitoneal (i.p.) injection of lipopolysaccharide (LPS) at 20 mg/kg body weight. At 2 and 0 h before the LPS administration, saline vehicle and riboflavin at dose of 1 (LB2) and 10 mg/kg (HB2) were delivered through i.p. injection. The survival rate of mice was monitored at intervals of 12 h for 7 days. ♦, The blood and organs were collected from mice at 0, 1, 6, 24, 48, 72 h post LPS administration from saline, LB2 and HB2 group, respectively.

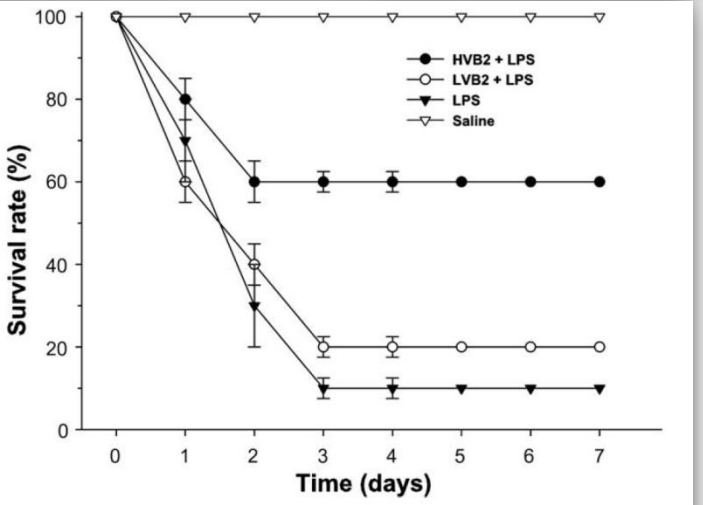


Fig. 2. The effect of vitamin B2 on the survival rate of septic mice. The study protocol was performed three times with 80 mice each. Eighty mice were assigned into one of four groups, saline vehicle, saline + lipopolysaccharide (LPS), low vitamin B2 + LPS (LB2), and high vitamin B2 + LPS (HB2) ($n = 20$). The results are expressed as mean ± SD ($n = 3$).

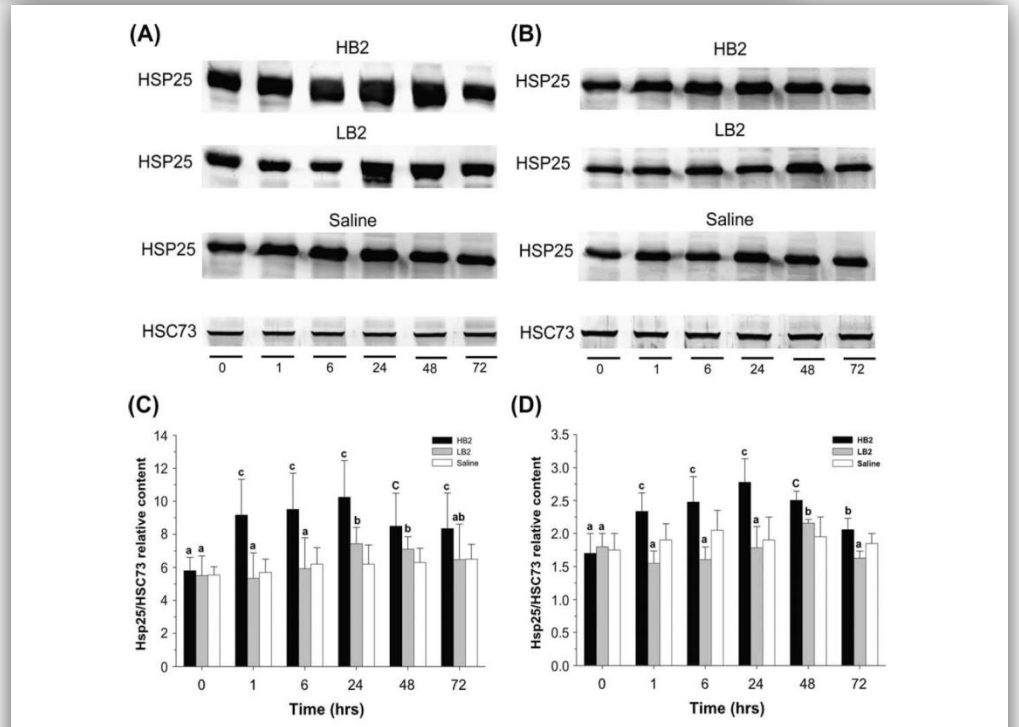


Fig. 3. The HSP25 expression in heart (A, C) and lung (B, D). HSC73 is used for normalization. The value of HSP25/HSC73 was obtained from three densitometric measurements. The results are expressed as mean ± SD ($n = 5$). $^{a-c}$Means with different letters differ, determined using a Bonferroni post test following a two-way ANOVA test with time and riboflavin as factors ($p < 0.05$).

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Riboflavin protects mice against liposaccharide-induced shock through expression of heat shock protein 25

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ABSTRACT

Riboflavin (vitamin B2) is a water-soluble vitamin essential for normal cellular functions, growth and development. This study aimed to investigate the effects of vitamin B2 on the survival rate, and expressions of tissue heat shock protein 25 (HSP25) and heat shock factor 1 (HSF1) in mice undergoing lipopolysaccharide (LPS) induced shock. Mice were assigned to four groups, saline vehicle, LPS, LPS plus low dose of vitamin B2 (LB2) and LPS plus high dose of vitamin B2 (HB2). Vitamin B2 (1 and 10 mg/kg BW) was administered intraperitoneally at 2 and 0 h before the i.p. administration of LPS. At the end of the experiment, the survival rate monitored was 10, 20, 60, and 100% for LPS, LB2, HB2, and saline mice, respectively. HSP25 expressions in the heart and lung were significantly enhanced in a time-dependent manner in the HB2 mice as compared to the saline mice ($p < 0.05$), but not altered in the LB2 mice. In the HB2 mice, plasma riboflavin concentrations reached 300 nM at 6 h post LPS and returned to the 0 h level at 72 h. The results showed that high dose of riboflavin could decrease LPS-induced mortality through an increased expression of HSP25.

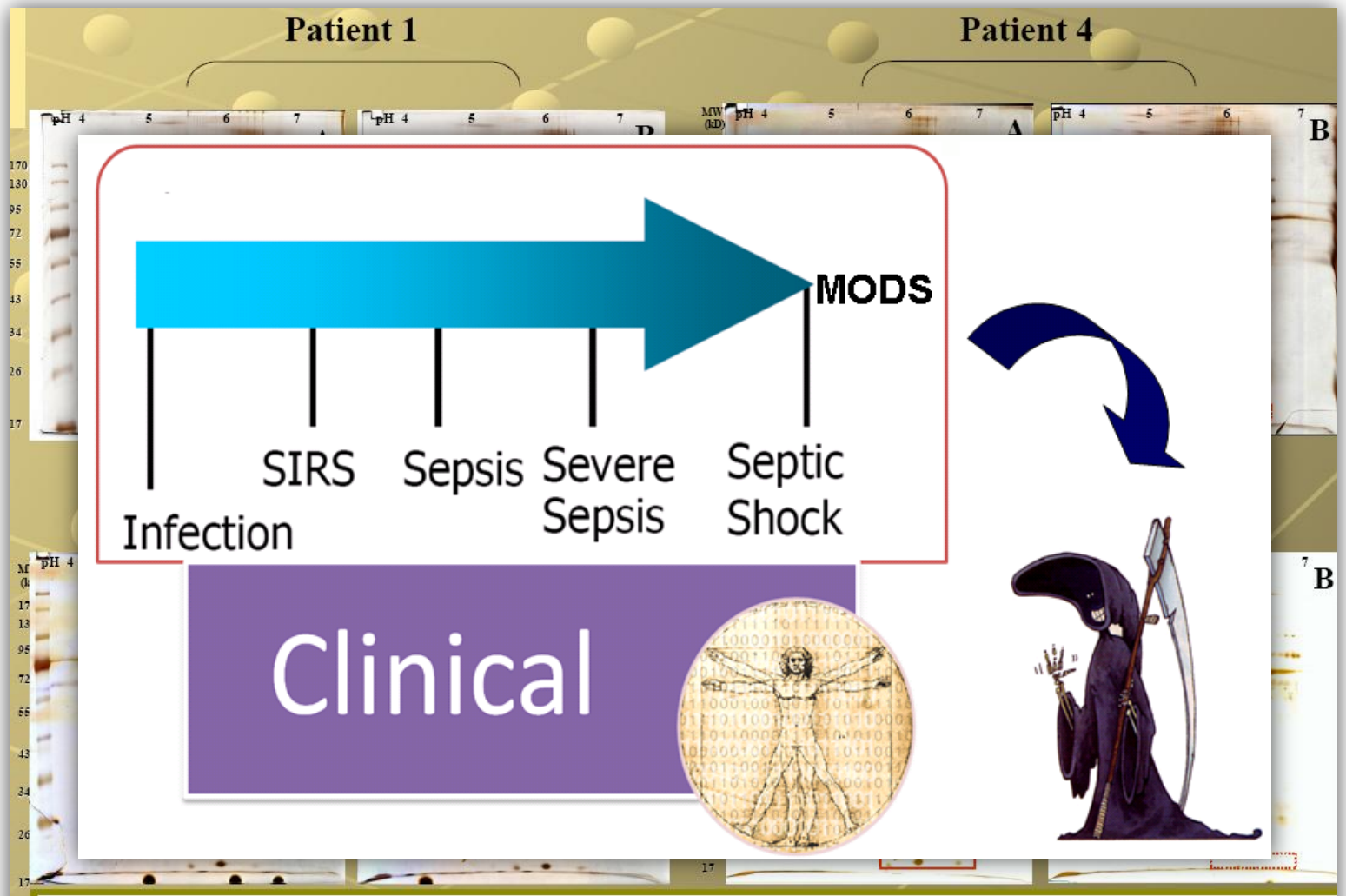

Figure 1. Typical two-dimensional protein map of serum. Protein separation was performed with immobilized nonlinear pH 4-7 gradient IPG strips and subsequent SDS-PAGE in 10% matrix of a decrease in molecular weight followed by silver staining. Spots marked by their numbers show the location of the proteins, which were differential expressed between 6 hour(A) and 7th day(B) time interval in septic patient 1、4、5、10 respectively.

國人營養生化指標分析-(2005~2008, B2/B3/B6, A/E/B-Caroteine)

D-2000: VB2+VB6 Series: 0039 Report Name: original System: Sys 1

D-2000 Elite HPLC System Manager Report

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Reported Date and Time: 2009/02/18 09:49 上午
Processed Date and Time: 2008/12/27 12:35 下午
Data Path: D:\李老師研究室\LEE\HPLC\VB2\DATA\0039\
Processing Method: run-1
System (acquisition): Sys 1
Series: 0039
Application(data): VB2+VB6
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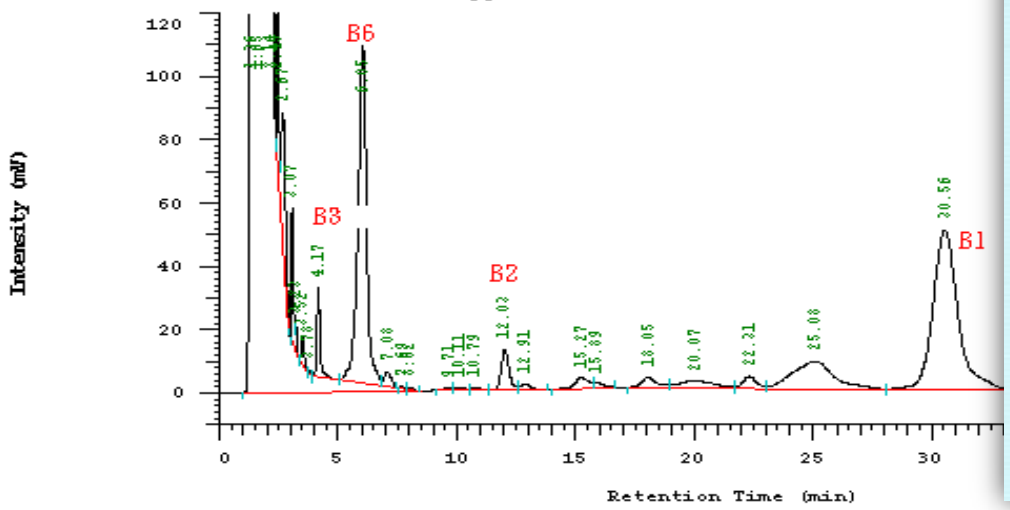
Sample Name: UNKNOWN002
Vial Type: UNK
Injection from this vial: 1 of 1
Volume: 100.0 ul
Sample Description:

Chrom Type: HPLC Channel : 2



Processing Method: run-1
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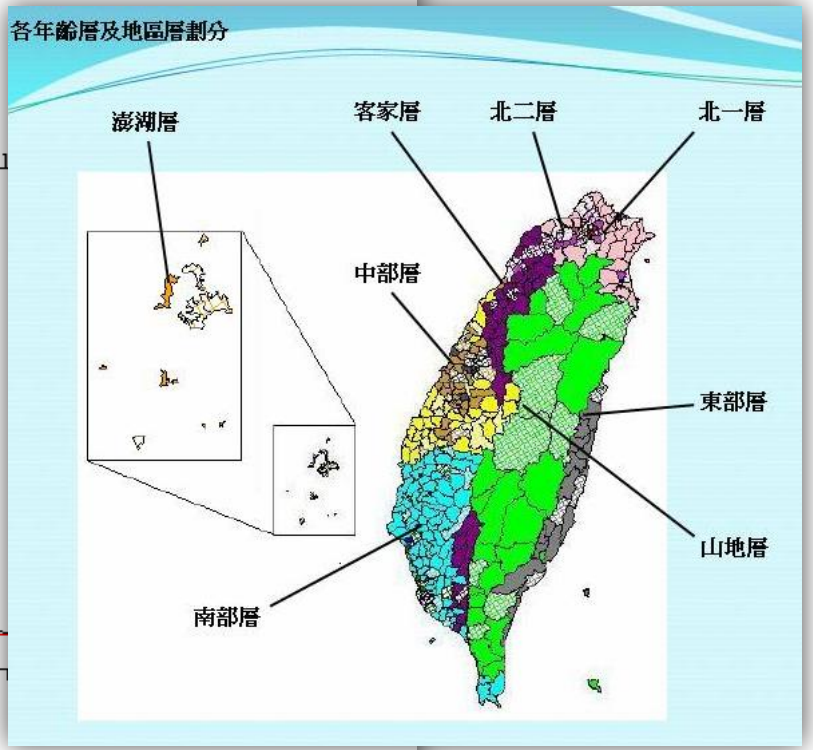
Method Developer: run
Method Description: 1 ml/min



專利地圖分析


首頁
台灣核准專利
台灣公開專利
美國核准專利
美國公開專利
歐盟核准專利
歐盟公開專利
中國專利
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登出
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下載提案構想書 新增提案
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查詢
找到 6 筆符合的資料
提案編號 中文名稱 填表人 提案日 提案狀態 發明人 目前進度
20100014 海藻活性物質萃取裝置 李信昌 2010/09/22 已受理 李信昌
20100013 蛋白質診斷標記尋找模組 李信昌 2010/09/22 已受理 李信昌
20100012 個人化健康飲食系統 李信昌 2010/09/22 已受理 李信昌
20100011 甘藷葉生物活性物質萃取裝置 李信昌 2010/09/22 已受理 李信昌
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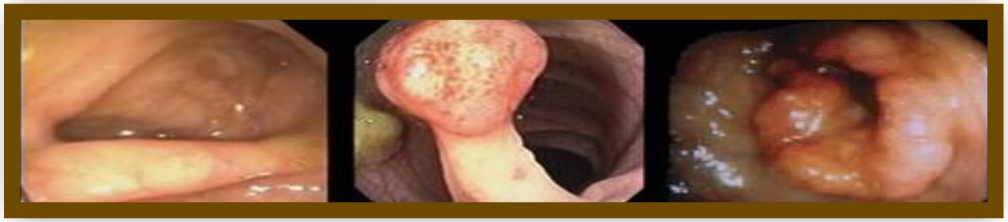
20100008 微生物檢驗裝置 李信昌 2010/09/17 退回修正 李信昌

素食者大腸結直腸癌評估

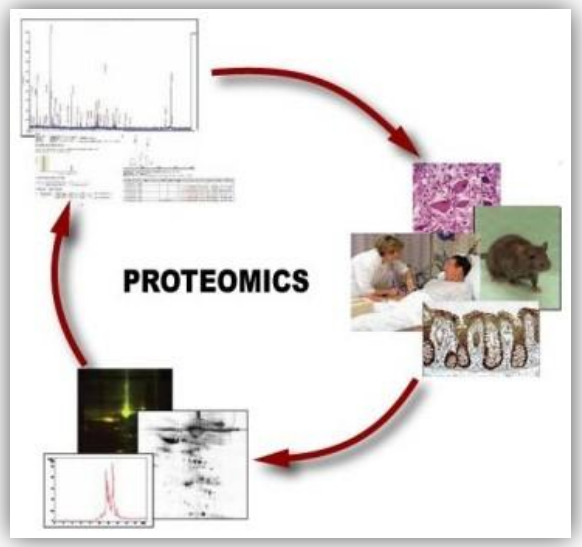
Figure 2. Example of Food Frequency Questionnaire

	Never	Once per week	2-4 per week	5-6 per week	Daily	Once per month	Once per 3 months	Once per year
Milk, yogurt, regular fat (1 cup)	○	○	○	○	○	○	○	○
Milk, yogurt, lowfat (1 cup)	○	○	○	○	○	○	○	○
Spinach, kale, other green leafy vegetables (1/2 cup)	○	○	○	○	○	○	○	○
Carrots (1 medium)	○	○	○	○	○	○	○	○
Beef (3 oz)	○	○	○	○	○	○	○	○
Rice, white (1 cup)	○	○	○	○	○	○	○	○
Rice, brown (1 cup)	○	○	○	○	○	○	○	○
Cookies (2 -2" diameter)	○	○	○	○	○	○	○	○
Ice cream, regular fat (1/2 cup)	○	○	○	○	○	○	○	○

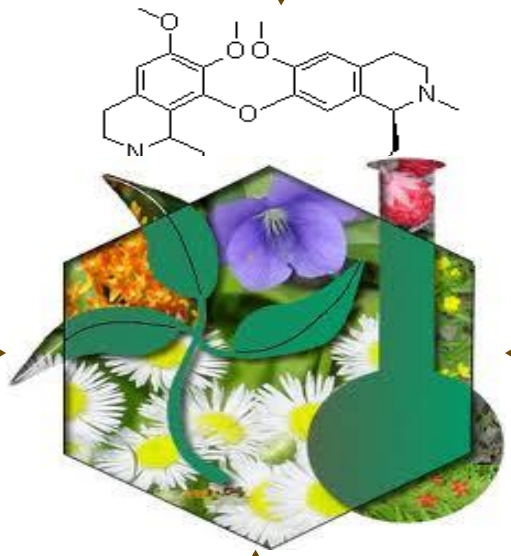
中央研究院國家基因型鑑定中心
National Genotyping Center at Academia Sinica

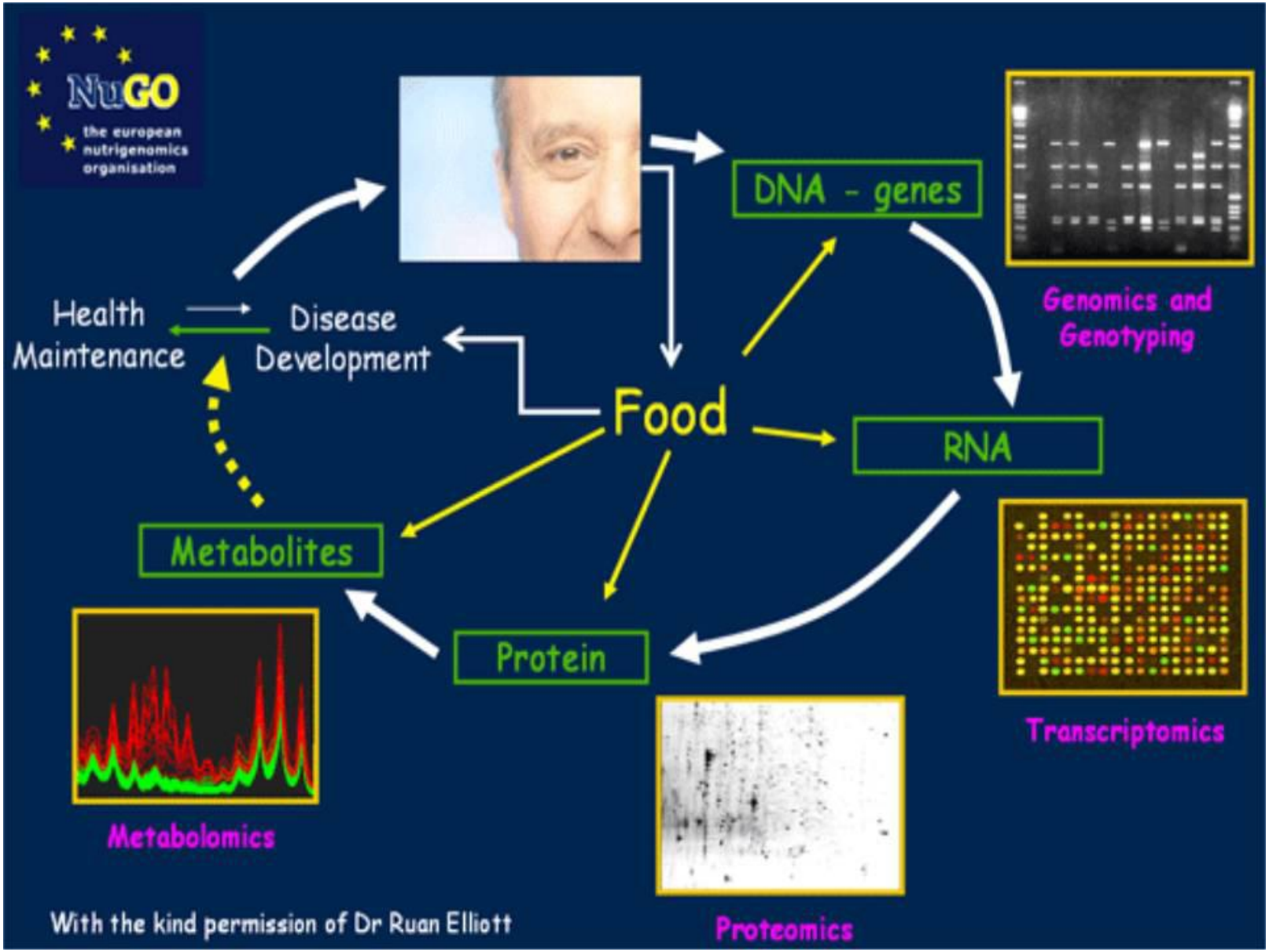


Normal → Adenoma → Carcinoma



代謝症候群保健食材評估

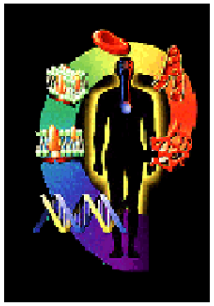



NuGO
the european nutrigenomics organisation
DNA - genes
Genomics and Genotyping
Health Maintenance
Disease Development
Food
RNA
Metabolites
Protein
Transcriptomics
Metabolomics
Proteomics
With the kind permission of Dr Ruan Elliott

Foods
Nutrition

Target Genes
Mechanisms
Pathways

Signatures
Profiles
Biomarkers



Molecular Nutrition
& Genomics

Nutritional
Systems Biology

- Identification of dietary signals
- Identification of dietary sensors
- Identification of target genes
- Reconstruction of signaling pathways

- Measurement of stress signatures
- Identification of early biomarkers

Small research groups
Small budgets

Large research consortia
Big money

Complexity

J Am Diet Assoc. 2006 Apr;106(4):569-76

誌謝

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黃小珍/林緯能/陳玉茹/周佳汶/詹詠晴

Thanks for attention