中國醫藥大學
CHINA MEDICAL UNIVERSITY

從食安風暴談起
–毒物科醫師的看法

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堅持品質 確保安全
正義
香豬油
正義
香豬油


CTTV
東森新聞


A1 頭條 二〇一四年十月九日 星期四 農曆甲午年九月十六日
請快下載《台灣蘋果日報》App
蘋果日報網站10月7日 總瀏覽量1930萬次
蘋果即時新聞瀏覽量1300萬次
用噁心病死豬煉製
頂新60油品今下架
摻飼料油
正義 香豬油
◀黃油給喜好企業的楊金山地下油廠，被發現大批凍存的下腳料。 陳宏銘攝
頂新味全董事長 魏應充
■魏應充的正義油品出包，維力清香油、香豬油與正興香豬油等60多項產品今將下架。 資料照
日蘋報果
APPLE DAILY
零售每份15元(離島18元)
訂報專線：0800-588-668
追思
訂閱獨享優惠
PERFECT經典真空保溫杯


全統 香豬油
飼料油、廢食用油
管理補破網
飼料油
進口報關：今起比照食用油須全面報驗，且輸入須先取得農委會許可並按月申報流向
製造、輸入動植物油脂業者：須取得中央或北、高兩直轄市主管機關的製造或輸入登記證；國內製造飼料油脂業者也須申報流向
廢食用油
廢棄物清理法將修法，擴大納管會產生廢食用油的業者，修法後列管事業將從900家增加到6000多家。環保署估計，每年可多掌握1萬3000噸廢油流向
製表：記者蔡穎、施文華
圖：資料照

頂新飼料油檢驗結果 VS 食用油脂衛生標準

檢驗種類	法規最高容許量	頂新豬油	頂新牛油
鉛	0.4ppm	0.73ppm 超標 1.8 倍	o.4ppm
汞	0.05ppm	沒驗出	沒驗出
鉻	零檢出	沒驗出	0.08ppm
銅	0.1ppm	0.54ppm 超標 5 倍	0.47、0.9ppm 超標 9 倍
酸價	2.5mg KOH/g	2.3mg KOH/g	5.8、6.7mg KOH/g 超標 2.6 倍

劣質豬油事件檢驗結果

103.9.8

檢體		檢驗結果					
		酸價 (mg KOH/g fat) (註1)	總極性物質 (%)	重金屬 (砷、鉛、汞、銅、錫、鉻)	苯駢芘 (ppb) (註2)	黃麴毒素 (ppb)	動物成分 (牛豬魚雞)
廢棄回收油 (編號 002)	胡信德工廠	2.8	未檢出	未檢出~正常範圍	0.9	未檢出	豬陽性 (極微量)
廢棄回收油 (編號 003)	胡信德工廠	2.7	未檢出	未檢出~正常範圍	6.6	未檢出	豬陽性 (極微量)
廢棄回收油 (編號 004)	胡信德工廠	3.0	未檢出	未檢出~正常範圍	6.0	未檢出	豬陽性 (極微量)
原料油	強冠 (油槽)	2.3	未檢出	未檢出~正常範圍	1.1	未檢出	陰性
全統香豬油		0.3	未檢出		0.7		

註：

1. CNS 2421 N 5069「食用豬脂」品質規定「酸價」項為 1.3 mg KOH/g fat。
2. 苯駢芘[benzo(a)pyrene]含量在歐盟食用油脂之容許量標準與我國監測指標值為 2 ppb。

戴奧辛污染物	毒性當量因子
2,3,7,8-TeCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
2,3,7,8-TeCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001

有害人體健康嗎？　會中毒嗎？

表 1.2　皮衣及相似皮製品用皮革副原料耗用量

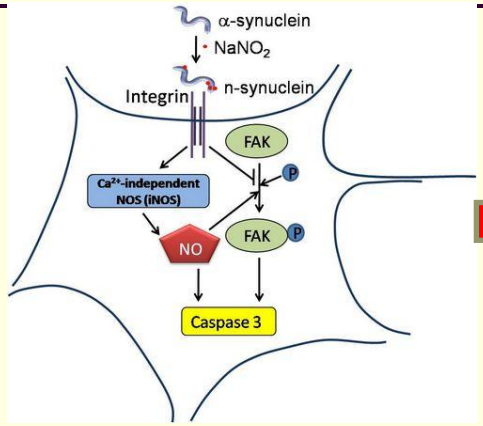
物料名稱	每 100 kg牛鹽濕皮耗用原料量(1)	鞣製面革 100ft^{2}耗用原料量(2)
石灰	3.8~5.6kg	1.95~2.80kg
人造單寧	2.5~7.1kg	1.25~3.60kg
栲膠	3.9~8.8kg	2.00~4.45kg
硫化鈉	2.5~3.9kg	1.25~2.00kg
鉻鞣精	8.6~11.2kg	4.30~5.95kg
洋糠精	0.7~1.4kg	0.35~0.70kg
硫氫化鈉	0.8~2.4kg	0.40~1.25kg
脫灰劑	1.4~2.9kg	0.74~1.55kg
油脂(硫化油)	9.0~16.0kg	4.55~8.10kg
蟻酸鈣(鈉)	2.6~5.0kg	1.30~2.60kg
樹脂	0.8~2.9kg	0.42~1.50kg
助劑	0.3~1.0kg	0.15~0.52kg
氨水	0.5~1.1kg	0.25~0.56kg
脫脂劑	1.2~2.8kg	0.60~1.40kg
蟻酸	2.0~4.8kg	1.05~2.40kg
鈦白粉	0.8~1.5kg	0.40~0.75kg
染料	2.4~6.8kg	1.20~3.40kg
塗料	0.5~1.6kg	0.26~0.85kg
工業鹽	8.4~11.0kg	4.20~5.95kg
硫酸	1.2~2.3kg	0.60~1.20kg
鹽酸	1.2~2.0kg	0.60~1.05kg
純鹼	1.0~1.2kg	0.50~0.60kg
溶劑	0.6~2.2kg	0.30~1.15kg

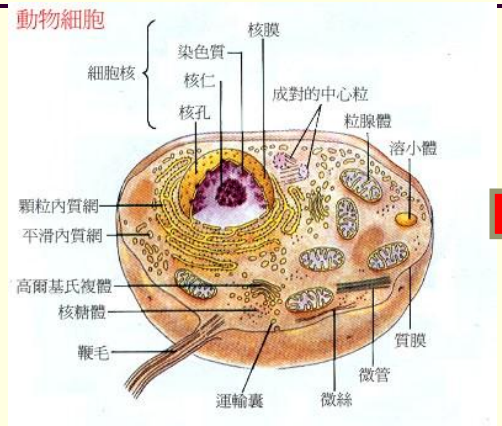
註：(1)100kg 約等於 4 隻(55~60 磅/隻)

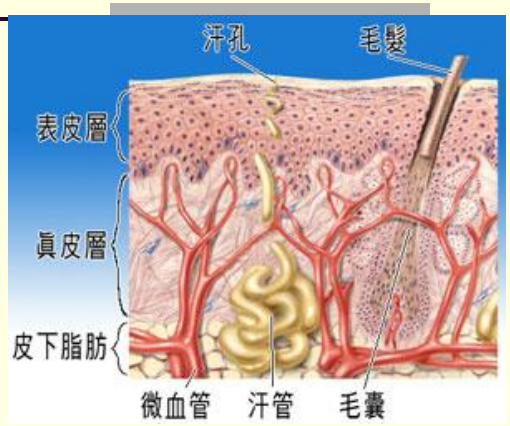
(2)100ft^{2}約等於 2 隻(即 4 片，6kg/片，厚度 1.6~1.8mm)

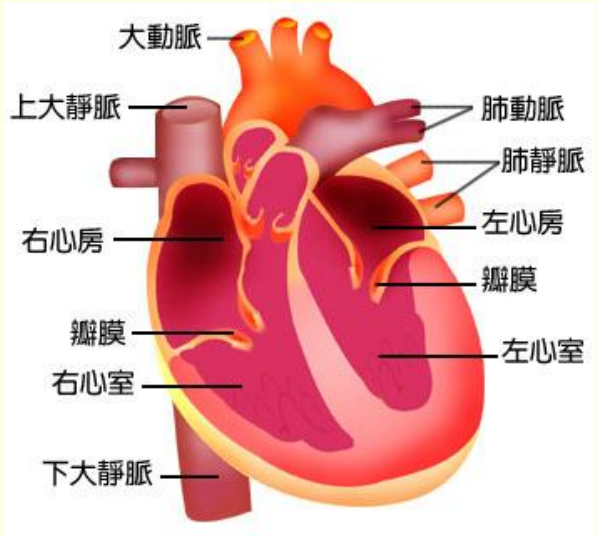
中毒的定義

- 任何含有毒素的物質，不管固體、液體、氣體的型態，經由口服食入、 呼吸道吸入、注射或皮膚接觸，而進入人體，破壞人體器官，損害健康甚至造成死亡。


α-synuclein
NaNO2
n-synuclein
Integrin
FAK
Ca2+-independent NOS (iNOS)
P
NO
FAK
P
Caspase 3


動物細胞
核膜
染色質
細胞核
核仁
核孔
成對的中心粒
粒腺體
溶小體
顆粒內質網
平滑內質網
高爾基氏複體
核糖體
鞭毛
質膜
微管
運輸囊
微絲

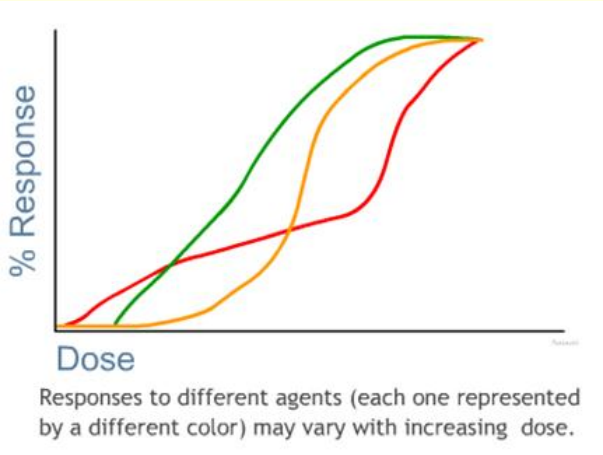

汗孔
毛髮
表皮層
眞皮層
皮下脂肪
微血管
汗管
毛囊


大動脈
上大靜脈
肺動脈
肺靜脈
右心房
左心房
瓣膜
瓣膜
左心室
右心室
下大靜脈

Paracelsus (現代毒物學之父)：所有物質都是毒物，沒有例外，劑量是毒物與否的唯一決定性因素。 藥物 *or* 毒物

致毒物

任何物質在生物體會產生不良反應的，稱之。



Responses to different agents (each one represented by a different color) may vary with increasing dose.

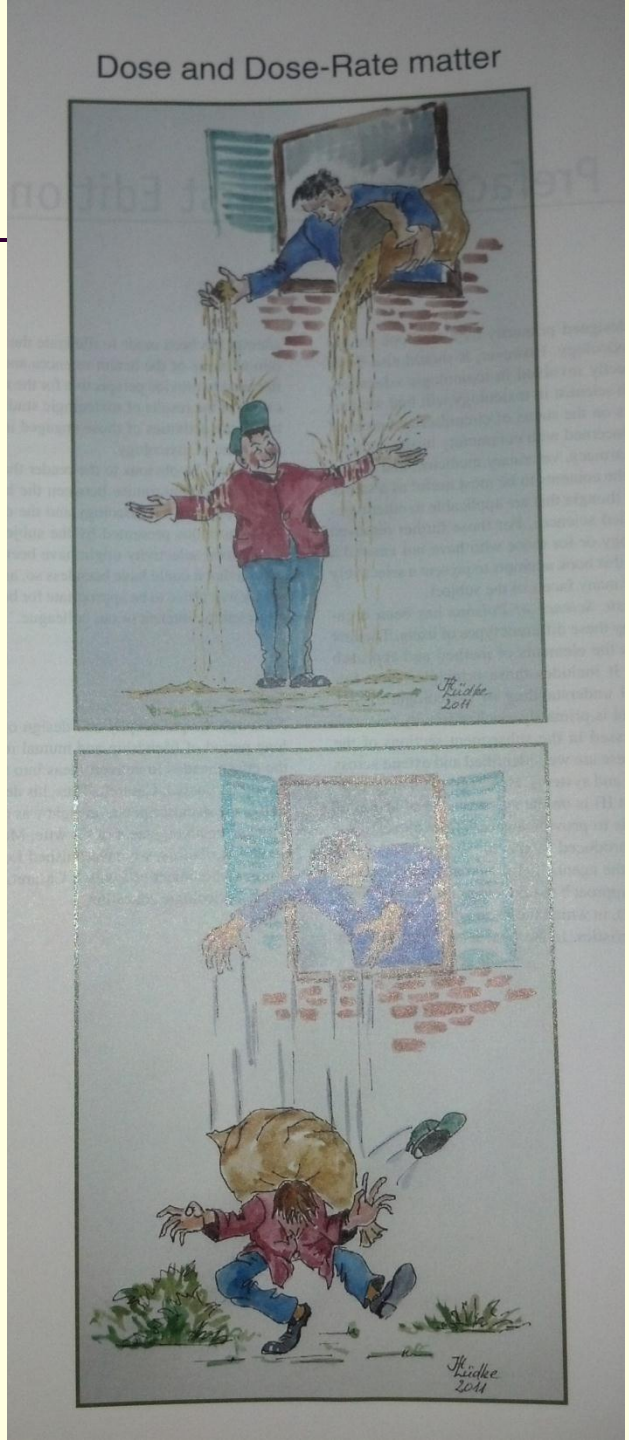
Philippus Theophrastus Bombastus Von HOHENHEIM PARACELSUS (1493-1541)

Casarett and Doull's TOXICOLOGY

The Basic Science of Poisons

"What is there that is not poison? All things are poison and nothing (is) without poison. Solely the dose determines that a thing is not a poison."

Paracelsus (1493–1541)



部分代表性毒化物的半數致死劑量(動物)

毒化物	半數致死劑量 LD_{50}, mg/kg
酒精	10,000
氯化鈉(食鹽)	4,000
Ferrous sulfate	1,500
Morphine sulfate	900
Phenobarbital	150
巴拉刈農藥	50
氰化鉀	5
番木鱉鹼	2
尼古丁	1
d-Tubocurarine	0.5
河豚毒素	0.10
戴奧辛	0.001
肉毒桿菌毒素	0.00001

表一：世界各國飲用水中總三鹵甲烷的限值

國　家	限值（mg/l）
美　國	0.1
日　本	0.1
加　拿　大	0.35
比　利　時	0.1
芬　蘭	0.1
中華民國台灣省	0.15
中華民國台北市	0.1

慢性暴露容許標準?

某種傷害=危害1+危害2+危害3+危害4+危害5+------

亞氯酸鹽未觀察到不良反應之劑量(NOAEL)=2.9 ***mg/kg BW/day***。(動物實驗)

每日容許攝取量(TDI, Tolerable Daily Intake)為30 ***μg/ kg BW/day***。

---WHO

致癌性毒化物評估

致癌性風險評估，顧名思義就是針對具有致癌性的毒化物進行評估，例如多氯聯苯(polychlorinated biphenyls)、戴奧辛(dioxin)、無機砷(inorganic arsenic)及甲基汞(methylmercury)等。在人體或動物會造成癌症的物質被認為具有**非閾值效應**，亦即並沒有安全暴露的水平。任何暴露都具有一些風險，當暴露增加，致癌反應的機率也增加(參見圖3-3)。

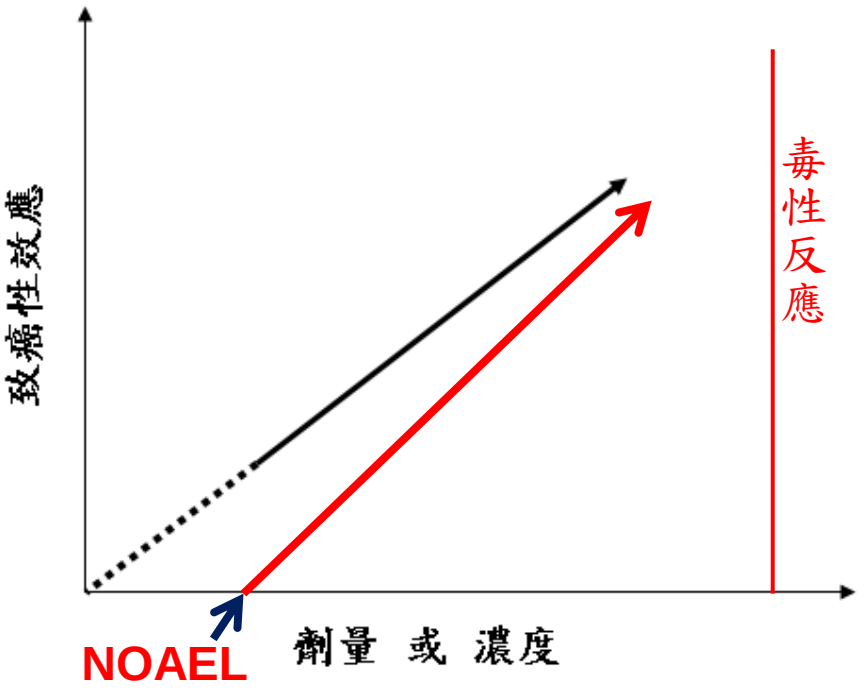


圖3-3. 劑量-致癌性效應關係圖

致癌風險之推估-總致癌性風險

總致癌風險為所有暴露途徑致癌風險之總和（常用基準值=10^{-6}, 百萬分之一）。

$$Risk_t = \sum_{i=1}^{n} Risk_i$$

$Risk_t$ = 單一暴露途徑致癌風險
$Risk_i$ = 第 i 個毒物個別致癌風險

國際癌症研究中心(IARC)將致癌物質分為四類

分 類	證 據	例 子
1. 人類致癌物	充份（人類）	石綿、砷、鈹、鎘、鎳、黃麴毒素、鉻、氯乙烯單體、苯、環氧乙烷、甲醛、戴奧辛、檳榔、酒精、多氯聯苯、香菸苯并蒎benzo(a)pyrene
2A. 對人類可能為致癌物	有限（人類） 充份（動物）	無機鉛、四氯丹、瘧疾
2B. 對人類懷疑為致癌物	充份（動物） 無（人類）	氯仿、DDT、多溴聯苯
3. 致癌證據不足	不足（動物）	氟化氫、阿特靈
4. 無任何證據	無	

Cigarette—as example

- 香菸看似無害. 燃燒時會放出約4,000 chemical--a dangerous cocktail
- more than 70 cancer-causing chemicals
- hundreds of other poisons.
- nicotine, a highly addictive drug
- additives designed to make cigarettes taste nicer and keep smokers hooked

Cancer-causing chemicals in tobacco smoke

- Tar - a mixture of dangerous chemicals
- Arsenic - used in wood preservatives
- Benzene - an industrial solvent, refined from crude oil
- Cadmium - used in batteries
- Formaldehyde - used in mortuaries and paint manufacturing
- Polonium-210 - a highly radioactive element
- Chromium - used to manufacture dye, paints and alloys
- 1,3-Butadiene - used in rubber manufacturing
- Polycyclic aromatic hydrocarbons - a group of dangerous DNA-damaging chemicals
- Nitrosamines - another group of DNA-damaging chemicals
- Acrolein - formerly used as a chemical weapon
- Other chemicals

Alcoholic beverages

- Cancer of the oral cavity and pharynx
- Cancer of the larynx
- Cancer of the oesophagus
- Cancer of the liver
- Breast cancer
- Cancer of the stomach
- Cancers of the colon and/or rectum
- Cancer of the pancreas
- Cancer of the lung
- Cancer of the urinary bladder
- Cancer of the endometrium
- Cancer of the ovary
- Cancer of the uterine cervix
- Cancer of the prostate
- Cancer of the kidney
- Cancer of the lymphatic and haematopoietic system
- Cancer at other sites

Aflatoxin 黃麴毒素

Table 3. Estimated HCC incidence attributable to aflatoxin, by WHO region.

WHO region/country	Reference	Aflatoxin exposure (ng/kg body weight/day[a])	Estimated annual HCC (per 100,000) HBsAg-negative	HBsAg-positive
Africa				
Democratic Republic of Congo	Manjula et al. 2009[b]	0.07–27	0.0007–0.27	0.02–8.10
Ethiopia	Ayalew et al. 2006[b]	1.4–36	0.01–0.36	0.42–10.8
The Gambia	Hall and Wild 1994; Shephard 2008	4–115	0.04–1.15	1.20–34.5
Kenya	Hall and Wild 1994; Shephard 2008	3.5–133	0.04–1.33	1.05–39.9
Mozambique	Hall and Wild 1994	39–180	0.39–1.80	11.7–54.0
Nigeria	Bandyopadhyay et al. 2007; Bankole and Mabekoje 2004[b]	139–227	1.39–2.27	41.7–68.1
South Africa	Hall and Wild 1994; Shephard 2003	0–17	0–0.17	0–5.10
Tanzania	Manjula et al. 2009[b]	0.02–50	0.0002–0.50	0.06–15.0
Zimbabwe	IPCS/WHO 1998	17.5–42.5	0.18–0.43	5.25–12.8
In general[c]	Hall and Wild 1994; Shephard 2008	10–180	0.10–1.80	3.0–54.0
North America				
Canada	Kuiper-Goodman 1995	0.2–0.4[d]	0.002–0.004	0.06–0.12
United States	IPCS/WHO 1998	0.26	0.003	0.08
In general[c]		0.26–1	0.003–0.01	0.08–0.3
Latin America				
Argentina	Etcheverry et al. 1999; Solovey et al. 1999[b]	0–4	0–0.04	0–1.20
Brazil	IARC 2002; Midio et al. 2001; Oliveira et al. 2009; Vargas et al. 2001[b]	0.23–50	0.002–0.50	0.07–15.0
Mexico	García and Heredia 2006; Guzmán-de-Peña and Peña-Cabriales 2005; Torres et al. 1995[b]	14–85	0.14–0.85	4.20–25.5
In general[c]		20–50	0.20–0.50	6.0–15.0
Eastern Mediterranean				
Egypt	Anwar et al. 2008[b]	7–57	0.07–0.57	2.1–17.1
Iran	Hadiani et al. 2009; Mazaheri 2009[b]	5–8.5	0.05–0.09	1.50–2.55
Pakistan	Munir et al. 1989[b]	7–50	0.07–0.50	2.10–15.0
Sudan	Omer et al. 1998	19–186	0.19–1.86	5.70–55.8
In general[c]		10–80	0.10–0.80	3.00–24.0
Southeast Asia				
India	Vasanthi 1998	4–100	0.04–1.00	1.20–30.0
Indonesia	Ali et al. 1998; IARC 2002; Noviandi et al. 2001[b]	9–122	0.09–1.22	2.7–36.6
Thailand	Hall and Wild 1994; Lipigorngoson et al. 2003[b]	53–73	0.53–0.73	15.9–21.9
In general[c]		30–100	0.30–1.00	9.00–30.0
Western Pacific				
Australia	NHMRC 1992; Pitt and Tomaska 2001	0.15–0.18	~0.002	~0.05
China	Li et al. 2001; Qian et al. 1994; Wang and Liu 2007; Wang et al. 2001[b]	17–37	0.17–0.37	5.10–11.1
Malaysia	Ali et al. 1999; IARC 2002[b]	15–140	0.15–1.4	4.5–42
Philippines	Ali et al. 1999; IARC 2002; Sales and Yoshizawa 2005[b]	44–54	0.44–0.54	13.2–16.2
Republic of Korea	Ok et al. 2007; Park et al. 2004	1.2–6	0.01–0.06	0.36–1.80
In general[c]		15–50 (except Australia and New Zealand)	0.15–0.50	4.5–15.0
Europe				
Eastern Europe	Malir et al. 2006[e]	3.5–4	0.04	~1.20
Southern Europe	Battilani et al. 2008; Giray et al. 2007[f]	0–4	0–0.04	0–1.20
Western Europe	IARC 2002	0.3–1.3	0.003–0.01	0.09–0.39
In general[c]		0–4	0–0.04	0–1.2

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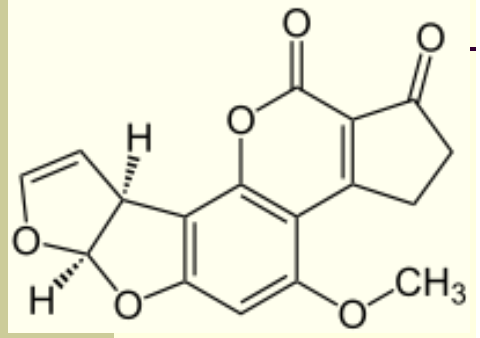


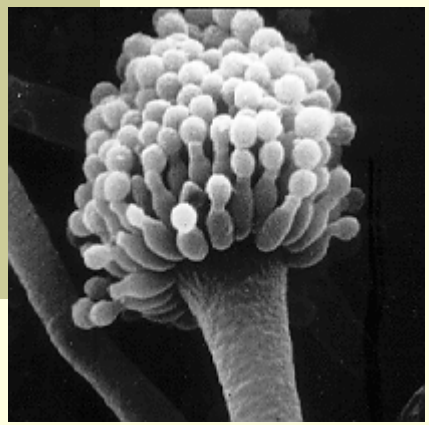


Table 4. Estimated annual global burden of HCC cases attributable to aflatoxin exposure in HBsAg-positive and HBsAg-negative populations.

WHO region/country	Population (millions)[a]	Annual HCC cases	
		HBsAg-negative	HBsAg-positive
Africa			
Democratic Republic of Congo	68	1–173	1–551
Ethiopia	85	11–288	21–643
The Gambia	1.7	1–17	3–117
Kenya	38	11–450	44–2,270
Mozambique	21	73–361	111–1,200
Nigeria	149	1,800–2,940	8,200–13,400
South Africa	48	0–79	0–255
Tanzania	41	1–195	1–554
Zimbabwe	13	19–50	68–249
Total region	755	2,150–9,300	9,230–50,600
North America			
Canada	33	1	1
United States	300	8	1–5
Total region	333	9	2–5
Latin America			
Argentina	40	0–16	0–5
Brazil	190	4–930	3–969
Mexico	109	152–924	14–83
Total region	562	589–2,980	84–2,060
Eastern Mediterranean			
Egypt	81	51–452	37–1400
Iran	66	33–56	4–9
Pakistan	172	116–832	119–851
Sudan	41	58–717	140–5,950
Total region	569	446–3,720	341–13,200
Southeast Asia			
India	1,150	438–11,200	331–16,200
Indonesia	237	203–2,820	160–4,340
Thailand	63	307–439	461–1,100
Total region	~1,734	1,740–17,300	1,460–27,600
Western Pacific region			
Australia	21	0–1	0–1
China	1,300	1,990–4,430	5,300–14,400
Korea	50	5–29	6–45
Malaysia	28	40–372	63–588
Philippines	90	333–462	594–2,330
Total region	~1,740	2,710–6,510	6,310–21,200
Europe			
Eastern Europe	290	94–114	61–244
Southern Europe	144	0–56	0–121
Western Europe	183	5–24	1–7
Total region	617	99–184	62–372
Total (world)	6,280	7,700–40,000	17,500–115,000
Total annual HCC cases attributable to aflatoxin worldwide		25,200–155,000	

[a]Data from Central Intelligence Agency 2009.

	HBV alone	AFB_1 alone	HBV and AFB_1
	RR (95% CL)*	RR (95% CL)	RR (5% CL)
[65]	4.8 (1.2 -19.7)	1.9 (0.5 - 7.5)	60.1 (6.4 - 561.8)
[64]	7.3 (2.2 - 24.4)	3.4 (1.1 - 10.0)	59.4 (15.6 - 212)
[80]	17.4 (3.6 – 143.4)	0.3 (0 - 3.6)	70.0 (11.5 – 425.4)
[54]	17.0 (2.8 - 103.9)	17.4 (3.4 - 90.3)	67.6 (12.2 - 373.2)
[59]	3.3 (1.3 - 8.3)	32.0 (4.0 - 255.8)	40.7 (12.7- 130.9)

Table 1. Findings in five studies comparing the risk of HBV infection alone, dietary exposure to AFB1 alone, and the two risk factors together in the genesis of HCC. * Relative risk (95% confidence limits).

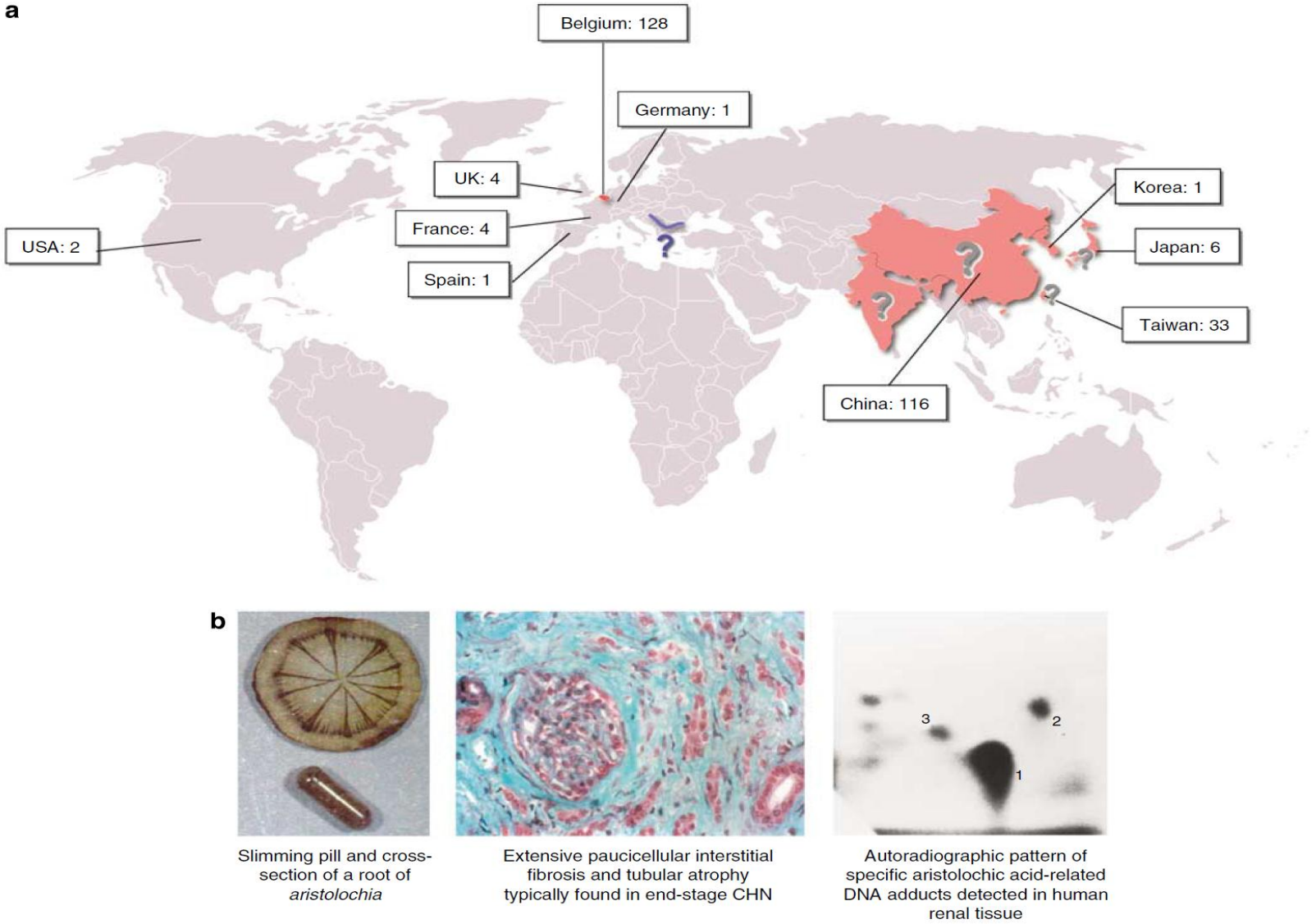


Figure 1 | Aristolochic acid nephropathy: a worldwide problem. (**a**) Counting cases of CHN/AAN around the world reported in the literature.[1–4,6–23] (**b**) CHNA/AAN is a rapidly progressive interstitial nephritis leading to end-stage renal disease and urothelial malignancy, which was originally reported in Belgium in the context of the intake of slimming pills containing powdered Chinese herbs (*A. fangchi*). (**c**) The true incidence of AAN is largely unknown and probably underestimated, as numerous ingredients known or suspected to contain AA are used in traditional medicines in India and Eastern Asia (see Tables 1–3 for more details). (**d**) Finally, another reason to suspect a higher number of AAN cases is based on the hypothesis that the exposure to seeds of *Aristolochia clematitis* comingled with wheat grain during the annual harvest could be responsible for BEN.

Aristolochic acid-associated urothelial cancer in Taiwan

Aristolochic acid, a potent human carcinogen produced by Aristolochia plants, is associated with urothelial carcinoma of the upper urinary tract (UUC). Following metabolic activation, aristolochic acid reacts with DNA to form aristolactam (AL)-DNA adducts. These lesions concentrate in the renal cortex, where they serve as a sensitive and specific biomarker of exposure, and are found also in the urothelium,where they give rise to a uniquemutational signature in the TP53 tumor-suppressor gene. Using AL-DNA adducts and TP53 mutation spectra as biomarkers,we conducted a molecular epidemiologic study of UUC in Taiwan, where the incidence of UUC is the highest reported anywhere in the world and where Aristolochia herbal remedies have been used extensively for many years. Our study involves 151 UUC patients, with 25 patients with renal cell carcinomas serving as a control group. The TP53mutational signature in patients with UUC, dominated by otherwise rare A:T to T:A transversions, is identical to that observed in UUC associated with Balkan endemic nephropathy, an environmental disease. Prominent TP53 mutational hotspots include the adenine bases of 5′AG (acceptor) splice sites located almost exclusively on the nontranscribed strand. A:T to T:Amutations also were detected at activating positions in the FGFR3 and HRAS oncogenes. AL-DNA adducts were present in the renal cortex of 83% of patients with A:T to T:A mutations in TP53, FGFR3, or HRAS. We conclude that exposure to aristolochic acid contributes significantly to the incidence of UUC in Taiwan, a finding with significant implications for global public health.

Table 2. AL-DNA adducts and *TP53* mutations in UUC cases from Taiwan

Cases	All subjects	Males	Females	Males vs. females
Cases with AL-DNA adducts	89/148 (60%)	45/82 (55%)	44/66 (67%)	$\chi^2 = 2.12$ $P = 0.1454$
Cases with *TP53* mutations	84/151 (56%)	34/82 (41%)	50/69 (72%)	$\chi^2 = 14.59$ $P = 0.0001$
Cases with *TP53* A→T transversions	47/151 (31%)	17/82 (21%)	30/69 (43%)	$\chi^2 = 9.04$ $P = 0.0026$
Cases with AL-DNA adducts and *TP53* A→T transversions	38/148 (26%)	14/82 (17%)	24/66 (36%)	$\chi^2 = 7.13$ $P = 0.0076$
AL-DNA adduct-positive cases with *TP53* A→T transversions	38/89 (43%)	14/45 (31%)	24/44 (55%)	$\chi^2 = 4.99$ $P = 0.0254$
TP53 A→T transversion cases with AL-DNA adducts	38/45 (84%)	14/17 (82%)	24/28 (86%)	$\chi^2 = 0.091$ $P = 0.7629$

www.pnas.org/cgi/doi/10.1073/pnas.1119920109

癌病對個人及社會的衝擊！

101年十大死因

1	惡性腫瘤	28.4%
2	心臟疾病(高血壓性疾病除外)	11.1%
3	腦血管疾病	7.2%
4	肺炎	6.1%
5	糖尿病	6.0%
6	事故傷害	4.5%
7	慢性下呼吸道疾病	4.1%
8	高血壓性疾病	3.2%
9	慢性肝病及肝硬化	3.2%
10	腎炎、腎病症候群、腎病變	2.8%

101年十大癌症死因

1	氣管、支氣管和肺癌	19.7%
2	肝和肝內膽管癌	18.6%
3	結腸、直腸和肛門癌	11.8%
4	女性乳房癌	4.4%
5	口腔癌	5.9%
6	胃癌	5.5%
7	前列腺癌	2.7%
8	胰臟癌	3.7%
9	食道癌	3.6%
10	子宮頸及部位未明示子宮癌	1.5%

資料來源：衛生署　製表：記者邱宜君

死亡時鐘 101年國人10大死因

死亡原因	平均每日死亡人數	每隔多久1人死亡
所有原因	421	3分25秒
惡性腫瘤	120	12分2秒
心臟疾病	47	30分41秒
腦血管疾病	30	47分31秒
肺炎	26	56分25秒
糖尿病	25	56分37秒
事故傷害	19	1小時16分28秒
慢性下呼吸道疾病	17	1小時23分5秒
高血壓性疾病	14	1小時45分24秒
慢性肝病及肝硬化	14	1小時45分38秒
腎炎、腎病症候群及腎病變	12	2小時1分28秒

資料來源：衛生署　製表：黃天如

可控制的致癌危險因子

- 食物 35%
- 香菸 30%
- 酒精 5%
- 職業 4%
- 陽光 3%
- 病毒 5%
- 其他 12%

某種傷害=危害1+危害2+危害3+危害4+危害5+----

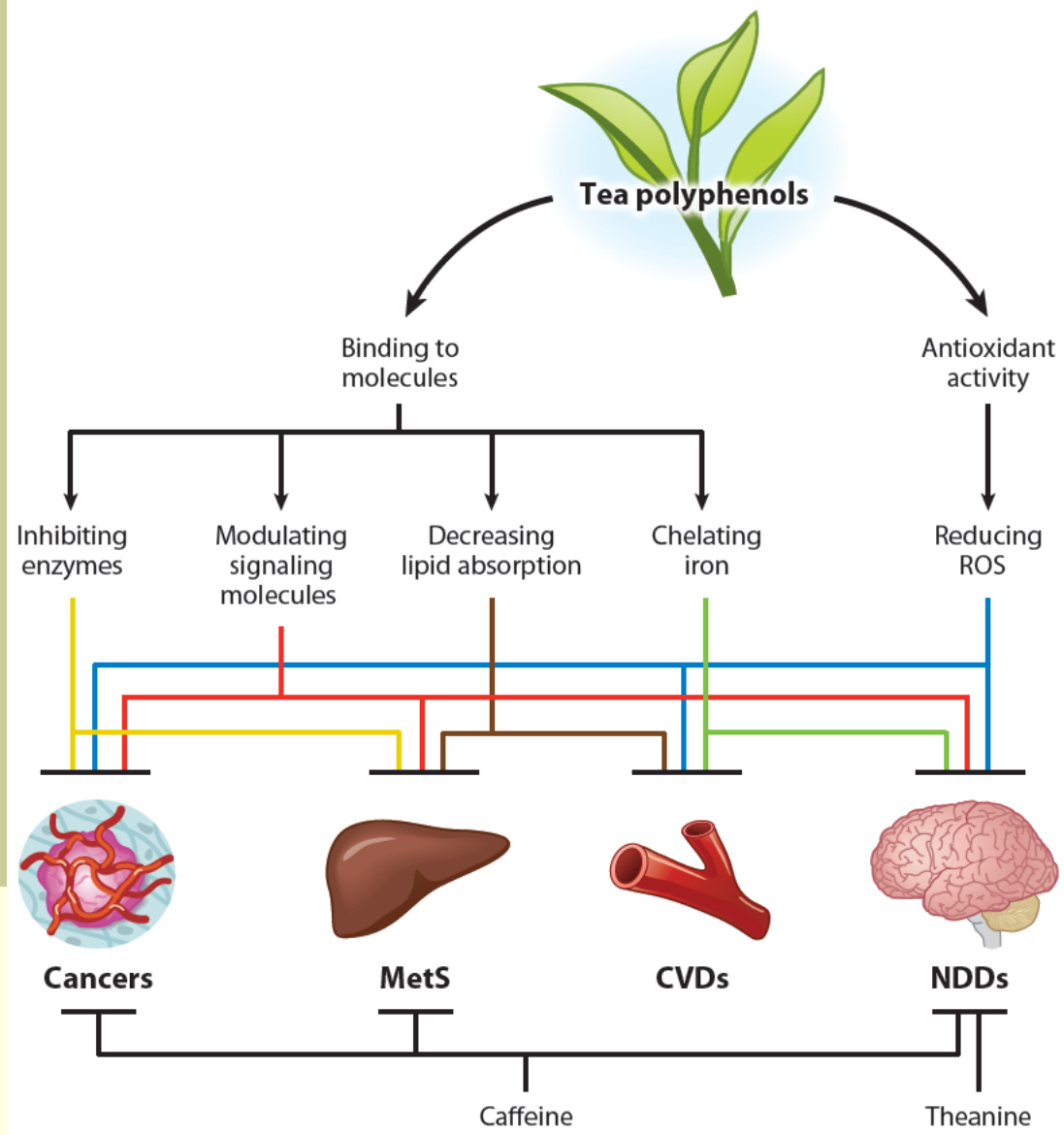
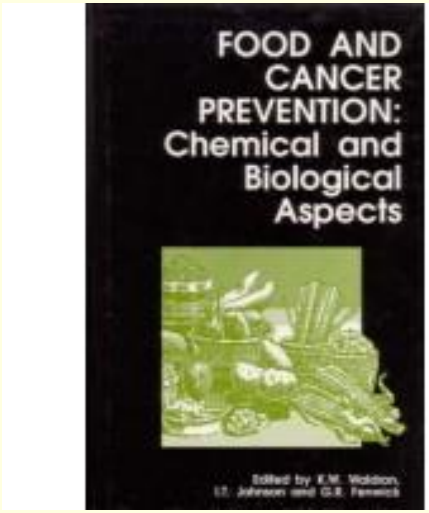
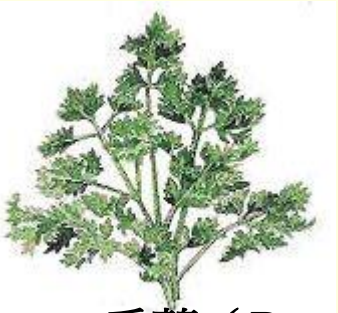


Figure 3

The proposed mechanisms by which tea constituents (polyphenols, caffeine, and theanine) prevent chronic diseases. Abbreviations: CVDs, cardiovascular diseases; MetS, metabolic syndrome; NDDs, neurodegenerative diseases; ROS, reactive oxygen species.



- **A regular diet including apiaceous vegetables (傘形科蔬菜)such as carrots, parsnips(美國食用芹菜), celery and parsley, may reduce the carcinogenic effects of aflatoxin (Food and Chemical Toxicology 44 (2006) 1474–1484)**



香菜（**Parsley**）

芫荽

茴香

Cruciferous Vegetables

- Arugula (芝麻)
- Bok choy
- Broccoli
- Brussels sprouts
- Cabbage
- Cauliflower
- Collard greens
- Horseradish(辣根)
- Kale
- Radishes
- Rutabaga(大頭菜)
- Turnips(蘿蔔)
- Watercress(豆瓣)
- Wasabi

Cruciferous Vegetables and Cancer Prevention

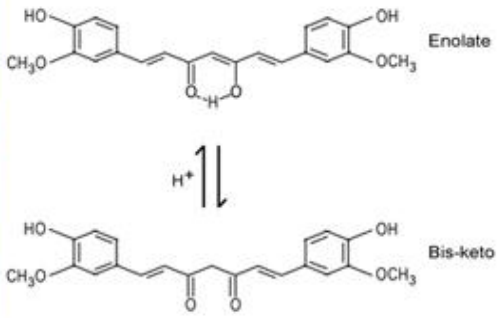
- Cruciferous vegetables rich in nutrients, including several carotenoids (beta-carotene, lutein, zeaxanthin); vitamins C, E, and K; folate; and minerals, and chemicals known as glucosinolates(芥子油苷). They also are a good fiber source .
- Glucosinolates break down into several biologically active compounds that are being studied for possible anticancer effects.
- Some of these compounds have shown anticancer effects in cells and animals, but the results of studies with humans have been less clear.

Glucosinolates in cruciferous vegetables

- Glucosinolates are broken down to form biologically active compounds such as indoles, nitriles, thiocyanates, and isothiocyanates during food preparation, chewing, and digestion.(Hayes et al. *European Journal of Nutrition* 2008;47 Suppl 2:73-88)
- protect cells from DNA damage.
- inactivate carcinogens.
- have anti-inflammatory effects.
- induce cell death (apoptosis).
- inhibit tumor blood vessel formation (angiogenesis) and tumor cell migration (needed for metastasis).

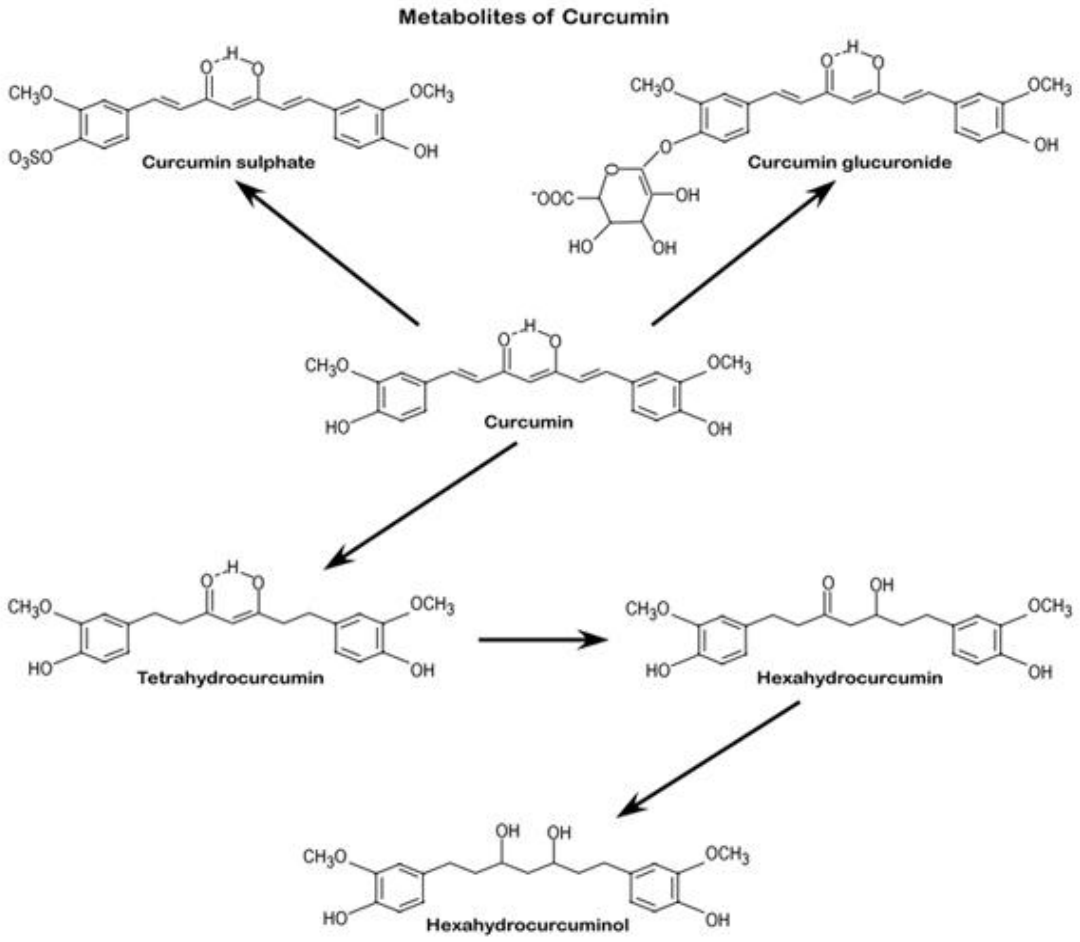
A

Turmeric Plant and Rhizome

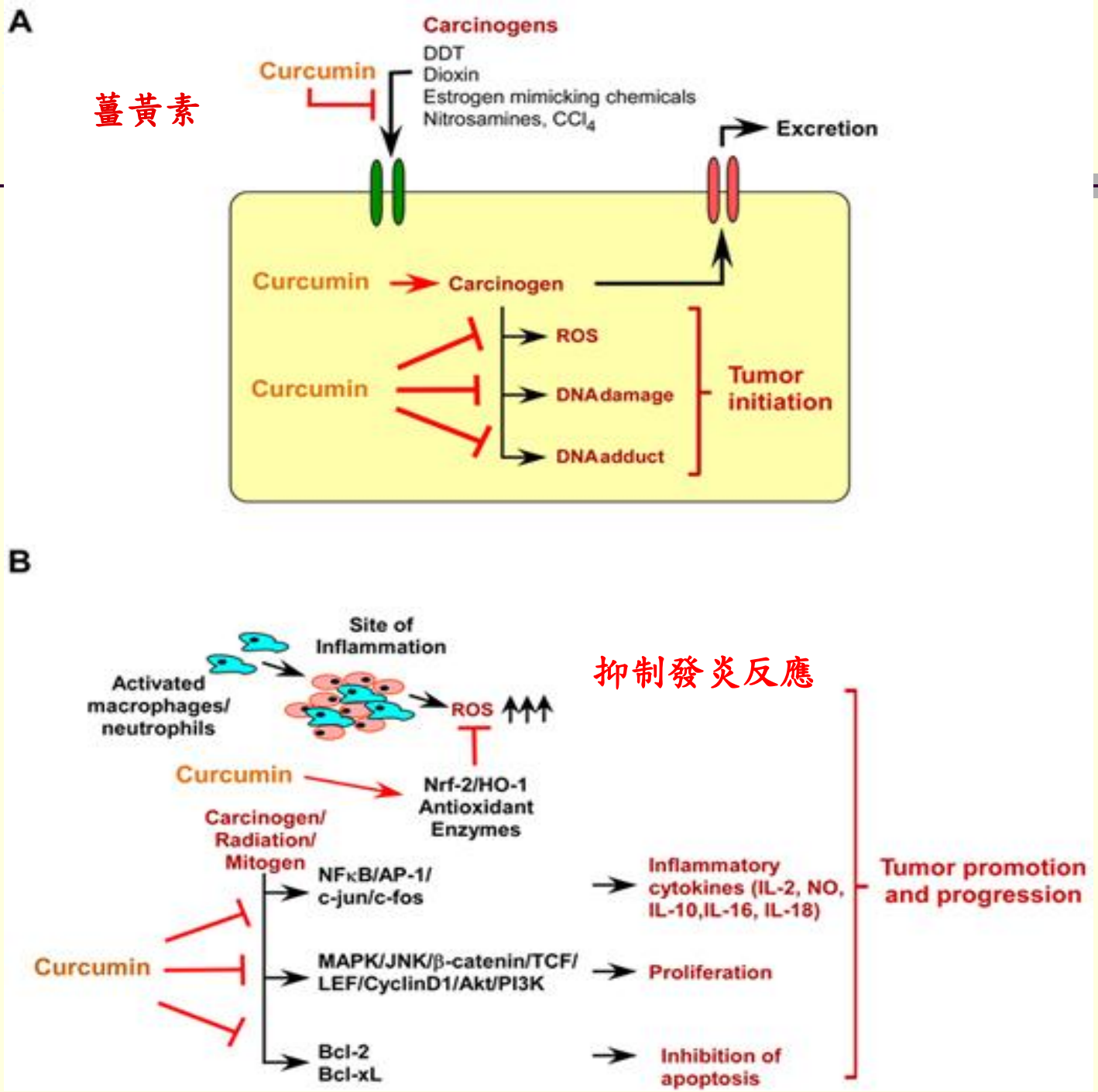


Curcumin

B



薑黃素


A
Carcinogens
DDT
Dioxin
Estrogen mimicking chemicals
Nitrosamines, CCl_4
Curcumin
Excretion
Curcumin
Carcinogen
Curcumin
ROS
DNAdamage
DNAadduct
Tumor initiation
B
Site of Inflammation
Activated macrophages/ neutrophils
ROS
Curcumin
Nrf-2/HO-1 Antioxidant Enzymes
Carcinogen/ Radiation/ Mitogen
NFκB/AP-1/ c-jun/c-fos
Curcumin
MAPK/JNK/β-catenin/TCF/ LEF/CyclinD1/Akt/PI3K
Bcl-2 Bcl-xL
Inflammatory cytokines (IL-2, NO, IL-10,IL-16, IL-18)
Proliferation
Inhibition of apoptosis
Tumor promotion and progression

抑制發炎反應

The dose makes the poison.

Paracelcus (1493-1541,現代毒物學之父)：Everything is a poison, there is nothing which is not. Only the dose differentiates a poison from a remedy.

Table 3. Rodent Carcinogens in the Natural Chemicals Present in Roasted Coffee

Carcinogens: N=21	acetaldehyde, benzaldehyde, benzene, benzofuran, benzo(*a*)pyrene, caffeic acid, catechol, 1,2,5,6-dibenzanthracene, ethanol, ethylbenzene, formaldehyde, furan, furfural, hydrogen peroxide, hydroquinone, isoprene, limonene, 4-methylcatechol, styrene, toluene, xylene
Noncarcinogens: N=8	acrolein, biphenyl, choline, eugenol, nicotinamide, nicotinic acid, phenol, piperidine
Uncertain:	caffeine
Yet to test:	~1,000 chemicals

Source: Carcinogenic Potency Database (http://potency.berkeley.edu); Gold and Zieger, *Handbook of Carcinogenic Potency.*

Errors of omission. *The major causes of cancer (other than* smoking) do not involve exposures to exogenous chemicals that cause cancer in high-dose tests; rather, **the major causes are dietary imbalances(飲食均衡), hormonal factors(荷爾蒙), infection(感染) and inflammation(發炎), and genetic factors(遺傳). Insufficiency of many vitamins and minerals, which is preventable by supplementation, causes DNA damage by a mechanism similar to radiation.**

Ames, B.N. and Gold, L.S. Cancer Prevention and the Environmental Chemical Distraction In: Politicizing Science: the Alchemy of Policymaking. Michael Gough ed., Hoover Institute Press, Stanford, California. pp 117-142.

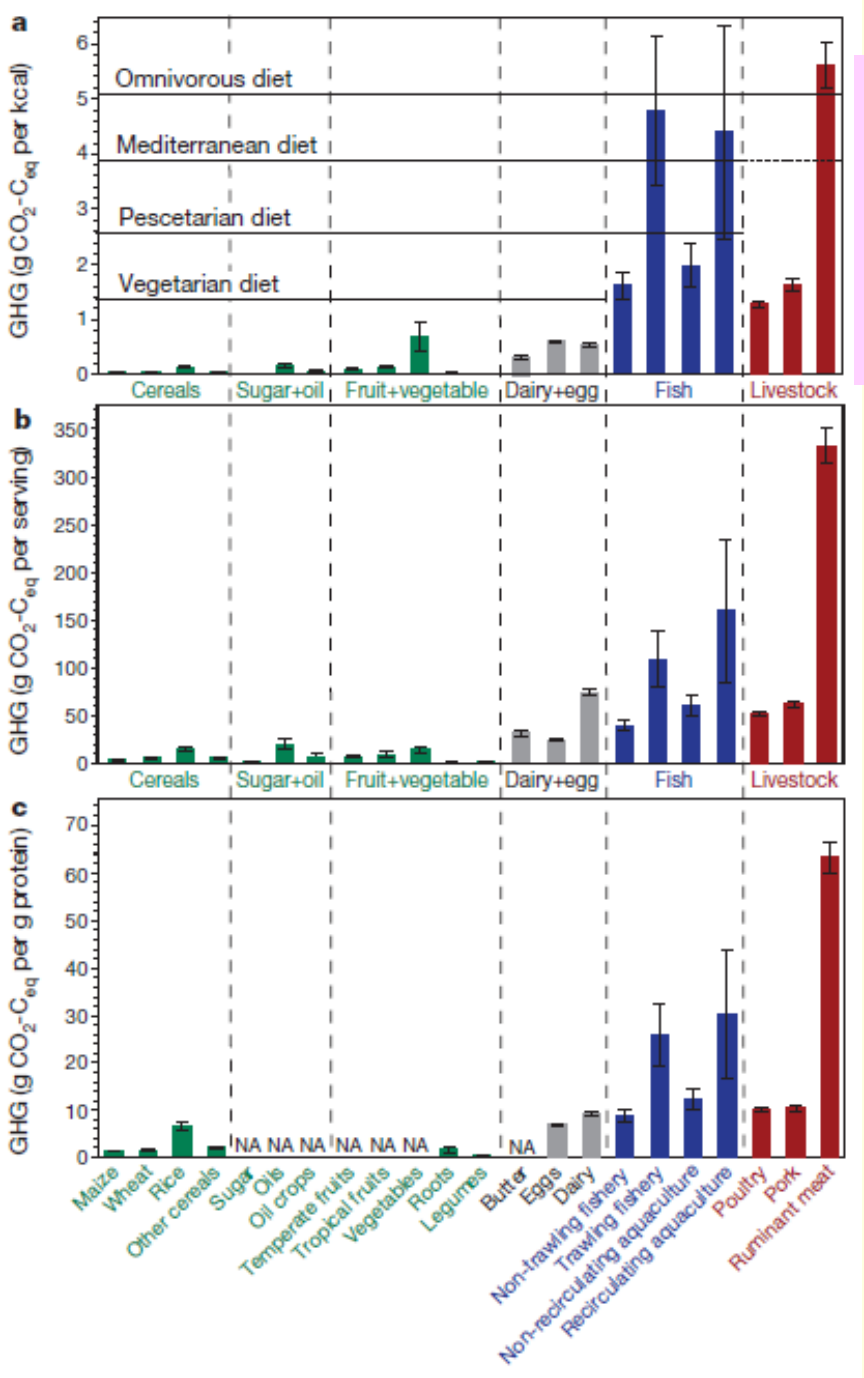


Figure 1 | Lifecycle GHG emissions (CO2-Ceq) for 22 different food types.The data are based on an analysis of 555 food production systems: a, per kilocalorie; b, per United States Department of Agriculture (USDA)-defined serving; c, per gram of protein. The mean and s.e.m. are shown for each case. Extended Data Tables 1–3 list data sources, items included in each of the 22 food types and show the mean, s.e.m. and number of data points for each bar, respectively. NA, not applicable.

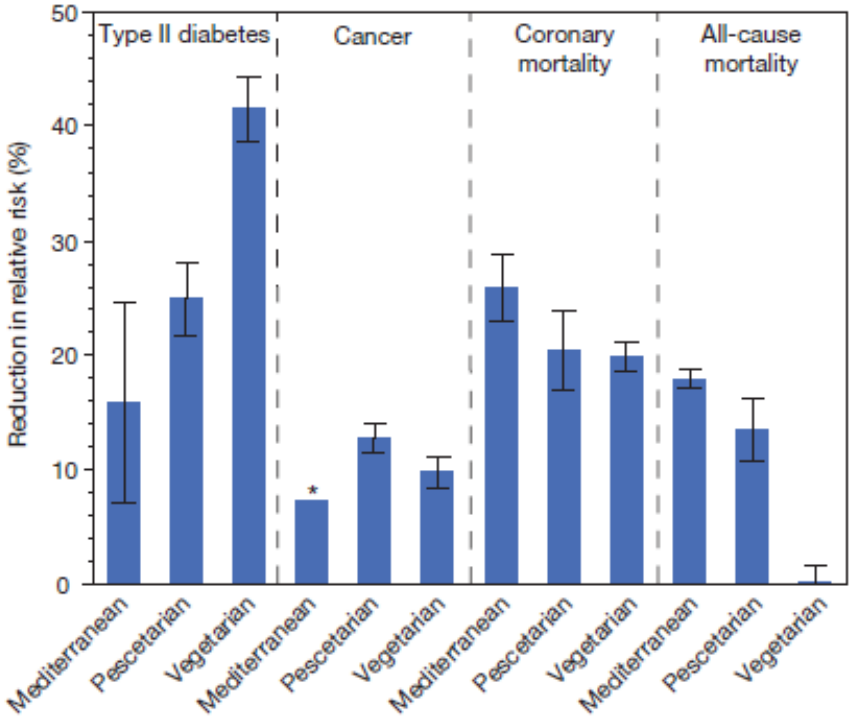
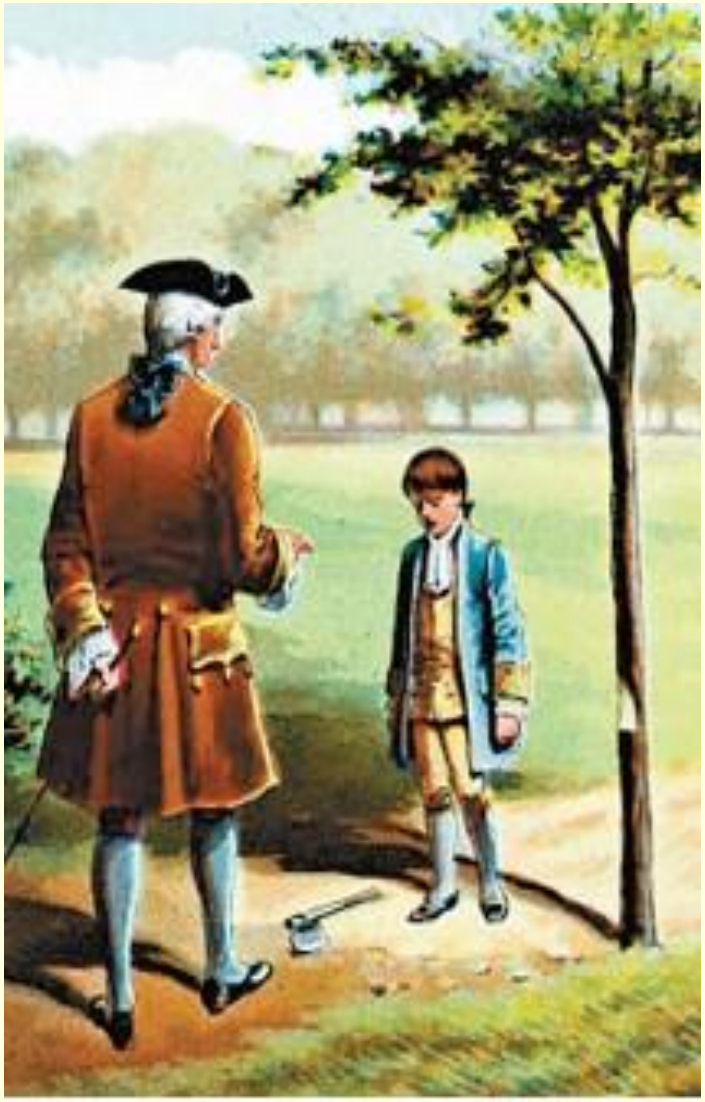


Figure 3 | Diet and health. Diet-dependent percentage reductions in relative risk of type II diabetes, cancer, coronary heart disease mortality and of all-cause mortality when comparing each alternative diet (Mediterranean, pescetarian and vegetarian) to its region's conventional omnivorous diet (Methods). Results are based on cohort studies[32–39]. The mean and s.e.m. values shown are weighted by person-years of data for each study. Number of studies for each bar are, from left to right, 3, 2, 2, 1, 2, 2, 4, 2, 5, 13, 2 and 4. *Cancer in Mediterranean diets is from a single study so no s.e.m. is shown.


誠信




馬總統，你的鼻子是不是
又變長了?!
提名
恐龍
大法官
馬英九
說謊
他要看報紙
才知道自己說謊!!
2011.4.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THE LONG NOSE
PINOCCHIO
政見跳票
施政無方
馬英九

謝謝各位的用心聆聽
歡迎批評指教