

I. 가

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, 가 가 가 가 가
1% , '95
가 가 가 가 가
가 가 가 가 가
가 가 가 가 가
2000 2004 가 1
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1).
가 가 가 가 가
가 가 1 5
, 5 가 2001

1)

II.

1.

1) 50

가

가 , 50

가가

- 2001 7 44,481 35,083 9,398 (26.8%) ,
0.46% 0.42% 0.04% 가 가

< 3 > 2001 7

	()	()	()	(%)
2001 7	9,682,607	44,481	1,398	0.46
2000 7	8,437,260	35,083	1,363	0.42
	1,245,347	9,398	35	0.04

- 2001 7 , 50 (30,541) (782)
68.7% 61.6% , 7.1%
7.5% 가 가

< 4 > 2001 7

	()	()	()	()	(%)
	764,213	9,682,607	44,481	1,398	0.46
50	737,824 (96.5%)	4,292,959 (44.3%)	30,541 (68.7%)	782 (55.9%)	0.71
50	26,389 (3.5%)	5,389,648 (55.7%)	13,940 (31.3%)	616 (44.1%)	0.26

50

2)

. . 가 2000

51%

- (27.9%), (13.8%), (11.9%), (8.1%), (7.7%)

2.

1) 50

가

50% 50 , 가 2),

- 2000 3,414 2,732 59.2%가 가 , , 1999
51% 50 3)

< 5 > ('96 '00)

	'96	'97	'98	'99	'00
(%)	8.77	8.11	6.79	7.45	7.27
()	1,529	2,119	1,838	2,732	3,414

가 . 가

4)

- 2000 1,666 815 137% 가,
37%가 가 2001

< 6 > ('99 '01 6)

									가 (%)
2001 6	2,668	575	2,093	905	412	493	1,037	151	40.2
2000	3,414	933	2,481	815	394	421	1,666		59.2
1999	2,732	1,174	1,558	344	161	183	1,214		-

2) '98

IMF

3)

. 1999

. 2001.7

4)

가

(VDT)

가

가, , , , 가, , , , 가,

2)

2001 933 1,174 241 (20.5%)

< 7 > ('99 '01 6)

							†
2000	933	425	251	17	21	28	191
1999	1,174	736	204	24	57	22	131

† :

- : 157 ('97) 192 ('98) 234 ('99) 257 ('00)

, 1999 575 , 157 (27.3%),
298 (51.8%), 82 (14.3%), 38(6.6%) 5)

, '00 1,645 '99 1,276
369 (28.9%) 가 , 가가 가 6)

- 20% 10%

< 8 >

2000	1,645 (100%)	266 (16.2%)	159 (9.6%)
1999	1,276 (100%)	245 (19.2%)	148 (11.6%)
	369	21	11

- , '99 (80.7%) (14.3%) 95.0%

5) 198 , 11 , 149 , 6 , 60 ,
6 , 1 , 66 , 25

6) : 536,239 ('99) 628,916 ('00) 247,627 ('01 6)

III.

1. CLEAN 3D

1)

50 (740,000) 4,312,000 5
가 가

- 162,000 (22.7%) 3D

< 7 > (2001.6)

		50 ()	30
()	767,117	740,384(96.5%)	47,107
()	9,815,688	4,312,229(43.9%)	6,274,969

- 50 0.59% (0.38%) 1.55 , ()
) 68.2%(56.2%) 가 91%가 50 가

- 11,928 가 , 50 46.5%¹¹⁾

- , 12)

《 『 CLEAN 3D ^{anger} ^{irtiness} ^{ifficulty} 』 》

(CLEAN)	(3D)
- Danger () :	()
- Dirtiness () :	()
- Difficulty () :	()

11) (2,152), (2,003), (1,115), (784), (690)

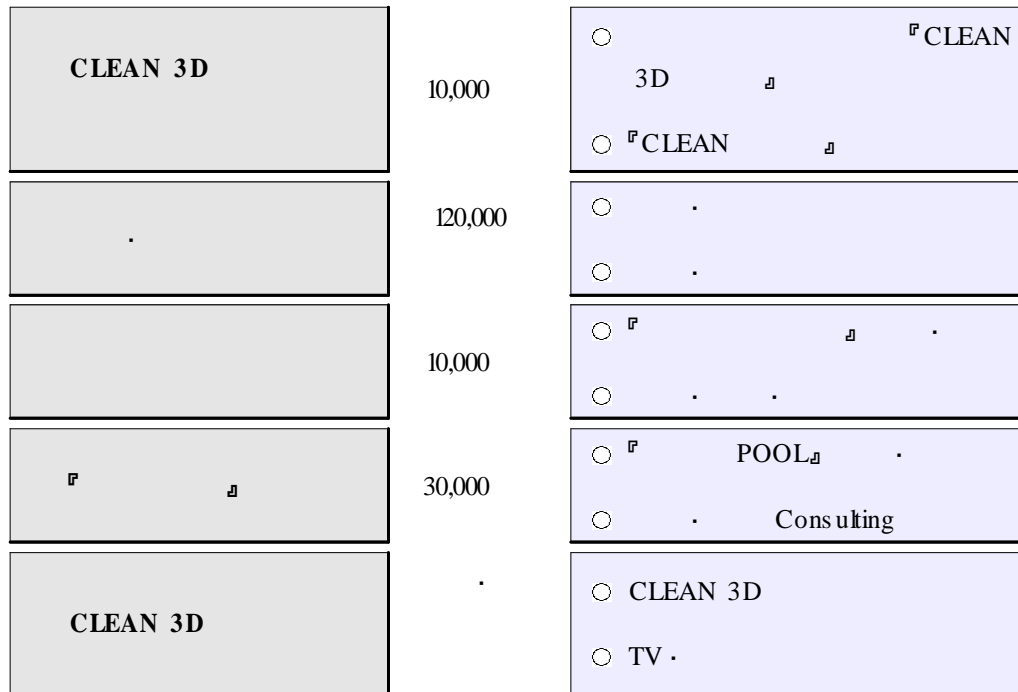
12) 2001.4.1 300 3D 2.74% 5 1.28% 2.14 < :
>

- '98 50 () 73%(82%)
 13)

- .

- 가 14)

2)



< 1 > CLEAN 3D

가. CLEAN 3D

CLEAN 3D

: 3D 10,000

13) 0.37% 0.21% 1.8 (1,173) 57%(668), ()
 14) '99 5 가 28%

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- 5 가 15)

- CLEAN 3D 17 () 16)

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- CLEAN 3D 1

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CLEAN 3D

- CLEAN . . 가

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CLEAN

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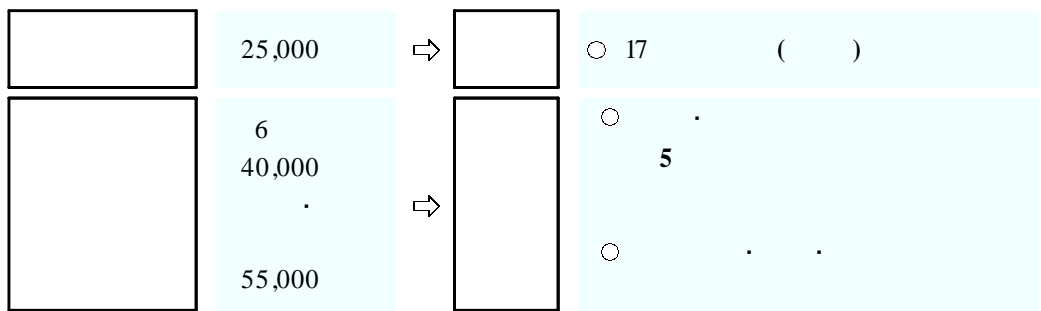
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- . , . CLEAN 3D

15)

16) . ,

- 가
- : 50 120,000
- (25,000) : 2 가 5
- (95,000) : 6 17)



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- 18) 19)
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- 17) , , , , ,
 18) · 6 (3 1)
 19)

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- 『 POOL』 . 20)
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20) .

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2000 (421), 2,481 (545) '99 59.2% 가 , (349), (1,666)

- 2001 2/4 2,093 (1,493) 40.2% 가

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- : 가,

3 23) , ('01.8 1,995)

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21

가

27) , 가

28) 가 (101)

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		31		
		92	2001. 4. (113)	
		7		가
		2001		
		2001		
		43		
		2001		

2)

가

가

29)

29)

가

가

가

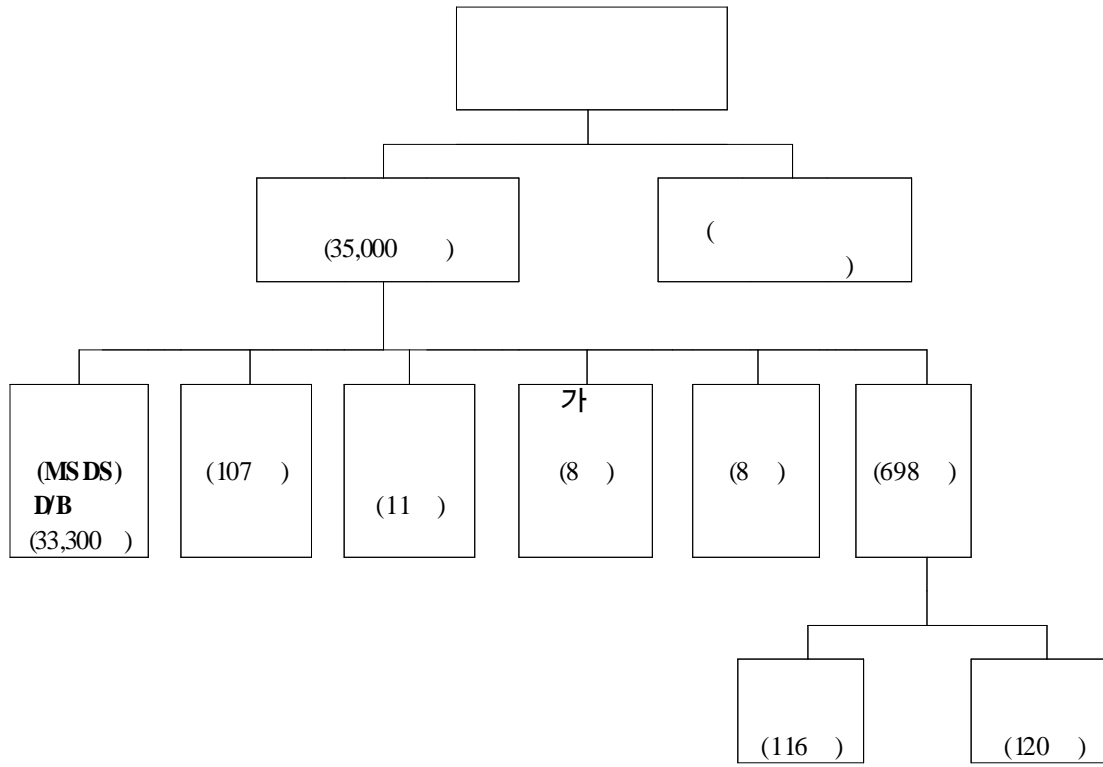
6.

1)

MSDS

698 , 가

116 .



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(2001 6)

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30) 1986 3

- (MSDS) 31)

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- (1 3), (1 3) 33)

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- ILO UNEP() ‘ , ’

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ILO 가

-) 9 , '02 2 3 가 가 (162 17

31) , , .

32) .

33) 1 3 , , .

7.

1)

가.

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: 6 1 , 6

- '00 / : 25,338 /25,075 (99%)

- '00 / : 25,338 /6,440 (25%)

34)

- 2000 95% .

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- 가 35)

< 9 > 2000

		6,440	5,637	803
	()	13,065	11,601	1,464
		478	349	129
		230	171	59
	. .	191	148	43
		11,369	10,271	1,098
		64	47	17
		149	115	34
		13	8	5
		85	492	81

34) . 가 (92)

35) 111 (15)

2)

가.

가

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- 가 (.)³⁶⁾

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가

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- . 38)

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36) , , .

37) 가

38) '00 87%

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(STEL) (Ceiling) . 가 , (Action level) 39)

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(consulting)

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2002

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가

가

50%

44)

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- 41) ('97), . ('98), ('99),
('00)
 - 42) Full-time ('00 12) : 488 , 2,003 , 162 ,
195 , 2,088
 - 43) ('00 12) : 1,900 , 105 ,
81 , 111

< 10 >

	'92	'93	'94	'95	'96	'97	'98	'99	'00
	1,328	1,418	918	1,120	1,529	2,119	1,838	2,732	3,414
	25	26	23	57	32	44	64	108	127
	10	6	9	48	13	24	32	50	52

'00. 12. 31

354

가

-

9.6% 34

, 46

가

가 50.0% 23

2)

-

20%

10

가

44)

가

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1, 2, 3, 4, 5, 1, 6, 7
 5, 8, 9, 10, 8, 11
 1, 2, 3
 4, 5, 6
 7, 가 8, 9
 10, 11

1.

90 가 4-5
 가 가 .
 , 가 (, 1996; , 1997;
 ,1997; , 1997), 가
 가(, 1998)가 , 가 (,
 1998; . 1999; , 1999; ,2000; , 2000; , 2001). 1,2
 (,
 1999; , 2000),
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 (epidemiological surveillance)
 , , 가
 . ' (surveillance)'
 ' (monitoring)'
 (epidemiologic surveillance)'
 , 가
 , 가 , , , , ,
 가 가 . ,
 . (Centers for Disease Control and prevention,
 CDC) ' , 가
 , ,
 ' (CDC, 1986).
 , 가
 (Thacker Berkelman, 1988).

가 90 가

가 . 가 , 가

가 가 ,

95% (, 2000).

가 가 가

(Mass Health Screening Tool) . 가 가

가 . 가

가 , 가 ,

가 가 ,

가 ,

가 가

가 가

(National Institute for Occupational Safety and Health, NIOSH) 1987

(Sentinel Event Notification System for Occupational Risks, SENSOR)(Baker, 1988; Baker,1989)

. 1986

(Halperin Ordin, 1996).

(Wu , 1996), 90

가

(Ross ,1995; Ross , 1997). 1998 (EPIDERM), (SWORD),

(MOSS), (SIDAW), (OSSA) (SOSMZ)

(OPRA)가 2000 가 20000

(Baker, 1989).

(6.2%)
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 130 17
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 1998, 1999, 2000 가
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 (16) (40) 97-65
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1.

Occupational Diseases	1998 N(%)	1999 N(%)	2000* N(%)	Agent	Industry/process/occupation
C o n t a c t dermatitis	40(31.1)	15(15.3)	13(8.9)	Irritant(e.g. cutting oil, solvents, phenol, Acid, Alkali, detergents) Allergen(Nickel, dye formaldehyde, rubber)	metal processing industry, Leather tanning, printing industry, wood processing industry, Nickel plasters, Foam workers
Occupational Asthma	27(20.9)	19(19.4)	33(22.6)	TDI, reactive dye, grain dusts	Polyurethane, adhesive, paint workers, Wood workers, dye manufacturer, feed handlers, grain handlers
Cumulative trauma disorder	53(41.1)	57(58.2)	67(45.9)	cumulative trauma	VDT workers, mail sorters, poultry processing industry, machine assemblers, welder, simple laborers, Meat packers
o t h e r respiratory diseases	3(2.3)	3(3.1)	6(4.1)	coal dust, Manganese	welders, mining, milling, cement industries
hematologic diseases	6(4.6)	-	2(1.4)	Organic solvents(toluene, trichloethylene), Lead ,etc	Leather tanning, printing industry, wood processing industry, electric industry
liver disease	-	2(2.0)	21(14.3)	organic solvents(toluene, phenol)	printing industry, wood processing industry,
r e n a l diseases	-	1(1.0)	-	diclomethane	chemical industry
neurologic disease	-	1(1.0)	1(0.7)	Organic solvents (TCE, Toluene)	dye industry, wood processing industry
Occupational ** cancer			2(1.4)	alachlor,butachlor, silica	pesticide industry, silica processing industry
e t c (M e t a l fume fever)			1(0.7)	Zn	welders
Total	129(100.0)	98(100.0)	146(100)		

*2000 1 1 - 12 15 (),

** 2000

2. 2000

K	55	()	
K	43	()	
K	44	()	
K	44	()	
J	51	()	
K	30	())
Y	44		
J	60		
J	39	()	(TCE)
K	48		

3.

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	9		4	1	2		2
	31	3	8	6	3		11
	61		7	5	12	10	27
	2				2		
()	6		5	1			
	17	14			3		
	1		1				
	1		1				
	1			1			
()	1			1			
	130	17	26	15	22	10	40

3.

가

가 (4), (, 2000; , 2001).

가 3가

1998 1 가

(Feasibility test) 가

1999 9 1 가 , , , 2000 5

가, , , (,),

(,), (http://www.ohis.net/)

가 CGI(Common Gate Interface)

가 (web database)가

가 [Sentinel Health Event (Occupational)]

(, 1999).

1998 4

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(, 1999).

2000

2001

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(<http://www.ohis.net/>)

가

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(under report)

2000 5 (), , 1999
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Clinic) (OEM Clinic; Occupational and Environmental
가 ,
가
.

(Occupational Sentinel health Events)

가 . 가
1999 1 (, 1999) 2000 2 (, 2000)

가

()

가 가 , 가

- (2). 1997;110: 37-49^b
- (3). 1997;111: 14-27^c
- (4). 1997;112: 17-27^d

, 1999

Baker EL, Honchar PA, et al. Surveillance in occupational illness and injury: concept and content. *AJPH*, 79:9-11, 1988

Baker EL. IV. Sentinel Event Notification System for Occupational Risks (SENSOR) : The Concept. *AJPH*, 79(Supp):18-20, 1989

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CDC, Case definitions for public health surveillance, *MMWR* 1990: 39; 1986.

KOEC(Korean associations of occupational and environmental clinic). <http://www.koec.org> , 2000

Halperin W, Ordin DL. Closing the Surveillance. *Am J Industrial medicine* 29:223-224,1996

OHIS(Occupatoional Health Information System). <http://www.ohis.net> ,1999

Ross DJ, Sallie BA, Mcdonald JC. SWORD '94: surveillance of work-related and occupational respiratory disease in the UK. *Occup med*;45(4); 175-178,1995

Ross DJ, Sallie BA, Mcdonald JC. SWORD '96: surveillance of work-related and occupational respiratory disease in the UK. *Occup med*;47; 377-381,1997

Thacker SB, Berkelman RL. Public health surveillance in the United States. *Epidemiol Rev* 1988;10:164-169

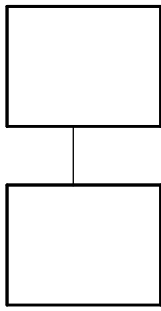
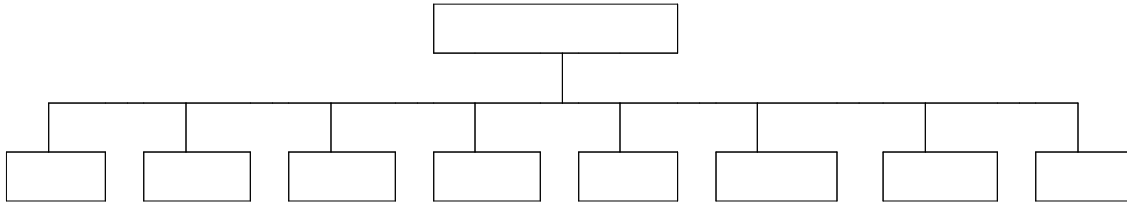
Wu TN, Liou SH, Wang JD, et al. Establishment of Work-related Diseases Surveillance System in Taiwan, Republic of China. *Preventive medicine* 25: 725-729,1996

▪ ▪

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- 3) ,
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- 6) ,
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 (Trigger finger) 4
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가, , 가
가. ,
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1) Guyon

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-Guyon , Tinel 가
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-Froment 가

2)

- (1,2,3) , ,
- / , Tinel 가
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-Phalen 가

3)

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4) ()

- (4,5) , ,
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4.

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		()		()	
			CTD(CTS)		()

(<http://oemdoctor.org>)

5. , 가

(http://oemdoctor.org)

6.

(9)

가 10 47 . 가 9 , 가 27 ,
5 1 10 가
가 5 가

7 5 1

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11 가 , 6 , TDI 3 , () 1 , 1 .

9 6 , 1 .

13 가 6 .

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(4 - 9)

	47	7	11	9	13	8
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7.

1) 가

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 (joint conference)
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2)

3), (,) 가 가 (, 가

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8.

- . 1999. . , 2000 , 2000.
1998- 15
(1). 1997;109:14- 21
- Baker EL, Honchar PA, Fine LJ. I. Surveillance in occupational Illness and Injury; Concepts and Contents. AJPB 1989 ;79(supp):9- 11.
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- 1), 2), 3), 4), 1), 5), 6), 2), 7),
- 7), 8), 9), 10)
- 1), 2), 3),
- 4), 5), 6),
- 7), 8), 9),
- 10)

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2.

가

1)

(National Institute for Occupational Safety and Health; NIOSH)
 NIOSH ABLES(Adult
 Blood Lead Epidemiology and Surveillance), SENSOR(Sentinel Events
 Notification System for Occupational Risks), NOMS(National
 Occupational Mortality Surveillance),
 BLS(Bureau of Labor Statistics), NOES(National Occupational Exposure
 Surveillance), OHNAC(Occupational Health
 Nurses in Agricultural Communities), FACE(Fatality Assessment and
 Control Evaluation program) (3).
 (NIOSH) SENSOR
 (4). SENSOR NIOSH
 NIOSH 가
 NIOSH SENSOR 가
 . 1997 SENSOR 12가
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NIOSH

The NOISH Surveillance Strategic Plan

The NOISH Surveillance Strategic Plan is based on a long-range vision of a comprehensive occupational surveillance program involving a coordinated set of complementary surveillance systems. (5)

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|--|
| <p style="text-align: center;">NIOSH Surveillance Strategic Goals</p> <ol style="list-style-type: none">1. Advance the usefulness of surveillance information at the Federal level for prevention of occupational illnesses, injuries, and hazards.2. Strengthen the capacity of State health departments and other State agencies to conduct occupational surveillance3. Strengthen surveillance of high-risk industries and occupations, and of populations at high risk, including special populations.4. Promote effective occupational safety and health surveillance conducted by employers, unions, and other non-governmental organizations.5. Increase research to improve occupational surveillance |
|--|

Physicians' Reporting Activities)

가

7

6

ODIN(Occupational Disease Intelligence Network)

Manchester

(6)(1).

ODIN

가

3-4

HSE가 2

25

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HSE

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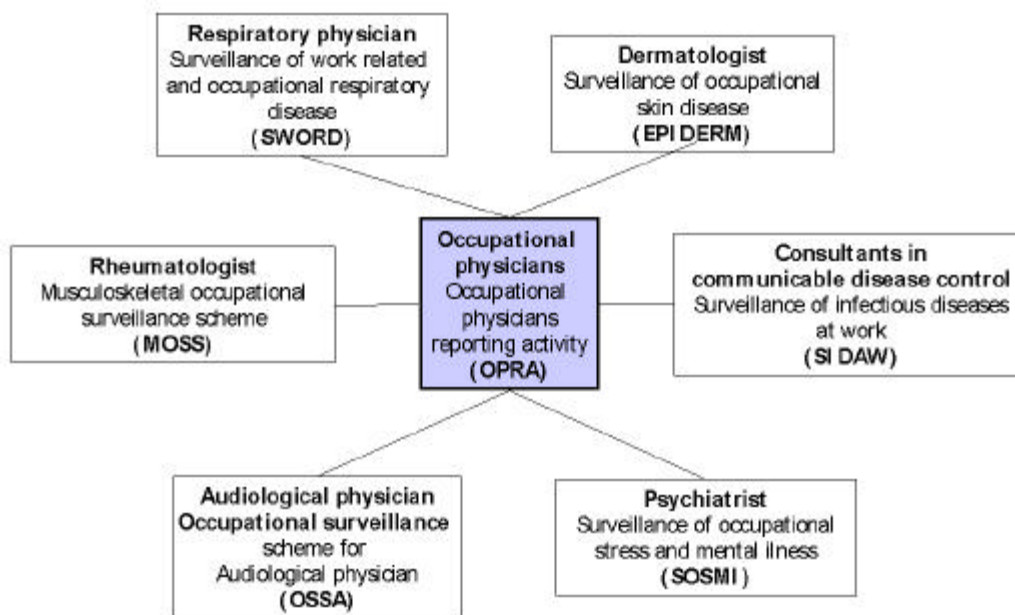


Fig 1. Occupational disease surveillance schemes in Occupational Disease Intelligence Network(ODIN)

3.

1)

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가

가

3)

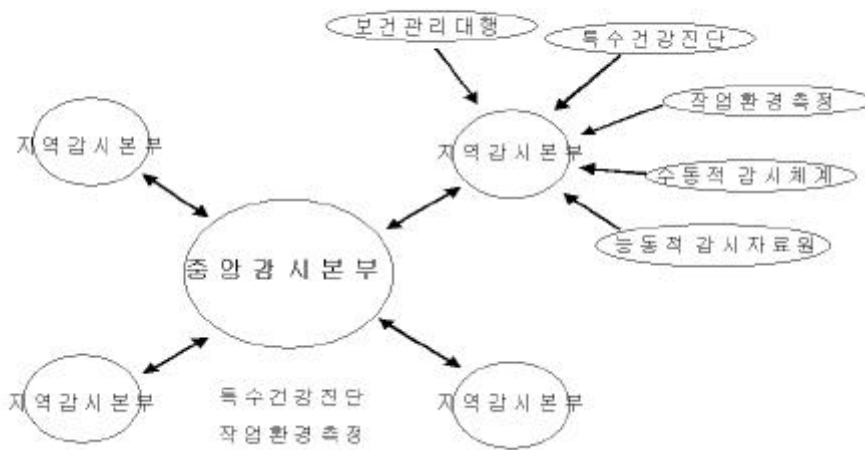
가

가

가

1

그림 1. 한국적 직업성질환경감시체계



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4.2 .

5. 가 .

5.1 가 .

5.2 .

5.3 .

4.

1. , . 13(1); 2001
2. , , , , , , , , , , , . 13(2); 2001
3. , , , . 13(1); 2001
4. Baker EL. Sentinel Event Notification System for Occupational Risks (SENSOR): the concept. Am J Public Health. 1989;79 Suppl:18-20.
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6. Cherry NM, Recent Advances: Occupational Disease, BMJ 1999;318(22):1397-9.
7. HSE (Health & Safety Executive), RIDDOR 1995, HSE, 1999.

-

MMPI

1.

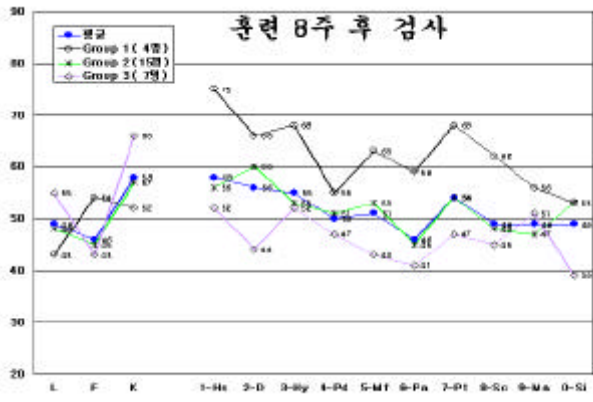
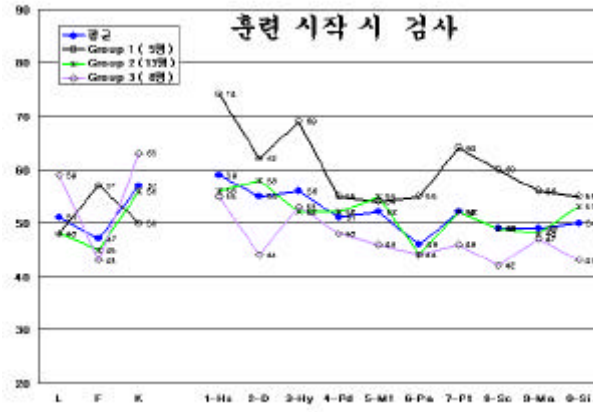
가
가

2.

2000
8
2 556 MMPI (Minnesota Multiphasic Personality Inventory)
32 6 26
MMPI K (T-score) 가
K-Means 3가 T 가 T

3.

30-45 36.8
8 T (Group)
1 1-3 7() 가
(somatoform disorder) 5
(19%), 4 (15%) 3 가 3 1
V 8 (31%), 7 (27%)
2 13 (50%),
15 (58%)



4.

가 ?,
 ?,
 MMPI 1(Hs, hypochondri-
 asis), 2(D, depression), 3(Hy, hysteria) 가 , 가
 1 1-3-7-8
 , MMPI 가
 , MMPI 가 가

가 가

0 10

1.

가

2.

(PRS: pain rating score) 365 (Visual analog scale) 1991
가
Kappa

3.

가 가 , 가
가 가
Kappa 0.88, 0.84, 0.89, 0.89
0.89
0.975(P<0.001)
Pearson
0.603(P<0.01)

4.

가 가
가

_____ 1) . 1) . 1) . 1) . 1)
 2) . 3) . 3) . 4)
 1)
 2)
 3)
 4)

1.

가

2.

가 69 17
 , 28 ,

3.

(5) , 가 26.09% (18) 29.41%
 , 가 '7 ,
 , 'Tinel test , 'Phalen test ,
 가 가 , 가 가 .
 , 'Visual Analogue Scale (VAS: 10) 4

4.

가 가 26.09% 29.41%
 가 1996 OSHA가

1) . _____ 1) . 1) . 4) . 2) . 2) . 3) .
1) , 2) .
3) , 4) .

1.

가

가

가

2.

2000 10 24 , 11
35 (40.3 , 3.94)
가
가 (Sonace 9900, Medison) 7.5 MHz
linear probe ,

Student's t-test

3.

3.9 4 4
가 41.7% 가
100 40.0 (29.2 ,
63.6)

가 9.00(95% : 0.95- 109.1), 가 7.20(95% : 0.81- 79.4)
 (p<0.05, ² test for trend). 가 가
 가 (p<0.05).
 가 (p<0.05).
 /) (/)가 0.17 0.17 (p<0.05).
 (×) 300 mm² 300 mm² (p<0.05).
 가 0.17 12.1(95% : 1.38- 106.6) 가
 9.87(95% : 1.23- 79.5) 가
 (p<0.01).
 , -0.280, -0.356, -0.257
 (p<0.05; p<0.01; p<0.01).
 0.284, 0.365 (p<0.05; p<0.01).
 /) 0.257, 0.437, 0.417 (p<0.05; p<0.01; p<0.01) (/)
) -0.262, -0.382 (p<0.05; p<0.01).
 (× (-))가 0.280
 (p<0.05). (×)
 -0.244, -0.280 (p<0.05).

4.

가 가

1

_____ . . .

1.

, , , Preiser's disease 가 가

2.

42 가 2001 4 m

B , 1996 가

1983 1986 , 1988
1990 1.5 Kg , 1991 1994
20 Kg , 1995 2001
0 20 , 50 ,
가 가

3.

, Preiser's disease 가

90

10

가
가

가

가

가

()
가

_____ .¹⁾ .²⁾ .

1)
2)

1.

2,408 가 1,000 (2000) , 144 1
가 가 가 가 .
4

2.

가 1 가 1 11 83 94
(), , (karasek JCQ, 1985),
, (NIOSH) .

3.

00 36 , 1 18 5 30 ,
(4-11) (18) 10 , 75 ,
(12-3) 3 , 27 . 18
6 (15-55) , 6 (15-55) , 6 (5-15)
55 kg
33.5 , 6.9 . 68%가
, 2.3 , 가 79%
가 75% .
47.0

, 39.7 가 “ 가 ” 가 .
 가 “ 가 ” 가 .
 57%가 가 ,
 53%가 . 40%가 ,
 가 가 39% .
 69%, 64%, 55%, 54%
 가 82% .

4.

,
 가 .
 4 .

_____

1.

가, , 가 가 가

2.

88 , 168 , 56 , 97 ,
88 , 93 35
Evaluation System , 가 Korean Performance

3.

가 가 가 가
가 가 가 가
가 가 가 가

4.

가 가 가 가 가 가

_____ . . .

1.

가가 . 가
가 .
가 , , ,
가 , .

2.

85 (45 , 40) 35 (23 , 12)
Swedish Performance Evaluation System
가 ,
Benton , , ,
가 , 가 .

3.

3 1 , 가 가 가
가 가 가 .
Benton 가 ,
- .

4.

가 , 1 1 3 가
가 3 가 .

가

가

가

.

1) . 1) . 1) . 2) . 3)
 1) , 2)
 3)

1.

2.

2001 7, 8 “ 가” 가
 (Korean version of computerized neurobehavioral test battery)
 (simple reaction time) 5 , (digit symbol) 9 , (digit span)
 7 , 가 (finger tapping) 3 , 가
 SAS 8.0 , 가
 t-test, ²-test, GLM

3.

354 (20) (51)
 283 table.1

Table 1. Mean performance score of the neurobehavioral core test battery (n=283)

	MEAN	STD	MAX	MIN
Mean of Simple reaction time(msec)	273.1	43.3	198.0	426.0
STD of Simple reaction time(msec)	64.2	26.4	26.0	197.0
Digit symbol(msec)	2697.7	1073.6	1598.0	10464.0
Digit span	7.9	1.9	3.5	12.5
Mean of finger tapping speed (right)	64.8	8.5	40.0	93.0
STD of finger tapping speed (right)	1.8	1.8	0.0	17.0
Mean of finger tapping speed (left)	59.0	8.4	25.0	84.0
STD of finger tapping speed (left)	1.7	1.6	0.0	12.8

STD standard deviation

136 , 147 , 가
 , 가
 10 , 77 , 40.4 . 25 (59) , 26-50
 (146) , 51 (78)
 가 (p<0.0001).
 가 0 , 19 , 12.1 . (9 , 47) ,
 - (9-15) , (16) ,
 가 , (p<0.001) (128
) , (155) , (p=0.042), (p=0.075),
 가 (p=0.0015) 가
 (63) , (42) , (178) , ,
 , 가 (p>0.20),
 (72) . (95) . (54) . (62)
 가 가 (p<0.0001),
 가 (p=0.3250).

table 2 .

Table2. Multivariate Analysis (regression coefficient, (standard error))

	gender	age	years of education	alcohol consumption	smoking	familiarity to computer
Mean of simple reaction time (msec)	0.031 (0.0657)	0.044 (0.0466)	-0.150** (0.0408)	0.065 (0.0577)	-0.035 (0.0399)	-0.001 (0.0304)
STD of simple reaction time (msec)	0.037 (0.0571)	0.047 (0.0405)	-0.149** (0.0354)	0.049 (0.0501)	-0.085* (0.0347)	0.034 (0.0264)
Mean of digit symbol (msec)	-0.147 (0.0749)	0.514** (0.0532)	-0.342** (0.0465)	0.063 (0.0658)	0.005 (0.0456)	0.189** (0.0347)
Maximum of digit span	-0.087 (0.0976)	-0.376** (0.0693)	0.371** (0.0606)	-0.027 (0.0857)	-0.029 (0.0593)	-0.158** (0.0452)
Mean of finger tapping speed(right)	-2.778** (1.0673)	-3.911** (0.7580)	2.674 (0.6626)	-1.447 (0.9378)	0.466 (0.6489)	-2.291** (0.4944)
Mean of finger tapping speed(left)	-2.313* (1.1739)	-2.999** (0.8337)	1.864* (0.7287)	-0.872 (1.0314)	1.442* (0.7137)	-1.898** (0.5438)

**p<0.01

*p<0.05

가 가

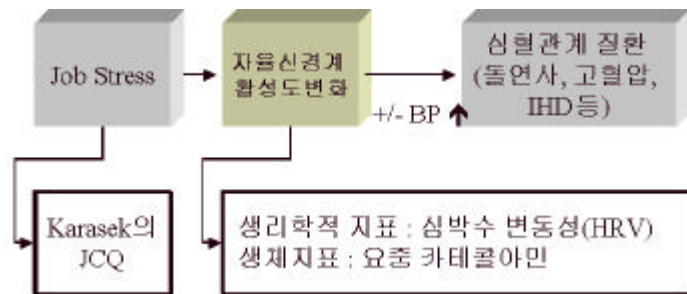
- 1) .
- 2) .
- 3) .
- 1) .
- 1) .
- 1) .
- 2) .
- 3) .

1.

가
(; Heart
rate variability, HRV) () 가

2.

134
(4 3) (Karasek's
Job Content Questionnaire, JCQ)' 가 ,
(, ,) 가 shift 3



1.

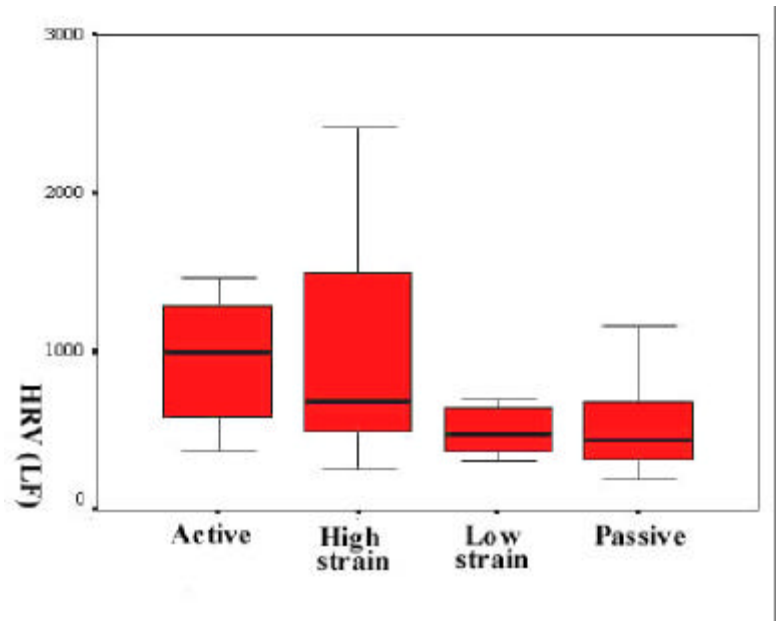
(49) , , 5 5

power spectrum density curve
0.04~0.15 Hz (low frequency band) , 0.15~0.3Hz (high

frequency band) spectral power . HPL
with ECD .

3.

29 , 25 44 . 134
77.9% 가 , 63.6% 가 .
가 (Job strain model) 4
(high strain) 31 , (low strain) 25 ,
63 , 20 .
4
(p-value=0.493)
(: -0.336 p-value:
0.001), (<60.5) ,
4 ()
- (Between Group) p-value 0.52 .



2. (60.5 n=64)
(JCQ , , ,
4) 가 (
) (, ,)
Random effect model (mixed model for repeated measured data)

1) 1) 2) 2)
1)
2)

1.

가 , 가 ,
(needle-stick injury)가 가 .
가 .

2.

2000 7 2 1
695 가 .
2000 3 6 가 4 ,

3.

가 4 443 200 45.1% , 1
11.5%, 2 10.6%, 3-4 10.2%, 5 12.8% .
200 1 4 , 1000 1 5,377 .
(multiple logistic regression
analysis) , 16
95.5%가 , 12.6%
413 93.2%, 311 70.2%,
379 85.6%가 .

4.

B (endemic area) , AIDS

가

1

_____ . . .

1.

(multiple chemical sensitivity)

, 가 1 ,

2.

58 가 6 7 , , , , , ,

, 30 7 8 .

2

, 가 . 98 Benzene, Toluene, Xylene Chloroform.

- Ig E , 가

FEV₁ 84% methacholine histamine

2 , 20% 가 (PEFR)

가

가

3.

가 , 가
가 .

가 가

_____

1.

effect가 가 health
 health effect
 health 가

2.

1999 7 9 8 30 , 2000 7 10 8 31 2
 308가 ,
 가 .

3.

1)

			Mean ± SD*	*	*
	0 4	13	2.29 ± 0.43	1.59	3.11
	5 8	58	2.52 ± 0.89	1.21	5.06
	9 12	57	2.06 ± 0.70	0.85	4.03
	13 15	49	2.06 ± 0.57	1.10	3.75
		93	2.38 ± 0.75	1.10	5.06
		84	2.06 ± 0.71	0.85	4.28
		177	2.23 ± 0.74	0.85	5.06

* Blood lead level : $\mu\text{g/dL}$

가 (mean difference 0.1541 $\mu\text{g/dL}$, 95%CI 0.061 0.2472, p=0.0001).

2) 가

Dependent variable	Independent variable	correlation	p- value
Log [PbB Child]	Log [PbB Father]	0.20116	0.0130
Log [PbB Mother]	Log [PbB Father]	0.23256	0.0005
Log [PbB Child]	Log [PbB Mother]	0.35353	<0.0001

가 , , ,
가 가 .

3) Final Model for Log [PbB Child]

	β	95% CI		P
Child age	-0.0225	-0.0366	-0.0083	<0.0001
Child sex	-0.1655	-0.2595	-0.0716	0.0006
Log [Mother Blood lead]	0.3349	0.1790	0.4907	<.0001
Log [Father Blood lead]	0.2644	0.0814	0.4474	0.0046

, , , 가
. , , , .

4.

Table 2 3 가
가
5 8 group 가 가 ,
physical activity가 가

N- Methylfo rma m ide (NMF)

1.

_____
 .
 (Dimethylformamide, DMF)
 가
 - (N-Methylformamide, NMF)
 .

2.

22
 2001 2 () 7 ()
 , B , C
 , ,
 SPSS

3.

22 40.8 , 80.6 16
 (72.7%), 6 (27.3%) 20 1 (4.5%), 30 8 (36.4%), 40 9
 (40.9%), 50 4 (18.2%) . 3.2 °C (-1.9 ~ 7.6 °C),
 26.5 °C (24.1 ~ 31.0 °C) 35.4%, 84.5% .
 22 19 (86.4%) 3 (13.6%)
 2 (9.1%), 2 (9.1%), 18 (81.8%)
 22 7 (31.8%)
 15 (68.2%)
 . Aspartate aminotransferase (AST) Alanine
 aminotransferase (ALT) 가 γ-glutamyl
 transferase (γ-GTP) (p < 0.05). γ
 -GTP 가 (p < 0.05) .

ppm
 31.23 ± 2.38 mg/g creatinine
 (p < 0.05).
 creatinine) (1 : 6.97)
 (Body Mass Index, BMI)
 가 .

-

11.55 ± 2.79 ppm, 13.78 ± 2.46

-

96.09 ± 3.12 mg/g creatinine
(ppm) - (mg/g)

2.6 .

-

4.

가 3 가
가

가

가 .

가

- , , -

_____ 1) . 2)
1)
2)

1.

1999 1 1
, ,

가 .

2.

1998 1) (1999 , 2000) 2) (1999 ,
2000) 3) (1999 , 2000) 3 , ,
-> 3) -> 4) -> 5) -> 6) -> 6) 1) -> 2)
, , 가 .

3.

1998 56.7 % 1999 34.0 % 가 2000 37.7%
가 . 1998 5732 57% 2444 1999
62% 2000 . 2 , ,
, 가 ,
가 . 가
2 , , , 가 .

Effects of genetic polymorphisms in metabolic enzymes on the relationships between 8-hydroxydeoxyguanosine levels in human leukocytes and urinary 1-hydroxypyrene and 2-naphthol concentrations

Yong-Dae Kim · Heon-Kim · Chul-Ho Lee · Hong-Mei Nan · Jong-Won Kang

Department Preventive Medicine, College of Medicine, Chungbuk National University

1. Objective

This study was designed to investigate the relationship between environmental exposure to polycyclic aromatic hydrocarbons (PAHs) and oxidative stress, and to evaluate the effects of cigarette smoking and the genetic polymorphisms of *CYP1A1*, *CYP2E1*, *GSTM1*, *NAT2* and *UGT1A6* on the relationship.

2. Methods and Materials

The subjects of this study were 105 healthy Korean males without occupational exposure to PAHs. Blood and urine samples were analyzed from each subject, and smoking habit assessed. The 8-hydroxydeoxyguanosine (8-OHdG) level in leukocytes, and urinary 1-hydroxypyrene (1-OHP) and 2-naphthol concentrations, were measured using high-performance liquid chromatography methods. Genetic polymorphisms of *CYP1A1*, *CYP2E1*, *GSTM1*, *NAT2* and *UGT1A6* were identified using PCR or PCR-RFLP methods. Correlations between the 8-OHdG level, and urinary 1-OHP and 2-naphthol concentrations were statistically analyzed. The effects of cigarette smoking and of each of the genetic polymorphisms on the correlations were also evaluated.

3. Results

The 8-OHdG level in leukocytes showed significant correlation with the urinary 1-OHP concentration in all subjects and in smokers, and with the urinary 2-naphthol level in non-smokers. The 8-OHdG level was significantly higher in smoking rapid acetylators than in smoking slow or intermediate acetylators, and in individuals with the *UGT1A6* wild-type than in those with the *UGT1A6* mutant genotype. Significant positive correlations between 8-OHdG in leukocytes and urinary 1-OHP concentrations were found in subjects with every genotype of the *CYP1A1* and *CYP2E1* genes, with the *GSTM1* null-type, with the *NAT2* genotype of a rapid acetylator, and with the *UGT1A6* wild-type, respectively. The urinary 2-naphthol level significantly correlated with the 8-OHdG level only in subjects with the *GSTM1* null-type. In multiple regression analyses, including the polymorphisms of *CYP1A1*, *CYP2E1*, *GSTM1*, *NAT2*

and *UGT1A6* genes and urinary 1-OHP or 2-naphthol concentration, only the urinary 1-OHP level significantly correlated with that of 8-OHdG in leukocytes.

4. Discussion

In conclusion, there is a significant correlation between the 8-OHdG level in leukocytes and the urinary 1-OHP concentration in the population not occupationally exposed to PAHs. This relationship is affected by genetic polymorphisms in PAH metabolic enzymes.

1) . 1) . 1) . 1) . 1) . 1) . 2) 3)
 1)
 2)
 3)

1.

가 , 가
 가

2.

2000
 가 가 160 (84.7%)
 189
 189
 Andersen
), 가 (

3.

1) 160 34%가 , 66%가
 2) .(P<0.05)

3) (72.0%) .(P<0.01) 가 , (39.3%) 가 ..

4) , 가 (58.2%) (74.1%) ,
.(P<0.05) 가 .

3) 가
. (P<0.01) , , , 가 .

4) , (74.2%)가, 가 , 가
가 . (P<0.05)

5) , .(P<0.05) , ,
.

4.

· , , 가
가 .

가

1) . 1) . 1) . 1) . 1) . 3)
 1)
 3)

1.

2000 2,528 955 , ,
 545 , 364 46 가 가 ,
 가 가 ,
 . 1999 ‘ ’ .
 가 ,
 1 가

2.

15,000 가 2000 1 1 12 31
 5,230 , 가 . 가 8,285
 210 2001 155
 ,
 2000 2001 가 , BMI, ,
 HDL , LDL paired t-test .

3.

1)
 13,515 5,458 (65.9%), 2,650 (32.0%)
 177 (2.1%) , 4,343 (83.1%), 854 (6.3%) 33
 (0.6%) . 20 3 (1%), 20-29 가 62 (1%), 20-39
 125 (2%), 40-49 18(3%) 50 2 (9%) .

2)
 210 86 2000
 2001 130.93 ± 18.89 mmHg 125.81 ± 14.91 mmHg,
 85.64 ± 14.81 mmHg 81.08 ± 9.23 mmHg, BMI가 24.98 ± 3.20 24.58 ± 3.08, 가

267.73 ± 42.68 mg/dℓ 244.42 ± 43.99 mg/dℓ, HDL 가 49.02 ± 10.74 mg/dℓ 48.07 ± 9.93
 mg/dℓ, LDL 가 182.50 ± 41.76 160.85 ± 45.82 , HDL
 2001 가 2000 (p<0.05).

3)

210 67 2000
 2001 132.99 ± 21.48 mmHg 127.46 ± 18.20 mmHg,
 86.94 ± 15.47 mmHg 81.49 ± 10.08 mmHg, BMI가 25.20 ± 4.19 25.03 ± 4.29,
 가 265.79 ± 51.99 mg/dℓ 250.64 ± 52.56 mg/dℓ, HDL 가 52.21 ± 13.96 mg/dℓ
 51.64 ± 11.67 mg/dℓ, LDL 가 183.69 ± 48.99 164.86 ± 50.04 . BMI HDL
 2001 가 2000
 (p<0.05).

4.

가 가
 , , .
 .
 가
 ,
 가

1) . _____ 1) . 2)
1)
2)

1.

가 ,
가 ,
3 가 ,
가
1

2.

, 가 ,
thermocouple

3.

32 , , ,
D ,
1996
20 25 , 3 4 ,
2
30 ,
2000 2 21 , 11 ,
가
thermocouple 가 ,

4.

가
가
1999
5,345
10
가

5.204, 2.674, 0.364 (Table 2). 가

Table 1. Results of univariate logistic regression analysis

Variables	Parameter estimate	Odds ratio (95% CI)	p-value
Age (< 40 years)*	1.4057	4.078 (1.315 12.653)	0.0150
Cold exposure severity*	0.9980	2.713 (1.201 6.127)	0.0164
Protective clothes	-0.4383	0.645 (0.221 1.884)	0.4228
Alcohol drinking (1 2 times/week)	0.5978	1.818 (0.519 6.373)	0.3502
Smoking	-0.5008	0.606 (0.130 2.831)	0.5243
Exercise (1 2 times/week)	0.7419	2.100 (0.653 6.749)	0.2129
Salt intake	-0.0451	0.956 (0.305 2.996)	0.9383
Milk intake*	-0.8259	0.438 (0.199 0.965)	0.0406
BMI (< 24kg/m ²)	0.2103	1.234 (0.686 2.219)	0.4824
Family history of hypertension	0.3314	1.393 (0.405 4.789)	0.5990

(* : p<0.05)

Table 2. Results of multivariate logistic regression analysis

Variables	Parameter estimate	Odds ratio (95% CI)	p-value
Age (< 40 years)*	1.6494	5.204 (1.440 18.812)	0.0119
Cold exposure severity*	0.9834	2.674 (1.080 6.618)	0.0335
Exercise	0.3444	1.411 (0.606 3.288)	0.4250
Milk intake*	-1.0095	0.364 (0.141 0.942)	0.0372

(* : p<0.05)

4.

가 가

1)2) . 3) . 3) . 4) . 4) . 2) .
 1) .
 2) .
 3) .
 4) .

1.

328

2.

가

4

가
 가

3.

85 - 101 dBA

가

OR=6.07(1.77- 20.79),
 OR=2.57(0.64- 10.31),
 OR=3.26(1.47- 7.25),
 OR=18.40(5.11- 66.28),
 OR=4.28(1.71- 10.75),
 OR=8.12(2.03- 32.53)
 가

4.

가

(synergistic effect)가

, , , , ,

.

25

25

2/3

33 1977 11 P 21

1998 2 K 1977 1998

9 6 30%,

60%, 10% 1977 1992 P

가

1992 1998 K

가 1977 1993

가

1993 가

가

K 1 0.01 2.24 mg/m³

0.34 mg/m³ , 2 0.01 1.25 mg/m³ 0.33

mg/m³, 3 0.01 2.17 mg/m³ 0.29 mg/m³

17.25 µg/m³ 96.41 µg/m³ 36.66 µg/m³

S 1 0.01 1.36 mg/m³

0.27 mg/m³ , 2 0.01 1.36 mg/m³ 0.32 mg/m³

15.59 76.90 µg/m³ 32.05 µg/m³

0.2 mg/m³ 136 (

) 45 (31.0%)

2.2-8.0 m/sec 가 가

가

1-hydroxypyrene 2-naphthol 1-hydroxypyrene

12.20 ng/ml(1.87 ng/MØ), 2-naphthol 16.77 ng/MØ(4.15 ng/

MØ), 1-hydroxypyrene 5.97 ng/MØ(0.65 ng/MØ),

2-naphthol 21.99 ng/MØ(3.18 ng/MØ)

4.

1999

Comet

T-, B-

DNA

1) . 1) . 1) . 2) . 1) .
1) .
2) .

1.

DNA DNA , B- T-
DNA 가 DNA .
T-,B- DNA 가 DNA 가 .

2.

1 41 , 41
가 , DNA 가
comet assay . Macs(magnetic cell sorting)
T-,B- , Comet 4.0 olive tail moment, tail
DNA %, extent tail moment 3가 tail parameter 100

3.

39.55±55.67, 0.12±0.22(ug/g creatinine)
Comet 3가 tail parameter olive tail moment(TM) tail
DNA(%) 가 가 ,
extent tail moment B- 가 B-
TM 3.86±0.71, 1.51±0.39 . T- 1.75±0.29,
1.47±0.41, 3.61±0.75, 2.60±0.59 .
(P<0.001). B- TM , T-
tail parameter
spearman B- 3가 tail parameter 0.7
, T- Tail DNA(%) Olive tail moment 0.3 ,
tail extent moment 0.36, tail DNA(%) 0.40, Olive tail moment 0.55

4.

comet DNA
T- 4 B- 3-4 5
TM B-
B- 가 TM DNA 가
B- 가 B-
B- DNA

UGT

1) . 1) . 2) . 3) . 1) . 1) . 1)
 1)
 2)
 3)

1.

Uridine diphosphate glucuronosyltransferase(UGT) ,
 (polycyclic aromatic hydrocarbon; PAH) 1-hydroxypyrene(1-OHP) 2-naphthol
 가 , 가

2.

PAH 88 PAH 123
 , PAH , HPLC
 PAH 1-OHP 2-naphthol, glucuronized form
 DNA PCR UGT1A6 UGT1A1, UGT2B7
 1-OHP 2-naphthol , UGT

3.

1-OHP 2-naphthol
 가 2-naphthol glucuronized form 가
 , 가 2-naphthol
 glucuronized form 가 . UGT1A6 wild type P1,
 P3 P4 P2 . UGT1A1
 [TA] [TA]₆(wild type) [TA]₇, [TA]₈
 [TA]₅ . 1-OHP UGT1A6가 wild type
 가, P1 P3, P4 UGT1A1
 가 6/6 가 6/7 6/8, 7/7
 . 2-naphthol UGT2B7 Y²⁶⁸/Y²⁶⁸ H²⁶⁸/H²⁶⁸,
 H²⁶⁸/Y²⁶⁸ . UGT1A6 UGT1A1, UGT2B7

1-OHP

glucuronized form 가 *UGT1A6* *UGT1A1*
, 2-naphthol glucuronized form 가 *UGT1A6*
. 1-OHP 2-naphthol 가
가 .

4.

UGT1A6 *UGT1A1*
UGT1A6 PAH pyrene naphthalene
. *UGT1A1* PAH pyrene
. *UGT2B7* PAH 가 naphthalene
.

(4)

가

1.

2.

가 2 가 36 4 44

Ewing Clarke 가
(Fall in systolic BP on Standing),
30% 1 가 (Rise in diastolic BP on
sustained handgrip) 2가 1 6
(Respiration(bpm)), 가 15
가 R-R 30 가 R-R
30:15 (Standing 30:15ratio) 2가

A549

가

Silica triggered cellular signaling without immediate increase of ROS in A549 cells

가

1.

가 (ROS) Rat2

ROS silica
ROS
가 A549 cell
NF- B silica가 redox

Cu/Zn-superoxide dismutase(SOD), catalase, peroxiredoxin(Prx)

2.

human lung adenocarcinoma A549 cell line(ATCC, CCL185) C6 cell 37 , 5% CO₂ chamber
1.0mg/ml 가
Prx I, PrxII, Cu/Zn-SOD, thioredoxin(Trx), catalase, I B-immunoblot analysis
가 DCFH-DA 5 μM 5 DCF
Fura-2/AM 37 45

3.

A549 cell type ROS H₂O₂
가 DCF , 가 Rat2 C6 cell line silica
가 NF- B I B-

A549 cell redox status
. PrxI PrxII가 , Prx
thioredoxin catalase Cu/Zn- SOD
가
A549 cell silica 가

4.

silica A549 cell ROS 가
. NF- B Prx

() 1

_____

1.

, , 가 (lead),
가

2.

52 1 6 가
20 X-
5 , 3
-antitrypsin FVC: 54%, FEV₁: 22%, FEV₁/FVC: 29%,
DLCO: 51 % (stage III)
가 가

()
가

3.

가

1) . 2) . 1) . 3) . 2) . 2)
 1)
 2)
 3)

1.

2.

216 19,272 ,
 1995 1999 ,
 74,371 ,
 1995 35 (1960
) 9,429 , 32,851
 1995 1999 1997
 35 .

3.

9,429 (38,148) 73 , 32,851 (116,408)
 81 , , , , ,
 10 191.4, 69.6 ,
 91, 38 , , , ,
 가 100 (40-44 (SMR 114)
 65-69 (SMR 113), 45-49 (SMR 300) 60-64 (SMR 125)).
 40-54 가 100
 10 .
 1995 , , , 3.5 (95% ; 2.346-5.069),
 3.1 (95% ; 1.287-7.344), 4.5 (95% ; 2.261-9.106), 3.5 (95% ; 1.367-9.154)

(p<.001).

229.2, 73.4 10 209.0, 62.6 ,
103, 25 .
4.7 .

4.

가
가
가 100 , 40 , 50
가 100 ,
가 105
가
가

-

1) . _____ 2) . 2) . 1) . 3)
 1)
 2)
 3)

1.

2.

2001 3 11
 3 31 240 , 211

(self-recorded questionnaire)

Levenstein (1993) 10 (1995)
 30 (1976)
 7 21

3.

211 , 가 110 (52.1%)
 , 가 117 (55.5%) , 52.74 (4 4)

(80.4%), / (77.4%), (49.4%), / 가 (81.9%) 가
(45.7%) .
, ,
, 1 .

4.

가 .

가 .

가

가 $\frac{\text{가}}{\text{가}}$ 가 $\frac{\text{가}}{\text{가}}$ 가 $\frac{\text{가}}{\text{가}}$ 가 $\frac{\text{가}}{\text{가}}$ 가

1.

(1)
(2)
가 , RULA

2.

7 /
(extension), (flexion), (bent)
RULA
RULA

3.

- 1) 1 가 , , 가 98 cm, 68 cm, 76 cm, 2 178 cm, 60 cm, 83 cm
2 가 가 1 1
(arm-rest)가 , 2 32°
가 . 1, 2 가 .
- 2) RULA 1 , 가 6.57±0.53, 6.86±0.38, 2
6.14±0.90, 6.00±1.00 , 1 6.71±0.49, 6.71±0.49, 2가 6.14±
0.90, 6.00±0.82 . RULA , 1, 2 .
- 3) RULA 1 6, 7 , 2 7 , 6 , 5
, 4 .

4) RULA

, , ,

5) RULA
RULA

, , , , ,
, , , .

4.

가 가 8 RULA
가 가
가 8
8 , .

86.67

1

2

(p=0.013).

(p=0.069)

(p=0.013)

가

(p>0.05).

가

가

(p<0.05),

(p>0.05).

(PWI)

가

(p<0.01),

(p<0.01).

가가

,

가

(p<0.01).

가

,

PWI

,

4.

1

가

.

,

,

- , -

.

.

.

가
, -가
가
가
가 가

4.

가 (3.51, 3.29)

가
가

1

_____

1.

syndrome) (Patellofemoral 가 .

2.

1 6 가 30 , 3 가 (de Quervain's disease) X- . OSHA 가 가 11 가 가 .

3.

(femoral condylar ridge)

JCQ

가

1) . 2) . 2)
 1)
 2

1.

가
 가
 JCQ) 가 (Job Contents Questionnaire, 49
 , JCQ 가
 가 , JCQ 가

2.

2001 8 -9 84 JCQ
 49
 (decision latitude), (job demands), (social support), (job
 insecurity) 5 가 가
 (job dissatisfaction) (self-identify through work)
 0 (), 1 () , 5
 20
 가

3.

27.3
 (22-42), 5.4 (0.25- 19), 47.4 (40-70) ,
 0.54 (SD 0.19), 17.2 (SD 1.6)
 1 , ,
 가 ,

demands), (job insecurity), (psychological job
 가 가
 (decision latitude), (customer relationships), (coworker support)
 (supervisor support)

Variables	Job dissatisfaction		Self-identify through work	
	correlation coeff.	p	correlation coeff.	p
Age	-0.17	0.15	0.25	0.04
Employment year	-0.24	0.04	0.32	0.01
Annual salary	-0.28	0.02	0.18	0.15
Decision latitude	-0.30	0.01	0.47	0.00
Job demands	0.26	0.02	-0.01	0.90
Job insecurity	0.23	0.03	0.05	0.67
Work hours/week	0.16	0.19	-0.04	0.76
Coworker support (a)	-0.22	0.05	0.34	0.00
Supervisor support (b)	-0.33	0.00	0.33	0.00
Social support (a+b)	-0.35	0.00	0.40	0.00
Exposure to hazards	0.20	0.07	-0.06	0.62
Customer relationships	-0.06	0.58	0.34	0.00

Table 1. Correlation of job satisfaction indices with other variables

Dependent variable	Independent Variable	β Estimate	95% Confidence Interval	p
Job dissatisfaction	Job insecurity	0.013	(0.003, 0.023)	0.01
	Job demands	0.014	(0.005, 0.023)	0.00
	Annual salary(million won)	-0.009	(-0.017, -0.0016)	0.02
Self-identity	Self-identity	-0.051	(-0.077, -0.024)	0.00
	Decision latitude	0.10	(0.05, 0.16)	0.00
	Customer relationships	0.21	(0.01, 0.40)	0.04
	Coworker support	0.43	(0.09, 0.77)	0.02

Table 2. Results from multivariable regression models

4.

JCQ 가 가

가

_____

1.

가 2 , , , 13 . 1

2.

1) : (styrene monomer)
13 1
2 10
3 7

2) :
A. :
 , , , 가 1 ,
가
IgE 가

B. 가: 가
ACGIH TLV-TWA 4 , ,
5 , , , 가
가 4 가

3.

13 11.3 , 7 12
3.5 2 , 5 .
 , , 가
10 71% 1 7%, MAST allergy
test 3 23%, IgE 가 8 61.5%, 8 61.5%,
13 100% . 13
84.6% 11 , 2 . 7
 , , , 1 , ,
1 2 .

4.

2 7 6
1 2
5% 20% 가
가

Effect of antioxidants on Survival of Paraquat Poisoning

_____ ^{1),2)} . 1) . 2) . 2)
1)
2)

1. Background and Purpose

Paraquat (1,1'-dimethyl-4,4'-bipyridium dichloride) is an effective herbicide that had low chronic toxicity because of its rapid deactivation upon soil contact. However, it has become notorious throughout the world as a potent human poison. In spite of the decreasing trend of the agricultural population in Korea, the incidence of paraquat poisoning is rapidly increasing. Treatments of paraquat intoxication examined experimentally or clinically include those that could prevent the accumulation of paraquat in the lung by various polyamines and D-propranolol, increase efflux of paraquat from the lung by cyclophosphamide and D-propranolol, and reduce the consequences of the redox cycling. The intracellular enzymatic antioxidant defense provided by superoxide dismutase, catalase, and glutathione peroxidase is present only at smaller concentrations in human plasma. In contrast, proteins whose primary biological function is related to the transport of iron and copper ions provide antioxidant protection by sequestering these transition metals in forms incapable of stimulating free radical reactions. Vitamin C is qualitatively the single most important plasma antioxidant. Thus, vitamin C has been used as a choice of antioxidants *in vitro*, but clinical use is still inconclusive. It has been known to protect all classes of lipids from oxidation under a number of relevant types of oxidant stress while other non-enzymatic antioxidants such as vitamin A, vitamin E, glutathione, bilirubin, and urate merely lower the rate of oxidation or act in a more restricted, local environment. Therefore, the measure of total antioxidant status (TAS) after addition of vitamin C can account for total biological antioxidant capacity. The current study was conducted to evaluate the effect of vitamin C on outcomes of paraquat poisoning. Additionally, administration of supplementary vitamin C was assessed as to its potential usefulness in the treatment of paraquat poisoned patients.

2. Materials and Methods

A clinical intervention trial was designed for the study. 148 paraquat poisoned patients were included according to the inclusion and exclusion criteria during 1999. During a half of the study year, regular dose of vitamin C (800 mg/day) was administered in 49 subjects (control group). In another half of the year, mega-dose of vitamin C (3000 mg) was administered each 8 hours for seven days and TAS was measured in a hour after injection in 99 subjects (experimental group).

Vitamin C was purchased from Handok Pharmaceuticals Co., Seoul, Korea (made under license from Hoechst AG., Frankfurt, Germany). In order to avoid exposure of vitamin C to oxygen in the air, all vitamin C was injected using vinyl bags instead of glass bottles. Intravenous injection rates of vitamin C were 100 mg/min. TAS was measured using a commercial kit (BTSR, Randox Lab. Ltd., UK) using Hitachi R 7150 (Hitachi Ltd. Tokyo) according to the manufactures instruction. The principle of this measurement is based on the quenching of the ABTS [2,2'-Azino-di-(3-ethylbenzthiazolline sulphonate)] radical cation, which is produced by the interaction of ABTS with ferryl myoglobin radical species, generated by the activation on metmyoglobin with H₂O₂.

3. Results

No differences in age, sex, and time interval for treatment were found between the groups ($P > 0.05$). Estimated amount of ingested paraquat and urine paraquat test did not show significant differences as well ($P > 0.05$). Overall fatality was insignificantly higher in the control group (49.0% vs. 42.9% $P > 0.05$). Survival time was not significantly different (10.6 ± 6.6 days for control, 11.3 ± 8.1 days for experimental group). Means of 7 consecutive TAS levels ranged 2.60 - 2.88 mmole/L without significant variation. Log rank test showed a significant difference in survival between groups ($P < 0.001$). Adjusted OR of vitamin C on fatality was significantly lower in experimental group (Adjusted OR = 0.18, 95% CI = 0.05 - 0.72, $P = 0.02$). In Cox's proportional hazard model, adjusted hazard ratio was significantly lower (HR = 0.30, 95% CI = 0.14 - 0.63), $P < 0.01$, controlling for age, sex, and exposure amount of paraquat.

4. Discussion

Vitamin C is most frequently used as a free radical scavenger, even though there are no guidelines for the adequate dosage. The effect of vitamin C on the survival was the focus in the current study and thus our hypothesis was that high doses of vitamin C might affect improvement of the clinical outcome. In conclusion, vitamin C is a useful and effective antioxidant in humans; thus, vitamin C can be used as a free radical scavenger in acute stage of lipid peroxidation by paraquat. When used in the clinical application, because of a close relation of vitamin C with TAS at relatively high levels, high doses of vitamin C can be applied to reduce free radicals for patients with acute paraquat intoxication. However, further controlled randomized study is recommended to clarify our hypothesis in the clinical fields.

가

가 _____ 가

1) , 2)

1) , 2)

1.

2.

26 가

3.

가

147.52 ± 57.34 ppm 134.55 ± 52.44 ppm

1.51 (0.53) g/L 0.49(0.14) g/L

가 4 (, 13:00)

가 가

가 89.3% 가

Y(

, g/L) = 0.007 × (, ppm) + 0.665

4.

가

Fit Test

Posturographic findings in workers exposed to styrene

Kyung Jong Lee · Keu Weon Lee · Kyoo Yup Jang
Kwang Jin Lim · Cheol Woo Bang

Department of Occupational and Environmental Medicine
Ajou University Medical Center
Suwon, Korea

1.

,
posturography
, styrene ?
, posturographic variables

2.

Styrene 4 25
20
styrene , phenylglyoxylic acid(PGA) 가,
(, ,),
Styrene PGA
Static posturography Accusway System (AMTI, USA) 30
. Center of pressure(COP) 가 area length .

- 1) EO : eyes open, standing on bare platform.
- 2) EC : eyes closed, standing on bare platform.
- 3) FO : eyes open, standing on a piece of 10 cm foam placed over the platform.
- 4) FC : eyes closed, standing on a piece of 10 cm foam placed over the platform.

3.

Sway variables 가 sway area sway length가 가
sway variables 가
가 .

4.

posturography styrene
 subclinical state

가가 ,
.

1

_____ . . .

1.

epichlorohydrin diglycidyl ethers of bisphenol A(DGEBA)

. , 가

2.

60

COAT'

'POLY
가 2 3

가

'POLY COAT'

diglycidyl ethers of

bisphenol A(DGEBA), butyl glycidyl ether, 2-methylpropan-1-01

'POLY COAT' 가
24 48

. Petroleum

1%

'POLY COAT' 48
가

3.

가

1

_____ 1) . 1) . 1) . 2)
 1) , 2)

1.

2001

2.

3.

35

가

. 1989

(chronic peripheral neuropathy)

, 1997

가

(foot drop),

(thenar muscle)

(hypothenar muscle)

(steppage gait ;

)

, 가

, 가

Hippuric acid

O-cresol

_____ 1) . 2) . 1) . 1) . 1) . 1) . 1) .
 1) .
 2) .

1.

가 ,
 o-cresol ,

2.

P 2001 8 20 24 34
 , benzoic acid , 30
 가 (G.C.
 Hewlett Packard 5890) , (Before shift on Monday),
 (End shift on Monday) (End shift on Friday) 1
 NIOSH(1995) `Method No 8301` (HPLC)
 , o-cresol
 (1997) ` o-cresol` SAS for window
 V8.1

3.

1) 34 43.8 , 19.2 .
 2) 46.41, 54.64ppm, 57.68ppm, 62.39ppm,
 58.82ppm .
 3) (0.298) ,
 (0.532) p<0.01, (0.730) p<0.01,
 (0.654) p<0.01 .

- 4) (0.322) ,
 (0.646) $p < 0.01$, (0.732) $p < 0.01$,
 (0.656) $p < 0.01$.
 5) o-cresol (0.409) $p < 0.05$,
 (0.613) $p < 0.01$, (0.601) $p < 0.01$, (0.647)
 $p < 0.01$.
 6) o-cresol (0.144), (0.083)
 (0.440) $p < 0.01$,
 (0.454) $p < 0.01$.

4.

o-cresol
 .
 o-cresol ,
 가 .
 ,
 가
 가
 .

가

____. 1) . 2)
1)
2)

1.

,
(under diagnosis) 가 가 (over diagnosis)
가

2.

,
, Kor J Int Med, J Kor Med Sci
(Clin Exp Allergy, J Allergy Clin Immunol, Annl Allergy)
가

3.

1978 가 가
46 19 21 .
. 1999 ,
가 ,
, Tertraphthaloyl chloride (1).

1.

	가)	5(1), 17(1)
		10(1)
	가)	15(2)
	가	13(4)
	가	3(1)
가		6(1), 15(2)
	가	7(2)
		16(4)
	()	1997
		1997
Cellulase		1997
(, ,)		1993
가		Clin Exp Allergy 1995
(Lysozyme, Peptidase)		J Allergy Clin Immunol 1995
Biodiastase		9(2)
가		20(4)
		18(2)
Isocyanate	TDI	가 , , , , 4(1), 4(2), 5(2), 6(2), 9(4), 1992, 1994
	TDI & MDI	11(1)
Acid anhydride	Phthalic anhydride	34(7)
	Methyltetrahydrophthalic anhydride	11(5)
Latex		1996
	, 가	6(2)
		16(2), 20(2)
	(Black GR)	1990,
	(Cationic red GRL)	32(11)
		1994
	(welding flux) 가	9(1)
		5(2)
	amoxicilin	12(2)
	cimetidine	Ann Allergy 1995
	cephalosporin(7-ADCA)	17(4)
	,	12(2)
	,	Ann Allergy 1992
	,	Kor J Int Med 1986
		J Kor Med Sci 1995
	Tetraphthaloyl chloride	19(2)

4.

Effect of Silica on the Activity of Phospholipase D in the Rat2 Fibroblasts

가 가 가
가 가 가

1. Objectives

The purpose of this study is to characterize and investigate the signaling pathways related to the activation of PLD in silica-stimulated rat2 fibroblasts

2. Methods

The silica-induced phospholipase D (PLD) activities were assayed as accumulation of [³H] phosphatidylethanol ([³H]PEt) was examined in [³H] palmitic acid-labeled rat2 fibroblasts with or without various activators and inhibitors.

3. Results

Silica stimulated the accumulation of labeled [³H]PEt in a time- and concentration-dependent manner. This Silica-induced PLD activity was partially attenuated by the pretreatment with U73122 (phospholipase C inhibitor), genistein (protein tyrosine kinase inhibitor), PD 98056 (MEK inhibitor) and mepacrine (phospholipase A2 inhibitor). But, sphingosine (protein kinase C inhibitor) and DPI (NADPH reductase inhibitor) had not affect the PLD activation by silica. Silica also increased the PLA2 activity about four fold, which imply that the PLD activity is more influenced by the mobilization of PLA2 than other signaling mediators. The PLD activity also partially inhibited calcium chelator EGTA or/and BAPTA/AM compared to silica.

4. Conclusions

The silica-stimulated phospholipase D activity is present in the rat2 fibroblasts and is modulated by combination of various signaling mediators

Diesel Exhaust Particles (DEPs)- Induced Phospholipase D Activation in Raw 264.7 Cells

1) . 3) . 2)
2) . 2) . 2) .
가 1)
가 2)
가 2)

1. Objectives

To evaluate the signaling pathways related to the diesel exhaust particles (DEPs)- induced PLD activation in Raw 264.7 cells

2. Methods

The DEP-induced phospholipase D (PLD) activities were assayed as accumulation of [³H] phosphatidylethanol ([³H]PEt) was examined in [³H] palmitic acid-labeled RAW 264.7 cells. In order to characterize the signaling pathways which increase the activity of PLD in DEP-stimulated Raw 264.7 cell, we tested the effect of various inhibitors on DEP-induced PLD activity in Raw 264.7 cells.

3. Results

DEP stimulated the accumulation of labeled [³H]PEt in a time- and concentration-dependent manner. DEP-induced PLD activity was affected by extracellular and intracelualr Ca²⁺. Also, DEP-induced PLD activation was partially inhibited by pretreatment with PLA2 inhibitor (mepacrine), PTK inhibitor (genistein), PLC inhibitor (U73122), PKC inhibitor (sphingosine) and CaM kinase inhibitor (KN62).

4. Conclusions

The DEP stimulate PLD activation in Raw 264.7 cells. The DEP-induced PLD activity is regulated extracellular and intracellular calcium. PLA2, PLC, PKC, CaM kinase and PTK may play a role in PLD activation by DEP in Raw 264.7 cells.

Auto regulation of Quartz-induced iNOS by iNOS-derived Hydrogen Peroxide in Rat2 fibroblast

가
가

1. Objectives

This study was performed to investigate molecular mechanism regulating Nitric oxide synthase (NOS) that was induced by -quartz in Rat2 fibroblast.

2. Methods

-quartz-induced Nitric Oxide (NO) and H₂O₂ formation and -quartz-induced iNOS protein expression in rat2 fibroblast were monitored. With iNOS inhibitor (L-N⁶-(1-iminoethyl)lysine hydrochloride, L-NIL) or antioxidant (catalase), we observed NO and H₂O₂ formation and iNOS protein expression in Rat2 fibroblast stimulated with -quartz.

3. Results

-quartz stimulated iNOS-induced NO and H₂O₂ formation in Rat2 fibroblast. L-NIL inhibited H₂O₂ formation and iNOS protein expression by -quartz in Rat2 fibroblast. Pretreatment with catalase blocked autoinhibitory pathway of iNOS by iNOS-induced H₂O₂, therefore H₂O₂ and NO production and iNOS protein expression were increased in Rat2 fibroblast stimulated with -quartz

4. Conclusions

-quartz-induced iNOS stimulated H₂O₂ formation in Rat2 fibroblast. INOS-induced H₂O₂ by -quartz play an important role in autoinhibition pathway for regulating iNOS function in Rat2 fibroblast

Cytotoxic Activity of Yellow Sand (Asian Dust Storm) with the Reference to SiO₂ and TiO₂

가 가 가
가 가 가

1. Objectives

This study was designed to evaluate the toxic potential of yellow sand (Asian dust storm) and understand the complicated biological processes involved in the pathogenesis of respiratory effect by yellow sand.

2. Methods

The toxic potential of yellow sand was evaluated by measurement of activity as a Fenton catalyst of dust, cytotoxicity of dust to alveolar epithelial cells (RLE-6TN) *in vitro* and nitrite, H₂O₂ and TNF release from dust stimulated RLE-6TN cells. In addition intracellular calcium was measured in dust-stimulated RLE-6TN cells.

3. Results

Mean aerodynamic diameter of yellow sand prepared without grinding was about 3 μm and irradiated by cobalt 60 for sterilization. Major element of yellow sand was Si (27.7 \pm 0.6 %), Al (6.01 \pm 0.17 %) and Ca (5.83 \pm 0.23 %) in order.

The cytotoxicity of yellow sand was measured as 67.5 % of SiO₂. And this increased cytotoxicity is regarded as the increased fenton activity, ROS (reactive oxygen species) and lipid peroxidation, not RNS (reactive nitrogen species) generation. Also yellow sand increased the release of TNF comparing with TiO₂.

4. Conclusions

The yellow sand-stimulated radical generation in lung epithelial cell may play a important role to understand the toxicity of yellow sand.

(NO)

1.

(exhaled air) NO(nitric oxide)가 가 NO
 NO
 NO 가

2.

2000 7 4 8 24 493 NO
 eco physic CLD 77 AM sp analyzer 100ml/s 2
 plateau NO
 (sex, age, body weight, height,
 arm span, smoking, FVC, FEV1, FEV1/FVC, asthma, wheezing history)
 . data NO log
 . age 10

3.

1. NO (ppb)

	n	mean	std	min	max
total	493	7.83	5.15	0.35	45
male	258	8.33	5.18	1.2	45
female	235	7.28	5.06	0.35	34.85

2. NO (ppb)

	10	10	20	30	40	50	60	70
n	48	65	36	96	116	89	36	8
mean	5.88	9.20	7.29	6.99	8.08	8.48	8.52	7.29
std	5.20	6.51	4.33	3.39	3.84	6.85	5.01	4.89

NO 493 NO 7.83ppb . 258 NO
 8.33ppb 235 NO 7.28ppb . 10 (9.20), 50 (8.48)
 10 (5.88) 가 .

3. (proc GLM)

Parameter	Estimate	Standard Error	Pr > t
Intercept	1.8001	0.5156	0.0005
male	0.0995	0.0617	0.1076
female	0.0000	.	.
arm span	0.0023	0.0031	0.4523
60< age	0.2740	0.1589	0.0853
50-60	0.3132	0.1516	0.0394
40-50	0.3549	0.1495	0.0180
30-40	0.2626	0.1535	0.0877
20-30	0.2658	0.1735	0.1263
10-20	0.4719	0.1460	0.0013
age<10	0.0000	.	.
FEV1/FVC	-0.0073	0.0036	0.0457

P-value 0.3 sex, arm span, age, FEV1/FVC
 10 FEV1/FVC가 P-value 0.05
 NO 10 10 (P=0.0013), 40 (P=0.0180),
 50(P=0.0394) 10 NO 30
 (P=0.0202).

4.

FEV1/FVC가 NO

NO

가

1.

가 ,

SiO₂ (Braunstein , 1977; Morfeld , 1997).

SiO₂ (IARC, 1997) (IARC)

SiO₂ SiO₂가 silica-stimulated cell (reactive oxygen species, ROS) , ROS (IARC, 1997; Shi , 2001), (Allam , 1987). SiO₂

2.

(,) 60

50 110

(Gilian, USA) (NIOSH method 0500), (NIOSH method 0600) (NIOSH method 7602) , NIOSH(1994)

110 lymphocytes

Flow cytometer(Coulter Ltd., USA)

(CD3+, CD4+/CD8+, NK cell, CD4+CD45RA+ CD4+CD45RO+) , serum serum

hydroxyl radical(OH), hydrogen peroxide(H₂O₂) lipid peroxide(LPO) superoxide dismutase(SOD) . serum (IgA, IgG, IgM) Behring Nephelometer Analyzer II(DADE Behring Co, USA)

, Version 7.5 SPSS

(SPSS Inc., USA) t-test

3.

35.4 , 43.4

(p<0.01),

가 . 4.38(SD, 6.07)
 1.49(SD, 1.92) mg/m³ , 0.038(SD, 0.057) mg/m³ .
 serum OH 45.8(SD, 19.5) 67.3(SD, 75.2) nmol/mg protein , H₂O₂
 14.0(SD, 1.46) 17.3(SD, 2.91) nmol/mg protein , LPO 11.6(SD, 6.17)
 11.6(SD, 4.04) nmol/ml serum, SOD 177.5(SD, 311.47),
 107.7(SD, 117.83) U/ml serum . OH, H₂O₂ LPO ,
 SOD , H₂O₂
 (p<0.01). serum IgA, IgG IgM , IgA IgM
 , IgG 973.2(SD, 181.20),
 769.9(SD, 259.31) mg/dl (p<0.01). NK cell, CD3+(total
 T-cell activity), CD4+, CD8+, CD4+CD45RA+ CD4+CD45RO+
 .
 (, ,) ()
 ,) ,
 , 1) 가 가 SOD ,
 가 . 2) OH(r=0.339, p=0.046),
 H₂O₂(r=0.72, p=0.001), OH(r=0.389, p=0.021), H₂O₂(r=0.770, p=0.001)
 , LPO 가
 . 3) OH(r=0.302, p=0.078), H₂O₂(r=0.678, p=0.001)
 , LPO . 4)
 , IgG (r=0.339, p=0.046) IgG
 , IgA IgM
 . 5)
 , 가 .

4.

(inflammation) ,
 (fibrosis)가 ,
 . ROSs (Allan , 1987).
 가 가 OH
 H₂O₂ 가 ,
 가

가

_____ . . .
가 . . .

1.

가 3 A 1968 ,
1,777.38m² . B 1978 ,
가 3 1,424.13m² .
가 .
가 , ,

2.

, 5 52 2 27 8 79

3.

, ,
, 0.069 f/cc, 0.067 f/cc,

0.055 f/cc, 0.048 f/cc 0.047 f/cc ,
 0.052 f/cc, 0.033 f/cc .
 0.1 f/cc 3 (33.3%), 2 (25%)
 , A B 가

(p 0.05).

13m, 2m) (17m, 11m) 가 (

4.

가 가

1

1.

가

2.

5

가 가

가

2 (ileocecal valve) 가 4
24

가

()				
		2	4	11
24	($\mu\text{g/day}$)	4.4	2.8	2.7
	($\mu\text{g/dl}$)	0.5	0.4	0.4

3.

(0.05%)

가

가

가

가

- 1) .
- 1) . _____ 1) .
- 2) .
- 3) .
- 4) .
- 5)
- 1)
- 2)
- 3)
- 4)
- 5)

1.

가

2.

39

12

3.

- 1) : 79.5% 가 ,
3 1,2
68.5% . 40.6%가 ‘ , 59.4%가 ‘ ’
가 62.5% ,
- 2) : 가 3
3 가 12 6 , 3 .
2.5
- 3) : 가 ,

4) , 가 : 2.5 ± 1.2 , 2.5 ± 0.7 .

4.

1) 가 : 가가

2) () : 3 1, 2

3) 가 : 가 가

4) 가 : 가 가

5) 가 : 가 가

가

10.6% .
 , VDT 24.7% 3 , 7.1%가
 26.1% 1/4 . 0.6% , 40%가
 가 13.2%, 12.3% . 가
 158 가 . 83 3/4
 가 156 , 203
 가 4.4%, 6.9%, IUGR 0.6%,
 9.5%, IUGR 1.9%, 0.5% . 5.7%,
 3.8%
 , , , , , ,
 , , , , , , ,
 , , , VDT, , , ,

4.

가 8.7 ,
 7.9
 가 2.7 가
 preliminary study .

Associations of Blood Pressure and Hypertension with Blood Lead, Tibia Lead and Patella Lead and Polymorphisms in the Vitamin D Receptor and δ -Aminolevulinic Acid Dehydratase Genes

_____ ¹⁾ . ¹⁾ . ¹⁾ . ¹⁾ . ¹⁾

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¹⁾

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Department of environmental health science, Division of Occupational health, Johns Hopkins School of Hygiene and Public Health³⁾

1. Objectives

To evaluate the relations of lead exposure indices, that is, blood lead, tibia lead and patella lead, and the effects of VDR and ALAD genotype on the relationship between blood pressure and these lead exposure indices. No prior studies have compared and contrasted associations of tibia lead, patella lead, and blood lead with blood pressure, or evaluated effect modification by ALAD or VDR genotype on these associations.

2. Methods

This study is a cross-sectional analysis of the third year of data from a three-year longitudinal study of the health effects of occupational inorganic lead exposure workers in Korea. The third year data were the focus of the analysis because patella lead was collected during that year, and no prior studies have evaluated associations of patella lead, ALAD and VDR genotype, and blood pressure. Analysis included 399 lead workers who had completed the third visit, through January 2001. Collected or measured variables were a standardized interview for demographics, medical history, and occupational history, blood pressure, blood specimen, spot urine sample, tibia lead concentration and patella lead concentration by X-ray fluorescence (XRF), DMSA chelatable lead and ALAD and VDR genotype. Linear regression analysis was used to model systolic and diastolic blood pressure. Associations between ALAD and VDR genotype and hypertension were evaluated in contingency tables using odds ratios and 95% exact confidence limits. Logistic regression was used to model hypertension status.

3. Results

Mean systolic and diastolic blood pressures of the third year visit (n=399) were 121.8/16.3 and 74.1/13.0 mm Hg respectively, and the prevalence of hypertension (defined as systolic blood pressure < 160 mm Hg or diastolic blood pressure > 96 mm Hg or taking anti-hypertensive medications) was 8.3%. Blood lead, tibia lead and patella lead concentration were 33.9 \pm 16.6 g/dL, 34.2 \pm 38.5 g Pb/g bone mineral and 79.4 \pm 38.5 g Pb/g bone mineral respectively. The prevalence of the ALAD 1-1 genotype and the VDR bb genotype were 89.4% and 88.4% respectively. Tibia and patella lead concentrations of study subjects with ALAD genotype 1-1 were higher at the 3rd visit than ALAD 1-2 genotype (35.0 \pm 39.8 g Pb/g bone mineral vs. 26.0 \pm 21.8 g Pb/g bone mineral and 81.9 \pm 100.5 g Pb/g bone mineral vs. 58.3 \pm 46.1 g Pb/g bone mineral, respectively: p-value <0.05). Subjects with VDR Bb or BB genotype had significantly higher blood lead concentration than VDR bb genotype (38.5 \pm 19.4 g/dL vs. 33.3 \pm 16.2 g/dL : p-value <0.1). Tibia lead was the only positive predictor of diastolic blood pressure. ALAD genotype and VDR genotypes did not modify the relations of blood lead, tibia lead and patella lead with blood pressure. After adjustment, using logistic regression, for age, gender, BMI, tibia lead, and current alcohol use, the odds ratio for the association of VDR genotype with hypertension was 2.0 (95% CI=0.7, 5.3). Tibia lead was a predictor of hypertension status (odds ratio=1.011 [95% CI=1.0011, 1.0202]).

4. Discussion

The primary motivation for this work was to evaluate the relations of patella lead with systolic blood pressure, diastolic blood pressure and hypertension, and to evaluate how polymorphisms of ALAD and VDR genotype modified relations of lead exposure indices and blood pressure. We found no clear associations of patella lead and blood pressure. This result was opposite to our prediction, especially compared with our previous report with the same data about the relation of blood pressure and blood lead, tibia lead and DMSA chelatable lead using first year data from all 801 lead workers. In that previous report, blood lead, tibia lead and DMSA chelatable lead were associated with blood pressure and hypertension. The third year follow up study was finished last June, and the data are now going on analysis about 755 lead workers.

1.

가 , , ,
가

2.

2000 3 1,181 883(74.8%)
963 (83.2%)
1,316
SPSS

3.

1)

883 307 (34.9%), 312 (35.5%), 81 (9.2%),
179 (20.4%) 3 790 (89.5%),2 93
(10.5%) 484 (54.9%) , 5 가 265
(30.1%) , 가 130 (14.8%) 150
가 430 (48.8%), 150-200 438 (49.7%) 200 가 868 (98.6%)

2)

11-20 가 400 (52.7%) 21 260 (35.3%)
가 11 660 (88.0%) 1
11 -20 273 (42.9%) 21 239 (37.5%) 11 612
(80.4%) . 2 21 49 (8%), 11-20 가 223 (37.7%)
가 11 272 (45.8%) . 3 4
가 11 127 (25.0%),91 (19.6%) 가

가 1 가

21 가 128 (44.4%) 99 (37.2%), 3
(5.7%) 가 (P<0.05).

가 2 51 (6.8%), 2 -5 641 (84.9%) 5

692 (91.7%) 1 2-5 488 (63.2%) 2 84
(10.9%) 5 572 (74.1%) 2 2

27(3.5%), 2-5 378 (49.4%) 3 4

5 235 (26.6%), 173 (19.6%)

1 5

273 (96.4%) , 242 (91.3%) 33 (63.4%)

1 가 2

5 가 199 (73.4%) 가 169 (70.7%)

33 (63.5%) 2 60%

3 4 5 가

124 (52.3%), 96 (43.3%) 94 (50.8%), 61 (47.3%)

17 (47.2%), 16 (50.1%) 40%

3)

가 483 343 가 679 661 97.3%

409 251 61.3% 408

218 53.4%

134 (20.4%), 가 64 (21.0%), 61
(27.0%), 73 (36.5%)

248 (39.4%)

62 (33.7%) 14 (7.4%), 25 (11.6%), 가

34 (12.1%)

48 24

17 (7.7%), 55 (22.9%)

870 782 (89.9%) 274

35.4%

B 5 (2.5%) C

1 (0.6%) AIDS 2 (1.2%) 가

B 131 (58.8%) C 47 (27.3%)

AIDS 13 (8.6%)

4)

865 660 76.3%

205 (23.6%)

223 (75.1%) 250 (83.9%)

50 (72.5%)

867 249 (23.6%)

	638 (71.3%)			
	72 (24.0%),	88 (28.7%),	19 (23.8%)	
		가 30%		
	868	819 (94.4%)	가	
가				287 (95.0%),
296 (96.7%),	72 (90.1%)			90%
가			가	867
813 (93.8%)				
가 278 (92.0%),	284 (92.4%),	75 (94.6%)		90%
		867	285 (67.1%)	
	가 196 (64.9%)			195 (63.9%)
	63 (78.8%)			
60%				

1.

10 H 가

2.

, 가 , , 가

3.

35 25 10 . 1997 8

11 18 P
 가 μl 10 11 22 가 μl

10 , 10.5 g/dl . 11 23

가 K . 11 24 K

117/74 mmHg , 78 , 24 , 38.5 .

2 FB가 ,

98.5 $\times 10^3$ / μl blast 가 85% . 8.5 g/dl,

26.5%, 86,000 / μl . 21 U/ 53 U/ .

(Na-K) 135-4.3 mmol/ . 14.1 ,

(aPTT) 39.9 . 105 M/dl .

. 11 25 AML M2

myeloid lineage with CD7, CD19(+) . 11 26 philadelphia

bone marrow R/0 46 XY t(1:4)(p32:p16)

가

3-4 , 1 1 10

1997 11 27

1999 5 30

1,800 mRem (18

mSv)

(Probability of Causation) 5.4-6.1%

50% 가 1%

4.

, 가 가 가 , ,
 ,
 .
 가 가 가
 가 , 가

가

_____ . . 1) . 2) .
 ,
 1)
 2)

1.

가 가
 , 가 , .
 , 가 , 가 .
 , 가 .
 , 가 .
 .

2.

2001 5 , 228 (,)
 ,) (, ,)
 , 2000 .
 45-64 139 (45-54 : 99 , 55-64 : 40) .

3.

228 가 가 212 (93.0%) , 125 (59.0%), 81
 (38.2%), 6 (2.8%) . ()
 1) 53.9 , 57.4 48.2 .
 2) (BMI) 23.5 23.5, 23.3 , BMI 25
 , 29.6%가 31.2%, 27.2% (45-54
 : 39.4%, 55-64 : 37.5%).
 3) 191.9mg/dL 192.1, 190.8 ,
 260mg/dL , 2.9%가 4.0%,
 1.2% (45-54 : 6.1%, 55-64 : 2.5%).
 4) (140mmHg/ 90mmHg) 56.8 % ,
 61.6%, 49.4% (45-54 : 34.3%, 55-64 : 42.5%).

5) (126mg/dL) 8.3% , 12.3%,
5.6% (45- 54 : 13.1%, 55- 64 : 17.5%).

4.

가 , 가 .

3

_____ 1), 1), 2), 3)
 1)
 2
 3)

1.

1990 1990 가 가
 가 ,

2.

, 1995 1997 3
 , Proportional hazards model (Cox regression)
 Hazard ratios
 70 가 400
 가 가
 Ethnographic approach

3.

,
 1997 가 , 1995 1997

, 1990 가 ,
 . 가 가
가 . , IMF 가 .

4.

, 가 ,
(,) 가 가 ,
가 .