(Suzuki et al., 2008).

Benign breast disease is a likely risk factor (Dorjgochoo et al., 2008) and inflammation clearly could play an important role, with suggestions of COX-2 andTGF-beta1/TNF-beta contributions to the etiology of mammary neoplasia (Lee et al., 2005; Gao et al., 2007). Stress also might impact on risk, having "ikigai" (life power) decreasing (Wakai et al., 2007) and short sleep duration elevating the likelihood of breast cancer (Kakizaki et al., 2008). Whether melatonin could act as an intervening factor between light exposure at night and serum concentrations of estrogen remains unclear (Nagata et al., 2008).

With regard to screening, there have not been any randomised control trials of mammography in the region, although biennial mammographic screening starting at age 50 years is being recommended, for example in Korea (Woo et al., 2007), but not Hong Kong and other Asian populations with low breast cancer prevalence (Leung et al., 2002). In one trial of breast self-examination conducted in Shanghai, intensive instruction did not reduce mortality, but the control arm also had very early cancers so that the population was already well educated (Thomas et al., 2002). Earlier studies in Japan did point to screening reduction of mortality (Kuroishi et al., 2000). One problem is compliance and women in the contemplation stage need specific and correct knowledge delivered by diverse materials, reminders, and the inclusion of husbands as facilitators (Park et al., 2007). Breast surgery is relatively straight forward and surgical volume of hospitals does not appear to generally affect the 10-year survival rate from breast cancer significantly (Nomura et al., 2006).

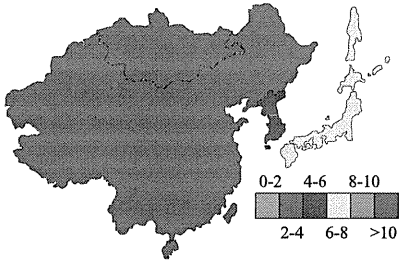

Figure 27. Ovarian Cancer Incidences/100,000 (Globocan, 2002: Ferlay et al., 2004)

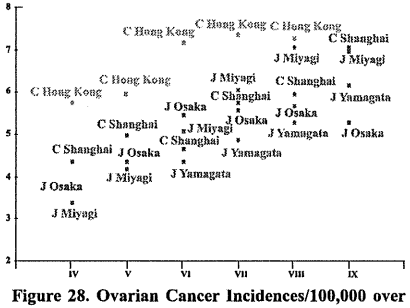

Figure 28. Ovarian Cancer Incidences/100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

Table 8. Ovarian Cancer Incidences: % Types

	Serous	Mucin	Endo	Clear	Adeno
Guangzhou	29.6	11.3	7.9	0.8	26.3
Hong Kong	24.4	16.2	17.2	12.5	10.2
Jiashan	17.0	17.7	4.3	0.0	40.4
Shanghai	4.1	1.9	3.4	1.9	7.2
Zhongshan	15.2	21.6	4.0	3.2	16.8
Busan	34.5	17.4	9.3	5.5	11.1
Daegu	30.7	19.5	8.3	6.7	8.3
Daejeon	35.8	8.8	10.8	4.1	14.2
Gwangju	30.7	19.6	8.0	2.5	13.5
Incheon	31.8	21.6	11.7	6.0	11.3
Jejudo	24.3	13.5	8.1	2.7	13.5
Seoul	34.2	15.7	11.9	7.4	10.0
Ulsan	28.8	16.3	6.7	2.9	8.7
Aichi	33.7	14.6	12.6	12.6	14.1
Fukui	46.9	13.5	13.0	6.8	6.8
Hiroshima	30.7	20.1	8.9	16.0	13.0
Miyagi	19.2	13.0	12.6	19.0	19.0
Nagasaki	29.2	14.6	15.1	10.8	16.2
Osaka	29.7	13.2	9.8	12.4	17.3
Yamagata	29.7	9.9	11.8	12.9	21.7

Data from Curado et al., 2007

Ovarian Cancer

Ovarian cancer is relatively infrequent in China and Mongolia and moderately common in Korea and Japan (see Figure 27). However, there is considerable variation across registries and Hong Kong has high incidences. With the exception of this registry, rates are generally increasing (see Figure 28).

One problem is that there are various subtypes and these have differences in risk factors (Sagae et al., 2003). In Japan, clear cell lesions may be more prevalent than elsewhere (see Table 8). One study in low incidence Taiwan showed marked differences in the distribution of histologic subtypes compared with high-incidence populations (Yen et al., 2003). Nevertheless there is an overall link with exposure to estrogen, and full-term and incomplete pregnancies, lactation, and oral contraceptive use are protective (Zhang et al., 2004a; 2004c). Although a CYP1A1 polymorphism was not related to ovarian malignancies in one Japanese study (Sugawara et al., 2003), intake of soy and isoflavones is inversely related to risk (Zhang et al., 2004d; Sakauchi et al., 2007), while hormone replacement therapy increases the chances of developing an ovarian cancer (Zhou et al., 2008).

Overweight is a factor in Japanese (Niwa et al., 2005), especially with change of BMI after 20 years of age (Hirose et al., 1999). The situation 5 years before diagnosis appears important in Chinese (Zhang et al., 2005). Sedentary behaviour also elevates the risk (Zheng et al., 1993; Zhang et al., 2004e), decline being observed with increasing duration of strenuous sports and frequency of activity-induced sweating among pre-menopausal women, and moderate post-menopausal activity (Zhang et al., 2003).

Consumption of foods low in fat but high in fibre,

carotene and vitamins appears to be protective in China (Zhang et al., 2004a), with a beneficial role for carotenoids (Zhang et al., 2007). Fresh vegetables and fruits appear to exert preventive influence, while animal fat and preserved (salted) vegetables (Zhang et al., 2002b), as well as dried or salted fish (Sakauchi et al., 2007) are potential risk factors. Silica dust may also increase the risk of ovarian cancer in Shanghai workers (Wernli et al., 2008) and a smoking link was reported in Japan (Niwa et al., 2005).

Regarding screening, annual gynecological examination (sequential pelvic ultrasound and serum CA125 testing) does allow detection of early-stage ovarian cancer in asymptomatic postmenopausal women (Sato et al., 2000; Kobayashi et al., 2008), but the degree of significance is problematic.

Corpus uteri

Endometrial cancer is relatively uncommon in China, Mongolia and Korea, but a little more frequent in Japan (see Figure 29). However, there is considerable variation within registries within China and the rates in Hong Kong and Zhongshan are high. Across the region, rates are gradually increasing (see Figure 30).

The most important risk factors appear to be estrogen exposure related, including early age at menarche, late age at menopause and nulliparity (Xu et al., 2004; Wernli et al., 2006), while pregnancies, including induced abortion, generally reduce the risk (Xu et al., 2004). Oral contraceptive and IUD use also protect against endometrial cancer (Tao et al., 2006). Breastfeeding may reduce (Okamura et al., 2006) while long-term tamoxifen and ever-use of sex hormones may promote the development of lesions in Japanese women (Khan et al., 2006; Yamazawa et al., 2006). Findings for polymorphisms in hormone metabolizing, receptor and sex hormone-binding globulin genes are also in line with an estrogen exposure etiology (Iwamoto et al., 2003; Kataoka et al., 2007; Tao et al., 2006; 2007) and soy foods may be protective (Xu et al., 2004b; 2007a). However, pregnancy history, menopause age, BMI and presence of diabetes mellitus or hypertension were not related to endometrial tumour development in one study (Nakamura et al., 2006).

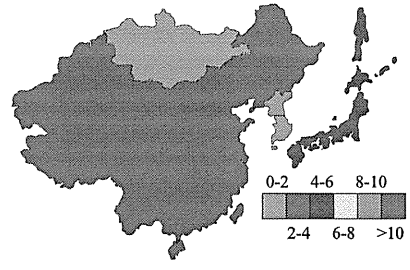

Figure 29. Endometrial Cancer Incidences/100,000 (Globocan, 2002: Ferlay et al., 2004)

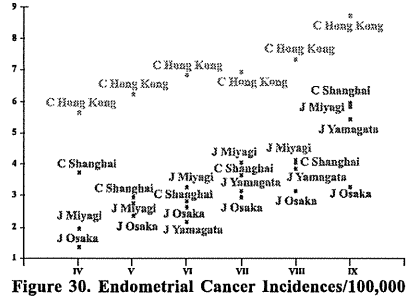

Figure 30. Endometrial Cancer Incidences/100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

Hormone involvement is also suggested by a positive link with adult but perhaps not adolescent obesity (Shu et al., 1993b; Xu et al., 2002; Wen et al., 2008). In particular, upper-body fat deposition is associated with an increased risk of endometrial cancer (Xu et al., 2005) and adult weight gain, especially during the peri-menopausal period, should be avoided (Xu et al., 2006b). Obesity-related insulin resistance and proinflammatory effects may play important roles (Wen et al., 2008) and both lower intensity lifestyle activities like walking and doing household chores and intentional exercise can reduce endometrial cancer risk (Matthews et al., 2005). Those with sedentary jobs or reporting sedentary life-styles were earlier found to have a somewhat increased risk (Shu et al., 1993a).

Diets rich in animal fat and animal protein may play an important role in the etiology of endometrial cancer. (Shu et al., 1993b; Xu et al., 2006a), while protection is afforded by dietary fiber, retinol, beta-carotene, vitamin C, vitamin E and vitamin supplementation (Xu et al., 2007b), with positive contributions for dark green/dark yellow vegetables, fresh legumes, and allium vegetables but not fruit (Tao et al., 2005). Folate intake is beneficial, interacting with MTHFR polymorphisms (Xu et al., 2007; 2008). Coffee consumption is also reported to protect against endometrial cancer in general (Hirose et al., 2007; Shimazu et al., 2008), as well as the endometrioid adenocarcinoma in postmenopausal women (Koizumi et al., 2008). Tea consumption demonstrated an inverse association in one study (Xu et al., 2007a). Paradoxically, cigarette smoking and alcohol may also significantly reduce the risk of endometrial cancer (Hosono et al., 2008; Zhou et al., 2008).

A pointer to toxic agent effects was provided by the reported link with textile industry exposure in Shanghai (Wernli et al., 2008) but expression of s major DNA repair protein was found to be without effect in Korea (Jo et al., 2007).

Cervix uteri

The incidence of cervical cancer is generally low in China, moderate in Japan and somewhat higher in Korea and Mongolia (see Figure 31). Rates are generally decreasing (see Figure 32), with some exceptions like in the elderly in Korea (Jo et al., 2007), this also being observed for mortality (Shin et al., 2008). However, it remains an important health problem among women in both China and Mongolia (Shi et al., 2008), Taiwan and Hong Kong (Tay et al., 2008) and Korea and Japan (Konno

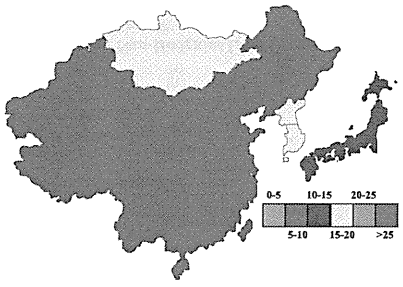

Figure 31. Cervical Cancer Incidences/100,000 over Time (Globocan, 2002)

et al., 2008). In Taiwan, interest focused in the 90's on an increasing trend in recent cohorts (birth after 1963) with a possible role of female sex hormones as an age effect, promiscuous sexual activity as a period effect and change in reproductive behavior as a cohort effect (Wang and Lin, 1996; 1997). The ratios of SCC to AC are shown in Table 9 for selected registries. Typically, adenocarcinomas account for 15-20% of the total burden.

The major risk factor is well recognized to be exposure to high-risk human papilloma virus (HPV) strains. The prevalence of high risk forms of the virus in Asian populations is well documented (Ghim et al., 2002; Anh et al., 2003; Shin et al., 2003). Levels in Shanghai are now the same as the worldwide rate, viral load predicting cervical lesions overall (Zhang et al., 2008). Both cervical cancer and cervical intraepithelial neoplasias (CIN) are highly influenced by HR-HPV viral load (Zhao et al., 2004). Seroprevalence correlates with genital HPV exposure in young Korean women, but its meaning in young men is unclear (Clifford et al., 2007). HPV status and histologic grade are independent predictive risk factors for progression and may be useful in the management of CIN (Konno et al., 1998).

Rate of high-risk HPV ranges from about one third in early CIN to almost 100% in carcinoma. HPV 16 is the most prevalent type in squamous lesions, followed by HPV types 58, 52 and 59 then HPV18 in Sichuan (Wu et al., 2008), 52, 58, 31 and 39 in Shenzhen City (Wu et al., 2007), 52 and 58 in Shenyang (Li et al., 2006), 58, 52, 33

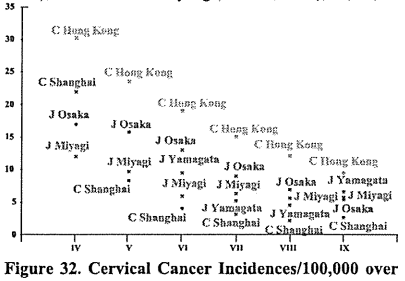

Figure 32. Cervical Cancer Incidences/100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

Table 9. Cervical Cancer Incidences: SCC/AC Ratios

	SCC	AC	Ratio	Total Number
Guangzhou	76.2	12.1	6.3:1	265
Hong Kong	75.6	16.7	4.5:1	2,193
Jiashan	87.9	9.1	9.7:1	33
Shanghai	18.1	6.2	2.9:1	629
Zhongshan	63.2	22.6	2.8:1	133
Busan	84.9	9.7	8.8:1	1,693
Daegu	85.1	10.5	8.1:1	1,149
Daejeon	82.3	7.1	11.6:1	678
Gwangju	76.5	15.2	5.0:1	532
Incheon	85.8	9.0	9.5:1	1,305
Jejudo	82.5	9.5	8.7:1	137
Seoul	84.5	10.0	8.5:1	4,644
Ulsan	86.9	8.5	10.2:1	260
Aichi	74.0	16.8	4.4:1	208
Fukui	72.8	13.6	5.4:1	191
Hiroshima	82.2	11.9	6.9:1	489
Miyagi	72.4	16.3	4.4:1	449
Nagasaki	79.4	13.1	6.1:1	627
Osaka	76.8	15.2	5.1:1	1,713
Yamagata	78.2	16.4	4.8:1	298

Data from Curado et al., 2007

and 18 in Shanxi (Dai et al., 2006) and 16, 18, 58, 52, 33 and 31 in Hong Kong (Liu et al., 2008). After HPV 16, types18, 31, 51,52 and 58 are most common in Japan (Sasagawa et al., 2001), 18, 31, 33, 35, 35, and 58 in Okinawa (Maehama et al., 2002) and 18, 58 and 33 in Korea (Lee et al., 2007). Guangdong appears exceptional in that the most predominant genotypes in cancers are HPV 52 and 58 (Lin et al., 2008). HPV-18 may more common than -16 in cervical adenocarcinoma, from data in Yanbian, northern China (Zhao et al., 2008).

In addition to HPV infection, age at first sexual intercourse and number of live births are associated risk factors in Hubei (Cai et al., 2008), multiple sexual partners, cervical inflammation, and public bathing in Shanxi (Zhao et al., 2006), multiple full term pregnancies, early age at first intecourse in Korea (Yoo et al., 1997) and early age at first pregnancy and multiparity in Japan (Yoshikawa et al., 1999). In Mongolia, lifetime number of sexual partners and induced abortions were shown to be directly associated with HPV DNA and/or seroprevalence (Dondog et al., 2008) and sexually transmitted infections are common, being found in 53% of female attendees of an urban STD clinic (Garland et al., 2001).

Smoking is a well established risk factor (Hirose et al., 1996; 1998; Matsumoto et al., 2003; Odongua et al., 2007) and lifetime ETS exposure is a major determinant for contracting cervical neoplasms among nonsmoking women in Taiwan (Wu et al., 2003). Chlamydia trachomatis infection were revealed may also be a significant risk factor for CIN (Matsumoto et al., 2003). Diet is also an important influence and daily intake of fruit and frequent consumption of boiled or broiled fish are protective (Hirose et al., 1996; 1998)

Health services clearly need to emphasize education about cervical cancer prevention while concentrating on screening (Woo et al., 2005; Jo et al., 2007; Jun et al., 2009). In Taiwan, with about 50% compliance with

screening guidelines, cancer knowledge is the most significant factor determining attendance (Liao et al., 2006). In China's rural areas with low-resource settings visual inspection with Lugol's iodine can be one of the primary screening tests if the screening frequency is to be increased (Li et al., 2006). HPV testing is another alternative and in Japan, sensitivity and the positive predictive value for detecting CIN proved superior to cytology in one investigation (Inoue et al., 2006). Based on comparisons of HPV testing and Pap with the existing healthcare infrastructure in China, however, it was concluded that refinement of primary HPV screening using centralized labs is needed (Belinson et al., 2001).

For the future the great long term hope is being placed on vaccination, although there remain barriers other than simply high monetary cost, including uncertain length of vaccine effectiveness, low perceived risk of HPV infection, no immediate perceived need of vaccination, anticipated family disapproval and fear of the pain of injection (Kwan et al., 2008). It has been estimated that the vaccine is cost-effective; for it to be affordable, however, even with financing assistance, vaccine prices may need to be even lower (Goldie et al., 2008).

Brain and Nervous Tissue Cancer

Brain and nervous tiisue cancers appear to be more prevalent in China and Korea than in Japan and Mongolia (see Figure 33). There does not appear to be any consistent trend over time, at least in the last 20 years (see Figure 34), although this might depend on the type and sex (Kaneko et al., 2002).

Risk of brain cancer increases with consumption of salted vegetables and salted fish (Guo et al., 1994; Hu et al., 1998; 1999a), as well as liquor intake and diseases related to the brain (Hu et al., 1998), while vegetables and fruit and intake of vitamin E calcium may be protective (Hu et al., 1998) Employment in textile industry maintenance jobs and exposure to wool products may be associated with an increased risk of brain tumors (Gold et al., 2008) and meningiomas were found to be positively associated with occupational exposure to lead, tin, cadmium and ionising radiation (Hu et al., 1999b). Patients with NAT2*7 alleles tend to have high-grade astrocytomas or glioblastoma multiforme (Liu et al., 2008). In Korea, significant increase in age-standardized rate ratios of cancers for high-power vs. low-power sites, with the exceptions of total cancer and of brain cancer in women (Ha et al., 2003). Recently, no effects were established for mobile phone use and exposure to radio frequency electromagnetic fields (Takebayashi et al., 2008) .

Thyroid cancer

Thyroid cancer is of medium importance in Japan and Korea, but is rarer in China and Mongolia (see Figure 35), but appears to be on the increase (see Figure 36). This is in line with increasing obesity since body size in early life

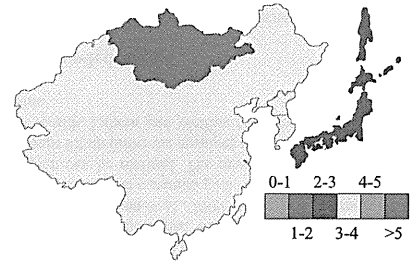

Figure 33. Male Brain Cancer Incidences/100,000 (Globocan, 2002: Ferlay et al., 2004)

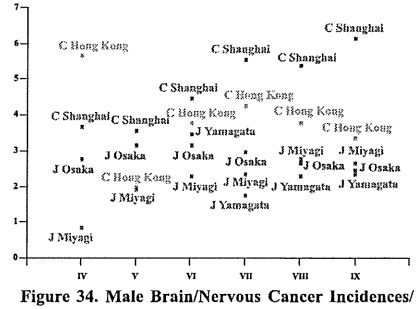

Figure 34. Male Brain/Nervous Cancer Incidences/ 100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

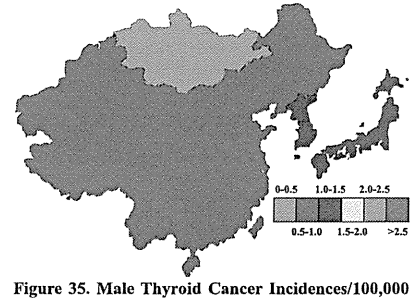

Figure 35. Male Thyroid Cancer Incidences/100,000 (Globocan, 2002: Ferlay et al., 2004)

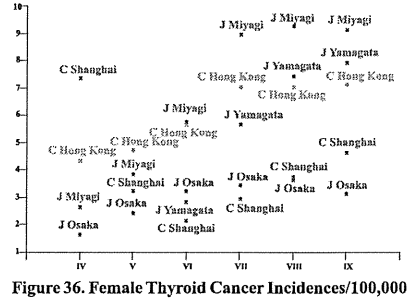

Figure 36. Female Thyroid Cancer Incidences/100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

as well as in adult is associated with an increased risk of papillary thyroid cancer (Oh et al., 2005; Suzuki et al., 2008). Despite the female predominance, there do not appear to be clear associations with reproductive factors (Wong et al., 2006). A history of goiter/nodules, CT examinations, and familial cancer are risk factors, while smoking and alcohol drinking, as well as tea consumption, may be protective (Zhe et al., 2006; Nagano et al., 2007).

Non-Hodgkins Lymphomas and Leukemias

Non-Hodgkins lymphomas (NHL) are more prevalent in Japan and Korea than China and Mongolia, in that order (see Figure 37), and are generally increasing in incidence (see Figure 38), while leukemias are moderately frequent throughout the region (see Figure 39), but possibly decreasing (see Figure 40).

The distribution of NHL subtypes may also be changing, as evidenced in Taiwan (Chang et al., 2008), and risk factors include HBV or HCV infection (Ohsawa et al., 1999; Kim et al., 2002; Matsuo et al., 2004c), with influence for polymorphisms in folate-metabolizing genes (Hishida et al., 2003; Matsuo et al., 2004a) and p53 (Hishida et al., 2004). DNA repair may play a limited role in lymphomagenesis independent of the histological subtype (Matsuo et al., 2004), while regular alcohol consumption is associated with reduced risk, with no change observed for smoking (Matsuo et al., 2001).

With leukemia, environmental pollution with rare-earth elements and organophosphorus pesticides may be important (Wu et al., 2003) as well as working exposure to benzene, synthetic fiber dust, radioactive materials, and toluene (Adegoke et al., 2003). Possible protective effects of green tea intake have been reported (Wu et al., 2003; Zhang et al., 2008). While higher mortality rates for leukemia were noted in some age groups in areas near AM radio broadcasting towers (Park et al., 2004), findings in Japan do not support any leukaemogenic impact of nuclear power plants (Yoshimoto et al., 2004)

Childhood cancers

Data on time trends are very limited and surveys do not appear to have been conducted recently (Ajiki et al., 1994). Paternal preconception smoking is related to a significantly elevated risk of childhood cancers, particularly acute leukemia and lymphoma (Ji et al., 1997; Liu et al., 1997), with possible modification by the CYP1A1 genotype (Lee et al., 2009). Maternal exposure to organic solvents during periconception might also be important (Sung et al., 2007) and DNA repair may exert an influence (Wang et al., 2006). There is also some evidence for leuemia risk increase associated with residential proximity to high-voltage power (Mizoue et al., 2004) or AM radio transmitters (Kim et al., 2002) , and high MF exposure (Kabuto et al.,2006). Furthermore, childhood brain tumors are associated with paternal use of hard liquor prior to the pregnancy (Hu et al., 2000).

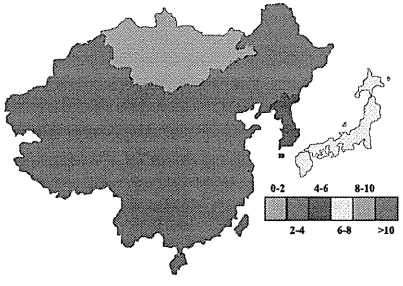

Figure 37. Male Non-Hodgkins Lymphoma Incidences/100,000 (Globocan, 2002: Ferlay et al., 2004)

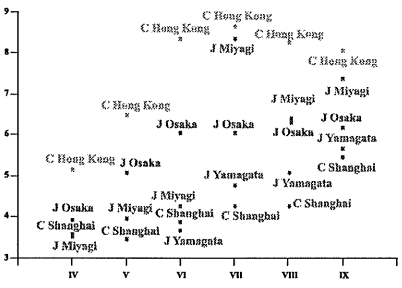

Figure 38. Male Non-Hodgkins Lymphoma Incidences/100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

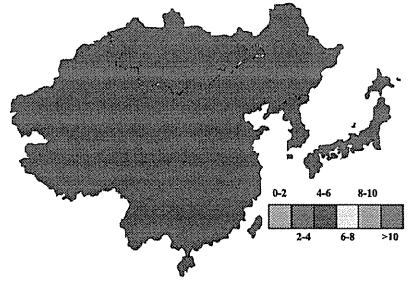

Figure 39. Male Leukemia Incidences/100,000 (Globocan, 2002: Ferlay et al., 2004)

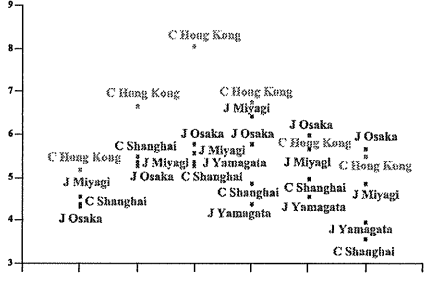

Figure 40. Male Leukemia Incidences/100,000 over Time (Waterhouse et al., 1982; Muir et al., 1987; Parkin et al., 1992; 1997; 2002; Curado et al., 2007)

Future Perspectives

From the present overview, it is clear that a great deal of information has already been generated regarding cancer prevalence and risk factors in North-East Asia, as stressed aearlier in a recent reveiw (Park et al., 2008). The various collaborative efforts within countries, like the Japan Collaborative Cohort Study (Tamakoshi, 2007), the Research Group for the Development and Evaluation of Cancer Prevention Strategies in Japan, the Korean Multicenter Cancer Cohort Study including a Biological Materials Bank (KMCC-I) (Yoo et al., 2002) and other cohorts in Korea (Yoo et al., 2005) and elsewhere will guarantee that new information will continue to be produced. The focus will continue to be on genetic-environment interactions as at the Hospital-based Epidemiologic Research Program at Aichi Cancer Center (Tajima et al., 2000; Hamajima et al., 2001). Genotype-phenotype links will also be stressed (Shin et al., 2008).

It is to be hoped that the collaboration started between China, Korea and Japan with the Korean-Japan-China (KOJAC) (Tajima et al., 2009) study will expand in the future so that the common goals of cancer control in the region can be realised. The new international research groups set up under the auspices of the Japanese Ministry of Health, Labour and Welfare should contribute to this aim.

Acknowledgment

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特集 消化器癌治療成績のさらなる向上に向けて

臨床家にとっての地域がん登録の意義，今後の展望

Significance and future direction of population-based cancer registry from clinician's viewpoint

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SOBUE Tomotaka

地域がん登録は，臨床家にとって直接診療に役立つ情報を提供する仕組みとは認識されにくいが，がん対策を正しく方向付け，効率よく実施するために必要ながん罹患情報を提供できる必須の情報インフラである．生存率は，地域がん登録，院内がん登録，臓器がん登録，それぞれに基づいて算出可能であるが，目的，対象とするがん患者の範囲，収集する情報がそれぞれで異なり，相互に補完すべき関係にある．一方，がん医療の質の均てん化の程度を検証するためには，生存率などの指標に加えて，標準治療実施割合など，直接診療に係わるデータが必要であり，追加的な仕組みを検討する必要がある．

I. がん対策とがん登録

2006年の「がん対策基本法」成立により，わが国においても国および都道府県レベルでがん対策に取り組む方向性が明文化された．諸外国をみても，国が主体となってがん対策に取り組むことにより，すでにがん死亡率の減少などの大きな成果を上げつつある国がある．WHOも国家的がん対策プログラム（National Cancer Control Programme）の推進を提唱している．その目的とするところは，第一に，がんの罹患率と死亡率を減少させることであり，第二に，がん患者とその家族のQOL（Quality of life）を向上させることである．予防・早期発見・診断・治療・終末期ケアからなる一連のがん対策において，証拠に基づいた戦略を系統的にかつ公平に実行し，限られた資源を効率よく最大限に活用することにより，上記2つの目的を達成させることが，その内容である．このがん対策を正しく方向付けるには，がんの実態を正確に把握する必要がある．がん登録はがんの実態を把握するための中心的な役割を果たし，がん対策を実施する上で必須の仕組みである．2007年に閣議決定された「がん対策推進基本計画」の中でも，がん登録は3つの重点的に取り組むべき課題のうちの1つにあげられた．

従来のがん登録は，異なった地域でがんの罹患率を計測して比較することにより，がんの原因を探索するといった研究的な目的が主体であった面があるが，近年の傾向としては，がん対策を実施するための情報インフラとしてがん登録を位置づけるように変化してきている．その意味では，がん対策を実施しないのであれば，必ずしもがん登録をすべての地域で整備する必要はないといえる．

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Key words：がん登録／がん対策／生存率

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II. がん登録の種類と機能

がん対策を進める上で，がんの実態を表す指標として，がんの死亡率（数），罹患率（数），および生存率がある．死亡率，罹患率は，住民数を分母とするが，生存率はがん患者を分母とする．定常的な状態では，死亡率／罹患率＝1－生存率の関係が成立する．死亡率（数）は，死亡診断書に基づく厚労省の人口動態統計により全数把握可能なシステムが確立している．罹患率（数），生存率は，がん登録により計測されるが，がん登録には，地域がん登録，院内がん登録，臓器がん登録の3種類がある．罹患率（数）を計測する唯一の仕組みが，地域がん登録である．

一方，生存率は，地域がん登録，院内がん登録，臓器がん登録のそれぞれで計測が可能であるが，目的，対象とするがん患者の範囲，収集する情報がそれぞれで異なる．

以下，地域がん登録，院内がん登録，臓器別がん登録について概説する．

1．地域がん登録

地域がん登録は，対象地域の居住者に発生したすべてのがんを把握することにより，がんの罹患率と地域レベルの生存率を計測する仕組みである．わが国では，県を単位として実施されており，罹患を把握する主な情報源は，①がんの診断情報を記録した医療機関からの登録票と，②対象地域におけるがん死亡情報である．両者を統合して，登録票のない患者（死亡情報のみの患者）の補完登録を行うとともに，登録精度を計測する．同じ患者の同じがんを誤って複数件計上することのないように，個人識別指標を照らし合わせて個々の患者（腫瘍）ごとに集約する．このための個人識別指標として，正確な生年月日，姓名，性別，住所情報の収集が不可欠である．

一方，対象地域に発生したすべてのがんを把握して初めて正確な罹患情報を計測できるので，登録時に個々のがん患者自身からの同意は確認せずに登録することが国際標準となっている．この点は，個人情報保護の観点からは本人同意原則に対立する行為となり，公衆衛生の観点からの社会全体の利益との調整が必要となる．アメリカ，カナダ，オーストラリア，北欧諸国などでは，法律でがんを届出義務のある疾患と定めることで，この問題を整理している．

わが国の地域がん登録は，1950年代より広島・長崎で開始され，1960年代に，府県を実施単位とする地域がん登録が，宮城，大阪などで始まった．その後，老人保健事業の成人病指導管理協議会「成人病登録・評価部会」の事業として地域がん登録が位置づけられ，補助金支給が開始されたことをきっかけに実施県が増加し，2008年度現在35道府県1市で実施されている．このようにわが国の地域がん登録は，諸外国に比べて開始時期は古いが，この間，国からの関与は研究班としての支援のみであり，法的基盤が弱く財政的支援が乏しい中で，医療関係者の篤志的努力により運営がなされてきため，登録精度が低いのが最大の欠点である（表1）．世界各国のがん罹患データを収集した「5大陸のがん罹患」最新巻において，わが国から掲載された7登録のみであり，その他の多くの登録は掲載されるための十分な登録精度を達成できていない．また，地域ごとに独自の工夫がなされたために，かえって作業手順の標準化が遅れており，罹患率の地域間比較・全国推計値の算出の際に障害となっている．標準化の促進と登録精度の向上が，わが国の地域がん登録の緊急の課題である．

地域がん登録の実際の作業内容などについては，以下のホームページを参照されたい．

- 国立がんセンターがん対策情報センターがん情報サービス＞医療関係者向け＞地域がん登録（http://ganjoho.jp/professional/registration/index.html）

表1 各種がん登録の特徴

	地域がん登録(県単位)	院内がん登録(施設単位)	臓器別がん登録(臓器単位)
目 的	地域のがん実態把握	施設のがん診療評価	全国のがんの詳細情報の収集
実施主体	都道府県(市)	医療機関	学会・研究会
登録対象	対象地域の全がん罹患例	当該施設の全がん患者	専門病院のがん患者
収集項目	診断，初回治療， 予後：標準25項目 (2004年)	診断，初回治療， 予後：必須・標準60項目 (2006年修正版)	臓器により異なるが，項目数は多い (100～300項目)
現 状	35道府県1市にて実施	全がん協加盟32施設 拠点病院375施設	19臓器ががん研究助成金研究班に参加
問題点	・罹患の把握漏れが多い ・標準化の遅れ ・予後調査未実施・負担大	・診療科単位の登録 ・医師による入力 ・腫瘍登録士の不足 ・標準化の遅れ ・不完全な予後調査	・個人情報の扱い ・不完全な予後調査

● 地域がん登録全国協議会
(http://www.cancerinfo.jp/jacr/)
● 厚労省第3次対がん総合戦略事業「がん罹患・死亡動向の実態把握の研究」班
(http://ncrp.ncc.go.jp/)

2．院内がん登録

院内がん登録は，当該施設でがんの診断・治療を受けた全患者について，がんの診断，治療，予後に関する情報を集約する仕組みである．当該施設における診療の実態を把握し，生存率を計測するなどの機能評価を行うとともに，地域がん登録への届出の役割も果たす．1つの施設内に留まるため，個人情報を第三者提供する際の個人情報保護法上の問題点はないが，病院の他の個人情報と同等かそれ以上の安全管理上の扱いが求められる．アメリカにおいては，院内がん登録を実施することが，アメリカ外科学会(American College of Surgeons)のがん委員会(Commission on Caner)によるがん専門病院認定(CoC Approval Program)の必須要件となっており，しかも，アメリカ腫瘍登録士協会認定の腫瘍登録士が医師の手を借りずに独自に新規症例を検索し必要な情報をカルテから抽出して入力する仕組みが標準的となっている．

わが国においては，施設全体をカバーする院内がん登録が存在しない施設が多く(診療科ごとに医師が自発的に登録している場合が多い)，また，院内がん登録が運営されている場合でも，医師以外の担当者(通常，診療情報管理士)の数が少なく，専門知識を備えていない場合が多いので，情報の入力を医師に頼る仕組みがほとんどであった(表1)．

2002年度から開始された「地域がん診療拠点病院」の指定要件に，院内がん登録システムの記述が含まれ，また，2006年度からは「地域がん診療連携拠点病院」(以下，拠点病院)と名称を変更してその指定要件には，標準登録様式に基づく院内がん登録を実施することが明記された．さらに2008年には，がん対策情報センターによる研修を受講した専任の院内がん登録の実務を担う者を1人以上配置すること，および，毎年院内がん登録の集計結果等をがん対策情報センターに情報提供することが明記された．今後，わが国のがん登録を整備するためのいくつかのステップの中で，地域がん診療連携拠点病院における院内がん登録を整備することが，第一に達成すべき課題であり，これを基盤として，地域がん登録，臓器がん登録の取り組みへと展開することが期待できる．

院内がん登録の実際の作業内容などについては，以下のホームページを参照されたい．

● 国立がんセンターがん対策情報センターがん情報サービス＞がん診療連携拠点病院向け＞院内がん登録(http://ganjoho.jp/hospital/cancer_registration/index.html)

3．臓器がん登録

臓器がん登録は，学会・研究会が中心となって，会員医師が所属する比較的大きな病院から学会・研究会の中央事務局にデータを集約することにより，全国規模の登録を実施する仕組みである．専門的な医師のいる病院に限られるため，症例に偏りのある危険性があるが，詳細な臨床情報が収集されているため，より適切な進行度分類のあり方の検討，詳細な治療法別の生存率の計測などが可能である．ただし，予後調査の方法が各医療機関によりさまざまであり，5年生存率を計測するために5年経過時点での生存確認ができていない例が多いなどの課題もある(表1)．また，個人情報保護法や疫学研究倫理指針などの制定により，個人情報の取り扱いについての再検討が求められてきたが，多くの臓器がん登録では，それぞれ目的にあった匿名化の方法を採用し，個人情報についての問題点を整理した上で，倫理審査の手続きを経て，登録作業を再開している．

4．生 存 率

地域がん登録による生存率は，当該地域に発生する全症例に基づく生存率を算出できるので，地域ごとの比較が可能である．しかし，詳細な病期・治療内容の情報を収集しないため，これらの要因別の分析はできない．すなわち，生存率に違いが観察されてもその要因を特定することは難しい．ヨーロッパ22ヵ国のがん生存率共同研究であるEurocare Studyでは，通常の地域がん登録で得られる情報に加えて，より詳細な臨床情報を追加的に調査したり(pattern of care study)，病理標本を集めて中央でレビューしたりして，要因を特定する研究が進められている．また，1人の患者が複数の医療機関で治療を受けた場合でも対応が可能である．

院内がん登録は，当該施設で診断された全がん患者に基づく生存率を算出できるので，施設ごとの比較が可能であり，また，病期をそろえての比較もできる．しかし，治療内容などの情報の詳細さは十分とはいえず，合併症の情報もないので，生存率の差の原因に言及するには，やはり追加的な情報の収集が必要となる．臓器がん登録は，病院内でも特定の診療グループの症例に限られることもあるので(たとえば手術症例のみ)，当該施設の全症例をカバーしているとは限らない．また，参加している施設が専門医の所属する施設に限られるため，それらの症例中での病期分布や組織型分布に偏りがあることは，常に念頭に入れておく必要がある．しかし，詳細な診療情報を収集しているので，詳細な要因別にみた生存率については，他のがん登録では得られない情報を提供することが可能であり，ステージ分類の改訂の際の根拠を提示できる．

III．わが国におけるがん登録の今後の方向性

わが国のがん登録システムの中で，諸外国に比べて決定的に遅れているのが地域がん登録の登録精度である．現状では，登録漏れの結果，罹患率として20%程度の過小評価となっていると推定される．登録精度を高めるためには，その基盤である院内がん登録を整備する必要があり，加えて，地域がん登録に関する標準的登録手順の整理，標準登録システムの開発，実務担当者の養成とその教育研究プログラムの開発が緊急の課題である．

2009年4月の指定で拠点病院の指定件数は375施設に拡大した．まずは，これらの施設において標準化された院内がん登録を整備することが必須である．このために，院内がん登録実務担当者の研修を行い，一定レベル以上の質のマンパワーを確保することが重要である．がん対策情報センターでは，初級，中級，指導者向け研修を提供してこれに対応している．また，地域がん登録，院内がん登録とも，厚労省研究班を中心に登録手順の標準化を進めつつある．地域がん登録については，

研究班で検討した標準方式による地域がん登録業務を実現するために設計された「標準データベースシステム」を放射線影響研究所情報技術部にて開発し，山形をモデル地区とした試験導入，評価，運用手順作成した，2008年までに，10県にて導入が完了し，さら導入を拡大しつつある．院内がん登録についても，標準的な院内がん登録ソフト「HosCanR」を研究班にて開発し，無償提供している．また，院内がん登録，地域がん登録の主要関係者が集まって，登録手順，定義についての合同委員会(Joint committee of Cancer Retistry)を定期的に開催して整合性を図っている．

地域がん登録の精度向上を，拠点病院の院内がん登録の普及によって実現するには，拠点病院の指定を拡大するか，拠点病院へがん患者さんを集約するか，拠点病院以外の病院への院内がん登録を普及するか，が必要であり，さらなる取り組みが必要である．

地域がん登録，院内がん登録，臓器がん登録の3種類のがん登録は，それぞれ目的，実施主体，登録対象，登録項目，収集時期などが異なるため単純に統合することはできないが，共通する部分も多く，相互に連携を深めて，効率の良い登録体制を構築する必要がある．臓器がん登録に対する医療機関側の情報源は各診療科が管理する診療科データベースであることが多いが(図1)，患者の基本情報について，院内がん登録とともに病院情報システムから抽出することで省力化が可能である．こうした診療科データベースは，個人情報保護の観点からのシステム管理が徹底されていない場合が多く，院内がん登録や病院情報システムと同レベルのシステム管理の必要性が高まってきている．

一方，多くの地域がん登録は，人口動態統計死亡データおよび住民票照会や本籍地照会による予後調査を実施しているが，これらの情報について院内がん登録を通じて臓器がん登録へ還元することで，医療機関における予後調査の負担を大幅に軽減できる．既存統計資料の有効活用をすることで，予後調査の際のデータ収集を効率的に進めることができる環境を整えることが重要である．さらに，がん医療の質の均てん化の程度を検証する

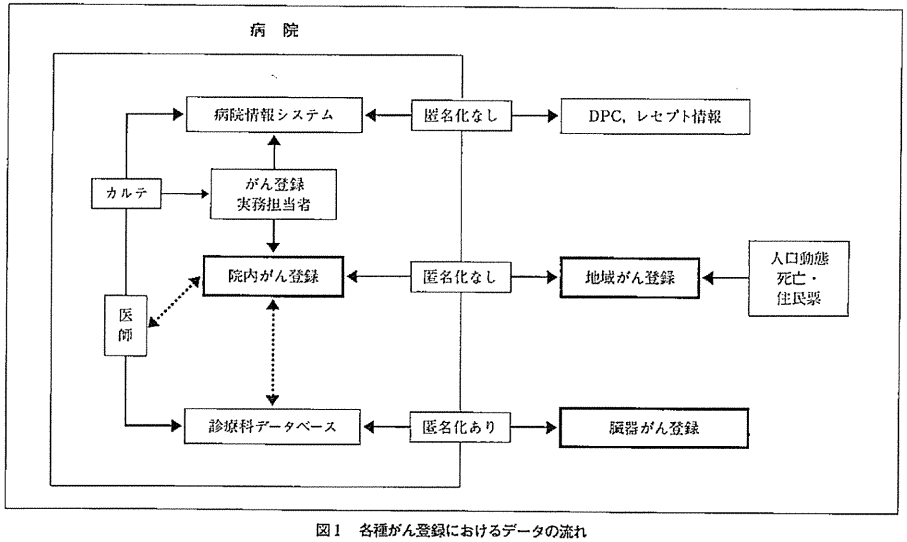

図1　各種がん登録におけるデータの流れ

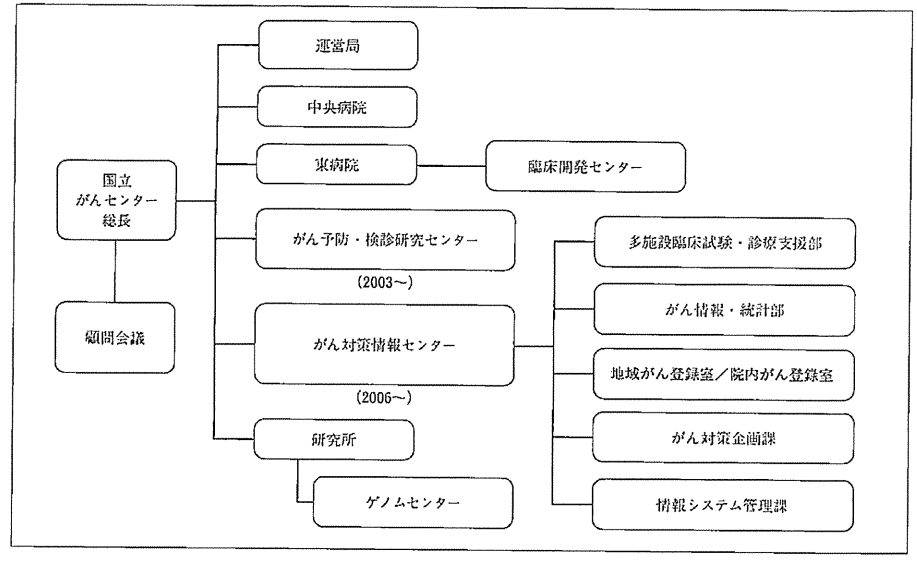

図2　国立がんセンターの組織図

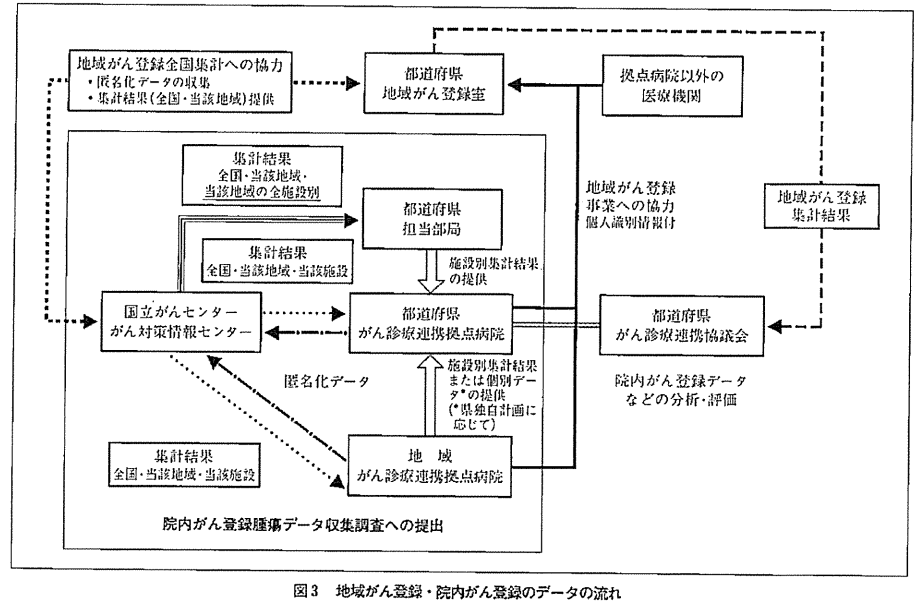

図3　地域がん登録・院内がん登録のデータの流れ

ためには，適切な対象に対して標準的な診断治療が実施されているかどうかのデータが必要であり，現在の地域・院内がん登録に含まれる項目だけでは，検証は難しく，サンプリング調査やデータベース間の照合などの追加的な調査が必要となる．

2006年10月に，国立がんセンターにがん対策情報センターが設置され，がん統計・情報部に地域がん登録室と院内がん登録室が設置された(図2)．当面，種々の研究班と連携しながら，地域がん登録については都道府県地域がん登録の罹患率全国集計(2004年より開始)を，院内がん登録については拠点病院からの院内がん登録腫瘍データ収集調査(2009年より開始)を行い，それぞれ，標準化と体制整備を支援すると共に，実務担当者の教育研修を行う，また，他のがん関連の統計を一元的に収集整理して，正確で役に立つがん統計情報の提供を行うことが想定されている(図3)．

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Original Article

Impact of Smoking and Other Lifestyle Factors on Life Expectancy among Japanese: Findings from the Japan Collaborative Cohort (JACC) Study

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ABSTRACT

Background: A number of lifestyle factors, including smoking and drinking, are known to be independently associated with all-cause mortality. However, it might be more effective in motivating the public to adopt a healthier lifestyle if the combined effect of several lifestyle factors on all-cause mortality could be demonstrated in a straightforward manner.

Methods: We examined the combined effects of 6 healthy lifestyle behaviors on all-cause mortality by estimating life expectancies at 40 and 60 years of age among 62 106 participants in a prospective cohort study with a 14.5-year follow-up. The healthy behaviors selected were current nonsmoking, not heavily drinking, walking 1 hour or more per day, sleeping 6.5 to 7.4 hours per day, eating green leafy vegetables almost daily, and having a BMI between 18.5 to 24.9.

Results: At age 40, we found a 10.3-year increase in life expectancy for men and a 8.3-year increase for women who had all 6 healthy behaviors, as compared with those who had only 0 to 2 healthy behaviors. Increases of 9.6 and 8.2 years were observed for men and women, respectively, at age 60 with all 6 healthy behaviors. When comparing currently nonsmoking individuals with 0 to 1 healthy behaviors, the life expectancy of smokers was shorter in both men and women, even if they maintained all 5 other healthy behaviors.

Conclusions: Among individuals aged 40 and 60 years, maintaining all 6 healthy lifestyle factors was associated with longer life expectancy. Smokers should be encouraged to quit smoking first and then to maintain or adopt the other 5 lifestyle factors.

Key words: lifestyle; smoking; cohort study; life expectancy

INTRODUCTION

Unhealthy lifestyle behaviors such as cigarette smoking,[1,2] excessive alcohol drinking,[3,4] physical inactivity,[5] too much or too little sleep,[6,7] low consumption of green leafy vegetables,[8,9] and overweight[10,11] are associated with increased risk of all-cause mortality. However, because these lifestyle factors are mutually related,[12,13] it is important to investigate their combined effects in a straightforward way. We recently reported the combined effects of modifiable lifestyle factors on all-cause mortality.[14] As compared with people with 0 to 2 healthy lifestyle behaviors (current nonsmoking, not heavily drinking, walking 1 hour or more per day, sleeping 6.5 to 7.4 hours per day, eating green leafy vegetables almost daily, and a body mass index (BMI) between 18.5 to 24.9), men and women with 6 healthy behaviors had 58% and 51% respective reductions in the risk of all-cause mortality.

Among these behaviors, smoking may be the best known risk factor. Studies have suggested that smoking has a larger impact on all-cause mortality than other factors. The EPIC-Norfolk Prospective Population Study found that the relative risks of all-cause mortality were 1.77 for smoking, 1.24 for physical activity, 1.26 for alcohol intake, and 1.44 for vitamin C level among men and women aged 45 to 79 years.[15] Similarly, ever-smoking women had a 1.66 relative risk in the Nurse's Health Study, whereas the risks associated with other factors (high BMI, physical activity, diet, and alcohol intake) were less than 1.35.[16] Thus, it is useful to investigate the discrete effects of smoking status and other lifestyle factors on all-cause mortality.

In order to motivate the public to make lifestyle modifications, it is necessary to show the combined impact of lifestyle factors on health in a manner that is easy to understand. Life expectancy indicates the theoretical number of years of life remaining at a given age, and is a well-known, comprehensive value for representing the health status of a population. The concept of life expectancy is understandable to the public and can be readily used by health providers to promote evidence-based health plans. Thus, our objective was to estimate life expectancies according to the number of healthy lifestyle behaviors at 40 and 60 years of age among participants in the Japan Collaborative Cohort (JACC) Study. We also separately estimated life expectancies for current smokers and current nonsmokers.

METHODS

Study subjects and data collection

The study design and methods of the JACC Study have been described previously.[17,18] Briefly, the cohort was assembled from 1988 to 1990 by using a self-administered questionnaire; it ultimately enrolled 110 792 subjects (46 465 men and 64 327 women) aged 40 to 79 years. The cause and date of death among the study subjects were identified by reviewing all death certificates in each area with the permission of the Director-General of the Prime Minister's Office (Ministry of Internal Affairs and Communications). Those who moved out of a study area were regarded as censored. We followed the subjects until the end of 2006, except in the 7 areas where follow-up was discontinued at the end of 1999 or 2003. The Ethical Board of Nagoya University School of Medicine, where the central office of the JACC Study is located, approved the entire study design.

Lifestyle variables

We selected 6 lifestyle behaviors: smoking status, alcohol consumption, walking duration, sleep duration, consumption of green leafy vegetables, and BMI. The selection of these lifestyle factors was based on the results of our previous cohort study and on an extensive review of other epidemiologic studies that had investigated the relationship between lifestyle factors and mortality; details of the selection and dichotomization of these lifestyle factors are described elsewhere.[14] Briefly, the potential health status of each factor was: current nonsmoking (including former smokers),[1,19] drinking no more than 1 *gou* (23 g of alcohol) per occasion or current nondrinking (including former drinkers),[4] walking 1 hour or more per day,[20,21] sleeping 6.5 to 7.4 hours per day,[6,7] eating green leafy vegetables almost daily,[8,22] and desirable weight for height (BMI between 18.5 to 24.9).[11,23] The number of healthy lifestyle behaviors for each subject was summed, with a higher number indicating a more healthful lifestyle.

The 62 106 subjects (27 582 men and 34 524 women) with complete information on all 6 lifestyle behaviors were considered eligible for the analyses.

Analysis

Age- and sex-specific mortality rates within 5-year age categories were calculated based on the person-year method according to the number of healthy lifestyle behaviors. Calculation of mortality was started at age 40 years and the last category was age 85 years or older. Because there was a small number of subjects included in the category at age 90 or older, mortality at age 85 years or older was calculated with the assumption that the ratio of mortality at age 85 to 89 years to mortality at age 85 years or older was the same in this cohort as in the general Japanese population. Thus, mortality at age 85 years or older in the study was calculated as $MR_{85+} = MR_{85-89} \times JMR_{85+}/JMR_{85-89}$, where MR_x indicates mortality at age x in the JACC Study, and JMR_x indicates mortality at age x throughout Japan in 2000. Chiang's abridged life table method[24] was used to calculate life expectancies with respect to the number of healthy behaviors. The fraction of all age categories of life was used to construct an abridged life table.[24,25] Those fractions were calculated from a complete life table for the year 2000 in Japan. Confidence intervals for life expectancies were also calculated using the formulae proposed by Chiang. Additionally, we estimated life expectancies according to the number of healthy behaviors stratified by smoking status (current smoking or current nonsmoking at baseline). The age-adjusted mortality rates according to the number of healthy lifestyle behaviors were calculated based on the 5-year age-specific mortality by sex, with the whole cohort considered as the standard population. We used SAS Ver. 9.1.3 (SAS Institute Inc., Cary, NC, USA) for analyses at the Aichi Medical University Computation Center.

RESULTS

During an average follow-up period of 14.5 years, a total of 10 843 deaths occurred by 2006 (6633 men and 4210 women). Death from any cancer accounted for 38.5% of deaths in men and 34.0% of deaths in women, and death from cardiovascular disease accounted for 27.6% and 32.8% of deaths in men and women, respectively.

At baseline, there were 16 363 current smokers: 14 663 men (53.2% of men) and 1700 women (4.9% of women). Current nonsmokers were older than current smokers among both men and women. Also, the former were more educated, had less mental stress, were more likely to have a spouse, and were more likely to eat breakfast every day. Among women, the

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Table 1. Age-adjusted mortality rates according to number of healthy lifestyle behaviors

	Men				Women			
	Number	Person-years	Deaths	Age-adjusted mortality (per 1000)	Number	Person-years	Deaths	Age-adjusted mortality (per 1000)
No. of healthy lifestyle behaviors								
0–2	5092	141967	2478	20.7	2277	39055	500	11.6
3	9147	125658	2133	16.9	9014	131480	1292	9.4
4	8969	85414	1464	14.9	13147	189491	1466	7.9
5	3694	31931	494	12.6	8287	120141	795	7.0
6	680	5587	64	8.9	1799	26550	157	6.2
Overall	27582	390504	6633	17.0	34524	506720	4210	8.3
No. of healthy lifestyle behaviors other than smoking								
Current smoker								
0–1	2908	40953	706	22.9	202	2668	44	19.1
2	4980	70007	1309	22.2	527	7171	103	14.5
3	4711	67370	1179	18.6	600	8708	91	11.7
4	1763	25546	443	18.1	322	4669	39	9.1
5	301	4315	80	18.7	49	—	9	—
Overall	14663	208184	3717	20.4	1700	23940	286	13.0
Current nonsmoker								
0–1	2184	31012	463	15.4	2075	29213	353	10.5
2	4167	58289	954	15.0	8487	122771	1201	9.3
3	4258	59873	1021	13.9	12547	184822	1427	7.8
4	1931	27617	414	11.6	7965	119413	786	7.0
5	379	5587	64	8.9	1750	26550	157	6.2
Overall	12919	182358	2916	13.9	32824	482760	3924	8.1

proportion of those with a history of stroke, myocardial infarction, or cancer was lower among current nonsmokers; however, the proportion was higher among men. With regard to the number of healthy lifestyle behaviors other than nonsmoking, both men and women with a higher number of healthy behaviors were more likely to eat breakfast every day than men and women with a lower number of such behaviors. The former were also more educated (except for smoking men), older and had less mental stress (in both smoking and nonsmoking men), and were more likely to have a spouse (in currently nonsmoking women).

Age-adjusted mortality rates by number of healthy lifestyle behaviors are shown in Table 1 (the mortality rate of smoking women with 5 healthy lifestyle behaviors could not be calculated because the sample size was too small). The rate varied from 8.9 to 20.7 per 1000 men and from 6.2 to 11.6 per 1000 women. Age-adjusted mortality rates in currently smoking men and women were 20.4 and 13.0 per 1000, respectively; among current nonsmokers, the rates were 13.9 and 8.1.

Table 2 shows life expectancies at 40 and 60 years of age. Life expectancies among 40-year-old men and women were 39.9 and 46.5 years, respectively, in those with 0 to 2 healthy behaviors, and 50.2 and 54.8 years in those with all 6 healthy lifestyle behaviors. Life expectancy rose as the number of healthy behaviors increased. The same life expectancy trend was observed in 60-year-old men and women. The differences in life expectancy between those with 6 healthy behaviors and those with only 0 to 2 such behaviors were 10.3 and 8.3 years in men and women, respectively, at 40 years of age, and 9.6 and 8.2 years at 60 years of age.

The trend toward longer life expectancy with a larger number of healthy behaviors was observed even when subjects were stratified according to smoking status. Among smoking women, the life expectancy of those with 5 healthy behaviors could not be calculated because the sample size was too small and no deaths were observed in their category at age 85 to 89 years. As compared with their currently nonsmoking counterparts, both male and female current smokers had shorter life expectancies at baseline. On average, the differences were 4.0 and 4.8 years at 40 years of age in men and women, respectively, and 3.5 and 4.2 years at 60 years of age. Among male smokers at ages 40 and 60, even when all 5 other healthy behaviors were present, their life expectancies were still shorter than those of nonsmokers with 0 to 1 other healthy behaviors. Similarly, smoking women with 4 of the other healthy behaviors had a shorter life expectancy at age 60, as compared with nonsmoking women with 0 to 1 healthy behaviors other than nonsmoking, although the life expectancy of the former at age 40 was only slightly longer (0.1 years) than that of the latter. Moreover, the differences between subjects with 5 healthy behaviors and those with 0 to 1 healthy behaviors were smaller among smokers than among nonsmokers, except for women at 40 years of age. The differences in life expectancy

Table 2. Estimated life expectancies according to number of healthy lifestyle behaviors

	Men				Women			
	Life expectancy at 40 years of age (95% CI)		Life expectancy at 60 years of age (95% CI)		Life expectancy at 40 years of age (95% CI)		Life expectancy at 60 years of age (95% CI)	
No. of healthy lifestyle behaviors								
0–2	39.9	(39.5–40.3)	21.9	(21.6–22.2)	46.5	(45.5–47.4)	28.1	(27.5–28.7)
3	42.3	(41.9–42.8)	23.9	(23.6–24.2)	48.0	(47.6–48.4)	29.1	(28.8–29.4)
4	43.4	(42.8–44.1)	25.1	(24.7–25.5)	50.1	(49.8–50.4)	30.9	(30.6–31.2)
5	44.7	(43.8–45.7)	25.9	(25.4–26.5)	50.9	(50.5–51.2)	31.5	(31.2–31.8)
6	50.2	(48.1–52.3)	31.5	(29.8–33.2)	54.8	(53.3–56.2)	36.3	(35.4–37.1)
Overall	42.1	(41.8–42.3)	23.8	(23.6–24.0)	49.5	(49.2–49.7)	30.4	(30.2–30.6)
No. of healthy lifestyle behaviors other than smoking								
Current smoker								
0–1	39.0	(38.3–39.7)	21.0	(20.4–21.5)	39.7	(36.8–42.7)	22.9	(20.8–25.0)
2	39.0	(38.5–39.6)	21.3	(20.9–21.7)	44.0	(42.0–45.9)	25.4	(24.1–26.7)
3	41.2	(40.6–41.9)	23.0	(22.6–23.4)	46.6	(45.0–48.1)	27.7	(26.3–29.1)
4	42.0	(41.0–42.9)	23.2	(22.5–23.9)	48.1	(45.9–50.3)	29.3	(27.5–31.0)
5	41.4	(39.3–43.4)	23.2	(21.4–24.9)	—		—	
Overall	40.1	(39.8–40.5)	22.1	(21.8–22.3)	44.9	(43.9–45.9)	26.4	(25.7–27.2)
Current nonsmoker								
0–1	43.4	(42.7–44.2)	24.5	(23.9–25.1)	47.9	(46.8–49.0)	29.3	(28.6–30.0)
2	43.6	(43.0–44.2)	24.9	(24.5–25.3)	48.1	(47.6–48.5)	29.2	(28.9–29.5)
3	43.9	(43.1–44.7)	25.8	(25.3–26.2)	50.1	(49.8–50.5)	30.9	(30.7–31.2)
4	45.3	(44.2–46.4)	26.4	(25.8–27.0)	50.9	(50.5–51.2)	31.5	(31.2–31.8)
5	50.2	(48.1–52.3)	31.5	(29.8–33.2)	54.8	(53.3–56.2)	36.3	(35.4–37.1)
Overall	44.2	(43.8–44.5)	25.6	(25.3–25.8)	49.7	(49.5–49.9)	30.6	(30.4–30.8)
All Japan (2000)[35]	39.1		21.4		45.5		26.9	

according to the number of healthy lifestyle behaviors in smoking men were 2.4 and 2.2 years at 40 and 60 years of age, respectively; the respective differences were 6.8 and 7.0 years in nonsmoking men.

DISCUSSION

The current study presents life expectancies according to the number of healthy lifestyle behaviors, using data from a large-scale population-based cohort followed for 14.5 years. The results showed that life expectancy rises as the number of healthy behaviors increases. The life expectancy of subjects maintaining 6 healthy lifestyle behaviors was 10.3 and 8.3 years longer than those with 0 to 2 healthy lifestyles in men and women, respectively, at age 40 years, and 9.6 and 8.2 years at age 60. Smoking status had the greatest impact on life expectancy. The life expectancies of male and female smokers who maintained all other healthy lifestyle factors were nevertheless shorter than those of their currently nonsmoking counterparts with only 0 to 1 healthy lifestyle behaviors. Furthermore, the increases in life expectancy associated with having more healthy behaviors were smaller among smokers than among nonsmokers, particularly in men.

Studies have investigated the combined effects of lifestyle factors on all-cause mortality by calculating life expectancies. Stamler et al reported that life expectancy for low-risk subcohorts (ie, those with favorable levels of cholesterol and blood pressure, nonsmokers, nondiabetics, and those with no MI or ECG abnormalities) was 5.8 to 9.5 years higher than that of others in 5 large US cohorts.[26] Based on an 11-year follow-up of UK cohorts aged 45 to 79 years, Khaw et al estimated that the mortality risk for individuals with 4 healthy behaviors (current nonsmoker, not physically inactive, moderate alcohol intake, and plasma vitamin C >50 mmol/l), as compared with individuals with none of these behaviors, was equivalent to being 14 years younger in chronological age.[15] Although the investigated lifestyle factors varied in these reports, they all observed differences in life expectancy between subjects with healthy lifestyle behaviors and those without them.

In Japan, the difference in life expectancy due to smoking status was previously calculated based on NIPPON DATA80 by Murakami et al.[27] Life expectancy in men and women aged 40 years was 42.1 and 45.6 years, respectively, in never smokers, 40.4 and 45.9 years in ex-smokers, and 38.6 and 43.4 years in current smokers, which represent slightly smaller differences in life expectancies between smokers and nonsmokers than those noted in our study. However, studies from other developed countries found larger differences in the life expectancies of smokers and nonsmokers. The life expectancies of 50-year-old never smokers and current

smokers were 32.6 and 26.3 years in men in the Whitehall Study, England.[28] In the Framingham Study, the differences in life expectancy between never smokers and always smokers were 8.7 years in men and 7.6 years in women at age 50.[29] While all these studies found longer life expectancies among nonsmokers than current smokers, the differences varied. The different circumstances of smokers may explain this variation. In Japan, at the time of our baseline survey, smoking was very common in Japanese men, and thus, some never-smoking men might have had health problems that compelled them to avoid smoking. Such unhealthy conditions among nonsmoking men might reduce the difference in life expectancies between smokers and nonsmokers. In contrast, smoking by women was not common at that time, especially among housewives. Thus, some smoking women might have been healthier than others.[27] This could have led to prolonged life span among smoking women, and might have diminished the difference in life expectancies between smokers and nonsmokers. These may partially explain why the differences in life expectancies in Japan were smaller than those reported in other developed countries. However, the prevalence of current smokers in our study was 53.2% in men, which was lower than the prevalence noted in NIPPON DATA80 (62.9%), which indicates a possible reduction in the number of unhealthy subjects among nonsmokers. This could account for the relatively larger differences in life expectancies among men in our study, as compared with NIPPON DATA80. The reason for the larger differences in life expectancies among women was not clear, as the prevalence of current smokers was lower in our study than in NIPPON DATA80 (4.9% and 8.8%, respectively). However, the distribution of other behaviors may be different.

This study revealed that the life expectancies of male and female smokers were shorter than those of nonsmokers with 0 to 1 healthy lifestyle behaviors, even when the former maintained all other healthy behaviors. In addition, the increase in life expectancy attributable to having a higher number of healthy lifestyle behaviors was smaller among current smokers than among nonsmokers, particularly in men. Two previous studies estimated the effect of smoking on life expectancies separately from other lifestyle factors. As compared with male never smokers reporting low physical activity, the expected age at death at age 65 for highly active male ever smokers was slightly older, but that for moderately active male ever smokers was younger[30]; the result was the same for women. Regarding obesity, Peeters et al found that the life expectancy of 40-year-old male smokers of normal weight was 1 year shorter than that of obese nonsmokers; although a similar such reduction in life expectancy was not observed among women.[31] These studies suggest that smoking has a somewhat larger impact on life expectancy than physical activity or obesity. Our results offer more evidence regarding the impact of smoking on health.

The lifestyle factors chosen in this study were current nonsmoking, not heavily drinking, walking 1 hour or more per day, sleeping 6.5 to 7.4 hours per day, eating green leafy vegetables almost daily, and BMI between 18.5 to 24.9. We previously found a clear reduction in mortality risk with an increasing number of these 6 healthy lifestyle behaviors.[14] These factors were selected based on the results from our previous cohort study[4,14,22,32-34] and a review of epidemiologic studies[1,3,6,8,11,20] that included data on associations between lifestyle factors and mortality. The lifestyle factors selected in this study are key elements of a healthy lifestyle included in *Health Japan 21*, a recent health promotion initiative of the government of Japan. Selected factors were dichotomized into healthy or unhealthy, and the healthy behaviors of each subject were summed. Therefore, the factors we selected for the study were sufficiently reliable, easily understood, and easy to calculate.

Our results should serve as a useful tool to help both the general public and health planners to understand the importance of maintaining a healthy lifestyle. Life expectancy is a readily comprehensible value that allows us to use differences in survival age to represent the combined risk impacts of different behaviors. With these easily understandable data, health planners are better able to encourage lifestyle changes in people. For a person who is considering a lifestyle change for health reasons, knowing how many years of life one might gain could be a powerful incentive.

The strengths of our study were 1) that it included data from a large cohort of subjects from all over Japan (including more than 10 000 deceased), and 2) its long follow-up period of approximately 14.5 years. These advantages allowed us to estimate life expectancies according to a number of healthy lifestyle behaviors. Moreover, we were able to assess the impact of smoking status on life expectancy separately from other lifestyle factors. As a result, it is clear that smoking has a larger impact than other lifestyle factors on life expectancy.

Our results show longer life expectancies that those noted in the Japanese complete life table at 2000[35]: 2.9 and 3.9 years longer in men and women, respectively, at the age of 40, and 2.4 and 3.5 years at the age of 60. People with health problems at baseline were not included in this study, and subjects who moved away from the study area for any reason were excluded from our analysis at that time. Therefore, age-specific mortality rates moved lower, which may explain why estimated life expectancies in this study were longer than those of the general population of Japan. Because such an overestimation might occur randomly, the rank of life expectancies among the lifestyle groups in this study should prove representative of the general population of Japan.

There were some limitations in this study that warrant discussion. First, as lifestyle status was self-reported, some measurement errors may have occurred. However, the JACC Study was a prospective cohort study, and misclassification of health status was more likely to be random. Second, data were only collected at baseline, and subsequent behavior changes could not be taken into account in our analyses. Kawado et al[36] compared baseline data with interim data collected approximately 5 years later in some of our participants, and found that the proportions of smokers and drinkers had decreased. If such behavioral changes occurred in other lifestyle factors, each change that resulted in a healthier lifestyle might diminish the differences between healthy and unhealthy groups at baseline, and the differences between estimated life expectancies might be diminished due to these changes. Third, we did not consider the effects of factors other than the 6 healthy lifestyle factors investigated in this report. Of course, not all differences in life expectancy were caused by these 6 lifestyle factors. However, because lifestyle factors are mutually related, it is possible that the number of healthy lifestyle behaviors is not merely a given number, and that estimated life expectancies in this study somewhat reflect the effects of other lifestyle factors.

In conclusion, we estimated life expectancy with respect to 6 healthy lifestyle behaviors, and found that men and women with all these behaviors had 10.3 and 8.3 years, respectively, of additional life expectancy at age 40 years, as compared with those with 0 to 2 such behaviors, and 9.6 and 8.2 years of additional life expectancy at age 60. Even though adoption of any of these behaviors would likely improve life expectancy, smoking status had the largest impact. Smoking cessation is thus the first and best way to extend life expectancy.

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