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The Cultural Revolution, Stress and Cancer

by

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Abstract

The link between mental stress and cancer is still a belief, not a well established scientific fact. Scientists have relied largely on opinions of cancer stricken patients to establish a link between stress and cancer. Such opinion surveys tend to produce contradictory statistical inferences. Although it is difficult to conduct scientific experiments on humans similar to those on animals, human history is replete with "experiments" that have caused enormous stress on some human populations. The objective of this exercise is to draw evidence from one such massive experiment, the Cultural Revolution in China. Cancer data from Shanghai analyzed through an age-period-cohort technique show very strong evidence in support of the hypothesis that mental stress causes cancer.

1. Introduction

There is widespread belief that stress causes cancer but scientific research has not established a definitive link between the two. After extensive surveys of the studies that assessed the link between psychological factors and cancer, Dalton et al. (2002) and Garssen (2004) reached the conclusion that the evidence in support of the hypothesis that psychological factors contribute to the development of cancer is extremely weak. The Cancer Council, NSW, Australia echoes the same conclusion: "For centuries people have wondered whether there is a link between stress and cancer, but we still don't have any solid evidence to say whether this is true or not. From the studies that have been done so far, stress does not seem to be an important risk for cancer." (Website, Aug 18, 2010)

Recent experiments on fruit flies (Wu et al., 2010) seem to provide new evidence on a direct link between stress and cancer. Nevertheless, how these results extend to humans remains to be seen. Scientific experiments on humans similar to those conducted on animals are not possible. As a result the assessment of the link between stress and cancer is mostly confined to opinions of cancer patients as to whether they were under prolonged stress before the onset of their cancers. Such opinion surveys tend to produce fragile inferences and tenuous conclusions.

Although it is difficult to conduct scientific experiments on humans similar to those on animals, human history is replete with "experiments" that have caused enormous stress on some human populations. However, keeping data records for scientific analysis was not part of these politically motivated oppressions and traumas imposed on human subgroups. Had there been data available from such historical events they would provide a rich source of information to examine the link between stress and cancer.

The objective our exercise is to focus on one such "human experiment", the Cultural Revolution in China and examine how it relates to cancer incidence. It is well documented (Thurston, 1984, 1985) that the Cultural Revolution that began in October 1966 with the objective of further propagating the socialist ideology and transforming the bourgeoisie lasted more than a decade and took violent forms, caused enormous social, political and economic upheaval subjecting large segments of the population to unbearable mental and physical stress. This was indeed a human experiment on stress of a massive scale.

We were motivated to this exercise by a newspaper article by a Singapore cancer specialist Dr. Ang Peng Tiam who wrote "I hardly ever make a statement without scientific proof. However, I believe there is at least one notable exception: Stress causes cancer, even though I cannot prove it" and drew attention to an increase in cancer incidence during the Cultural Revolution (Ang, 2008). In an intriguing account of traumatic experiences of some Chinese who have lived through the Cultural Revolution Thurston (1985) also draws attention to the link between stress and cancer. (It is worth reading Thurston, 1985, as a prelude to our exercise.) After searching through the literature we came across only one study that examined the impact of another major "human experiment", the Nazi Holocaust, on cancer (Keinan-Boker et al., 2009). The conclusion of this exercise was that the Israeli Jews who were potentially exposed to the Holocaust experienced higher incidence of all cancers later in their lives.

2. Data and Methodology

A good data set from some major cities of China that cover the Cultural Revolution period and beyond would be ideal for our analysis. The data set has to include not only the records of cancer incidence but also a set of control variables that helps us isolate the impact of the Cultural Revolution on cancer. A

major criticism that Garssen (2004) leveled against the studies he reviewed was not having enough control for age, demographic variables and other confounding factors.

After a considerable search we were able to compile a reasonably complete data set only for Shanghai. This data set includes cancer incidence and population at risk by age from 1973 to 2002. Data from 1978 to 2002 were taken from Cancer Incidence in Five Continents, Volumes II – IX (Doll et al., 1970; Waterhouse et al. 1976, 1982; Muir et al., 1987; Parkin et al., 1992, 1997, 2002; Curado et al., 2007). Data from 1973 to 1977 were taken from Gao and Lu (2007). Cancer incidence data are available in 5-year age groups running from age 0-4 to 85-89. We grouped annual population data by age and other annual data series to five year intervals to match the cancer age grouping. Total cancer incidence cases refer to cancers on all body sites excluding non-melanoma skin cases. Cancer cases are classified using International Classification of Diseases (ICD-10). Data on Shanghai annual aggregate income and final energy consumption by industry (see Section 3) are from China Compendium of Statistics 1949-2004 compiled by Department of Comprehensive Statistics (National Bureau of Statistics, 2005). For a comparison we also examine cancer incidence in Osaka Prefecture, Japan. Cancer and population data from 1963 to 2002 for Osaka are from Cancer Incidence in Five Continents (cited above). As a proxy for Osaka annual income growth we use the national gross domestic product growth, data taken from International Financial Statistics (International Monetary Fund, online data).

Our focus is to isolate the impact of the Cultural Revolution on the increase in cancer incidence in different birth cohorts as they pass through the Cultural Revolution during their working ages. For this we use a regression model. The methodology is an adaptation of the age-period-cohort (APC) analysis which is often used in demographic, epidemiological, and sociological studies in analyzing data classified by age and year. The most commonly used is a Poisson regression model of the form

$$\ln(E(C_{it})) = \ln(N_{it}) + \mu + \alpha_i + \beta_t + \gamma_k \tag{1}$$

where $E(C_{it})$ is the expected incidence of cancer cases at age i for $i = 1, 2, ..., I$ and time period t for

$t = 1, 2, ..., T$, N_{it} is the size of the population at risk, μ is the (adjusted) mean cancer rate, α_i is the

age effect, β_t is the time or period effect, and γ_k is the cohort effect for $k = 1, 2, ..., (I + T - 1)$ with

$k = I - i + t$.

For the purpose of our analysis model (1) needs to be re-formulated to overcome some difficulties

that it poses. First, model (1) suffers from the well known identification problem arising from the perfect

linear relationship age=period (recording year)-cohort (birth year). A number of researchers have

addressed the problem and so far a satisfactory general solution to the problem has not emerged. (See

Sociological Methods & Research, Vol. 36(3), 2008, for a review of various methods.) Yang et al. (2004,

2008) offers a purely statistical method which they termed the "intrinsic estimator" to solve the

identification problem. Despite the apparent promise of the method, we observe that it is far from

satisfactory as a general method. Winship and Harding (2008) offers what they call a mechanism

approach, which appears promising but requires a substantial amount data, a luxury for the problem at

hand. McKenzie (2006) shows that second differences over age, period, and cohort of (1) can be used to

isolate each of these effects without further assumptions; however, obtaining the original parameters

requires assumptions similar to those used in Generalized Linear Model methods applied to (1).

As Heckman and Robb (1985) have argued: "The age-period-cohort effect identification problem

arises because analysts want something for nothing: a general statistical decomposition of data without

specific subject matter motivation underlying the decomposition. In a sense it is a blessing for social

science that a purely statistical approach to the problem is bound to fail." (pp. 144-45). Obviously the

identification problem does not arise if one of the three categories in (1) is dropped from the model.

Clayton and Schiffers (1987a, 1987b) advocate using AP or AC models and resort to APC models only if the former does not provide a good fit. Instead of dropping a category, if data are available, the identification problem can easily be overcome by capturing the period effect through some actual determinants of the period effect. This is the approach we adopt in our exercise.

Second, the cancer rate is not that ideal for an analysis that focuses on cancer incidence in a major city. City populations are subject to large inflows of migrants. A large influx of young and healthy migrants in a given year may result in a reduction in the cancer rate despite an increase in the incidence of cancer cases. Moreover, if Model (1) is taken as a predictive equation, it underlies the assumption that a 1% increase in population size leads to a 1% increase in expected cancer incidence. Therefore, more meaningful results can be generated by letting the regression determine the coefficient of the population instead of constraining it to unity.

Third, in addition to the additive effects in Model (1), we may have to account for interaction effects. For example, a substantial increase in air pollution in a given year may affect a young and an old person differently. This creates an interaction effect between age and period in addition to their individual effects.

To account for the above considerations and also to have a sharper focus on our hypothesis we use the growth rate of cancer incidence as our unit of analysis and specify the model as

$$c_{it} = \phi n_{it} + \beta' x_t + \lambda' x_t^* + \delta_k + \varepsilon_{it} \tag{2}$$

where $c_{it} = 100 \ln(C_{it} / C_{it-1})$ and $n_{it} = 100 \ln(N_{it} / N_{it-1})$ are the growth rates of cancer incidence and population size respectively, x_t is a vector of variables to capture the period effect, x_t^* represent age-period interaction variables, $\delta_k = \gamma_k - \gamma_{k-1}$, and $E(\varepsilon_{it}) = 0$. We do not assume a specific distribution

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for the random term ε_{it} and suggest using robust standard errors if the least squares regression residuals indicate that ε_{it} is heteroscedastic and/or serially correlated (White, 1980, Newey and West, 1987).

Note that although taking time difference over given ages removes the constant and fixed age effects from (1), the age-period interaction effect remains in (2). In fact, if age, period, and cohort are represented by their underlying causal variables differencing over age, period, or cohort will not eliminate any of these effects.

Through preliminary estimates we observe that the age profile can be well represented by an age polynomial, which also helps in conserving the degrees of freedom. For illustrative purposes suppose only x_{1t} interacts with age, we can write Model (2) for estimation as

$$c_{it} = \overline{\delta} + \phi n_{it} + \beta' x_t + \lambda_1 age_i x_{1t} + \lambda_2 age_i^2 x_{1t} + \sum_{k=1}^{I+T-2} \delta_k^* d_k + \varepsilon_{it} \tag{3}$$

where d_k are centered cohort dummies and $\delta_k^* = \delta_k - \overline{\delta}$, $\overline{\delta}$ is the average of δ coefficients such that $\sum_k \delta_k^* = 0$. (The standard dummies that take value 1 for cohort k and 0 otherwise can be centered by subtracting the last dummy from each of the other dummies so that the last dummy drops out. Centered dummies remove the dependence of coefficient estimates on a reference group and introduce a constant term to the model so that OLS residuals sum to zero and R^2 is well defined.)

3. Results

Figure 1 plots the Shanghai overall cancer rate (per 100,000) by year. Apart from the upward trend associated with the period effect, the plot shows a spike in 1978 and then again in 1989. From these data it is hard to make any statistical inference on the impact of the Cultural Revolution.
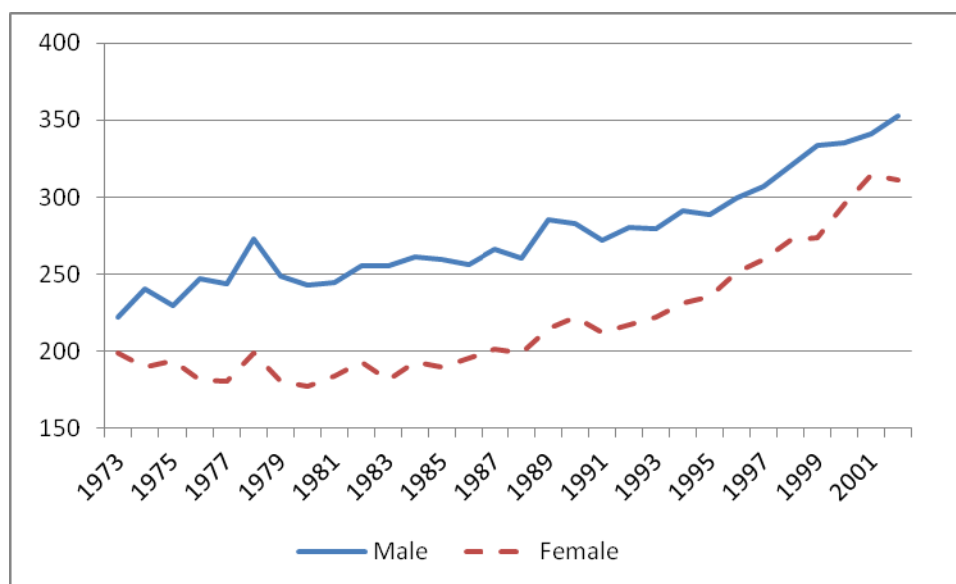

Figure 1. Shanghai cancer incidence rate per 100,000

To estimate (3) we considered two variables to capture the period effect, Shanghai aggregate annual income (gross domestic product, X_{1t}) and Shanghai aggregate annual energy consumption (X_{2t}) over the sample period. When income grows we can expect a reduction in cancer if higher income translates into better nutrition and healthcare by individuals. As income grows, however, environmental degradation and pollution also take place increasing the possibility of higher cancer incidence. In the absence of better data we use energy consumption as a proxy for pollution. These two variables tend to be highly collinear; therefore, we use in our regression the ratio of energy consumption to income,

which in growth rates is $x_{2t} - x_{1t}$, where $x_{1t} = 100\ln(X_{1t} / X_{1t-1})$ and $x_{2t} = 100\ln(X_{2t} / X_{2t-1})$. We

expect this ratio to have a positive relationship with cancer incidence. For interaction with age we tried

$t, x_{1t}, x_{2t}, x_{2t} - x_{1t}$ and observe that our main results remain unaffected by the choice but they have a

bearing on the other coefficient estimates. We present the results based on our pollution proxy x_{2t} since

it is a more meaningful choice to interact with age.

Table 1 presents the estimation results from Model (3) with some diagnostics for Shanghai total

cancer incidence growth by sex. The diagnostic statistics indicate mixed results for the male and female

models. Nevertheless, the RESET test does not indicate a functional form misspecification in both cases.

Therefore, mixed results on diagnostics are more reflective of data quality than anything else. Although

we have reported in the table the hetersocsedacity-autocorrelation consistent (HAC) standard errors,

we observe that they are very similar to the standard least squares standard errors, indicating that

heteroscedaticity and autocorrelations are not serious issues in our sample. Although normality is less

likely to hold for these data, our sample size is reasonably large (90 observations) for us to rely on the

Central Limit Theorem to use normal distribution for statistical testing.

Among the control variables, at the conventional levels of significance of 1%, 5%, or 10%, only

population growth is statistically significant in the female model. The population coefficient estimate in

both models is also very robust, not sensitive to different choices of interaction variables. The estimates

show that 1% growth in population leads to about 0.2 to 0.4 percent growth in cancer incidence. The

coefficients that represent the period effect and age-period interaction effect also carry the expected

signs.

Table 1. Estimation results from Model (3) for Shanghai cancer growth rate

	Male				Female			
	Coeff est	std error	t-stat	p-value	Coeff est	std error	t-stat	p-value
Constant	13.519	4.467	3.026	0.004	5.434	6.558	0.829	0.410
Population growth	0.242	0.147	1.649	0.104	0.441	0.194	2.273	0.026
Energy/Income growth	0.142	0.074	1.906	0.061	0.004	0.090	0.048	0.962
Age*energygrowth	-0.032	0.015	-2.137	0.036	-0.018	0.022	-0.846	0.401
Agesq*Energy growth	0.0004	0.000	2.442	0.017	0.0003	0.000	0.939	0.351
Birth Cohorts								
C1893-	22.816	7.396	3.085	0.003	10.598	9.934	1.067	0.290
C1898-	34.671	7.741	4.479	0.000	17.299	11.962	1.446	0.153
C1903-	23.195	6.565	3.533	0.001	8.593	9.527	0.902	0.371
C1908-	17.081	5.642	3.027	0.004	1.903	8.353	0.228	0.821
C1913-	14.246	8.331	1.710	0.092	17.180	13.375	1.284	0.204
C1918-	10.738	6.402	1.677	0.098	10.221	8.486	1.205	0.233
C1923-	7.593	5.509	1.378	0.173	7.038	7.339	0.959	0.341
C1928-	1.819	4.057	0.448	0.655	0.433	5.881	0.074	0.942
C1933-	-5.273	3.555	-1.483	0.143	-0.155	5.189	-0.030	0.976
C1938-	-34.815	7.007	-4.969	0.000	-21.507	8.625	-2.493	0.015
C1943-	-12.052	4.978	-2.421	0.018	-0.595	6.309	-0.094	0.925
C1948-	36.763	6.161	5.967	0.000	30.175	8.789	3.433	0.001
C1953-	40.049	6.278	6.379	0.000	31.399	8.656	3.627	0.001
C1958-	23.060	5.064	4.553	0.000	14.896	7.124	2.091	0.041
C1963-	-32.880	8.229	-3.996	0.000	-22.433	11.147	-2.012	0.048
C1968-	-46.809	9.984	-4.688	0.000	-42.420	12.380	-3.427	0.001
C1973-	-12.498	7.509	-1.664	0.101	-13.314	11.366	-1.171	0.246
C1978-	-0.098	5.600	-0.017	0.986	-5.087	9.278	-0.548	0.585
C1983-	41.844	8.766	4.773	0.000	41.930	14.397	2.912	0.005
C1988-	-5.854	10.462	-0.560	0.578	-11.398	9.986	-1.141	0.258
C1993-	-63.335	25.300	-2.503	0.015	-44.826	17.102	-2.621	0.011
C1998-	-60.263	-	-	-	-29.930	-	-	-
No of observations	90				90			
Mean of dependent	5.66				6.11			
R-squared	0.94				0.89			
S.E. of regression	11.26				14.23			
AR(1), F(1,70)	3.28			0.075	6.50			0.013
Hetero, F(25,64)	5.34			0.000	1.02			0.463
RESET, F(1,63)	0.10			0.753	2.00			0.148
Normal, Chi-sq(2)	1.50			0.470	8.49			0.014

Note: RESET=regression equation specification error test. Standard errors are heteroscedaticity and autocorrelation consistent. C1998 is minus the sum of other cohort coefficients.

Figures 2 and 3 plot the cohort effects (after adding back the constant term) for Shanghai and Osaka. As stated earlier we use Osaka for comparison (control case). (For Osaka we used only income growth for period effect.) Note that the cohorts are in five-year intervals and the figures show (as in Table 1) the beginning year of each cohort.

Shanghai cohort effects show two distinct humps for both males and females. The estimates pertaining to these humps are statistically significant (Table 1) and not sensitive to different specifications of Model (3). Regardless of whether we use age dummies or an age polynomial and with or without period controls these hump estimates remain roughly the same. The first hump in Figure 2 pertains to cohorts born between 1943 and 1962. These cohorts were in their young working ages during the Cultural Revolution. Having controlled for age, population, income and pollution effects and also observing the absence of a corresponding hump in Osaka we have to attribute these sharp increases in the growth of cancer incidence to the Cultural Revolution.

Exposure to traumatic events and psychological stresses of the Cultural Revolution must have affected the young adults more for them to become victims of cancer later in their lives. As Thurston (1985) observes: "Those who were middle-aged at the start of the Cultural Revolution had already experienced, at a minimum, the Japanese invasion, the civil war, the anti-rightist campaign, and the natural disasters of the "three bad years" from 1959-61." As for young people, Thurston observes that there were direct victims and there were those who began as active supporters of the Cultural Revolution and later became its victims. Both groups had to endure both physical and mental stresses.

What is further interesting to note in Figure 2 is the second hump that peaks for the cohort born over 1983-1987. The data show that this peak is due to a sharp increase in leukemia cases in the youngest age group 0-4 (see also Figure 4). This appears to be a ripple effect of the Cultural Revolution

itself. This suggests that a causal relationship between parental exposure to stresses of the Cultural Revolution and the incidence of leukemia in babies is very likely.
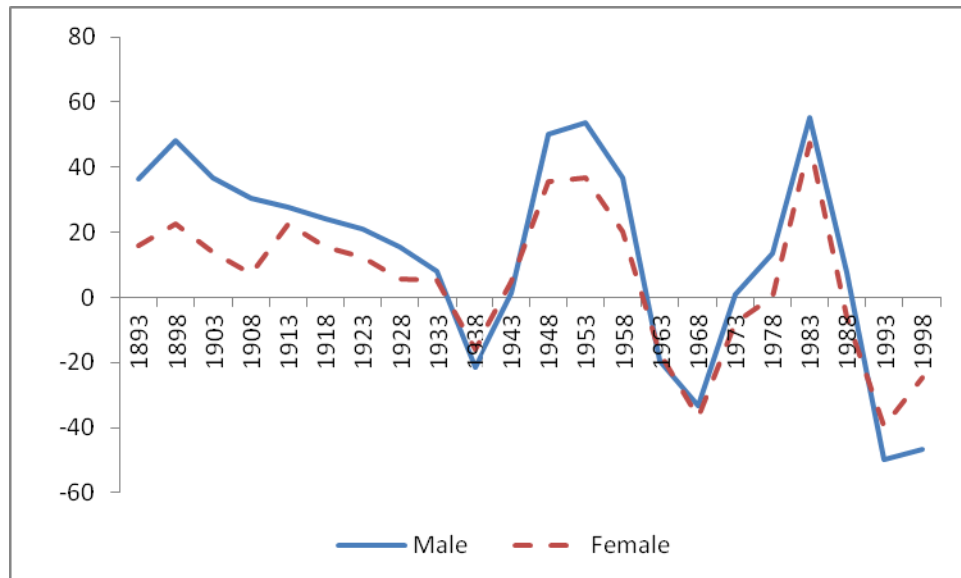

Figure 2. Growth (%) of cancer incidence in Shanghai by birth cohort
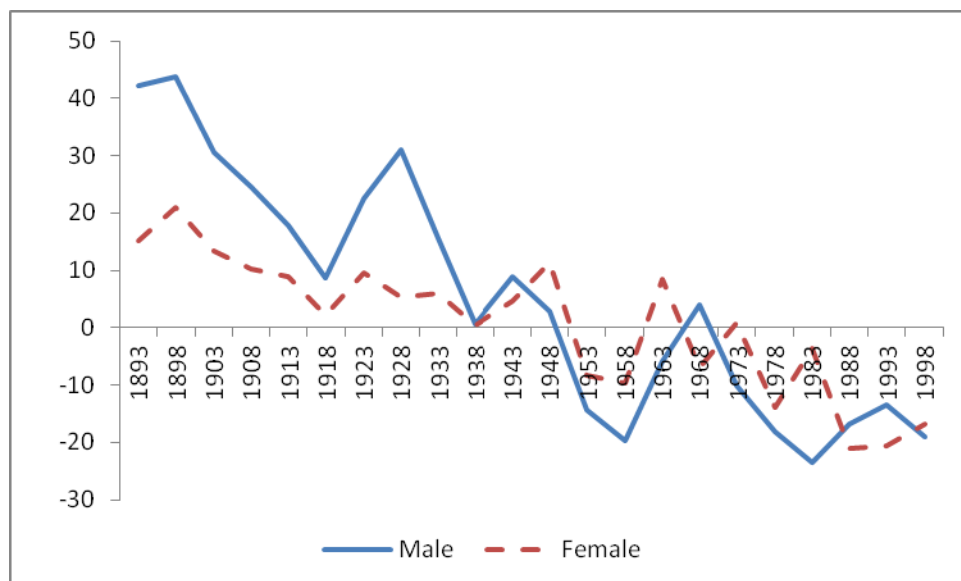

Figure 3. Growth (%) of cancer incidence in Osaka by birth cohort

It is also worth noting that the growth of cancer incidence in Osaka (Figure 3) shows a general downward trend with successive cohorts. However, there is a significant jump in cohort coefficients for those males born between 1923 and 1932. This jump is absent for females. Since these male cohorts were in their late teens and early twenties during World War II, we cannot exclude the possibility that the stress of Japan's role in World War II must have contributed to the jump in growth of cancer incidence in Osaka.

Whether mental stress is going to cause cancer in an individual depends on how the individual copes with stress. We can observe three types of responses by individuals to stress: 1. Some resort to external stimuli like alcoholic drinks and smoking, 2. Some simply suffer mentally and 3. Some manage stress by cultivating mental strength. Therefore, data at an individual level show large variations and make it difficult to measure the link between pure mental suffering and cancer, the aspect scientists are more interested in. However, data from events like the Holocaust, where the mode of oppression changed frequently without allowing much time for people to make mental adjustments to cope with the situation are likely to show the direct link, if any, between mental suffering and cancer.

We can shed some light on these aspects by applying the above cohort analysis to Shanghai data by type of cancer. A major limitation we face here is that for certain cancer types incidence numbers by age are so small or zero and as a result the estimated cohort effects are not very reliable. Therefore, we limit our analysis only to some illustrative cancer types. The estimated cohort effects for some of these cancer types are presented in Figure 4. End years in each chart in the figure are not the same because we had to drop some younger cohorts because of the above data problem.

Figure 4 shows that the hump observed in Figure 2, which we attributed to the Cultural Revolution, is present in each case though with some variation between males and females. To provide some

perspective to these charts it is useful to note the differences in cancer incidence by type between men and women. Over our observation period on average the incidence of liver and lung cancer among men was about 2.5 times and stomach and esophagus cancers about 2 times larger than that among women. The incidence of cancers in colon, rectum & anus, brain & central nervous system and of leukemia was very similar for men and women. Stomach cancer was the most frequent among both men and women till about mid 1980s but subsequently lung cancer becomes dominant among men and breast cancer among women.

In Figure 4 notable differences in the hump shape between males and females occur in liver cancer and to some extent in lung cancer. Since these cancers are more prevalent among men we may argue that the jump in the growth of cancer incidence is likely to have happened as a result of men seeking to more alcohol, other intoxicants and smoking to cope with stress induced by the Cultural Revolution. However, the presence of more pronounced hump shapes for women in the case of stomach and rectum & anus cancers, which are also more prevalent among men, precludes us from attributing the entire hump to changes in behavioral factors (alcohol and smoking). The picture becomes even clearer in charts for leukemia and prostate, testis, penis and breast cancer. Since leukemia incidence had been roughly similar in men and women very similar humps for leukemia cannot be attributed purely to the changes in behavioral factors. Similarly the jumps in prostate, testis and penis cancer in the case of males and breast cancer in the case of females cannot be attributed purely to changes in behavioral factors. Therefore, we cannot rule out the bearing that mental suffering had on the increase in cancer incidence among the cohorts who were exposed to the Cultural Revolution during their young working ages.
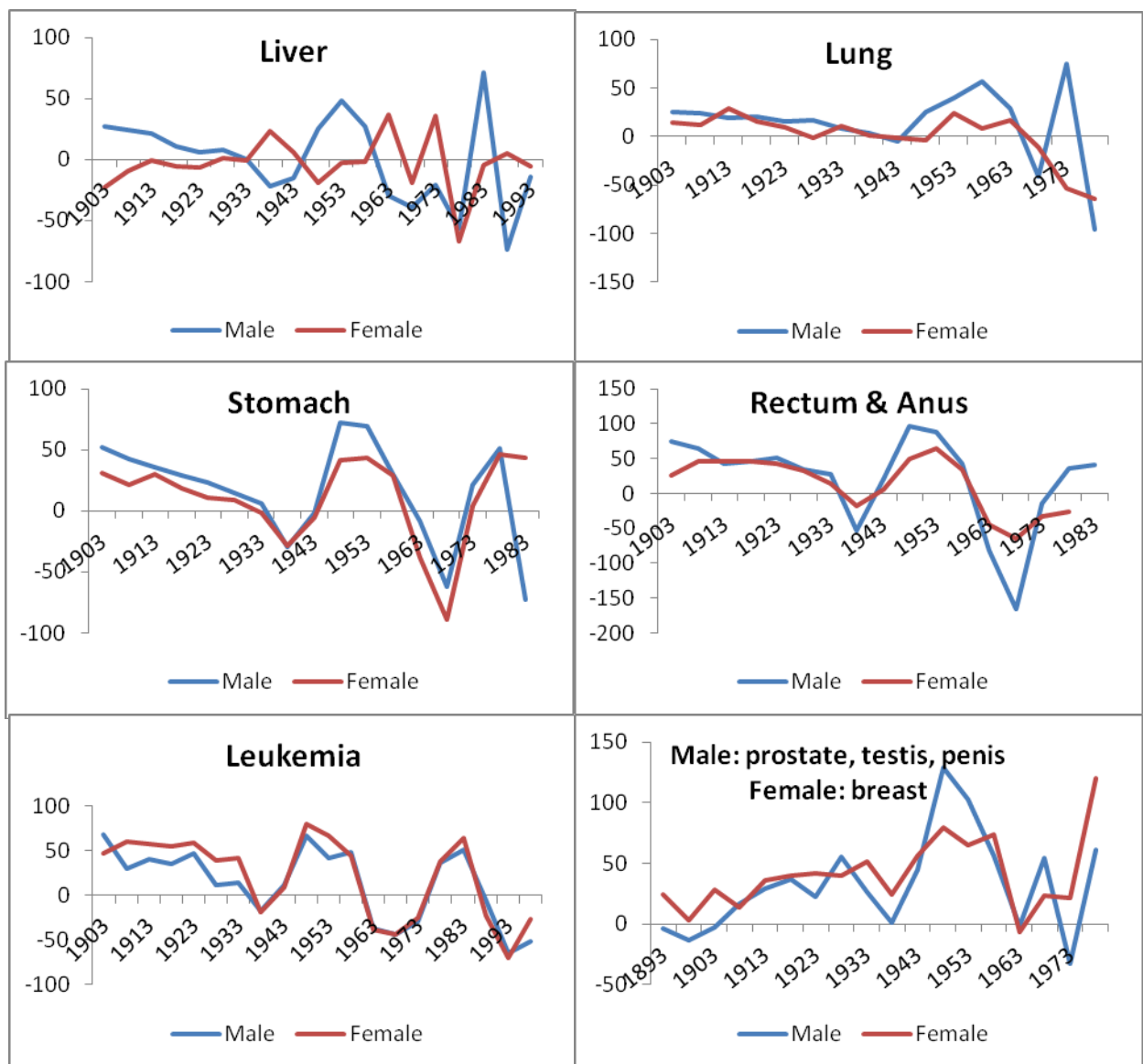
Figure 4. Growth (%) of cancer incidence in Shanghai by cancer type and birth cohort

4. Conclusion

Our analysis of Shanghai cancer data shows a very clear link between the Cultural Revolution and increase in cancer incidence. After controlling for population effect, age effect, period effect captured by a pollution proxy and income, and age-pollution interaction effect the regression estimates show a statistically significant sharp jump in cohort coefficients pertaining to those who were exposed to the Cultural Revolution during their young working ages. Some of this increase may be attributed to changes in behavioral factors such as increased alcohol consumption and smoking as means of coping with stresses caused by the Cultural Revolution. However, the similarity in the jump in the growth rates of incidence of different types of cancers between men and women clearly indicate a direct link between pure mental stress and cancer. As the effects of the Cultural Revolution withered away, growth of cancer incidence (adjusted for other effects) also came down.

Cancer has emerged as the top killer, surpassing heart disease, in some high-income countries. In terms of economic cost the American Cancer Society has ranked cancer as the top economic killer (John and Ross, 2010). Cancer was not a common disease in antiquity (David and Zimmerman, 2010). The rapid increase in cancer incidence in modern societies is largely attributed to increasing exposure to carcinogens and behavioral factors such as improper diet, smoking and a lack of physical activity. These are also the running themes of the papers on cancer prevention presented at the UIUC 10 World Cancer Congress held in Shenzhen, China in August 2010. We did not come across any paper in the conference that focuses on mental stress and its interaction with other factors as a trigger of cancer. Persistent mental stress in modern fast paced competitive societies is a common observation. In Japan, for example, deaths due to heart diseases have been much lower than that due to cancer probably because of healthy diets. However, age-adjusted cancer incidence rates for men and women in Japan have trended upward while the age-adjusted incidence rates due to coronary heart diseases do not show

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such an upward trend (Cancer Statistics in Japan, online and Kitamura et al., 2008). Therefore, the effect of mental stress on triggering cancer may not be isolated to events like the Cultural Revolution and cancer prevention needs a holistic approach to both mind and body management.

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