Education and cancer risk*

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Abstract

There exists a strong educational gradient in cancer risk, which has been documented in a wide range of populations. Yet relatively little is known about the extent to which education is causally linked to cancer incidence and mortality. This paper exploits a large social experiment where an education reform expanded compulsory schooling during the 1960s in Norway. The reform led to a discontinuous increase in educational attainment, which we exploit to estimate the effect of the reform on various cancer outcomes. We find little evidence that education affects overall cancer risk, as well as the most common cancer sites in isolation with two exceptions: The compulsory school reform lowered the risk of lung and prostate cancer for men. These protective effects are consistent with the idea that education reduced smoking behavior and increased the uptake of prostate cancer screening.

Keywords: Education, Causality, Health, Cancer
JEL-Codes: I12, I21

*We are grateful to Kjell Salvanes and Manudeep Bhuller for generously sharing the details of the education reform and education coding. The research on this paper was funded by the Norwegian Research Council (ES436293). Some of the data in this article are from the Cancer Registry of Norway. The Cancer Registry of Norway is not responsible for the analysis or interpretation of the data presented.

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1 Introduction

Cancer is a primary health risk. Over the last 30 years, the western world (including Norway) has witnessed a steady increase in cancer incidence and cancer mortality (for almost all cancer sites), for both men and women. By now, cancer is one of the major causes of death, and this is unlikely to change in the near future. On a brighter note, considerable progress in cancer survival has been made for many of the common cancer sites. Due to improved cancer prevention as well as cancer treatment, we have also seen an increase in cancer survival rates, which more than doubled in most western countries for most (but not all) cancer sites over the same period.

Nowadays, there are many public policies that aim to reduce cancer risks and increase cancer survival; among these are public health campaigns to encourage healthy behavior, taxation and subsidization to regulate healthy consumption, mandatory screening programs to detect cancer early, health insurance policies to improve access and coverage, financial assistance programs to cancer patients, and the funding of cancer research.

In this paper we concentrate on the question whether education reduces cancer risk. If more educated men and women have more resources to devote to preventive and curative health care, prefer longer and healthier lives, are more able to detect cancer early, and are better informed on how to seek and respond to the cancer treatments, we should see reduced cancer risks (and higher survival rates) among more educated men and women. Along similar lines, Grossman (2006), Cutler and Lleras-Muney (2010) and Brunello et al. (2016) argue that differences in resources, preferences, knowledge and behavior may explain why more educated men and women face lower health risks.

In order to get at the causal link between education and cancer risk empirically, we exploit a large compulsory schooling reform that happened 50 years ago in Norway. From 1960 to 1972, this education reform was gradually implemented in different

municipalities at different times. Before the reform, children had to attend school through the 7th grade. After the reform, children had to attend school through 9th grade, adding two years of compulsory education. Since the reform exogenously affected the educational attainment, these children form meaningful control and treatment groups; that is, some children spend two more years in school than other children similar to them on any other point but their birth year, and municipality of residence. In addition to extra years of compulsory education, the reform also standardized the curriculum and postponed tracking. In our empirical setup, we will follow these children into (later) adulthood and subsequently compare their differences in cancer mortality, overall cancer risk and cancer risks for the most common cancer sites. In particular, we will estimate the effect of the reform on the various cancer outcomes. If the reform raised the average educational attainment and human capital of children, we can interpret any negative reform estimate as a protective effect of education on cancer risk.

The data we use are a combination of multiple administrative registers in Norway. Educational attainment, taken from detailed education classifications, is the highest obtained schooling measured in nominal years of education. Municipality information comes from the 1960 Census, which collects information on municipality of residence at the time of the education reform. Cancer information comes from the Norwegian Cancer Registry, which holds records of any cancer diagnosis and, in case of death, whether cancer has been the leading cause. These registers are then matched using personal identification numbers of all Norwegian citizens, providing information about a child's year of birth, gender, municipality, educational attainment, cancer mortality, overall cancer risk and cancer risks for all cancer sites.

To preview the main results, we find little evidence that education affects cancer risk. This holds for all cancer sites together as well as the most common cancer sites in isolation, with two exceptions. Our estimates consistently show that the education reform lowered the risk of lung and prostate cancer for men. These protective effects

are consistent with the notion that education reduced smoking and increased the uptake of prostate cancer screening. Equally important is our finding that almost all the correlations we estimate between education, cancer mortality and cancer risk, aggregated across all cancer sites, are statistically significant and negative. It thus seems that unobserved endowments play a crucial role in explaining the educational gradient in cancer risk.

Our work relates to a small number of recent papers on the causal effect of education on cancer risks.[1] Palme and Simeonova (2015) estimate the effect of education on breast cancer risk. Using a compulsory schooling reform in Sweden (which is very comparable to the compulsory schooling reform we use in our paper), they find that breast cancer incidence and morality rates are significantly higher for women exposed to the reform. Lager and Torssander (2012) estimate the effect of education on overall cancer mortality as well as cause-specific cancer mortality using the same compulsory schooling reform. They find that mortality rates, defined as dying from cancer after age 40, fall for both men and women. When they look at mortality rates at four different cancer sites (lung, breast, lymph and other), they only find a reform effect for lung cancer for women; that is, women exposed to the reform are less likely to die from lung cancer. They do not observe any reform effect at the other cancer sites. Buckles et al. (2013) estimates the impact of college education on, among others, cancer mortality among US men. Using military draft lotteries in the 70s as instrument for college education, they find that large protective effects on cancer mortality (and on lung cancer mortality in particular).

Our work adds to this literature in three ways: it applies a comparable methodology with different data, which is useful when available evidence is scarce and not always consistent; it considers a wider set of cancer sites; and, it also examines

[1]There is much empirical work on the relationship between education and cancer risks. See Woods et al. (2006) for an overview of most of these studies, which mostly find that education associate negatively with cancer risks and associate positive with cancer survival. Because these associations do not make a distinction between selection and causation, their interpretation remains unclear

whether the cancer has spread to other locations (metastatic cancer), which is more likely to inflict serious health risks.

Our work also relates to a growing literature in economics on the causal link between education and later life health, in particular mortality. A majority of these studies make use of the compulsory schooling reforms as source of exogenous variation in education. The first causal study on education and mortality is of Lleras-Muney (2005). She uses differences in compulsory schooling laws across the various states in the United States and finds that education has a causal impact on mortality; that is, more education increases life expectancy.[2] Since then, other studies have examined comparable relationships using changes in compulsory schooling legislation, across regions, time or both, to determine whether education is helping us to be healthier (Clark and Royer, 2010; Gathmann et al., 2015; Meghir et al., 2013; Oreopoulos, 2006). The results of these studies are mixed. Oreopoulos (2006) shows that more educated men have a better health and live longer, while Clark and Royer (2010) report that education has little impact on later life health. Gathmann et al. (2015) even find a small negative effect of education on mortality for men. The findings of Meghir et al. (2013) lie somewhere in between. Their estimates indicate that more educated men experience reduced mortality up to the age of fifty, but that these life gains are offset by increased mortality later on in life. Meghir et al. (2013) also consider cancer mortality as some of their health outcomes, and find some evidence that deaths from preventable diseases (including lung cancer) increase slightly for men who are exposed to the reform.

The rest of our paper proceeds as follows. Section 2 describes the Norwegian education reform in more detail. Section 3 describes the data and Section 4 introduces the empirical strategy and shows how the reform can be used to get differences-in-differences estimates on the effect of education on cancer risk. Section 5 presents

[2]In a follow-up study, Glied and Lleras-Muney (2008) find that the survival advantage for more educated people is highest for diseases which treatments have experienced large technological progress.

results and Section 6 concludes.

2 Education and cancer risk factors

2.1 The compulsory school reform in Norway

In this paper we exploit an education reform that, among others, expanded compulsory schooling in Norway. The education reform (intended to raise the human capital of the Norwegian population) has been used before to identify the causal effect of education on, for example, earnings (Aakvik et al., 2010; Bhuller et al., 2016), IQ (Brinch and Galloway, 2012), educational attainment of the next generation (Black et al., 2005) or fertility (Monstad et al., 2008). Its institutional background has been well documented in studies of, for example, Lie (1973) and Lindbekk (1993). In the following, we build on these sources to provide a brief overview of the reform, and how the reform can be helpful in identifying the effect of education on cancer risk.

2.2 Institutional background of the compulsory school reform

In the 1960s and 1970s, an education reform extended compulsory education from seven to nine years. Prior to the reform, children started school at the age of 7 and finished compulsory schooling in 7th grade at the age of 14. After the reform, children started school again at the age of 7 but finished compulsory schooling in 9th grade at the age of 16. The educational reform also introduced a new comprehensive school. In the new comprehensive school children were kept together in one common school through 9th grade. All children were exposed to the same curriculum and faced the same level of instruction in two subsequent tracks: grades 1 to 6, which are regarded as lower primary education, and grades 7 to 9, which are regarded as upper primary. The new comprehensive school replaced the more selective system of lower secondary education where children were tracked into two parallel tracks with different levels of instruction: the academic track (*realskole*) prepared children for an

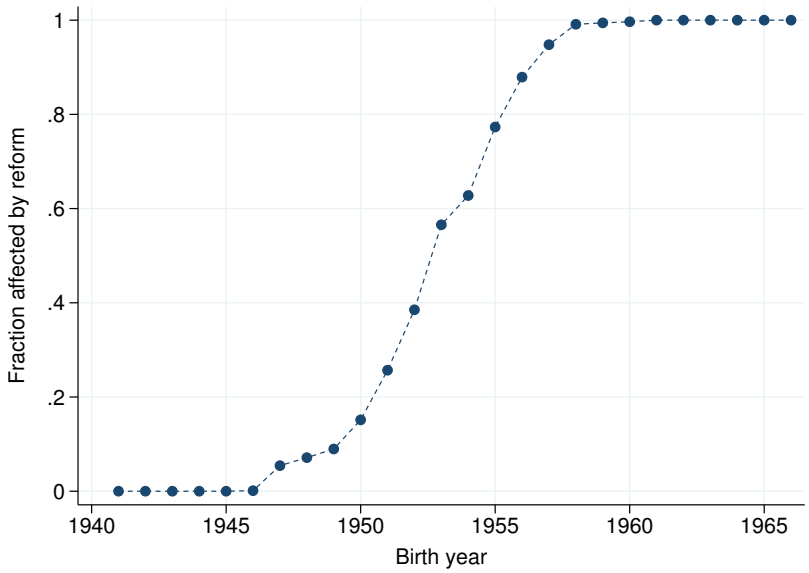

Figure 1. Cohort shares of reform exposed children by birth cohort.

academic gymnasium and subsequent university education and the vocational track (*fagskole*) prepared children for vocational training and general education outside the path to higher professional and theoretical education. The reform was aimed to improve the quality of primary education, in particular in more rural areas. This was done by establishing a common primary school act where the local governments of cities and rural communities were subject to the same minimum school requirements regarding course of instruction and school facilities.

The reform was implemented within a twelve year period. The reform began in 1960. The first cohort that could be affected by the reform was the cohort born in 1946. These children started school in 1954 and were exposed to the reform for at least two years. The reform was completed in 1975. The last cohort affected was the cohort born in 1961. In Figure 1 we plot cohort shares of reform exposed children by birth cohort. The figure illustrates the gradual roll out of the reform.

Prior to implementation, municipalities were asked to report on their population growth, tax revenues, on the local demand for education and school situation, including the availability of teachers, the number of required teachers for the nine year comprehensive school, and the available school buildings. A committee under

the Ministry of Education took these municipality characteristics into account when deciding on participation. Once approved, funding for extra teachers and school buildings was provided. Since the implementation of the reform at the municipality level placed economic and organizational demands on the local resources, economic subsidies were given to encourage implementation. These subsidies were granted to level differences between rich and poor municipalities and ensured that implementation was arguably representative across Norway (Lie, 1973).

2.3 The compulsory school reform treatment

In our empirical setup we treat the compulsory school reform as a natural experiment that assigned children to treatment and control groups, in a more or less random fashion depending on their year of birth and municipality of residence. With the treatment defined as exposure to the compulsory school reform, we follow these children into later adulthood and compare their cancer mortality rates, overall cancer risk and cancer risks for the most common cancer sites. Any observable cancer differences between the treated and control children can then exclusively be attributed to differences in reform exposure. In order to interpret an estimated reform effect as an education effect, however, we need to know more about what the reform treatment actually entails.

The reform introduced three institutional changes to the education system at the same time. The reform increased mandatory education, standardized curriculum and postponed ability tracking. Each of these three changes may have had an impact on the educational attainment of these children, both in quantity and quality. The mandatory increase from seven to nine years of compulsory education, for example, raised the time children spent in school and possibly the skills (relevant to a good health) they have learned there. The standardization of the curriculum likely improved the average level of quality of the schools (Black et al., 2005). The delayed tracking was aimed to raise equality of opportunity; it is unclear, however,

how tracking has affected the educational attainment of children. Some studies find that early tracking is beneficial for all students (Duflo et al., 2011), while other studies find a small positive effect of postponed tracking, possibly depending on family background (Meghir and Palme, 2005; Pekkarinen et al., 2013).

Additionally, the reform may have induced other changes in the education system affecting the educational outcomes of children (and possibly their later life outcomes as well); among these are the reform-induced changes in teacher quality and classroom composition. The reform increased the demand for teachers due to increased school access. If new and inexperienced teachers are more likely to teach those children affected by the reform, the reform was accompanied with a fall in teacher quality with possible long run consequences (Chetty et al., 2011). Another, typically overlooked, feature of the reform is that the reform changed the classroom composition of children. Since the reform forced all children to stay in school for two additional years, the peer composition of each child individually changed. It is not clear how this affects classroom quality. It is possible, for example, that children who would obtain a university degree before the reform did worse because of increased exposure to potential dropouts. And reversely, children who would leave school in 7th, 8th or 9th grade before the reform may do better because of increased exposure to more academically orientated children.

The reform effect is thus a composite reform effect on cancer risk, incorporating not only the increase in years of mandatory education, but also the anticipated (and unanticipated) changes in school, teacher and classroom quality that likely matter for the human capital accumulation of children. While our empirical framework is limited in its ability to disentangle the composite reform effect, it is still possible to interpret the composite reform effect as an education effect. Our empirical framework gives us reform estimates that measure the effect of education on cancer risk if we assume that the reform raised the educational attainment and human capital of children. With the positive reform effect estimates on IQ, educational attainment

and earnings (Aakvik et al., 2010; Bhuller et al., 2016; Black et al., 2005), we believe that this interpretation assumption is not too difficult to accept.

3 Data

The data we use are a combination of multiple administrative registers in Norway. The baseline sample is drawn from the Norwegian Population Register. This register contains information on all Norwegian citizens who were alive in 1954. In our analysis we focus on cohorts affected by the Norwegian education reform based on year of birth and municipality of residence (at the school going age). In the Norwegian Population Register we select the cohorts born between 1941 and 1966.[3] From the 1960 Census we collect information on municipality of residence at the time of the compulsory schooling reform.

Education information comes primarily from the Norwegian Education register which was established in 1970. This register contains detailed information on the highest education level obtained by each individual in 1970 and onwards. We follow Bhuller et al. (2016) and measure the highest educational attainment in nominal years of schooling based on detailed education classifications.

Cancer information comes from the Norwegian Cancer Registry, which holds records of any cancer diagnosis and, in case of death, whether cancer was the leading cause. At the time of writing this cancer registry contained individual level data from 1954 to 2014. Reporting to the cancer registry is mandatory (and done by clinicians and pathologists), and the completeness of registrations for solid tumors is close to 100 percent (Cancer Registry of Norway, 2007; Larsen et al., 2009). Information is available on the date of diagnosis, location of the tumor (encoded by ICD-10), stage at diagnosis (metastasis), the date the death certificate was issued (if the patient has died) and whether cancer was the main cause of death.

[3]Following (Bhuller et al., 2016) we extend the sample with pre- and post-reform cohorts. This allows us to better approximate linear trends prior to reform implementation in municipalities that adopted the reform early.

Table 1. Cancer incidence, by gender

	Men	Women
Cancer type:		
- Colorectal	0.011	0.010
- Skin	0.011	0.012
- Lung	0.009	0.008
- Leukemia	0.010	0.007
- Prostate	0.026	—
- Testicular	0.005	—
- Breast	—	0.038
- Cervical	—	0.013
- Other	0.040	0.038
All cancers	0.100	0.115
Metastatic cancers	0.014	0.015
Cancer mortality	0.027	0.028
N	725,778	697,363

Table 1 shows the prevalence of cancer mortality, overall cancer incidence, and cancer incidence for the most common cancer types in our sample encoded by the first three digits of the ICD-10 (International Classification of Diseases, 10th revision) codes. Because we work with samples in which the average age when diagnosed with cancer is quite young, the cancer rates are quite low.[4] Yet we see the common cancer patterns (albeit much smaller), with breast cancer being the most common cancer type among women and prostate cancer for men. Overall about 10 percent of the women in our sample and 11 percent of the men are diagnosed with cancer, and a bit less than 3 percent of them die from cancer. We also consider metastatic cancer as one of our outcome variables. About 1.5 percent of both men and women in our sample are diagnosed with metastatic cancer.

[4]In our sample (which includes the 1941-1966 birth cohorts), average and median ages at diagnosis are 53 and 56. When not excluding any birth cohorts, average and median ages at diagnosis are 66 and 68 years

4 Empirical strategy

We examine three related questions concerning the relationship between educational attainment, the education reform and cancer risk. We start by examining the impact of the education reform on educational attainment. While the reform represents in principle a composite effect, it is important to establish that it indeed increased the educational attainment of those affected.

We then examine the extent to which various cancer risks (observed up to the age 68) vary with education. We first estimate the education gradient in cancer risk, and then explore how these correlations compare (and contrast) to those reported elsewhere in the medical literature. After that we turn to the causal impact of the education reform on cancer risk.

The first model provides an estimate of the impact of the reform on educational attainment. Since different municipalities adopted the education reform at different times, we estimate a standard differences-in-differences regression model

$$educ_i = \beta_1 reform_i + \beta_2 female_i + \phi_m + \omega_t + v_i \tag{1}$$

where education $educ_i$ depends on whether the individual is exposed to the reformed education system $reform_i$, gender $female_i$, school municipality ϕ_m, year of birth ω_t, and the random error term v_i, which captures unobserved endowments and characteristics that have an impact on educational attainment. In our model we treat the education reform as our source of exogenous variation in education and assume that v_i is uncorrelated with $reform_i$ conditional on the other control variables. The parameter β_1 can therefore be interpreted in a causal fashion, measuring the average change in average years of education due to reform exposure.

We use Cox Proportional Hazard (CPH) models to analyze cancer incidence and mortality rates. Cancer risks are right censored, which may lead to a censoring bias, and Cox models take right censoring into account, exploiting all time variation in the

data, while at the same time allowing the baseline risk to vary non-parametrically across municipalities. For time to cancer diagnosis/death we thus use the following proportional hazard specification

$$\lambda(s) = \lambda_m(s) \cdot \lambda_t \cdot \exp(\delta_1 reform_i), \tag{2}$$

with exposure time s, a municipality specific baseline hazard λ_m, a cohort specific relative risk factor λ_t, and explanatory variables defined above. We will estimate these proportional hazard models separately for men and women.

The key assumption in differences-in-differences estimation is that reformed and non-reformed municipalities experience common trends; that is, in the absence of the reform the outcome variable should evolve similarly in reformed and non-reformed municipalities. If this assumption is violated, these specifications will give us inconsistent estimates of the reform effect.

The common trend assumption may be violated if the roll-out of the reform across municipalities is not exogenous, but correlates in a systematic manner with municipality characteristics that affect cancer risk. To investigate this possibility we first correlated municipality characteristics in 1960 with cancer risk. We have information about population size, density, educational attainment, per capita income, labor market outcomes such as unemployment, welfare take-up and disability rates (all measured in 1960). For ease of comparison we standardized these variables in the population on municipalities, and Figure A1 shows that many of these factors are indeed significant predictors of cancer risk.

In a next step we assess whether the timing of the reform correlates with these municipality baseline confounders. We follow Bhuller et al., 2016 and regress a dummy that is one in the first reform year on time dummies, and time dummies interacted with baseline covariates. This allows us to check whether, in any given year, the municipalities that introduced the reform are different from the other municipalities.

These results are shown in Figure A2. Here we do not see any systematic pattern between the timing of the reform and the municipality characteristics.

The main threat to identification comes from the timing of the reform being confounded by municipality specific trends in cancer risk. While the previous checks show that the timing does not systematically correlates with baseline confounders, the analysis below also reports estimates from specifications that control for trends that are allowed to vary across municipalities as a function of year of reform. Reassuringly, the inclusion of these flexible trends will make our baseline estimates more precise, while the impact estimates barely change.

5 Results

5.1 The effect of the reform on education

The impact of the reform on educational attainment is significant and positive. The school reform raised the average number of years spent in school by about 0.19 (s.e. 0.02) years. This effect is primarily driven by the increased education of those men and women who would have dropped out in either 7th or 8th grade in the absence of the reform. Note that our reform estimates are remarkably stable across different samples and different specifications. We obtain very similar reform estimates when we look at men and women separately, and with and without municipality specific trends. As expected, the estimates are very similar to those reported in Bhuller et al. (2016).

The right panel of Figure 2 displays the effect of the reform on average years of education, after taking out municipality and birth year fixed effects. Time zero represents the year of reform implementation. Comparing the average years of education in pre- and post-reform years, we see that there is a discontinuous jump in the educational attainment of men and women around the time of the implementation of the reform.

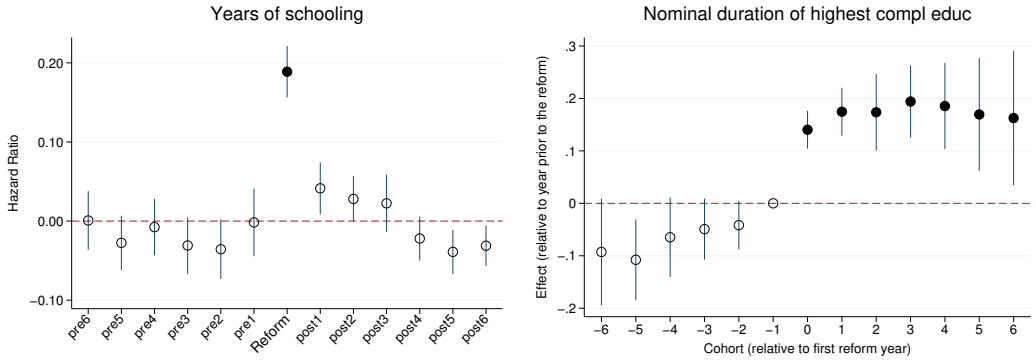

Figure 2. Schooling, Men and Women

The left panel of Figure 2 presents the point estimates from placebo checks that shift the reform, and thus pretend that the reform was implemented earlier or later than the actual reform year. In our pre-placebos we estimate treatment in models where we falsely assign treatment to men and women in our control group born up to six years before the first treated cohort. And reversely, in our post-placebos we estimate treatment effects in models where we falsely assign no treatment to men and women in the treatment group born up to six years after the first treated cohort. As can be seen in the graph, there are no indications of such (placebo) "reform effects" on educational attainment. The estimated placebo coefficients are close to zero and not statistically significant.

5.2 The association between education and cancer risk

Table 2 contains simple estimates of the relationship between education (measured in years) and cancer risks controlling for the exogenous variables municipality and year of birth. Results are presented for various cancer outcomes: cancer mortality, overall cancer risk and cancer risks for the most common cancer sites, including colorectal cancer, lung cancer, skin cancer, leukemia, breast and cervical cancer for women, and prostate and testicular cancer for men.

Hazard ratios lower than one indicate that education is associated with lower risk, while hazard ratios larger than one indicate elevated cancer risks for those with

Table 2. Cancer cancer risk and years of schooling – Hazard ratios, by gender

	Men		Women	
	(1)		(2)	
Cancer type:				
- Colorectal	0.99***	(0.004)	0.98***	(0.004)
- Skin	1.05***	(0.004)	1.03***	(0.004)
- Leukemia	1.00	(0.004)	1.00	(0.005)
- Lung	0.88***	(0.003)	0.88***	(0.004)
- Prostate	1.02***	(0.002)	—	
- Testicular	1.02***	(0.006)	—	
- Breast	—		1.02***	(0.002)
- Cervical	—		0.96***	(0.003)
- Other	0.97***	(0.002)	0.98***	(0.002)
All cancers	0.99***	(0.001)	0.99***	(0.001)
Metastatic cancer	0.93***	(0.003)	0.94***	(0.003)
Cancer mortality	0.93***	(0.002)	0.94***	(0.002)

Note: The sample includes all men (N= 725,778) and women (N = 697,363) born between 1941 and 1966. Reported in each cell are hazard ratios from separate models. All models are stratified at the municipality (of residence in the 1960s) level, and include dummies for birth cohorts. *** significant at 1%.

more education. More education is accompanied by a small but highly statistically significant reduction in overall cancer risk, both for men and women. The education gradient is somewhat steeper for metastatic cancer risk and cancer mortality risk, which are also reduced for individuals with more years of schooling.

Across the different cancer sites the picture is more mixed. For colorectal, cervical and other cancers more education is accompanied by small but highly statistically significant reductions in overall cancer risk. Lung cancer stands out here with larger reductions in cancer risk: the hazard ratio is about 0.88 (for both men and women) which implies that one extra year of education is associated with a 12 percent lower risk of being diagnosed with lung cancer.

In contrast, for four cancer types – skin, testicular, prostate and breast – the hazard ratios are significantly larger than one, which is consistent with previous research which also found positive associations between education and the risk of getting these particular types of cancer (Garner et al., 2005; Hemminki and Li,

2003; Pukkala and Weiderpass, 2002; Palme and Simeonova, 2015). For instance, the probability of being diagnosed with prostate and breast cancer increases by about 2 percent for each additional year of schooling. The only cancer type that is not significantly related to educational attainment, neither for men nor women, is leukemia.

These results are comparable to the estimates that have appeared in the medical literature. It is however difficult to interpret these estimates – both in terms of magnitude and in terms of direction – in a causal fashion as they are potentially confounded by correlations between individuals' educational attainment and unobserved cancer risk factors. In the next subsection we therefore turn to our reform estimates which test the hypothesis whether cancer risk and education are causally linked.

5.3 The effect of the reform on cancer risk

In Table 3 we present the hazard ratios for cancer incidence by reform exposure, separately for men (left panel) and women (right panel). The results in columns (1) and (3) are obtained by estimating equation (2) whereas we in columns (2) and (4) additionally include linear trends by year of reform implementation.

For most types of cancer there is no evidence that education affects cancer risk, as the reform estimates are not significantly different from one. For many cancer types the point estimates of the effect sizes are also small. For example, in contrast to Palme and Simeonova (2015), our reform hazard ratio for breast cancer is 0.99. Also for testicular and cervical cancer the hazard ratio is close to 1. The estimated hazard for all cancers is 0.99 for both men and women and relatively precisely estimated. When we pool these estimates we can rule out protective effects of the reform for overall cancer as small as 0.03.

There are two noticeable exceptions to the above. For men we estimate a lung cancer hazard ratio of 0.89 and a prostate cancer hazard ratio of 0.92, which are both significant at the five percent level and suggest that the reform had a significantly

Table 3. Education reform and cancer risk – Hazard ratios, by gender

	Men				Women			
	(1)		(2)		(3)		(4)	
Cancer type:								
- Colorectal	0.96	(0.05)	0.95	(0.05)	1.07	(0.06)	1.08	(0.06)
- Skin	1.04	(0.06)	1.04	(0.06)	0.98	(0.05)	0.98	(0.05)
- Leukemia	1.08	(0.06)	1.08	(0.06)	1.03	(0.07)	1.03	(0.07)
- Lung	0.89**	(0.05)	0.90	(0.06)	0.96	(0.06)	0.93	(0.06)
- Prostate	0.92**	(0.04)	0.93*	(0.04)	—		—	
- Testicular	1.01	(0.08)	0.98	(0.08)	—		—	
- Breast	—		—		0.99	(0.03)	0.99	(0.03)
- Cervical	—		—		1.01	(0.05)	1.01	(0.05)
- Other cancers	1.02	(0.03)	1.02	(0.03)	1.04	(0.03)	1.04	(0.03)
All cancers	0.99	(0.02)	0.99	(0.02)	1.01	(0.02)	1.01	(0.02)
Metastatic cancer	0.93	(0.04)	0.92*	(0.05)	1.05	(0.05)	1.04	(0.05)
Cancer mortality	0.95	(0.03)	0.94*	(0.03)	1.02	(0.03)	1.01	(0.04)
Trends			✓				✓	

Note: The sample includes all men (N= 725,778) and women (N = 697,363) born between 1941 and 1966. Reported in each cell are hazard ratios from separate models. All models are stratified at the municipality (of residence in the 1960s) level, and inlcude dummies for birth cohorts. Trends indicate linear trends by year of reform implementation. ** significant at 5%, * significant at 10%.

large and protective impact on lung and prostate cancer risk.

When we compare the correlational results in Table 2 to the causal effects of Table 3, we see that both sets of results suggest that an extra year of education will reduce the risk of lung cancer (and all the positive externalities associated with it). For lung cancer omitted variable bias does not change the direction of the effect. For prostate cancer, on the other hand, the direction of the effect changes from increased risk, 1.02 in Table 2, to reduced risk, 0.92 in Table 3. One explanation for this sign reversal may be that men who choose to take more education simply live longer and are then more likely to be diagnosed with prostate cancer. When controlling for the selection into education, the probability of being diagnosed with prostate cancer decreases with years of schooling.

One potential caveat when interpreting the results in Table 3 comes from the fact that we consider several hypotheses, and that the more hypotheses we test, the higher is the probability of rejecting zero while there is in reality no effect (i.e. a false positive). We rely on permutation type placebo tests and event graphs to investigate the possibility of statistically significant effect estimates in Table 3 being false positives.[5]

Figures 3 and 4 present the placebo- and event graphs for lung and prostate cancer for men, the only two cancer types where we find statistically significant effects.[6] All specifications control for birth cohorts and stratify the hazard by municipality.

As before, the placebo graph shows two different types of placebo checks. In the pre-placebos the reform is shifted back in time and untreated cohorts are assigned treatment status based on the original reform timing. For example, *pre3* in the left panel of Figure 3 indicates that the reform is moved three years backward in time. To ensure that the placebo reform is unconfounded by the original reform, actually

[5]Alternative approaches, such as the Bonferroni method, control for the family-wise error rate (the probability of having at least one false positive) by adjusting the critical values of the different hypotheses. These methods are however very conservative. Applied in our case, these methods turn out to be uninformative.

[6]Appendix Tables A3–A6 present placebo and event graphs for all outcomes separately for men and women.

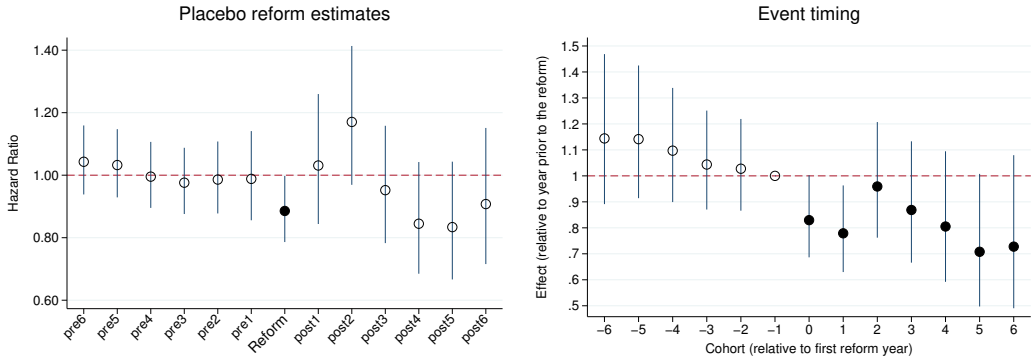

Figure 3. Lung cancer, Men

treated cohorts are omitted from the estimation. In contrast, in the post-placebos we pretend that treated cohorts are untreated by shifting the reform forward in time, again exploiting the original reform timing. As above, *post3* indicates that the reform is moved three years forwad in time. Here the untreated cohorts are dropped from the sample to ensure that the placebos are unconfounded by the original reform.

The placebo graphs in Figures 3 and 4 show that we consistently find no evidence of an effect in the placebo reforms. Note that the pre-placebo estimates, which falsely assign treated status to untreated cohorts, are much more precisely estimated than the noisy post-placebo estimates, which falsely assign untreated status to the treated. The only statistically significant effect we observe is the one for the actual reform, which we take as evidence that the effects for lung and prostate cancer are not false positives.

The event timing graph in Figures 3 and 4 investigate whether the timing of effect are consistent with a true effect. As above, the x-axis measures time in years to the reform implementation. When comparing pre- and post reform cohorts there is, as expected, a discontinuous (negative) drop in the risk of getting lung and prostate cancer that starts for the first affected cohort, which we interpret as additional evidence that the effects for lung and prostate cancer are not false positives.

The severity of the cancer depends not only on where the tumor is located, but also on whether the cancer has spread to other locations (or metastasize). We also

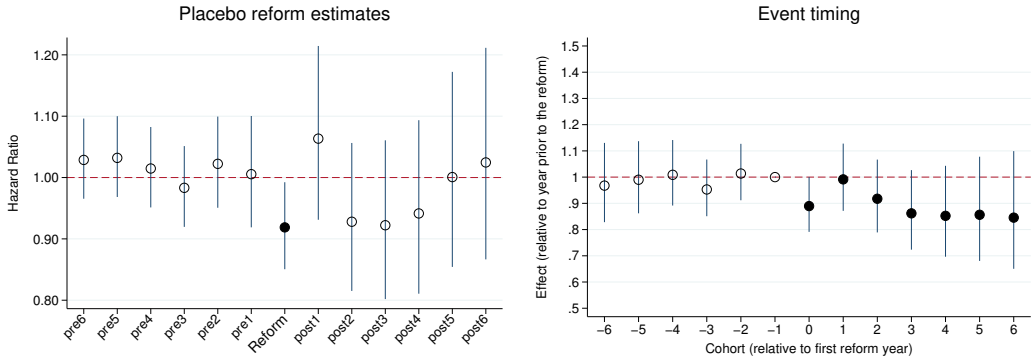

Figure 4. Prostate cancer, Men

investigate whether education and reform effects change when we consider the cancer risks of metastatic cancers, which are more likely to cause health problems. This is reported in the second lowest row of Table 3. For men, the hazard rate for metastatic cancer is about 0.93 and statistically significant in the specification where we control for reform year, which suggest that more educated men are less likely diagnosed with cancer that has spread out to other locations. As a comparison there is no effect of the reform on being diagnosed with cancer at all. For women, the hazard rates for metastatic cancers is fairly similar to the ones for all cancer, and not statistically significant.

In the last row of Table 3 we estimate the effects on dying from cancer. For men the hazard rate for cancer mortality is about 0.94 and statistically significant at the ten percent level when controlling for linear trends by year of reform implementation. For women, there is no effect on cancer mortality.

Although the effects on metastatic cancer and cancer mortality are marginally significant for men, the placebo tests and event graphs reported in the appendix are not conclusive, and we are therefore less confident about these estimates.

5.4 What is the effect of education on cancer risk?

We find little evidence that education has an impact on cancer risk across the board. This holds for all cancer sites together as well as the most common cancer sites in

isolation, with two exceptions: The compulsory school reform lowered the risk of lung and prostate cancer for men. It is important to understand why this is, and where these differences come from. In this section we discuss possible explanations.

Most medical scientists will a priori agree on the protective effect of education on lung cancer risk, and indeed the vast majority of empirical studies report a negative correlation between education (or a socioeconomic variation thereof) and lung cancer risk (Sidorchuk et al., 2009). These correlations are typically interpreted as evidence that poor health behavior, and harmful smoking in particular, is the leading cause of lung cancer (Peto et al., 2000). Of course, for this to be true we must assume that education causes men to smoke less or improves their working conditions. Evidence on this includes the work of De Walque (2007), who finds a negative effect of education on cigarette consumption (using the military draft lottery as a natural experiment). The weaker effect we find for women is not unexpected; at the time of the reform, women smoked considerably less than men (Lund and Lindbak, 2007).

In case of prostate cancer, we find that men who were exposed to the reform have a lower chance of getting prostate cancer. According to the American Cancer Society (cancer.org/cancer/prostatecancer) both diet and smoking are listed as potential risk factors which could be one explanation why taking higher education reduces the risk of prostate cancer. Another explanation may be that prostate cancer screening has become more and more common in Norway during the last ten to twenty years, and that education increases the likelihood of screening. While we are not aware of any studies looking at how education affects screening for prostate cancer, there is some evidence that individuals with a college degree are more likely screened for breast, colorectal and cervical cancer (Lange, 2011). As opposed to mammography screening – which is universal for all women in Norway between 50 and 69 – prostate cancer screening is opportunistic which may explain why we only find an effect of the education reform on prostate cancer and not breast cancer.

6 Conclusions

There is an extensive medical literature that finds strong negative associations between education and cancer risk. Important questions about the causal effect of education on cancer risk, however, remain largely unresolved. In this paper we try to provide some answers; that is, we investigate whether education has a protective effect on cancer risk. Our strategy uses the reform of the Norwegian education system, which has been implemented in different municipalities at different times, to establish causal effects of education on various cancer risks faced in middle and late adulthood. Our main finding is that we find little evidence that education has any impact on cancer risk. This holds for all cancer sites together as well as the most common cancer sites in isolation. There are two exceptions to these findings: Our estimates consistently show that the education reform lowered the risk of lung and prostate cancer for men.

When interpreting these results, four caveats should be kept in mind. First, the reform estimates are often small and for individual cancer sites also often relatively imprecisely estimated, with only significant reform effects estimates for lung and prostate cancer. Although this suggests some caution in the interpretation, our reform estimates rule out large gains in overall cancer risk from more and better education and across cancer sites we can rule out protective effects of the reform as small as 0.03. Second, we observe the affected birth cohorts up to age 53 to 67. To the extent that risks of developing (or dying from) cancer at later ages are driven by differences in education, our estimates do not capture this. Third, our results apply to Norway and may not generalize to other countries that have more costly education and health care. And last, the reform estimates we present come from children who are affected by the compulsory school reform; that is, children who stayed in school for at least one or two more years because of the reform. This means that our estimates do not necessarily measure potential protective gains of, for example, a college degree. A recent study by Buckles et al. (2013) considers

the impact of college education on, among others, cancer mortality among US men. They find large protective effects on cancer mortality (and on lung cancer mortality in particular). A comparable exercise using college opening reforms in Norway could be enlightening but is left for future research.

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A Extra results

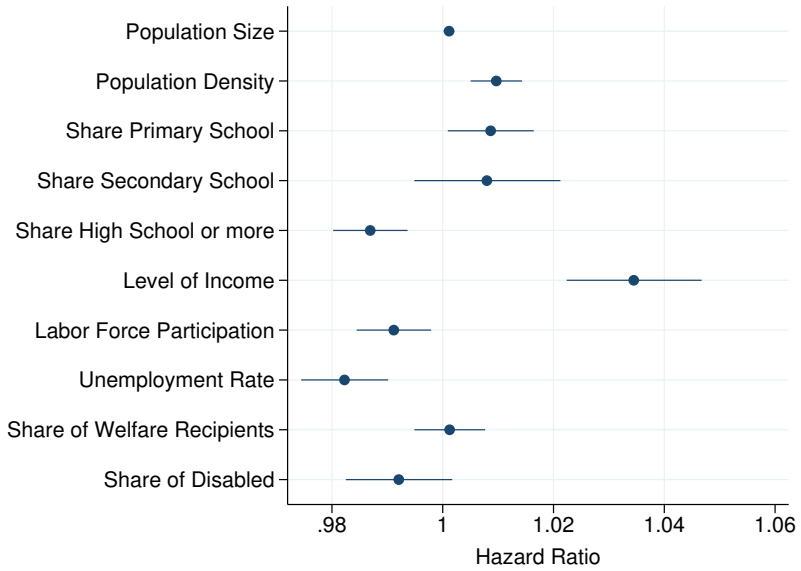


Figure A1. Municipality characteristics and cancer risk

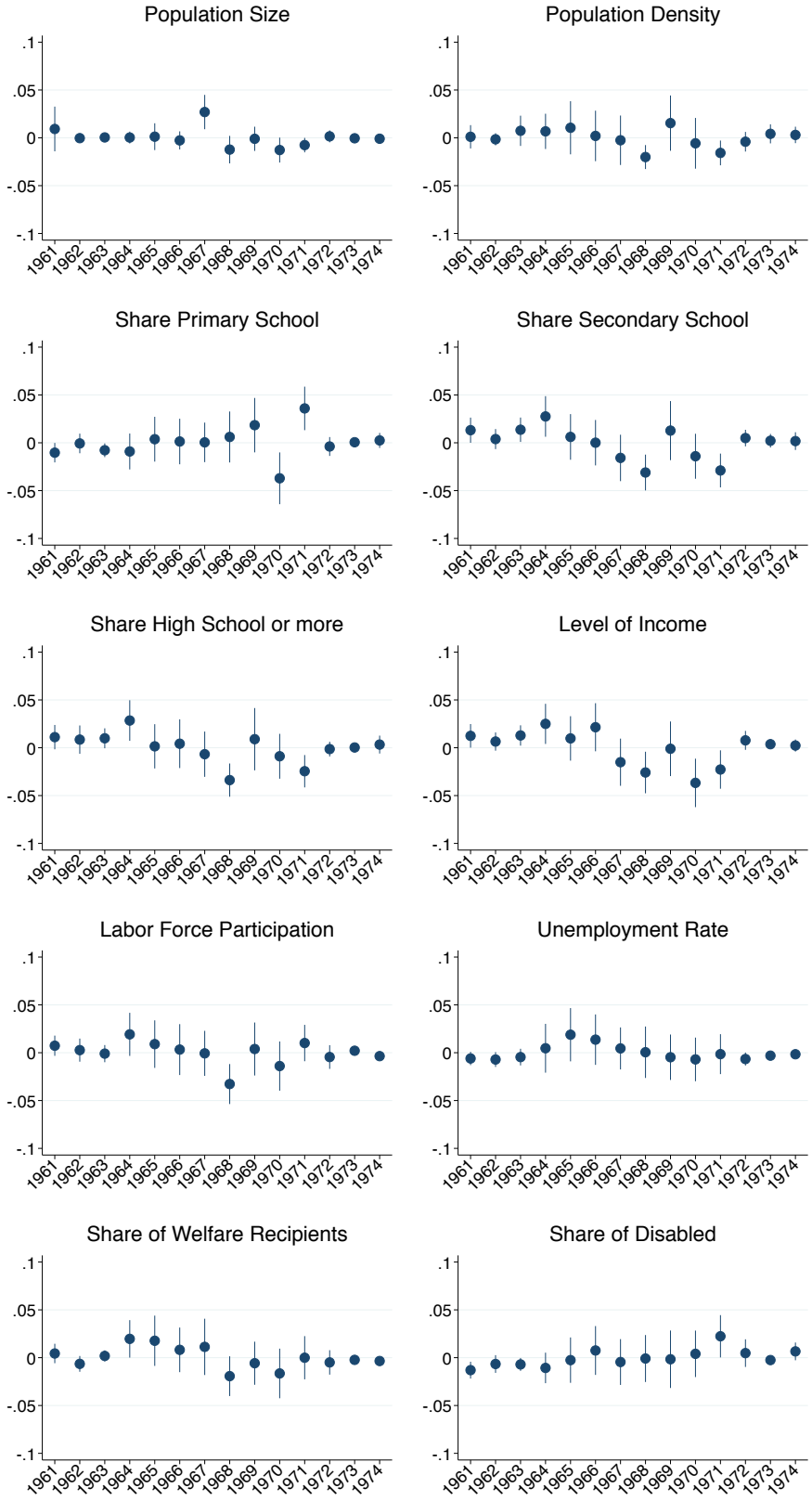


Figure A2. Timing of reform and municipality baseline characteristics

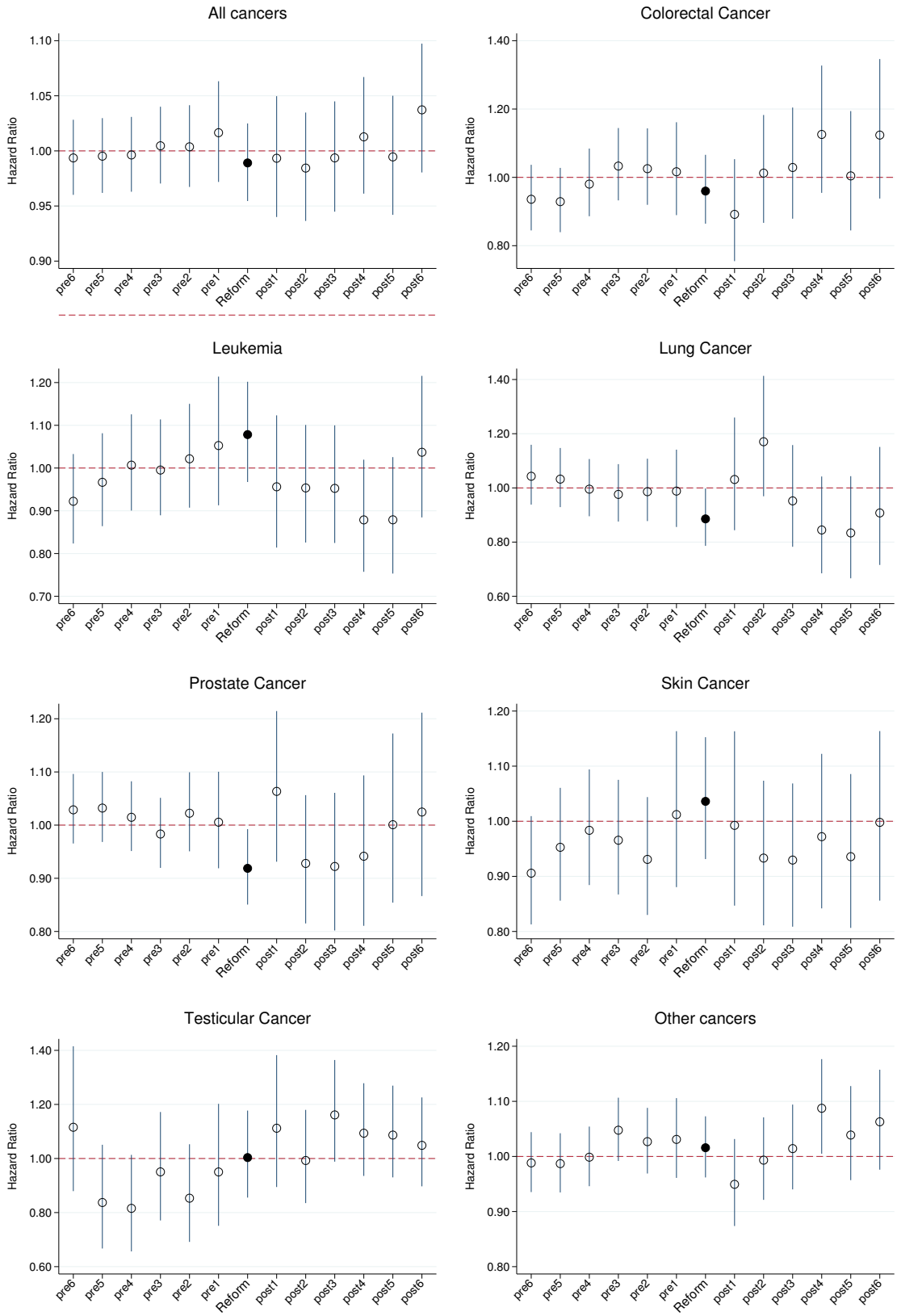


Figure A3. Placebos, Men

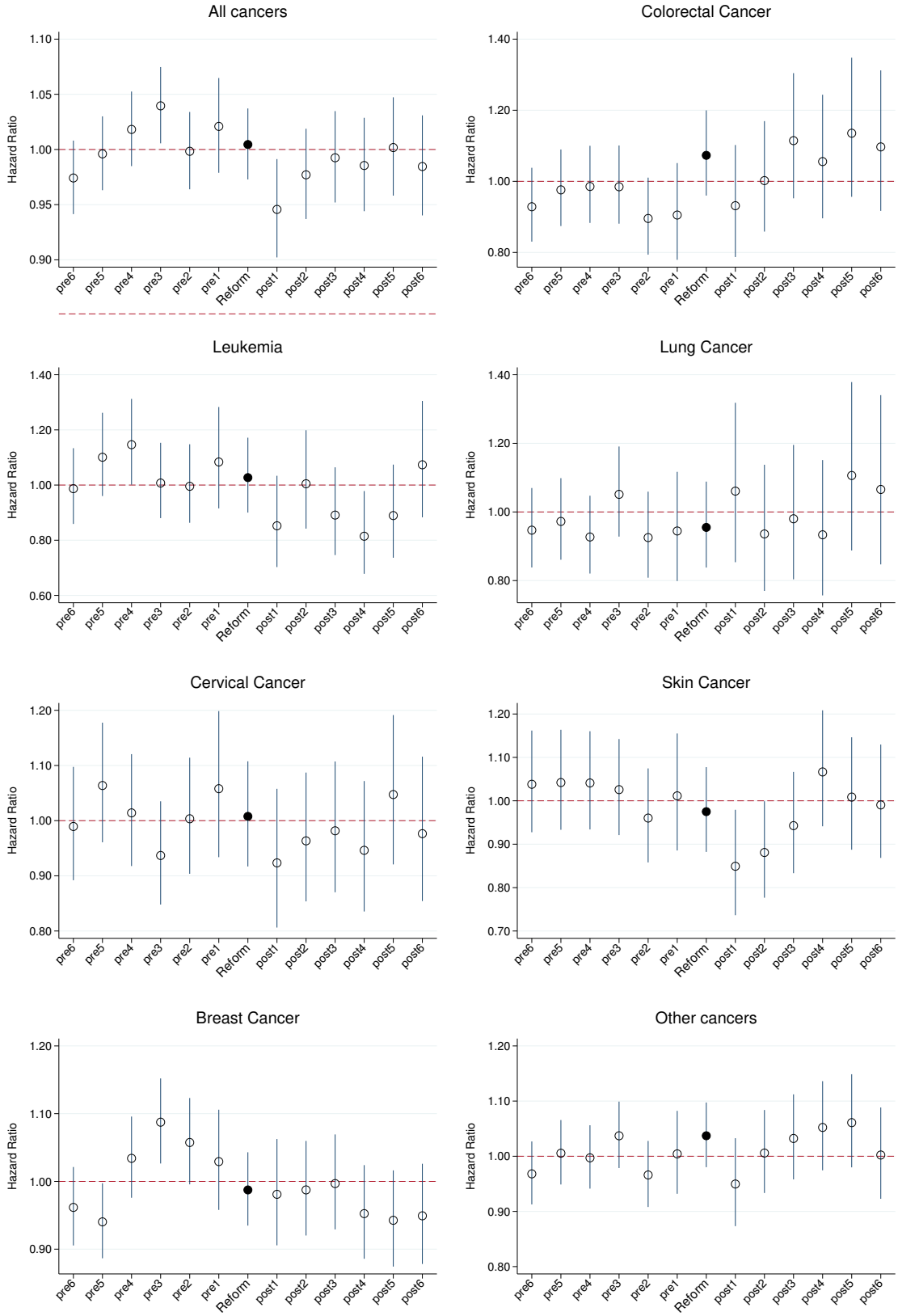


Figure A4. Placebos, Women

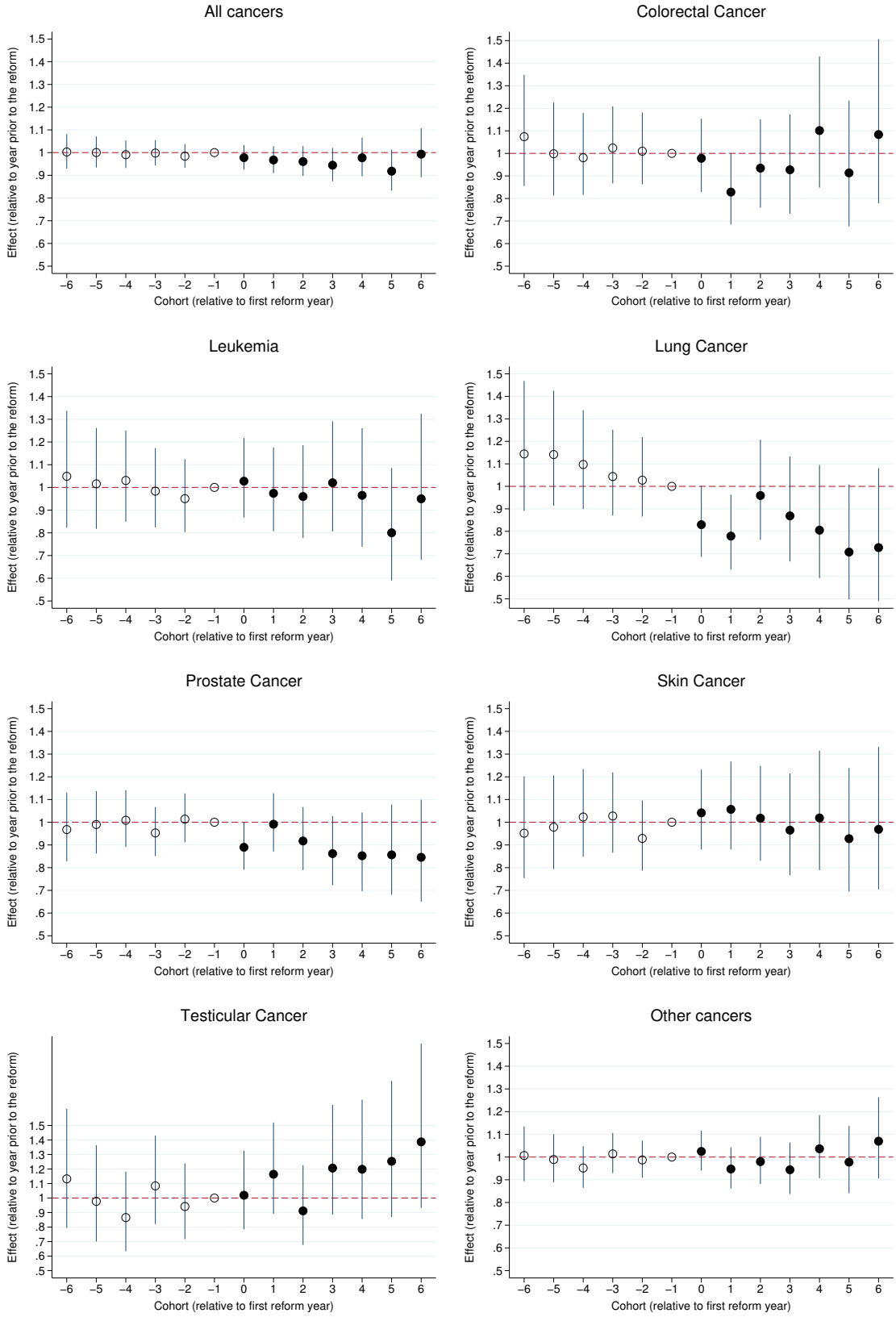


Figure A5. Timing, Men

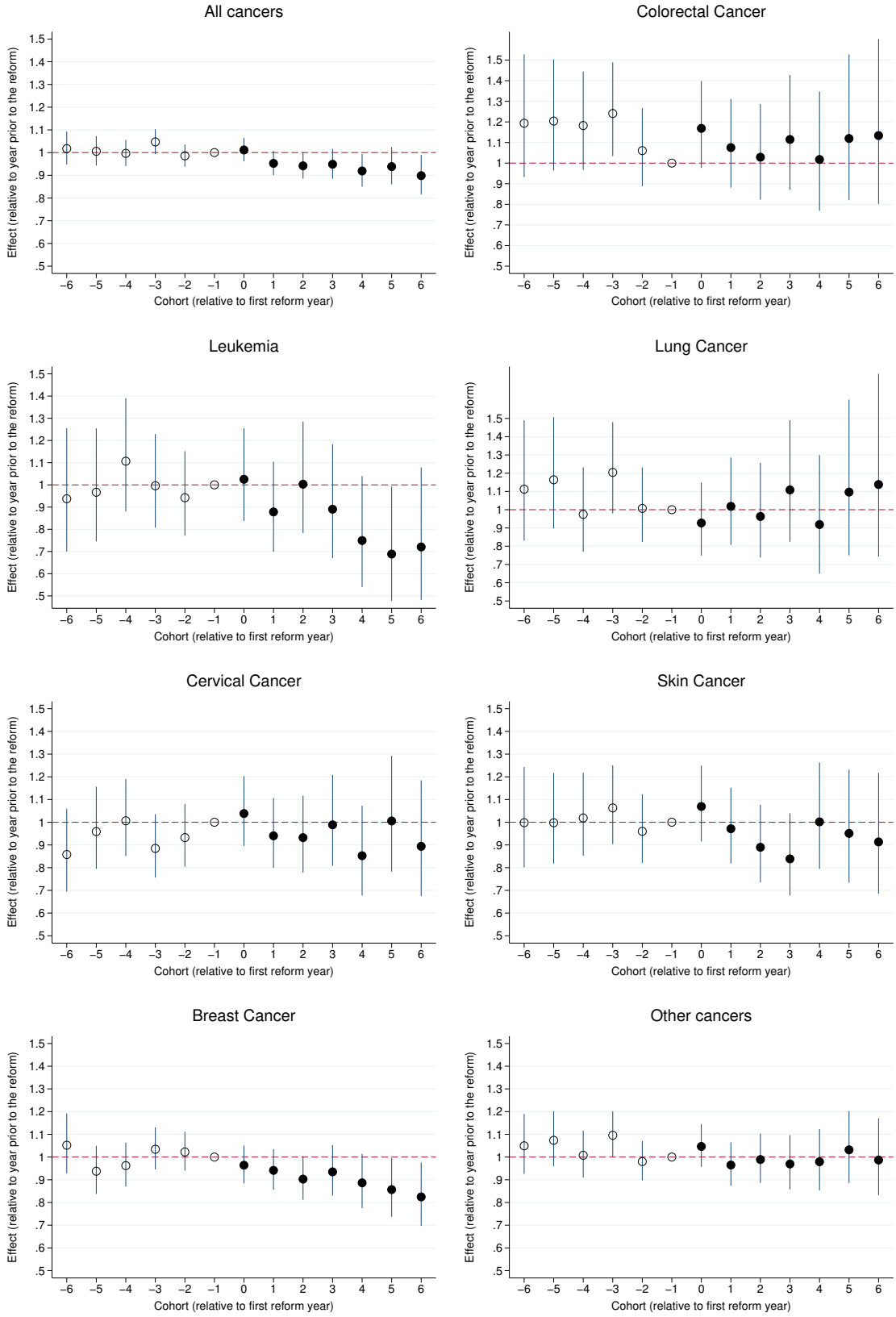
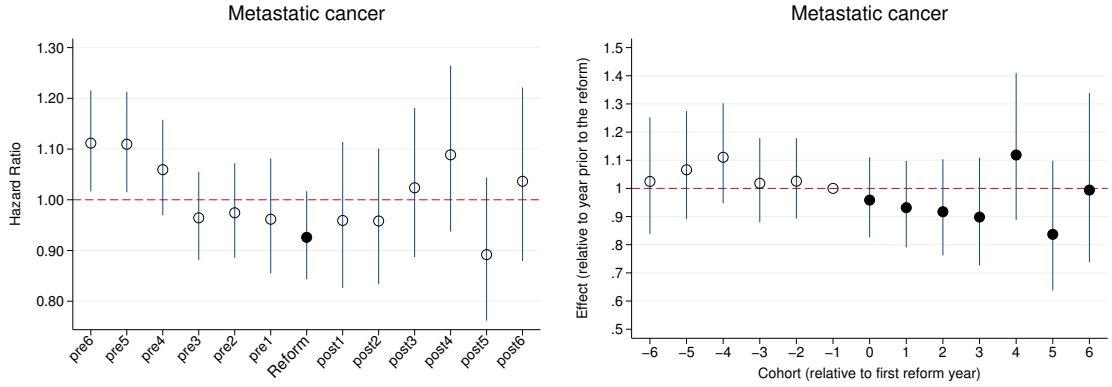


Figure A6. Timing, Women

A. Men



B. Women

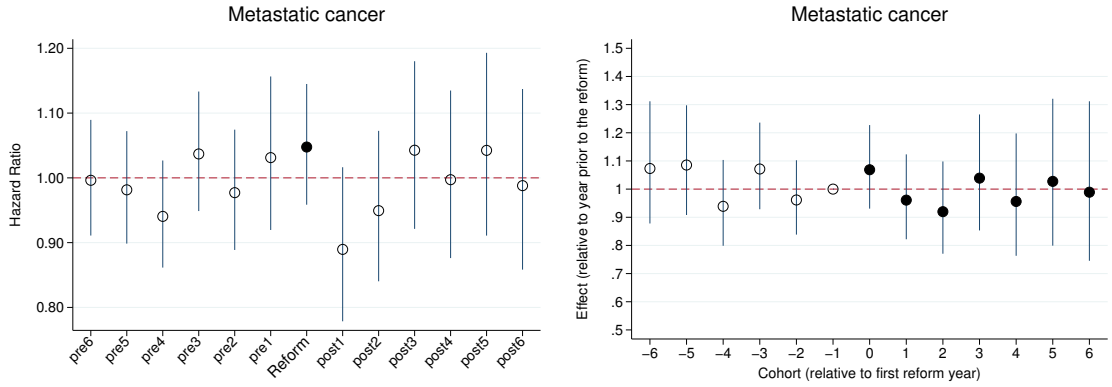
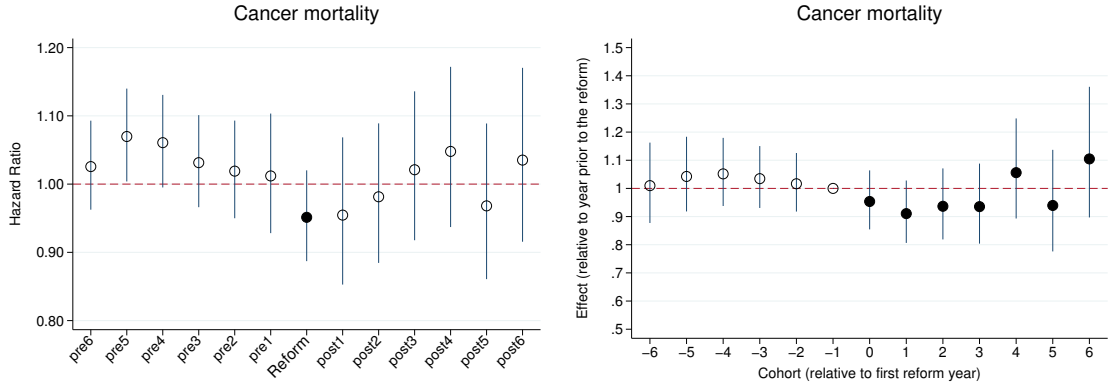


Figure A7. Placebo and Event graphs – Metastatic Cancer, By gender

A. Men



B. Women

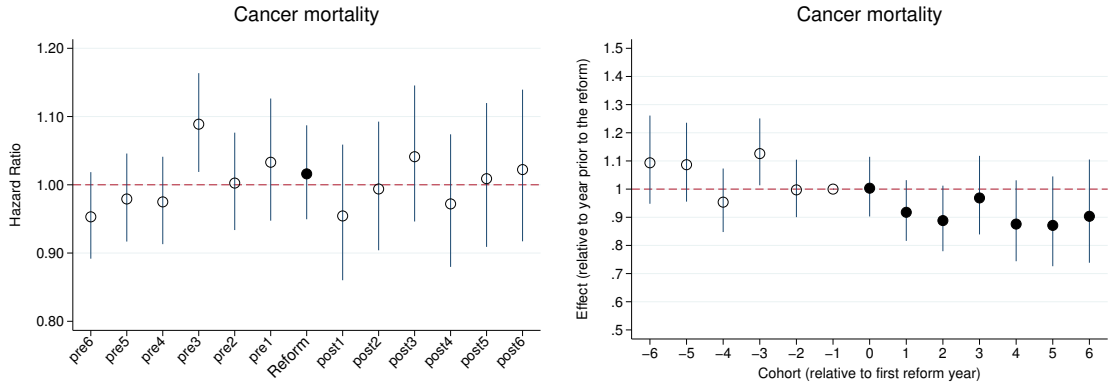


Figure A8. Placebo and Event graphs – Cancer Mortality, By gender