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Using panel econometric methods to estimate the effect of milk consumption on the mortality rate of prostate and ovarian cancer

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Working Paper Series: Business and Law

Working Paper No. 03

Using Panel Econometric Methods to Estimate the Effect of Milk Consumption on the Mortality Rate of Prostate and Ovarian Cancer

Tobias Hagen und Stefanie Waldeck

June 2014

Zusammenfassung

1. Einleitung

Die Harvard School of Public Health (Havard University) schreibt in Bezug auf die Frage, ob man seinen Kalziumbedarf über Milchprodukte decken sollte, dass der Konsum von Milchprodukten *wahrscheinlich* das Risiko von Prostatakrebs und *möglicherweise* das Risiko für Eierstockkrebs und erhöht. Neben zahlreichen Studien basierend auf Individualdaten (Befragungen), gibt es zu dem Thema drei Studien, die sich aggregierter Länderdaten bedienen (siehe die Übersicht im Appendix zum Paper). Dabei werden (Neuerkrankungs- oder) Mortalitätsraten von möglichst vielen Ländern verglichen und in Beziehung zu den durchschnittlichen Ernährungsgewohnheiten dieser Länder gesetzt. Bei aller Ungenauigkeit haben die zugrundeliegenden Daten den Vorteil, dass diese frei für jedermann bei der World Health Organization (WHO) sowie der Food and Agriculture Organization of the United Nations (FAO) verfügbar sind, und somit einem wissenschaftlichen Diskurs nichts im Wege steht. Zudem sind diese nicht von Messfehler betroffen, die bei Individualdaten dadurch entstehen, dass die Befragten unbewusst oder bewusst falsche Angaben in Bezug auf ihre Ernährung machen.

Allerdings sind die Kritikpunkte an solchen Querschnitts-Betrachtungen offensichtlich: Unterschiede in den Krebs-Mortalitätsraten zwischen Ländern hängen von sehr vielen Faktoren ab, so dass man bei so ermittelten Korrelationen wenig bis nichts bzgl. kausaler Effekte der Ernährung lernt. Auch wenn man im Rahmen einfacher Querschnitts-Regressionenanalysen für ein paar Variablen (wie das Durchschnittseinkommen, andere Nahrungsmittel oder die Sonneneinstrahlung) kontrolliert, werden jedem Kritiker immer weitere Faktoren einfallen, die der Forscher nicht berücksichtigt hat bzw. nicht berücksichtigen konnte (*confounding factors*).

Insofern versucht unsere im nachfolgenden vorgestellte Studie einen Beitrag zur Ermittlung des kausalen Effekts von Milchkonsum auf das Ausmaß der Mortalität infolge von Krebs zu leisten. Dabei bedienen wir uns ebenfalls der frei verfügbaren Länderdaten der WHO und der FAO. Anders als die bisherigen (uns bekannten) Studien versuchen wir jedoch nicht, den Effekt der Ernährung durch einen Vergleich zwischen den Ländern zu ermitteln, sondern durch einen Vergleich über die Zeit. Mittels aktueller panelökonometrischer Methoden, soll der Effekt der Ernährung auf die Mortalität aufgrund von Krebs dadurch identifiziert werden, dass getestet wird, ob eine Veränderung der Ernährung über die Zeit innerhalb der Länder mit einer Veränderung der Mortalitätsraten in den Länder verbunden ist. Unsere Analyse basiert dabei auf bis zu 50 Ländern und deren jährlichen Mortalitätsraten aufgrund von Prostatakrebs und Eierstockkrebs im Zeitraum 1990 bis 2008 sowie jährlichen Ernährungsdaten dieser Länder von 1961 bis 2008.

2. Daten und Definitionen

Die **Mortalitätsrate** aufgrund von Prostatakrebs bzw. Eierstockkrebs gibt die Todesfälle innerhalb eines Jahres pro 100.000 Einwohner an. Um solche Mortalitätsraten international vergleichen zu können, werden diese altersstandardisiert, um die unterschiedliche Altersstruktur in den Ländern zu bereinigen. Milch bedeutet hier Milch plus alle Milchprodukte jedoch ohne Butter. Butter wird in der Definition der FAO unter tierischem Fett geführt.

Außerdem ist es wichtig, zu verstehen, was mit **Effekt des Milchkonsums** gemeint ist. Wenn ein Land bspw. seinen jährlichen pro-Kopf Milchkonsum (unter gleichen sonstigen Ernährungsgewohnheiten) um 10 Kilogramm erhöht, dann bedeutet dies, dass

1. die **Gesamtkalorienaufnahme** in dem Land steigt. Es ist denkbar und wahrscheinlich, dass die Summe der eingenommenen Kalorien (unabhängig von der Quelle dieser Kalorien) einen eigenen Effekt auf die Mortalitätsrate hat.
2. der **Anteil der Milch an der gesamten Kalorienaufnahme** steigt. Dies ist der eigentlich interessierende Effekt: Welchen Effekt hat es – bei gegebener Gesamtkalorienzufuhr – wenn man einen höheren Anteil seines Kalorienbedarfs durch Milchprodukte deckt?

Wir sind an dem zweiten Effekt interessiert. Deshalb gehen alle Nahrungsvariablen als prozentualer Anteil an der gesamten pro-Kopf-Kalorienzufuhr der Länder ein.

Zudem wird die Mortalitätsrate im Jahr *t* (bspw. 2006) im Land *i* durch den durchschnittlichen jährlichen Milchkonsum (in Prozent der gesamten Kalorienaufnahme) in den Jahren *t*-24 bis *t* (also 1982 bis 2006) des selben Landes *i* erklärt, um zu berücksichtigen, dass längerfristige Ernährungsgewohnheiten relevant sein dürften.

3. Deskriptive Analysen

In Table 1 des Papers wird ein Überblick über die Daten gegeben. Die **Tabelle 1** ist ein Auszug davon. Für alle Länder wird jeweils das erste und das letzte Jahr der Stichprobe dargestellt. Bereits an dieser Stelle ist es möglich einige Regelmäßigkeiten zu identifizieren:

- In Ländern, in denen der Milch-Konsum sehr gering ist bzw. war (<3%), ist die Mortalitätsrate beider Arten von Krebs relativ gering (siehe Ägypten, Südkorea, Philippinen, Sri Lanka, Thailand).
- In Ländern, in denen der Milch-Konsum sehr hoch ist bzw. war (>11 %), ist die Sterblichkeit an Prostatakrebs deutlich überdurchschnittlich (siehe Australien, Finnland, Irland, Niederlande, Norwegen, Schweden, Schweiz). In geringerem Maße zeigt sich dieser Zusammenhang auch für die Mortalität aufgrund von Eierstockkrebs.

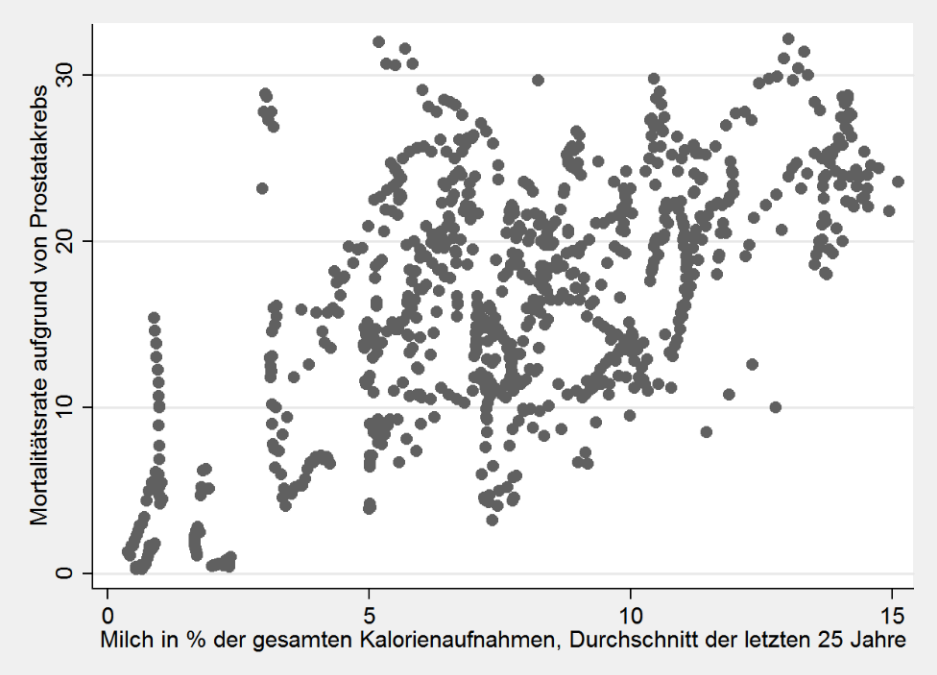
Tabelle 1: Variablen der ökonometrischen Analysen – Erstes und Letztes Jahr der Stichprobe – **Auszug aus Table 1 des Papers**

		Alters-standardisierte **Mortalitätsrate** aufgrund von **Prostatakrebs** pro 100.000 Einwohner	Alters-standardisierte **Mortalitätsrate** aufgrund von **Eierstockkrebs** pro 100.000 Einwohner	Tägliche **Kalorienaufnahme pro Person**, Durchschnitt der vorherigen 25 Jahre	Anteil **Milchprodukte** an der gesamten Kalorienaufnahmen in %, Durchschnitt der vorherigen 25 Jahre	Anteil von **Zucker** an der gesamten Kalorienaufnahmen in %, Durchschnitt der vorherigen 25 Jahre
		(1)	(2)	(3)	(4)	(5)
Australien	1990	23,0	6,7	3.123	11,2	16,5
	2006	18,4	4,6	3.098	11,1	14,5
Österreich	1990	22,4	9,1	3.303	9,9	12,3
	2008	16,5	5,8	3.618	9,0	12,0
Ägypten	1990	1,1	0,2	2.694	1,7	8,4
	2008	5,1	0,9	3.227	1,9	9,0
Finnland	1990	23,6	7,5	3.091	15,1	13,2
	2008	18,0	5,3	3.119	13,7	11,6
Frank-reich	1990	22,3	6,4	3.375	10,8	10,8
	2008	15,2	5,3	3.558	10,9	10,3
Deutsch-land	1990	20,5	8,4	3.272	7,6	12,3
	2008	15,3	5,8	3.436	8,4	12,6
Griechen-land	1990	10,6	3,4	3.317	9,1	8,6
	2008	12,9	4,5	3.579	10,3	8,9
Irland	2001	24,4	9,6	3.599	13,1	11,9
	2008	19,1	8,2	3.599	12,2	11,0
(Süd-) Korea	1990	1,3	1,1	2.950	0,4	4,6
	2006	6,0	2,4	3.030	1,0	9,3
Nieder-lande	1990	24,0	8,7	3.150	14,1	14,5
	2008	18,6	6,7	3.231	13,5	14,4
Norwegen	1990	27,9	8,2	3.139	13,6	13,1
	2008	25,5	7,5	3.315	11,0	12,7
Philippi-nen	1992	4,5	2,1	2.101	1,1	11,0
	2008	15,4	4,2	2.335	0,9	11,2
Polen	1991	12,4	7,4	3.453	10,2	12,7
	2008	16,4	7,8	3.384	8,3	12,9
Portugal	1990	17,9	3,7	2.998	4,5	8,6
	2008	17,8	3,4	3.421	6,6	8,4
Sri Lanka	1990	0,5	0,7	2.242	2,0	8,1
	2006	1,0	1,5	2.279	2,4	10,8
Sweden	1990	26,9	8,2	2.933	14,1	15,0
	2008	24,7	6,5	3.061	13,8	14,0
Schweiz	1995	24,8	6,5	3.409	11,9	13,5
	2007	18,0	5,4	3.386	11,7	14,7
Thailand	1990	0,3	0,2	2.060	0,5	5,8
	2006	1,8	1,3	2.306	0,9	9,5
Groß-britannien	1990	21,1	9,6	3.186	10,7	13,8
	2008	17,6	7,2	3.314	10,4	11,5
USA	1990	22,2	7,0	3.189	11,8	17,7
	2007	13,3	5,9	3.613	10,8	17,0

Anmerkungen: Dies ist ein Auszug aus Table 1 des Papers.
Durchschnitt der jährlichen Mortalitätsrate aufgrund von Prostatakrebs in dieser Stichprobe: 16,96
Durchschnitt der Mortalitätsrate aufgrund von Eierstockkrebs in dieser Stichprobe: 5,06

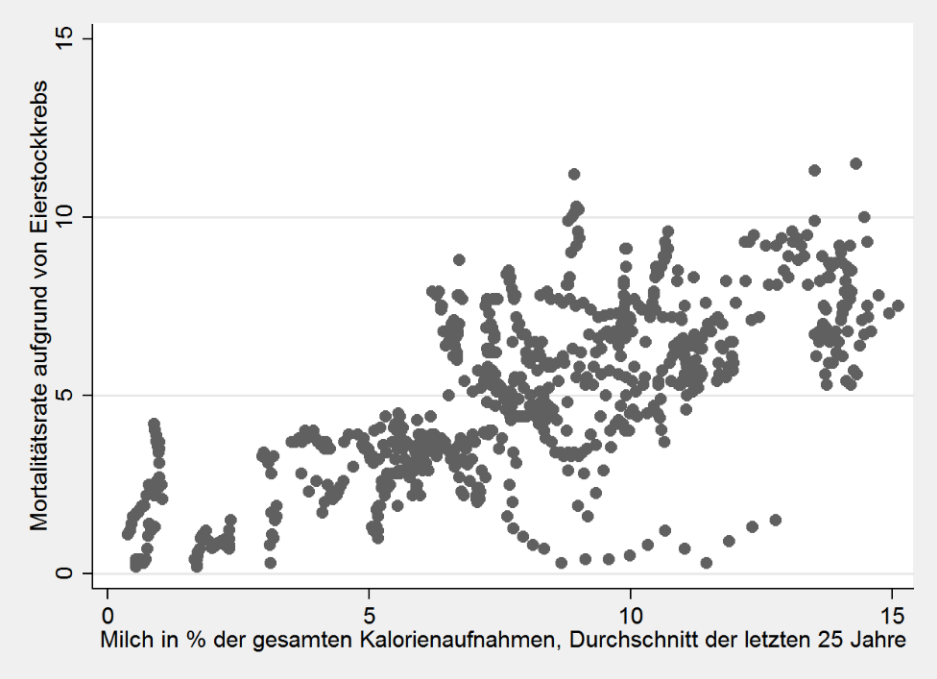
- In 26 der 49 Ländern in Table 1 des Papers steigt die Mortalitätsraten aufgrund von Prostatakrebs ab dem ersten Jahr bis zum letzten Jahr der Stichprobe. 17 von diesen 26 Ländern haben ihren Milchverbrauch erhöht – gemessen als Änderung der Werte in Spalte (4). In 3 dieser 26 Länder blieb der Milchverbrauch konstant und in 7 Ländern ist der Milchkonsum gesunken.
- In 23 der 49 Länder in Table 1 des Papers steigen die Mortalitätsraten aufgrund von Eierstock-Krebs ab dem ersten Jahr bis zum letzten Jahr der Stichprobe. In 14 von diesen 23 Ländern stieg der Milchverbrauch an, in 2 Ländern blieb der Milchkonsum konstant und in 7 Ländern sank der Milchkonsum.

Grafik 1 zeigt die Mortalitätsrate aufgrund von Prostatakrebs in den Jahren 1990 bis 2008 auf der Y-Achse und den durchschnittlichem Anteil der Kalorienzufuhr, der in den jeweiligen 25 vorangegangenen Jahren durch Milch gedeckt wurde, auf der X-Achse. Hier entspricht ein Punkt einem bestimmten Land in einem bestimmten Jahr. Der Korrelationskoeffizient zeigt mit 0,6423 eine mäßig hohe Korrelation. Die analoge Gegenüberstellung für Eierstockkrebs ist in Grafik 2 zu finden, wo der Korrelationskoeffizient mit 0,7259 sogar noch höher ist.



Korrelationskoeffizient (*p*-Wert): 0,6423 (0,000), Anzahl der Beobachtungen: 872

Grafik 1: Altersstandardisierte Mortalitätsrate aufgrund von **Prostatakrebs** 1990-2008 und Anteil von Milchprodukten an der Kalorienaufnahme in %

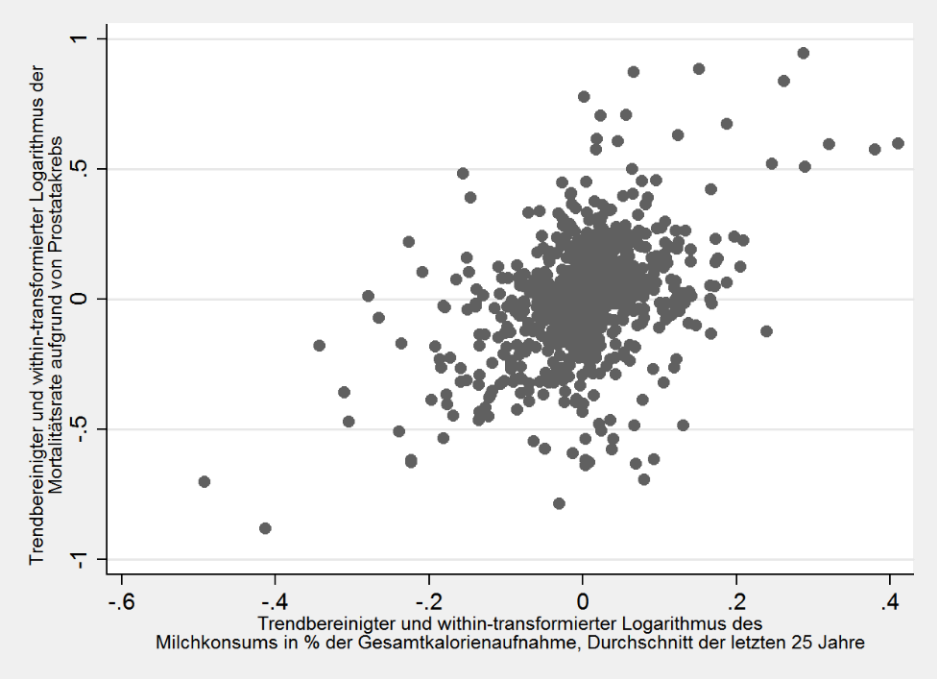


Korrelationskoeffizient (*p*-Wert): 0,7259 (0,000), Anzahl der Beobachtungen: 777

Grafik 2: Altersstandardisierte Mortalitätsrate aufgrund von **Eierstockkrebs** 1990-2008 und Anteil von Milchprodukten an der Kalorienaufnahme in %

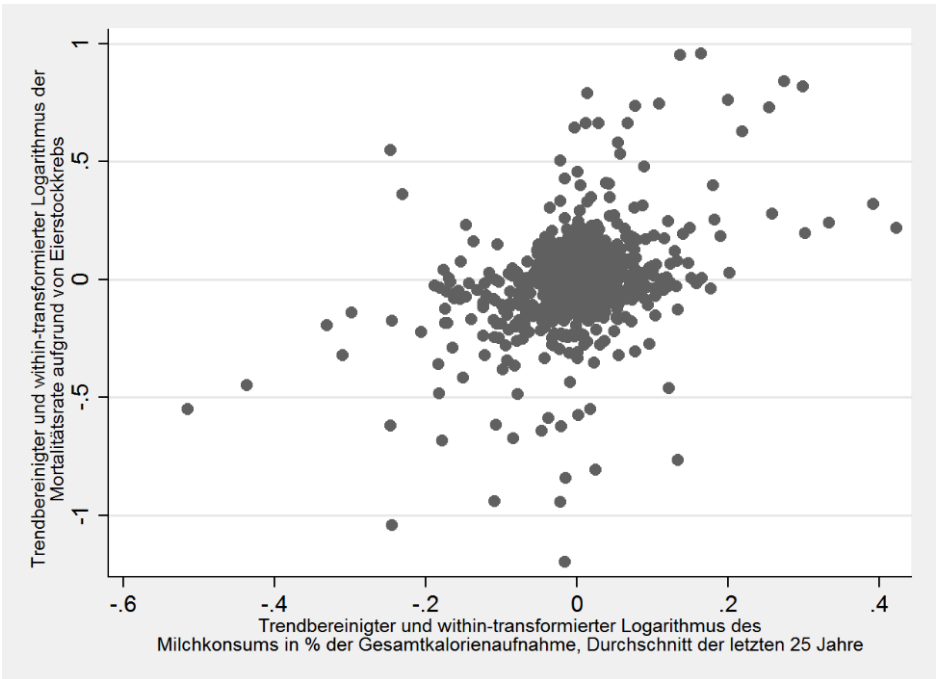
Diese Korrelationskoeffizienten beinhalten sowohl den Zusammenhang der beiden Variablen (Mortalitätsrate versus Milchkonsum) zwischen den Ländern als auch über die Zeit. Grafik 1 und Grafik 2 sind demnach Beispiele für den Versuch, den Effekt des Milchkonsums auch dadurch zu identifizieren, dass man Länder untereinander vergleicht. Wie in der Einleitung dargestellt, wird man aber bei dieser Strategie niemals plausibel machen können, dass man alle *"confounding factors*" (also mögliche Einflussfaktoren) kontrolliert hat. Deshalb wird hier – wie in der Einleitung dargestellt – der Effekt dadurch identifiziert, dass die Veränderungen der Variablen über die Zeit betrachtet werden, und analysiert wird, ob diese Veränderungen über die Zeit korreliert sind.

Dazu werden zunächst alle Variablen „natürlich logarithmiert“, da dies zu einer deutlich besseren Anpassung der Modelle an die Daten führt. Um nun weg vom Vergleich zwischen den Ländern, hin zu einer Analyse der zeitlichen Veränderungen zu kommen, werden alle Variablen „within“-transformiert, d.h. für jede Variable und jedes Land wird der Mittelwert über die Zeit subtrahiert. Darüber hinaus werden alle Variablen trendbereinigt, indem für jedes Jahr der Mittelwert aller Länder subtrahiert wird. Damit wird berücksichtigt, dass es über alle Länder hinweg zeitliche Trends, wie bspw. einen medizinischen Fortschritt, gibt.



Korrelationskoeffizient (*p*-Wert): 0,4405 (0,000), Anzahl der Beobachtungen: 872

Grafik 3: Trendbereinigte und within-transformierter Logarithmus der Mortalitätsrate aufgrund von **Prostatakrebs** 1990-2008 und Trendbereinigter und within-transformierter Logarithmus des Anteils von Milchprodukten an der Kalorienaufnahme in %



Korrelationskoeffizient (*p*-Wert): 0,3834 (0,000), Anzahl der Beobachtungen: 777

Grafik 4: Trendbereinigte und within-transformierter Logarithmus der Mortalitätsrate aufgrund von **Eierstockkrebs** 1990-2008 und Trendbereinigter und within-transformierter Logarithmus des Anteils von Milchprodukten an der Kalorienaufnahme in %

Das Ergebnis der Gegenüberstellung der so transformierten Variablen ist in Grafik 3 für Prostatakrebs sowie in Grafik 4 für Eierstockkrebs zu sehen. Jeder

Punkt kann nun als trendbereinigte prozentuale Änderung im Vergleich zum Mittelwert des entsprechende Landes interpretiert werden. So kann bspw. der am weitesten rechts liegende Punkt in Grafik 3 so interpretiert werden, dass in dem Land eine Steigerung der Milchkonsums um 40% im Vergleich zum mittleren Milchkonsum dieses Landes mit einer Steigerung des Mortalitätsrate um 50% im Vergleich zu mittleren Mortalitätsrate des Landes verbunden war. Wenn auch schwächer, bleibt die signifikant positive Korrelation erhalten (siehe Anmerkungen zu den Grafiken). Dies bedeutet, dass der Zusammenhang erhalten bleibt, auch wenn man nur die Korrelation der zeitlichen Veränderungen betrachtet und einen gemeinsamen Zeittrend eliminiert.

4. Panelökonometrische Methoden

Die deskriptiven Analysen lassen noch keine Aussage bzgl. der Kausalität zu. Um weitere Einflussfaktoren auf die Mortalitätsrate (aufgrund von Prostatakrebs oder Eierstockkrebs) zu kontrollieren, wird folgendes Paneldaten-Regressionsmodell spezifiziert, bei dem die Mortalitätsrate *mit* in Land i =1,..., N (49) und Jahr t (= 1990,...,2008) durch das Durchschnittseinkommen des Landes im Vorjahr (*GDPit*-1), die gesamte pro-Kopf-Kalorienaufnahme (*total*) sowie die Anteile verschiedener Lebensmittel an der gesamten Kalorienaufnahme (*pmilk, psugar* etc.) erklärt wird:

$$\ln(m_{it}) = \rho \ln(m_{it-1}) + \alpha_1 \ln(GDP_{it-1}) + \alpha_2 \ln(\overline{total}_{it}) + \beta_1 \ln(\overline{pmilk}_{it}) + \beta_2 \ln(\overline{psugar}_{it}) + ... + \lambda_t + c_i + u_{it}$$

Die Nahrungsmittel gehen als gleitende 25-Jahres-Durchschnitte ein, d.h.:

$$\overline{total}_{it} = \frac{1}{25}\sum_{j=0}^{24} total_{i,t-j} \qquad \text{für } t = 1990,...,2008$$

$$\overline{pmilk}_{it} = \frac{1}{25}\sum_{j=0}^{24} pmilk_{i,t-j} = \frac{1}{25}\sum_{j=0}^{24} \frac{milk_{i,t-j}}{total_{i,t-j}} \qquad \text{für } t = 1990,...,2008$$

Die erklärenden Nahrungsvariablen werden zuvor mittels statistischer Methoden aus einer Vielzahl von Variablen ausgewählt. Zentral für die Modelle sind die fixen Länder-spezifischen Effekte (c_i), die für unbeobachtete zeitkonstante Unterschiede zwischen den Ländern (wie bspw. unterschiedliche Gesundheitssysteme, genetische Unterschiede etc.) kontrollieren, sowie die fixen Zeiteffekte (λ_t), die unbeobachtete gemeinsame Zeiteffekte, wie bspw. den medizinischen Fortschritt, modellieren. Die Modellierung von c_i sowie λ_t entspricht der within-transformation sowie der Trend-Bereinigung in der deskriptiven Analyse in Abschnitt 3.

Alle erklärenden Variablen können mit diesen fixen Effekten korreliert sein, ohne dass dadurch die Modelle inkonsistent würden. Das Einbeziehen einer verzögert abhängigen Variable (*mit*-1) führt zu einem dynamischen Modell. Dies

soll den verzerrenden Einfluss möglicherweise ausgelassener Variablen (*confounding factors*) mindern.

5. Determinanten der Mortalitätsrate aufgrund von Prostatakrebs und der Mortalitätsrate aufgrund von Eierstockkrebs

In den folgenden Grafiken werden die Ergebnisse dreier panelökonometrischer Methoden dargestellt.[1] Um welche Art von Methoden es sich handelt wird ebenso wie weitere zentrale Parameter im Paper beschrieben. In den Grafiken werden die geschätzten Koeffizienten mit einem Punkt und die zugehörigen 95%-Konfidenzintervalle mit einer waagerechten Linie dargestellt. Überlappt eine waagerechte 95%-Konfidenzintervall-Linie die senkreche rote Null-Linie, ist der entsprechende geschätzte Koeffizient statistisch nicht von Null verschieden. Die geschätzten Koeffizienten der dynamischen Modelle sind in die hier relevanten langfristigen Effekte (Elastizitäten) umgerechnet.

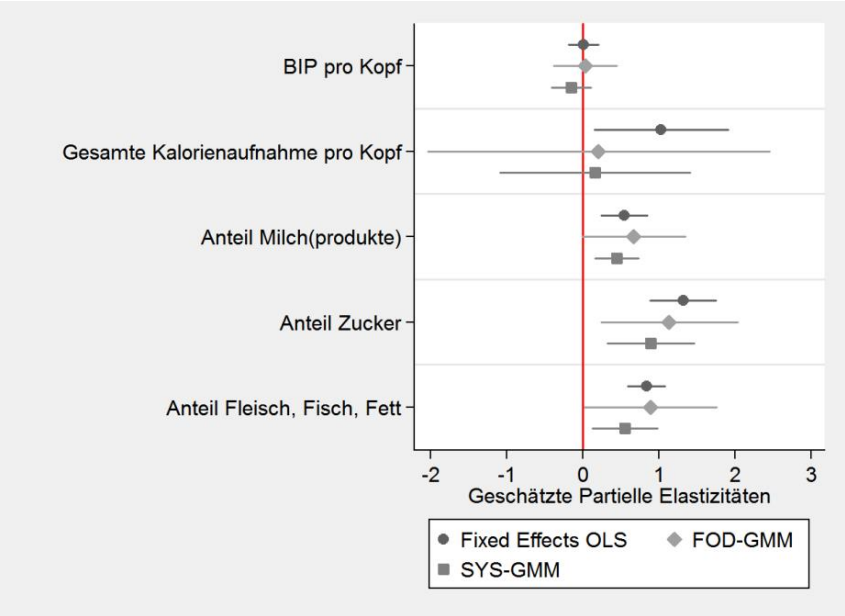
In Grafik 5 erkennt man bei den Ergebnissen aller drei hier vorgestellten Methoden ein ähnliches Bild: Ein Anstieg des Anteils von Milch an der gesamten Kalorienzufuhr innerhalb der vorangegangenen 25 Jahre erhöht statistisch signifikant die Mortalitätsrate aufgrund von Prostatakrebs. Besonders relevant sind zudem die Zuckeraufnahme sowie die Aufnahme anderer tierischer Produkte wie Fleisch, Fisch und Fett. Die Ergebnisse bzgl. der Gesamtkalorienaufnahme sind dagegen nicht eindeutig.

In Grafik 6 erkennt man in Bezug auf die Determinanten der Mortalitätsrate aufgrund von Eierstockkrebs ebenfalls einen eindeutig positiven (d.h. schädlichen) Effekt der Milch. Weitere schädliche Faktoren sind die Gesamtkalorienaufnahme sowie wieder der Anteil von Zucker.

Es ist wichtig zu betonen, dass der geschätzte positive (schädliche) Effekt des Milchkonsums bei einer gegebenen Gesamtkalorienzufuhr gilt. Wenn also ein Land bei einer geschätzten Elastizität von ca. 0,5 bspw. die Milch-Kalorien um 10% erhöht und dafür die Getreide- oder Gemüsekalorien um 10% senkt (beides dauerhaft innerhalb von 25 Jahren), dann erhöht diese nach diesen Ergebnissen die Anzahl von Todesfällen aufgrund von Eierstockkrebs um ca. 5%.

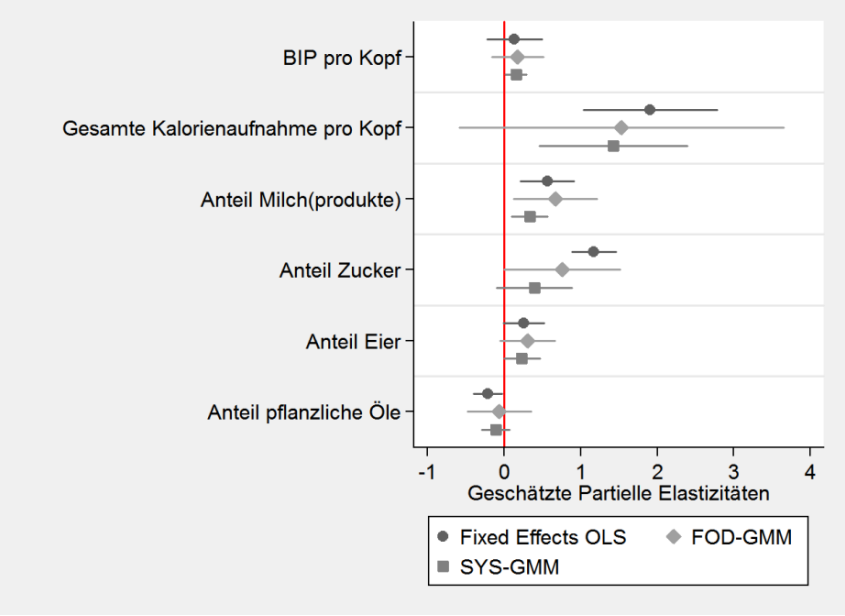
Es ist wahrscheinlich, dass die hier präsentierten Ergebnisse belastbar sind, da verschiedene Methoden (Fixed-Effects-OLS, Fixed-Effects-Quantile-Regression, GMM, SYS-GMM, Instrumentierung der Nahrungsmittelvariablen, corrected LSDV, Extreme Bounds Analysis) für verschieden Unterstichproben (Zeiträume, Ländergruppen) und erklärende Variablen (Gesundheitsausgaben anstatt Durchschnittseinkommen) zu ähnlichen Ergebnissen führen.

[1] Die Grafiken wurden mit der Stata-Routine „*coefplot*“ von Jann (2013) erstellt.



Grafik 5: Determinanten der jährlichen altersstandardisierten Mortalitätsrate aufgrund von **Prostatakrebs** 1990-2008 basierend auf aggregierten Länderdaten; aggregierte Ernährungsdaten 1966-2008.

Fixe Ländereffekte und fixe Zeiteffekte (Jahres-Dummy-Variablen) sind im Modell (implizit) enthalten, hier aber nicht dargestellt. Fixed Effects OLS: Spalte (5) von Table 7 im Paper. FOD-GMM: Spalte (3) von Table 8 im Paper. SYS-GMM: Spalte (5) von Table 8 im Paper**.**



Grafik 6: Determinanten der jährlichen altersstandardisierten Mortalitätsrate aufgrund von **Eierstockkrebs** 1990-2008 basierend auf aggregierten Länderdaten; aggregierte Ernährungsdaten 1966-2008.

Fixe Ländereffekte und fixe Zeiteffekte (Jahres-Dummy-Variablen) sind im Modell (implizit) enthalten, hier aber nicht dargestellt. Fixed Effects OLS: Spalte (7) von Table 15 im Paper. FOD-GMM: Spalte (3) von Table 16 im Paper. SYS-GMM: Spalte (5) von Table 16 im Paper

6. Simulationen zur Illustration der Ergebnisse

Um besser verstehen zu können, was die Ergebnisse nun *quantitativ* bedeuten, werden einige Simulationen durchgeführt. Dabei behaupten wir nicht, dass unsere Modelle für exakte Vorhersagen geeignet wären, sondern wir wollen die Ergebnisse besser verständlich machen.

Konkret versuchen wir, die folgende Frage zu beantworten: *Wie hoch wären die Mortalitätsraten zwischen 1991 und 2008 gewesen, wenn die Einwohner aller Länder weniger Milchprodukte konsumiert hätten*? „Weniger" bedeutet hier, dass die gegebene Kalorienzufuhr zu einem geringeren Anteil durch Milchprodukte gedeckt worden wäre und zu einem höheren Anteil durch pflanzliche Nahrungsmittel. Die Simulationen basieren immer auf der sog. Ceteris Paribus Klausel, d.h. alle anderen Variablen, wie das Durchschnittseinkommen, die Gesamtkalorienaufnahme, die fixen Ländereffekte (die Unterschiede in den Gesundheitssystemen, Genen etc. kontrollieren sollen) sowie die fixen Zeiteffekte (der gemeinsame Trend bzgl. des medizinischen Fortschritts) bleiben unverändert.

Tabelle 2 zeigt für drei unterschiedliche Szenarien die Simulationsergebnisse für Prostatakrebs. Im ersten Szenario gehen wir davon aus, dass der Anteil der Milch an der gesamten Kalorienaufnahme um ein Viertel reduziert wird. Auf der Grundlage des Models 1 sowie des beobachteten Mittelwerts der Stichprobe von 7,8 % (Kalorien aus Milch an der gesamten Kalorienaufnahme), führt eine Verringerung des Anteils der Milch an der Gesamtkalorienaufnahme um ein Viertel auf 5,85 % zu einer Reduktion der durchschnittlichen jährlichen Mortalitätsrate von 17,4 auf 16,0 pro 100.000 Einwohner. Diese jährliche Reduktion der Verluste von Menschenleben in Höhe von 1,4 pro 100.000 Einwohner entspricht einem Rückgang um 8%. Das Modell 2 prognostiziert dagegen nur einen Rückgang der Mortalitätsrate um 3%.

Im zweiten Szenario in Tabelle 2 wird davon ausgegangen, dass der Milchverbrauch um 50 % sinkt und damit Milch nur noch 3,9% (= 7,8% / 2) der gesamten Kalorienaufnahme ausmacht. Gemäß der Vorhersage unserer Modelle wird die Anzahl der Männer, die jährlich infolge von Prostatakrebs sterben, um 2,4 (Model 1) oder 3,9 (Model 2) pro 100.000 Einwohner reduziert. Dies entspricht einem Rückgang von 14% bzw. 23%.

Beim dritten Szenario in Tabelle 2 wird davon ausgegangen, dass alle Länder ihren Milchverbrauch auf 1 % der gesamten Kalorienaufnahme reduzieren. Dies ist etwa das Niveau, das für Thailand zu beobachten ist (siehe Tabelle 1). Der durchschnittliche Anteil der Milch in der Stichprobe ist 7,8 %. In Ländern wie den Niederlanden, Schweden, Finnland und Albanien ist der Anteil der Milch an der gesamten Kalorienzufuhr höher als 13% (siehe Tabelle 1). In der Schweiz, Irland und Rumänien sind es mehr als 11%. Tabelle 2 zeigt, dass diese deutliche Reduktion des Milchverbrauchs auf 1% der gesamten Kalorienzufuhr, die Zahl der Todesfälle aufgrund von Prostatakrebs um 30% bis 65% reduzieren würde.

Tabelle 2: Simulationen der jährlichen Mortalitätsrate aufgrund von **Prostatakrebs** 1991-2008 basierend auf zwei unterschiedlichen Modellen

	Beobachtete Mittelwerte in der Stichprobe		**Simulierte Mittelwerte basierend auf zwei Modellen**					
			Model 1: SYS-GMM FOD Table 8, Col(5)			**Model 2: Fixed Effects OLS** Table 7, Col (5)		
	Anteil der Milch	*Mortalitäts-rate*	*Mortalitäts-rate*	*Absolute Veränderung*	*Relative Veränderung*	*Mortalitäts-rate*	*Absolute Veränderung*	*Relative Veränderung*
Szenario	(1)	(2)	(3)	(4) =(3)-(2)	(5) =(4)/(2)	(6)	(7) =(5)-(1)	(8) =(6)/(1)
1. Reduktion Milch um 1/4	7.8%	17.4	16.0	*-1.4*	*-8%*	16.8	*-0.6*	*-3%*
2. Halbierung Milch	7.8%	17.4	15.1	*-2.4*	*-14%*	13.5	*-3.9*	*-23%*
3. Reduktion Milch auf 1% der Kalorien-aufnahme	7.8%	17.4	12.3	*-5.2*	*-30%*	6.1	*-11.3*	*-65%*

Tabelle 3 zeigt die analogen Simulationsergebnisse für die Sterblichkeit aufgrund von Eierstockkrebs. Zunächst einmal ist es wichtig zu beachten, dass der mittlere Anteil der Milch in Gesamtkalorien (7.9%) nicht identisch ist zu dem in Tabelle 2 (7.8%), da die Stichproben nicht deckungsgleich sind. Zweitens ist das Ausgangsniveau der Mortalitätsrate aufgrund von Eierstockkrebs mit jährlich 4,9 pro 100.000 Einwohner deutlich niedriger. Allerdings sind die *relativen Veränderungen* in der Mortalität infolge des verringerten Milchkonsums vergleichbar mit denen für Prostatakrebs.

Beispielsweise zeigt das dritte Szenario (Senkung der Kalorieneinnahme in Form von Milch auf 1%), dass damit jährlich 1,8 bis 3,4 Frauen pro 100.000 Einwohner weniger an Eierstockkrebs sterben würden. Diese absoluten Zahlen entsprechen einem jährlichen Rückgang von 30 % bis 65 %.

Tabelle 3: Simulationen der jährlichen Mortalitätsrate aufgrund von **Eierstockkrebs** 1991-2008 basierend auf zwei unterschiedlichen Modellen

	Beobachtete Mittelwerte in der Stichprobe		**Simulierte Mittelwerte basierend auf zwei Modellen**					
			Model 1: SYS-GMM FOD Table, Col(5)			**Model 2: Fixed Effects OLS** Table 15, Col (6)		
	Anteil der Milch	*Mortalitäts-rate*	*Mortalitäts-rate*	*Absolute Veränderung*	*Relative Veränderung*	*Mortalitäts-rate*	*Absolute Veränderung*	*Relative Veränderung*
Szenario	(1)	(2)	(3)	(4) =(3)-(2)	(5) =(4)/(2)	(6)	(7) =(5)-(1)	(8) =(6)/(1)
1. Reduktion Milch um 1/4	7.9%	4.9	4.7	-0.2	*-5%*	4.5	-0.4	*-9%*
2. Halbierung Milch	7.9%	4.9	4.3	-0.6	*-13%*	3.6	-1.4	*-27%*
3. Reduktion Milch auf 1% der Kalorien-aufnahme	7.9%	4.9	3.1	-1.8	*-37%*	1.6	-3.4	*-68%*

7. Fazit und Diskussion

Die bisherigen epidemiologischen Studien basierend auf Länderdaten nutzen nur Querschnittsanalysen und sind daher (nicht nur) aus der Sicht eines Ökonometrikers nicht geeignet, glaubhaft den kausalen Effekt von Milchkonsum auf Krebs zu identifizieren.

In den letzten 25 Jahren gab es im Bereich panelökonometrischer Methoden erhebliche Fortschritte. In unserer Studie werden diese Methoden auf diese Frage und auf Daten von bis zu 50 Ländern angewandt. Es wird die Mortalitätsrate aufgrund von Prostatakrebs und Eierstockkrebs der Jahre 1990 bis 2008 durch die durchschnittliche Ernährung in den Ländern in den Jahren 1966 bis 2008 sowie weiteren Einflussfaktoren erklärt.

In dieser Studie zeigt sich relativ deutlich, dass sich der im Querschnitt von Ländern gefundene positive Zusammenhang zwischen Milchkonsum und Sterblichkeit aufgrund von Krebs auch mittels Paneldaten robust nachweisen lässt, wobei hier die Identifikation des Effektes auf der Variation der Variablen über die Zeit basiert.

Um besser verdeutlichen zu können, was die Ergebnisse quantitativ bedeuten, wurden **Simulationen** durchgeführt. Dabei behaupten wir nicht, dass unsere Modelle für exakte Vorhersagen geeignet wären, sondern wir wollen die Ergebnisse besser dokumentieren.

- Eine nur mäßige Reduktion des Konsums von Milchprodukten um 25% (bei äquivalenter Erhöhung pflanzlicher Nahrungsmittel) würde die Todesfälle aufgrund beider Arten von Krebs um weniger als 10% senken.
- Eine Halbierung des Konsums von Milchprodukten (bei äquivalenter Erhöhung pflanzlicher Nahrungsmittel) würde die Todesfälle beider Arten von Krebs um 10% bis 30% reduzieren.
- Eine Reduktion des Anteils der Milch von durchschnittlich knapp 8% auf nur noch 1% der Kalorienaufnahme in allen Ländern (was ungefähr dem Niveau von Thailand entspricht), würde die Anzahl der Todesfälle für beide Arten von Krebs um 1/3 bis zu 2/3 senken.

Wie belastbar sind nun die Ergebnisse der Untersuchung? Dies hängt ganz entscheidend davon ab, inwieweit es ausgelassene zeitvarrierende Variablen gibt, die einen Einfluss auf die Mortalitätsrate haben und die fälschlicherweise nicht in den Modellen enthalten waren (*confounding factors*). Welche Art von confounding factors können hier möglich sein?

- Es ist zu betonen, dass alle nicht in den Modellen enthaltenen Variablen, die über den Analysezeitraum innerhalb der Länder konstant sind – genetische Unterschiede, Sonneneinstrahlung, große Unterschiede im Lebensstandard und bestimmte Aspekte der nationalen Gesundheitssysteme – von den fixen Ländereffekten in den Regressionsanalysen absorbiert werden.

Das ist einer der Gründe dafür, warum wir unsere Analysen auch für kürzere Zeiträume (1990-1999, 2000-2008) durchführen, da dies die Plausibilität dafür erhöht, dass solche confounding factors zeitkonstant sind.

- Es werden Variablen, die über alle Länder gleich wirken, wie z. B. medizinischer Fortschritt (bessere Diagnose und wirksamere Therapien) durch die fixen Zeiteffekte (Jahres-Dummy-Variablen) kontrolliert.
- Zeitlich variierende Unterschiede des Lebensstandards und bestimmter Aspekte des nationalen Gesundheitssystems werden durch die Einbeziehung des BIP pro Kopf in die Regressionsanalyse eliminiert.
- Es werden zusätzlich dynamische Modelle mit einer verzögerten abhängigen Variable genutzt, um weitere zeitlich veränderliche confounding factors (ausgelassene Variablen) zu kontrollieren.
- Es wird auf die durchschnittliche gesamte Kalorienaufnahme pro Person konditioniert und die Milch-Variable wird als Anteil der Milch an der gesamten Kalorienaufnahme spezifiziert. Dies sollte bereits zu einer eher konservativen Schätzung des Milch-Effektes führen.
- Falls darüber hinaus zeitlich variierende confounding factors relevant sind, die mit der Milch-Variablen korreliert sind, dann werden diese confounding factors dazu führen, dass eine Korrelation der Milch-Variablen mit dem Fehlerterm entsteht. Dieses Problem kann jedoch durch eine Instrumentierung der Milch-Variablen (und der anderen Lebensmittel) gemindert werden. Selbst nach Anwendung dieser Methoden finden wir in vielen Spezifikation einen deutlich positiven Effekt der Milch-Variable auf die Mortalitätsraten.

Demnach gehen wir mit einer hohen Wahrscheinlichkeit davon aus, dass confounding factors (ausgelassene Variablen) nicht zu einer maßgeblichen Verzerrungen unserer Ergebnisse geführt haben.

Using Panel Econometric Methods to Estimate the Effect of Milk Consumption on the Mortality Rate of Prostate and Ovarian Cancer

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ABSTRACT

Recently prostate and ovarian cancer has been related to milk consumption. However, existing observational studies based on country level data do not attempt to identify causal effects since they are only based on simple cross-sectional analyses. This paper takes a step toward estimating of causal effects of milk consumption on cancer by applying panel econometric models and by using the within-country variation of the mortality rates and food consumption instead of the between-country variation in a panel of up to 50 countries for 1990 to 2008. Possible methodological problems arising from omitted variables (confounding factors), heterogeneity, and outliers are carefully discussed and a wide range of recent panel econometric estimators are applied. The results indicate fairly well that milk consumption increases both the mortality rate of prostate cancer as well as the mortality rate of ovarian cancer. The estimated effects are also important in quantitative terms, i.e., a reduction in the consumption of milk products can reduce the number of people dying of prostate and ovarian cancer appreciably. Furthermore, the consumption of other animal food products as well as sugar seems to be harmful. For the mortality rate of ovarian cancer we find that total calories intake increases the mortality rate too.

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1. Introduction

Several studies have indicated a positive association between the intake of specific nutrients, like animal products and the incidence of malignant diseases; such as prostate cancer or ovarian cancer (Kushi *et al*. 1999; Larsson *et al*. 2004; Zhang and Kesteloot 2005; Allen *et al*., 2008). Moreover there is evidence that the consumption of specific food increase not only the incidence of those cancers, but also the mortality rate.

With regard to the effects of dairy intakes on the mortality rate, it has to be considered, that the results vary in dependence of the cancer type. We summarize the findings of several studies in Table A 1 in the Appendix. While milk consumption seems to be significantly positively correlated with prostate cancer mortality (Rose *et al*., 1986; Colli and Colli, 2006; Hebert *et al*., 1998), the undertaken studies, which examine the impact of milk intake on ovarian carcinoma incidence and mortality, are more heterogeneous (Kushi *et al*., 1999; Larsson *et al*., 2006; Mommers *et al*., 2006). Besides researches, which suggest a positive association between dairy diet and the mortality of ovarian cancer (Rose *et al.*, 1986) respectively the post-diagnosis survival time (Dolecek *et al.*, 2010; Nagle *et al.*, 2003), there are other studies, which have found "*no association between intakes of several specific dairy foods […] and risk of ovarian cancer*" (Genkinger *et al.* 2006: 371).

The Havard School of Public Health concludes with regard to the question whether milk should be a source of calcium, that milk consumption *possibly* increase the risk of ovarian cancer and *probably* increase the risk of prostate cancer.[2]

This paper builds on Ganmaa *et al.* (2002), Zhang and Kasteloot (2005) as well as Colli and Colli (2006) and uses country-level data. However, in contrast to these previous studies, we apply panel econometric models. By doing this, we hope to identify not only a simple correlation, but the causal effect from milk consumption to cancer. "*Econometrics may be defined as the social science in which the tools of economic theory, mathematics, and statistical inference are applied to the analysis of economic phenomena*." (Goldberger, 1964, p.1). Although at first glance country-level data seem to be less adequate than individual-level data for the research question, individual-level data on nutritional habits may be measured inaccurately since these data are usually based on self-reported information of the respondent in surveys.

Different from the previous studies based on country-level data, we do not attempt to identify the effect via the cross-sectional variation (between the countries) of the dietary practices and the

[2] Harvard School of Public Health. http://www.hsph.harvard.edu/nutritionsource/calcium-full-story/

mortality rate, but via the time variation within the countries. The difference is explained by the means of simple scatter plots in Section 3. Before doing that, we introduce our dataset in the following Section. Section 4 explains the econometric methods applied. In Section 5 and 6 we try to select the explanatory variables (milk and the control variables) for the model for prostate cancer and the model for ovarian cancer in a systematic way by applying Bayesian Model Averaging techniques as well as extended Extreme Bounds Analysis (EBA). Applying EBA is, furthermore, a way to gain insight into the robustness of the effects of milk consumption on both types of cancer. In Section 7 and 8 we will show the results of several econometric estimators. We will apply different methods on different samples and show that the results – a positive (damaging) impact of milk – on the mortality of both types of cancer is found. Section 9 applies a new method proposed by Oster (2013) to evaluate the possible effects of confounding factors on our results. In order to understand the estimated effects also in quantitative terms, but not with the intention to make exact predictions, we show in Section 10 some simulations, where we assume several scenarios for milk reduction and the resulting predicted changes in mortality rates. Section 11 discusses the results and concludes.

2. Data

Our dependent variable is the Mortality Rate of Prostate Cancer and Ovarian Cancer per 100,000 persons. The amount of deaths due to prostate and ovarian cancer was received from the World Health Organization (WHO) Mortality Database[3], which provides country-reported data on mortality by age, sex and causes from the annual report of national civil registration systems of deaths.

For the purpose of our examination we focus on countries, which are available in both the WHO Mortality Database and the Food Consumption Database[4] compiled by the Food and Agriculture Organization (FAO) of the United Nations. In order to consider the different age structures between several populations and to avoid age-related bias when comparing these populations, the age-standardize mortality rates are employed (see Boschi-Pinto *et al.* 2001). Taking these criteria into account, up to 50 countries, including 16 American, 21 European, 7 Asian, 4 African and 2 Oceania countries could be selected. We assembled the mortality rates for each of these countries from 1990 to 2008. There are some gaps in the time series of the mortality rates. Only for the descriptive analyses in Section 3 these gaps are filled by imputation based on simple

[3] http://www-dep.iarc.fr/WHOdb/WHOdb.htm
[4] http://faostat3.fao.org/faostat-gateway/go/to/home/E

linear regression of the mortality rate on a linear time trend separately for every country. This implies 57 imputed data points for prostate cancer and 44 for ovarian cancer.

The food data used in our research were derived from the FAO of the United Nations. By applying the Food Balanced Sheets of the FAOSTAT Database we obtained the required per capita consumption rates from 1961-2008 for the same countries selected in the WHO Mortality Database. The food items analyzed in our study were milk (including milk products and excluding butter), sugar (sweeteners), vegetable oil, animal fats (including butter), meat, fish and seafood, eggs, fruits, vegetables, pulses and cereals. Some of these food categories are pooled together based on statistical analyses or content considerations. For the purpose of the subsequent analysis we chose item units, which express the food intake as percent of total energy 'food supply (kcal/capita/day)'.

In order to analyze the selected country-level data from an economic perspective we used World Bank Open Database compiled from officially-recognized international sources. The global development indicators we chose, are the GDP per capita and total (=public + private) health expenditure per capita, both expressed in purchasing power parity and constant 2005 international Dollars.

3. Descriptive Analyses

In the following some simple descriptive – mostly graphical – analyses are presented. We start in Section 3.2 with prostate cancer and turn to ovarian cancer afterwards in Section 3.3. Before doing that Table 1 in the next section provides an overview of the variables in the first and the last year of the estimation sample for every country.

3.1 Overview

Some time series of the mortality rate of ovarian cancer start later – the corresponding values are marked (*,$^{\#}$,$^{+}$ see the notes below Table 1). It is important to distinguish between the annual variables in Columns (1), (2), (3), and (5) and variables that measure an average over 25 years (the current year and the 24 previous years) in Columns (4), (6), and (7). For example Table 1 shows that in Albania in 1990 10% out of the total calories intake of 2,656 kcal were based on milk. On average the proportion of milk was 7.8% in 1966-1990. In the econometric analysis we want to explain the current annual mortality rate from 1990 to 2008 by the 25-years average of food consumption.

When taking a closer look to the numbers in Table 1 one can already identify some "regularities":

- In countries in which milk consumption is / was very low (<3%) – such as Egypt, Korea, Philippines, Sri Lanka, Thailand – the mortality rate of both types of cancer is relatively low.
- In some countries in which milk consumption is / was very high (>11%) – such as Australia, Finland, Ireland, Netherlands, Norway, Sweden, Switzerland – the mortality rate of prostate cancer is clearly above average. To lesser extent this association can also be found for the mortality of ovarian cancer.
- In 26 out of 49 countries the mortality rates of prostate cancer increase from the first year to the last year in Table 1. In 17 out of these 26 countries milk consumptions increased (measured as a change in Column (6)). In 3 countries milk consumption stayed constant and in 7 countries milk consumption decreased.
- In 23 out of 49 countries the mortality rates of ovarian cancer increase from the first year to the last year in Table 1. In 14 out of these 23 countries milk consumptions increased (measured as a change in Column (6)). In 2 countries milk consumption stayed constant and in 7 countries milk consumption decreased.

TABLE 1: VARIABLES BY COUNTRY FOR THE FIRST AND LAST YEAR IN THE ESTIMATION SAMPLE

	Year	Age- standardized Annual Mortality Rate of **Prostate** Cancer per 100,000	Age- standardized Annual Mortality Rate of **Ovarian** Cancer per 100,000	**Total Food Intake** in kcal per Person per Day	**Total Food Intake** in kcal per Person per Day, Average of previous 25 years	Proportion of **Milk** in total calories intake in %	Proportion of **Milk** in total calories intake in %, Average of previous 25 years	Proportion of **Sugar** in total calories intake in %, Average of previous 25 years
		(1)	(2)	(3)	(4)	(5)	(6)	(7)
Albania	1990	11.0	1.3	2,656	2,576	10.0	7.8	6.2
	2004	10.0	1.5	2,819	2,753	18.7	12.8	6.7
Argentina	1990	17.0	*4.7	2,913	3,138	8.2	7.5	12.5
	2007	19.9	4.6	2,988	3,084	9.3	8.5	14.1
Australia	1990	23.0	6.7	3,177	3,123	11.3	11.2	16.5
	2006	18.4	4.6	3,206	3,098	10.4	11.1	14.5
Austria	1990	22.4	9.1	3,509	3,303	9.4	9.9	12.3
	2008	16.5	5.8	3,826	3,618	6.8	9.0	12.0
Brazil	1990	11.6	#2.6	2,721	2,559	5.6	4.9	17.5
	2008	17.4	3.1	3,177	2,861	6.7	6.1	15.8
Bulgaria	1990	10.5	4.4	3,133	3,540	6.4	6.2	10.0
	2008	13.6	6.2	2,802	3,040	8.2	8.3	10.4
Canada	1990	21.7	6.8	3,019	2,967	8.7	10.0	15.8
	2004	16.0	6.0	3,539	3,198	6.2	8.0	14.5
Chile	1990	17.8	*3.7	2,536	2,606	5.6	5.1	13.6
	2007	21.6	3.5	2,925	2,705	4.5	5.5	15.1
Colombia	1990	13.3	3.1	2,394	2,187	6.6	5.8	18.7

	2007	19.5	3.9	2,666	2,515	7.6	7.0	17.6
Costa Rica	1990	18.6	3.8	2,802	2,454	8.4	7.8	23.7
	2008	17.8	2.8	2,876	2,782	10.3	9.1	20.5
Cyprus	1999	6.7	1.9	2,710	2,651	9.9	9.0	11.0
	2008	13.7	5.1	2,665	2,705	10.1	10.1	12.3
Denmark	1994	24.9	9.9	3,284	3,126	7.3	8.8	14.9
	2006	25.5	8.3	3,392	3,262	9.6	8.8	14.0
Ecuador	1990	11.7	*2.1	2,131	2,112	6.9	7.1	17.0
	2008	15.9	2.3	2,271	2,213	7.0	7.1	10.2
Egypt.	1990	1.1	0.2	3,154	2,694	1.5	1.7	8.4
	2008	5.1	0.9	3,406	3,227	3.0	1.9	9.0
El Salva-dor	1990	4.0	1.3	2,318	2,063	4.4	5.0	15.4
	2008	9.3	1.9	2,587	2,436	7.2	5.5	15.5
Finland	1990	23.6	7.5	3,147	3,091	13.8	15.1	13.2
	2008	18.0	5.3	3,218	3,119	14.6	13.7	11.6
France	1990	22.3	6.4	3,515	3,375	11.3	10.8	10.8
	2008	15.2	5.3	3,598	3,558	10.2	10.9	10.3
Germany	1990	20.5	8.4	3,321	3,272	8.5	7.6	12.3
	2008	15.3	5.8	3,537	3,436	9.4	8.4	12.6
Greece	1990	10.6	3.4	3,539	3,317	9.7	9.1	8.6
	2008	12.9	4.5	3,656	3,579	11.4	10.3	8.9
Guate-mala	1990	4.1	0.8	2,350	2,124	2.5	3.4	15.5
	2008	15.5	1.9	2,226	2,207	3.5	3.2	15.9
Hungary	1990	20.4	7.9	3,702	3,457	6.3	6.2	11.5
	2008	16.3	6.8	3,495	3,483	6.8	6.7	13.2
Ireland	2001	24.4	9.6	3,716	3,599	12.8	13.1	11.9
	2008	19.1	8.2	3,588	3,599	10.5	12.2	11.0
Israel	1990	11.2	7.5	3,398	3,221	7.7	7.2	11.7
	2008	9.3	5.6	3,560	3,478	7.8	7.3	11.3
Italy	1990	15.3	5.4	3,584	3,439	8.0	7.2	8.8
	2008	11.3	4.9	3,612	3,588	8.0	7.7	8.2
Japan	1990	4.8	3.7	2,945	2,793	4.2	3.5	11.0
	2008	6.6	3.5	2,768	2,888	4.2	4.2	10.3
Korea	1990	1.3	1.1	2,956	2,950	0.9	0.4	4.6
	2006	6.0	2.4	3,124	3,030	1.2	1.0	9.3
Kuwait	1990	4.7	4.0	2,281	2,765	5.6	7.3	14.6
	2008	6.0	2.9	3,675	3,139	5.8	7.2	12.5
Mauritius	1990	8.5	3.1	2,725	2,537	5.8	5.1	17.2
	2008	14.2	3.2	2944	2,823	5.0	6.0	14.3
Mexico	1990	13.8	3.3	3,033	2,863	4.7	5.1	14.2
	2008	13.6	3.6	3,188	3,128	5.4	4.9	15.3
Nether-lands	1990	24.0	8.7	3,269	3,150	12.2	14.1	14.5
	2008	18.6	6.7	3,277	3,231	13.8	13.5	14.4
New Zea-land	1990	24.2	8.5	3,254	3,076	10.9	10.9	15.5
	2008	21.7	5.7	3,169	3,150	3.3	7.1	16.5
Norway	1990	27.9	8.2	3,154	3,139	11.5	13.6	13.1
	2008	25.5	7.5	3,475	3,315	9.5	11.0	12.7
Panama	1990	13.2	3.2	2,305	2,294	6.7	6.2	14.5
	2008	23.9	3.7	2,623	2,378	7.4	7.0	13.4
Paraguay	1990	9.0	2.8	2,423	2,479	4.0	3.2	8.4
	2008	17.8	2.6	2,540	2,566	4.8	4.5	9.4
Philipp-ines	1992	4.5	2.1	2,277	2,101	0.9	1.1	11.0
	2008	15.4	4.2	2,633	2,335	0.5	0.9	11.2
Poland	1991	12.4	7.4	3,292	3,453	9.9	10.2	12.7
	2008	16.4	7.8	3,363	3,384	7.0	8.3	12.9
Portugal	1990	17.9	3.7	3,393	2,998	6.1	4.5	8.6
	2008	17.8	3.4	3,614	3,421	7.7	6.6	8.4
Romania	1990	8.7	5.0	3,149	3,071	7.7	7.7	7.9
	2008	11.2	5.4	3,546	3,157	14.0	10.8	8.2

South Africa	2002	26.9	3.3	2,911	2,850	2.9	3.2	12.4
	2008	23.2	3.3	2,996	2,879	3.1	3.0	11.5
Spain	1990	17.1	4.0	3,279	2,976	7.0	8.3	9.6
	2008	12.5	4.3	3,232	3,279	6.5	7.7	8.8
Sri Lanka	1990	0.5	0.7	2,166	2,242	2.4	2.0	8.1
	2006	1.0	1.5	2,411	2,279	2.8	2.4	10.8
Sweden	1990	26.9	8.2	2,974	2,933	13.8	14.1	15.0
	2008	24.7	6.5	3,123	3,061	13.5	13.8	14.0
Switzer-land	1995	24.8	6.5	3,306	3,409	11.9	11.9	13.5
	2007	18.0	5.4	3,428	3,386	11.5	11.7	14.7
Thailand	1990	0.3	0.2	2,069	2,060	0.7	0.5	5.8
	2006	1.8	1.3	2,887	2,306	1.1	0.9	9.5
Trinidad & Tobago	1990	42.4	[+]6.1	2,635	2,707	5.8	6.6	17.5
	2006	65.8	7.0	2,716	2,718	7.4	6.7	20.1
United Kingdom	1990	21.1	9.6	3,244	3,186	10.8	10.7	13.8
	2008	17.6	7.2	3,453	3,314	10.0	10.4	11.5
United States	1990	22.2	7.0	3,507	3,189	10.9	11.8	17.7
	2007	13.3	5.9	3,794	3,613	10.0	10.8	17.0
Uruguay	1990	27.0	[*]4.5	2,509	2,762	9.8	10.4	11.9
	2004	29.8	4.7	2,768	2,722	8.6	10.4	11.6
Venezuela	1990	21.3	[#]3.2	2,394	2,483	5.0	6.9	16.7
	2007	24.4	3.7	2,692	2,494	4.9	5.5	15.8

Notes: [*]1997, [#]1996, [+]1999
Mean of Mortality Rate of Prostate Cancer over all countries and time periods of the sample: 16.96
Mean of Mortality Rate of Ovarian Cancer over all countries and time periods of the sample: 5.06

Table 2 shows a correlation matrix of the explanatory food variables and the dependent variables. All food variables are expressed in percentages of total calories intake. Furthermore, the food variables are moving averages of the preceding 25 years. The following insights can be gained from Table 2:

- Both mortality rates are positively correlated (0.585).
- Mortality of ovarian cancer is highly correlated with GDP per Capita (0.628). However, this correlation may be driven by the high correlation between total calories intake and the GDP per capita (0.656) on the one hand, and the high correlation between total calories intake and mortality of ovarian cancer (0.671) on the other hand.
- The proportion of milk in total calories is positively correlated with both mortality rates. The correlation with ovarian cancer (0.702) is larger than the correlation with prostate cancer (0.453). Furthermore, the proportion of milk is positively correlated with GDP per capita (0.606), that is, residents of richer countries consume a larger proportion of their calories intake in the form of milk products than residents of poorer countries.
- The proportion of milk in total calories is positively correlated with the proportion of meat and animal fats (0.656), and negatively correlated with the proportion of pulses (-0.444).
- Furthermore, the proportion of milk in total calories intake is positively correlated with total calories intake (0.543). That means, the more milk is consumed in a country the higher is to-

tal calories intake. On the one hand, this may reflect, that milk products such as cheese are high-calories food. On the other hand, the positive correlation may not be causal, but may be generated by the fact that milk is positively correlated with GDP per capita and GDP per capita is positively related to total calories intake.

TABLE 2: CORRELATION MATRIX OF DEPENDENT VARIABLES AND EXPLANATORY VARIABLES (*p*-values) FOR THE ESTIMATION SAMPLE 1990-2008 AND 49 COUNTRIES

	Mortality Rate of Prostate Cancer	Mortality Rate of Ovarian Cancer	GDP per Capita in PPP	Total Calories, 25	Prop. of Milk, 25	Prop. of Sugar, 25	Prop. of Meat and Fat, 25	Prop. of Eggs, 25	Prop. of Fish, 25	Prop. of Vegs & Fruits, 25	Prop. of Pulses, 25
Mortality Rate of Prostate Cancer	1.000										
Mortality Rate of Ovarian Cancer	0.585 (0.000)	1.000									
GDP per Capita in PPP	0.234 (0.000)	0.628 (0.000)	1.000								
Total Calories, 25	0.192 (0.000)	0.671 (0.000)	0.656 (0.000)	1.000							
Prop. of milk, 25	0.453 (0.000)	0.702 (0.000)	0.606 (0.000)	0.543 (0.000)	1.000						
Prop. of Sugar, 25	0.437 (0.000)	0.244 (0.000)	-0.013 (0.706)	-0.240 (0.000)	0.163 (0.000)	1.000					
Prop. of Meat and Fat, 25	0.413 (0.000)	0.765 (0.000)	0.544 (0.000)	0.624 (0.000)	0.656 (0.000)	0.007 (0.855)	1.000				
Prop. of Eggs, 25	-0.045 (0.206)	0.483 (0.000)	0.522 (0.000)	0.449 (0.000)	0.272 (0.000)	-0.107 (0.003)	0.420 (0.000)	1.000			
Prop. of Fish, 25	-0.087 (0.014)	0.008 (0.837)	0.329 (0.000)	0.011 (0.764)	-0.097 (0.006)	-0.240 (0.000)	-0.108 (0.002)	0.331 (0.000)	1.000		
Prop. of Vegs & Fruits, 25	-0.224 (0.000)	-0.238 (0.000)	-0.027 (0.441)	-0.173 (0.000)	-0.148 (0.000)	-0.151 (0.000)	-0.194 (0.000)	0.042 (0.236)	-0.017 (0.637)	1.000	
Prop. of Pulses, 25	-0.052 (0.146)	-0.525 (0.000)	-0.523 (0.000)	-0.513 (0.000)	-0.444 (0.000)	0.373 (0.000)	-0.567 (0.000)	-0.381 (0.000)	-0.348 (0.000)	-0.056 (0.115)	1.000
Prop. of Veg. Oils, 25	0.153 (0.000)	0.181 (0.000)	0.391 (0.000)	0.346 (0.000)	0.349 (0.000)	0.018 (0.615)	0.002 (0.954)	0.117 (0.001)	0.047 (0.182)	0.335 (0.000)	-0.061 (0.086)

Number of observations: 797 and 708 in case of Ovarian Cancer.

Obviously, the quality of the whole analysis is based on the quality of our dependent variables, the age-standardized mortality rates. Particularly, if the age-standardization of the mortality rates is not complete, our dependent variables are correlated with the age distribution of the countries' residents. Unfortunately, we do not have any detailed information on the age distribution in order to control for this. But we have an estimate of the life expectancy at birth by the World Bank. By plotting this variable against the age-standardized mortality rates we can get some indication whether there is a problem. As "South Africa" is an outlier driving the results towards "no correlation" (low life expectancy and high mortality rate), we exclude the country from the following figures. In case of prostate cancer we do not find any correlation (Figure 1). For ovarian cancer

we find a weak positive correlation (0.47). We conclude from this analysis, that there is no obvious evidence in favor of quality problems of the age-standardization of the mortality rates. Note that measurement errors in the mortality rates, which may even be correlated with the explanatory variables, do not bias our econometric results, as long as they are time constant, since it is this case they are absorbed by the country fixed effects (see Section 4).

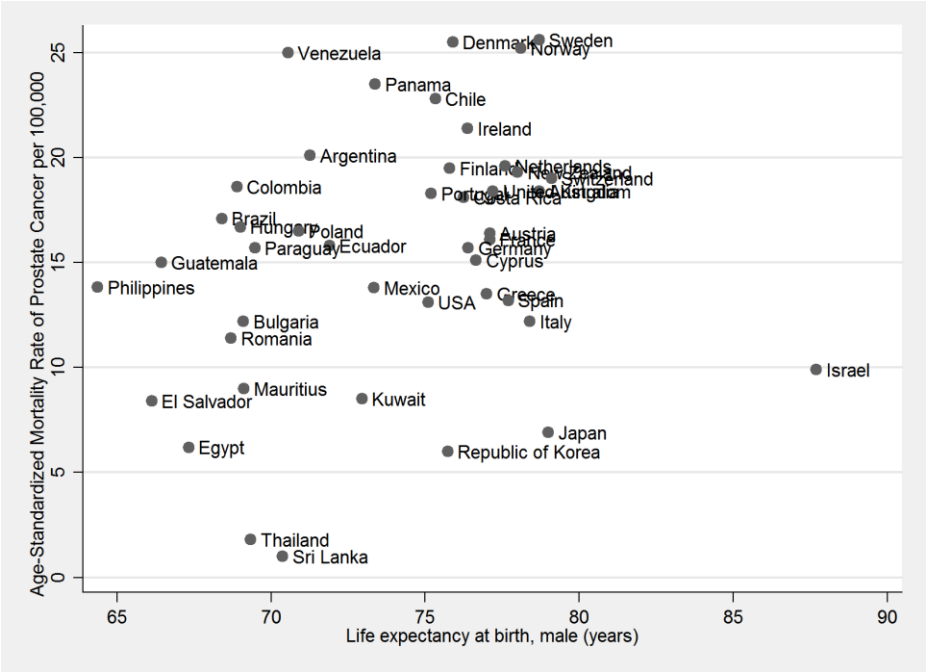


FIGURE 1: MORTALITY RATE OF PROSTATE CANCER VERSUS LIFE EXPECTANCY IN 2006
Notes: Correlation Coefficient (*p*-value): 0.2480 (0.1046); *N*=44

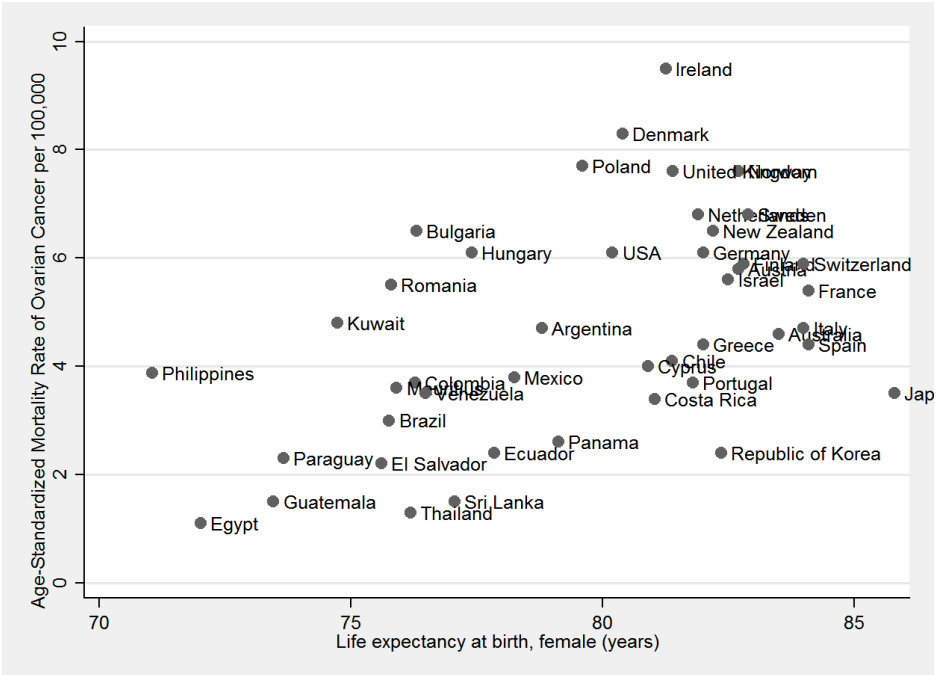
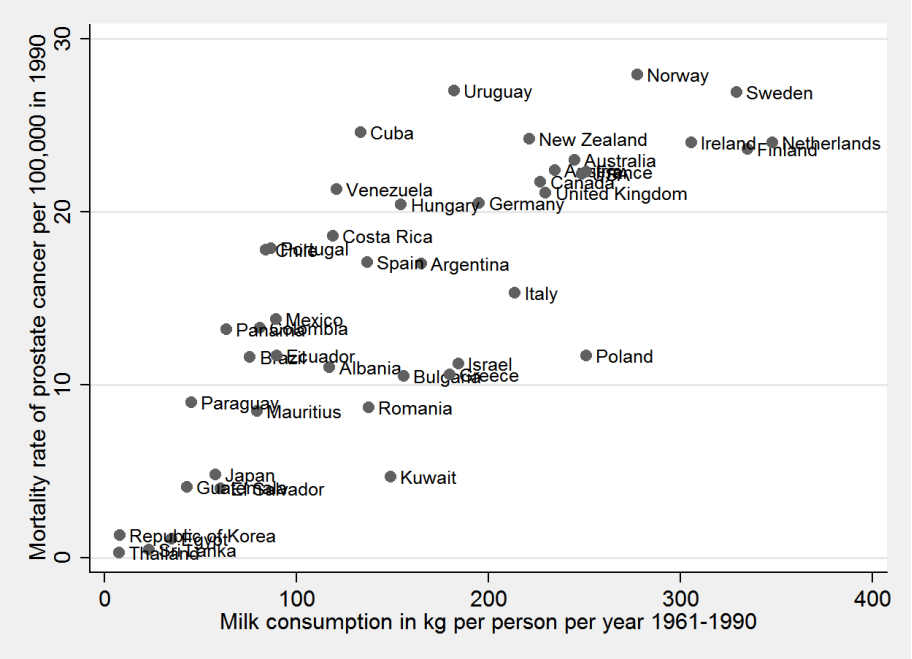


FIGURE 2: MORTALITY RATE OF OVARIAN CANCER VERSUS LIFE EXPECTANCY IN 2006
Notes: Correlation Coefficient (*p*-value): 0.4754 (0.0011); *N*=44

3.2 Mortality Rate of Prostate Cancer and Milk Consumption

Only in the following graphical representation – but not in the econometric analyses – we exclude Trinidad and Tobago. The reason is its very high mortality rate which makes it hard to show this country in the same graph with the other countries (see Table 1). We start with Figure 3 plotting the age-standardized mortality rates of prostate cancer in 1990 against average annual per-capita milk consumption in kg during the 30-years period 1960-1990. We use this as a starting point, since it is a replication of the Figure in Ganmaa *et al.* (2002). However, due to data restrictions the countries are not identical. In Figure 3 the well-known strong positive cross-country correlation between prostate cancer and milk consumption can be found (0.78).



Correlation Coefficient (*p*-value) is 0.7765 (0.000); N=44

FIGURE 3: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER AND AVERAGE PER CAPITA MILK CONSUMPTION (1961 – 1990)

As explained in greater detail in Section 4, one should distinguish between the total calories effect and the dietary composition effect of food consumption. For example, if we assume in Figure 3 that the higher mortality rate in Sweden in comparison to Thailand results causally from its higher milk consumption, this may be explained by the higher calories intake which is associated with the consumption of milk products as well as the higher proportion of milk products in total calories intake. We are only interested in the latter (the composition effect).

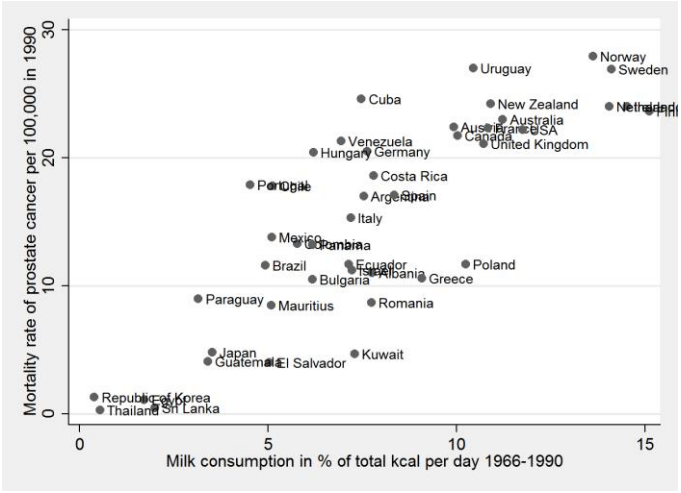
Figure 4, Figure 5, and Figure 6 present the association for three different time periods. The milk consumption is now expressed in terms of percentage of total daily calories intake. For ex-

ample 10% on the x-axis means that 10 percent of the daily calories intake derive from milk. Moreover, just as in the econometric analyses in Section 5 and 6 the average milk consumption of 25 years (instead of 30 years) is analyzed.

Starting with Figure 4 which presents the mortality rate in 1990 and the average milk consumption in 1966 to 1990, the correlation (coefficient 0.82) seems even stronger than in case of Figure 3 where milk consumption is expressed in absolute terms (coefficient 0.78). That means, the correlation seems to be driven by the composition effect rather than the calories effect – not the calories from milk are correlated with the mortality of cancer but the ingredients of milk. Of course this correlation has to be re-examined with multiple regressions. If one examines later time periods in Figure 5 and Figure 6 this very strong correlation seems to decrease from 0.63 in 2000 versus 1976-2000 to 0.43 in 2006 versus 1982-2006.[5] A closer look reveals that this decreasing correlation may be driven by medical progress since rich "high-milk-consumption" countries such as Sweden, Norway, Switzerland and the Netherlands were obviously able to decrease their mortality rates of prostate cancer.

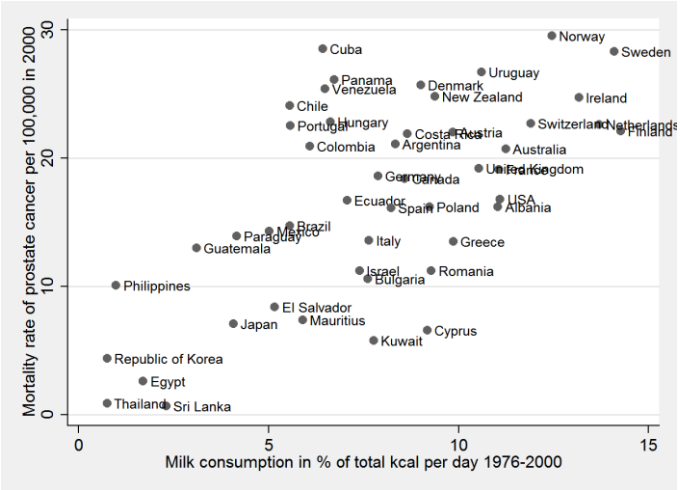
Figure 7 shows the mortality rate of prostate cancer and the calories from milk in total calories intake for the whole sample period 1990 to 2008 and all countries within one graph. Though Figure 7 proves an impressive positive between-countries correlation (0.64) again, this is not the identification strategy we are interested in. We want make use of the within-country variation of the variables over time to estimate the effect of nutrition on mortality of cancer. A first step into this direction is Figure 10 which shows separately for 49 countries association between milk consumption and cancer. For 32 out of these 49 countries the correlation coefficient is positive and statistically significant at the 10% level. For 9 out of the 49 countries the correlation coefficient is negative and statistically significant.

[5] We chose the year 2006 instead of the year 2008 (which is the latest available) because in 2006 we have 65 observations countries whereas in 2008 there are only 36



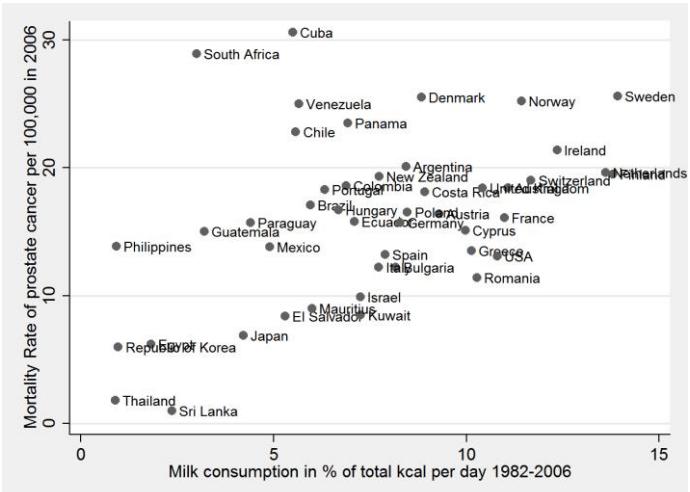
Correlation Coefficient (*p*-value): 0.8188 (0.000), N=63

FIGURE 4: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER 1990 AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE 1966-1990



Correlation coefficient (*p*-value): 0.6300 (0.000), N=69

FIGURE 5: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER 2000 AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE 1976-2000



Correlation Coefficient (*p*-value): 0.4270 (0.0031), N=65

FIGURE 6: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER 2006 AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE 1982-2006

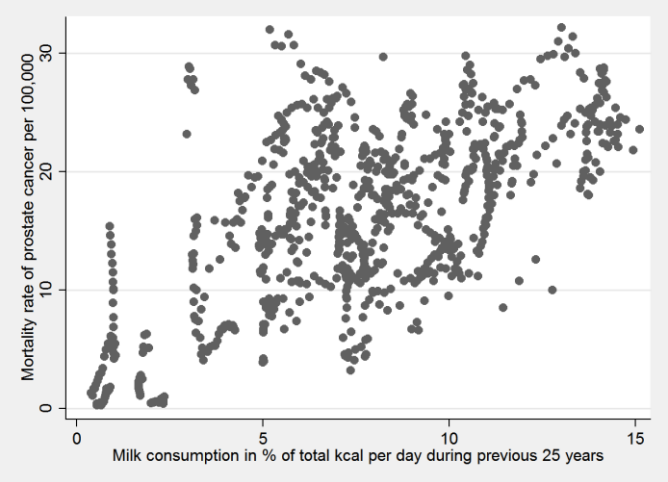
In Section 4 we will introduce our estimation strategy which assumes country fixed effects. These country fixed effects control for time invariant unobserved variables affecting the mortality rates. Examples for such unobserved variables are differences in the countries' health system, genetic differences, and geographic differences (solar radiation). Moreover we will use the natural logarithm of all variables since this leads to a better fit. One way to eliminate the fixed effects in variables is the so-called within transformation. Let x_{it} be a variable (such as the mortality rate or milk consumption) with the subscripts *i* denoting the country (*i*=1,…, *N*) and *t* indicating the year (*t*=1…T_i). The mean for every country *i*=1,…,*N* is $\bar{x}_i = T_i^{-1}\sum_{t=1}^{T_i} x_{it}$. The within-transformed variables in natural logarithms are $\left(\ln x_{it} - \overline{\ln(x_i)}\right)$ for every country *i* and year *t*. Figure 8 shows both variables transformed in this way for all countries within one graph. In Figure 11 all countries are shown separately. Each point in both graphs can now be interpreted as the change rate to the respective country mean $\bar{x}_i$. We see that a positive deviation from the country mean in milk consumption is often associated with positive deviation in the mortality rate and vice versa. Indeed, the overall correlation coefficient of 0.47 in Figure 8 is still statically significant. For 32 out of these 49 countries the correlation coefficient is positive and statistically significant at the 10% level (see Figure 11). For 9 out of the 49 countries the correlation coefficient is negative and statistically significant.

The dependent variable – the mortality rate of prostate cancers – covers the period 1990 to 2008. Of course, medical progress and common trends with regard to nutrition practices took place during this period. In order control for this medical progress and time trend we will include year dummy variables into our model which is presented in Section 4. This is called the two-way fixed effects model since it controls for unobserved country fixed effects as well as unobserved time fixed effects (see Greene 2011). Including year dummy variables is a way of detrending the variables without imposing a functional form assumption with regard to the time trend. In regression analysis transforming variables into deviations from time means is equivalent to the inclusion of time dummies (see Bond *et al.*, 2001).[6] The mean of the variable x_{it} for every year (*t*=1…T_i) is $\bar{x}_t = N_t^{-1}\sum_{i=1}^{N_t} x_{it}$, with N_t indicating the number of countries in year *t*. The time-detrended and within-transformed variables in natural logarithms are $\left(\ln x_{it} - \overline{\ln(x_i)} - \overline{\ln(x_t)}\right)$ for

[6] However, this statement is only correct for balanced panels. In our case of an unbalanced panel this transformation is not completely equivalent. Since this section is, however, a pure descriptive analysis which should be kept as intuitive as possible, we refrain from using more complicated ways of detrending.

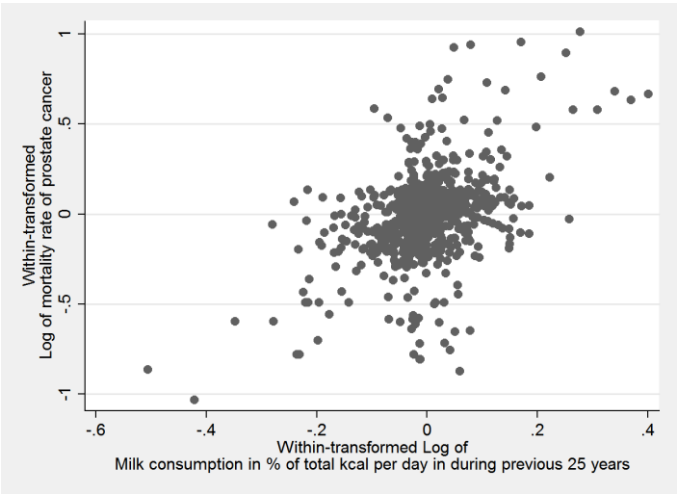
every country *i* and year *t*. Both variables transformed like this can be found in Figure 9 and Figure 12. In Figure 9 the overall correlation coefficient is 0.4405 and it is still significant at the 5% level. Figure 12 shows the results by countries.

Again for 30 out of these 49 countries the correlation coefficients are positive and statistically significant at the 10% level. After the detrending only for 4 out of 49 countries the correlation coefficient is negative and statistically significant. For example, while the correlation coefficient for Germany was statistically significant negative with -0.96 in Figure 11 it is now – after detrending – statistically insignificant with -0.16 in Figure 12.



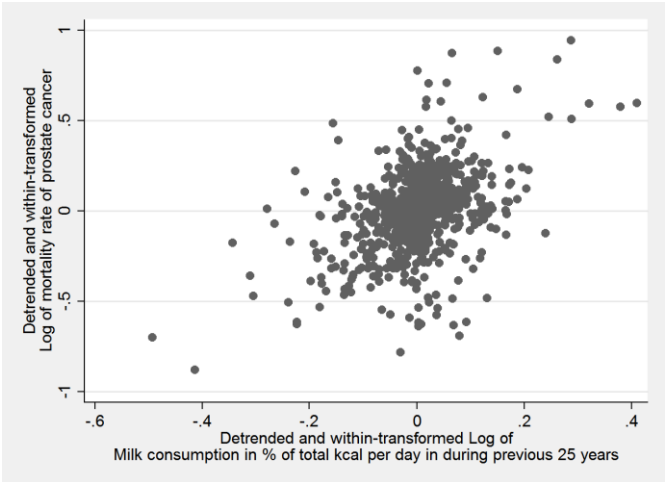
Correlation Coefficient (*p*-value): 0.6423 (0.000), Number of observations: 872

FIGURE 7: AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE



Correlation Coefficient (*p*-value): 0.4665 (0.000), Number of observations: 872

FIGURE 8: WITHIN-TRANSFORMED LOG OF MORTALITY RATES OF PROSTATE CANCER AND THE WITHIN-TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE



Correlation Coefficient (*p*-value): 0.4405 (0.000), Number of observations: 872

FIGURE 9: DETRENDED WITHIN-TRANSFORMED LOG OF MORTALITY RATES OF PROSTATE CANCER AND DETRENDED WITHIN-TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK

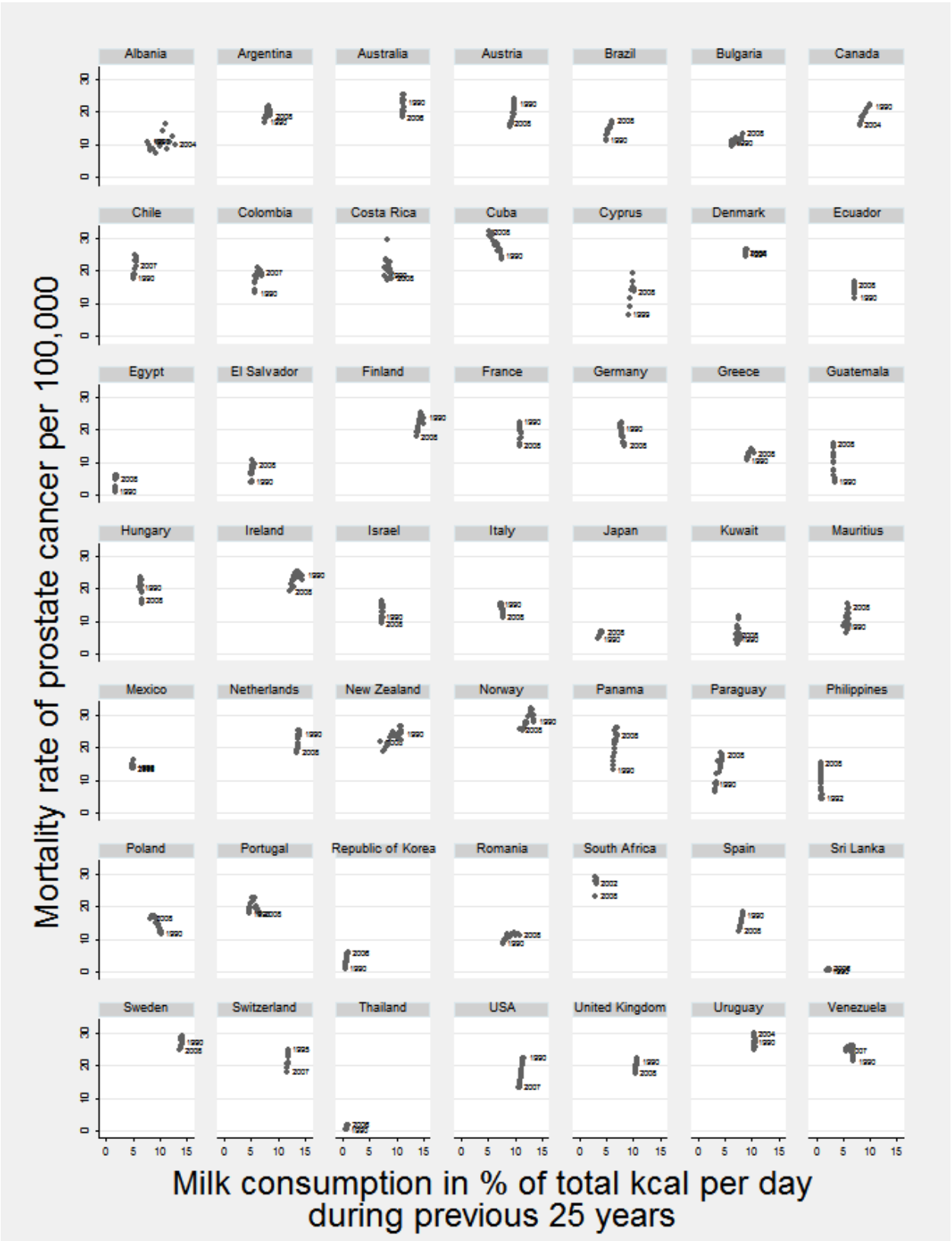


FIGURE 10: AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE DURING THE PREVIOUS 25 YEARS

Notes: Number of countries statistically significant positive correlation coefficient: 31 out of 49
Number of countries with statistically significant positive correlation coefficient: 9 out of 49

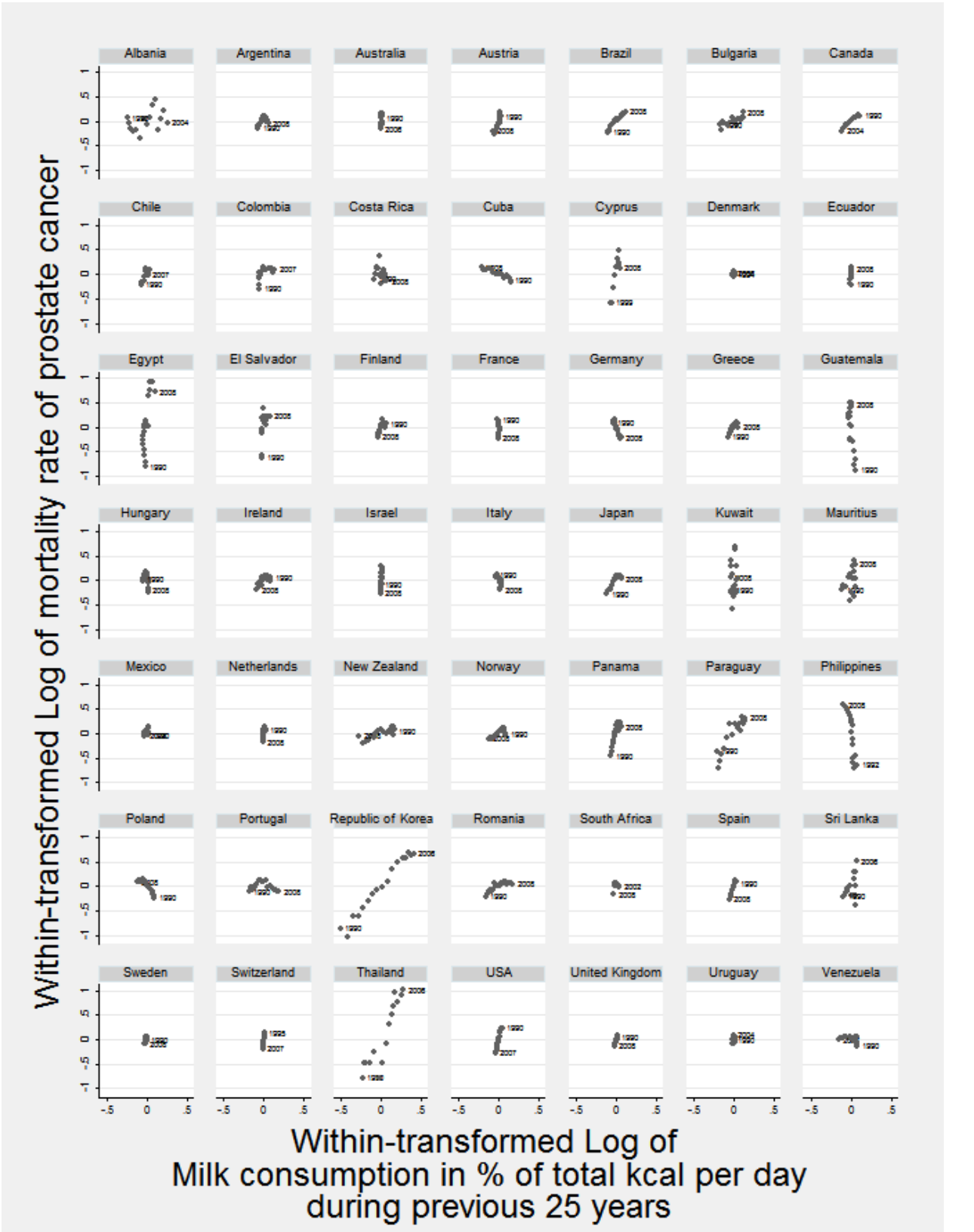


FIGURE 11: WITHIN-TRANSFORMED LOG OF AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER AND THE WITHIN-TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE DURING THE PREVIOUS 25 YEARS

Notes: Number of countries statistically significant positive correlation coefficient: 32 out of 49
Number of countries with statistically significant positive correlation coefficient: 9 out of 49

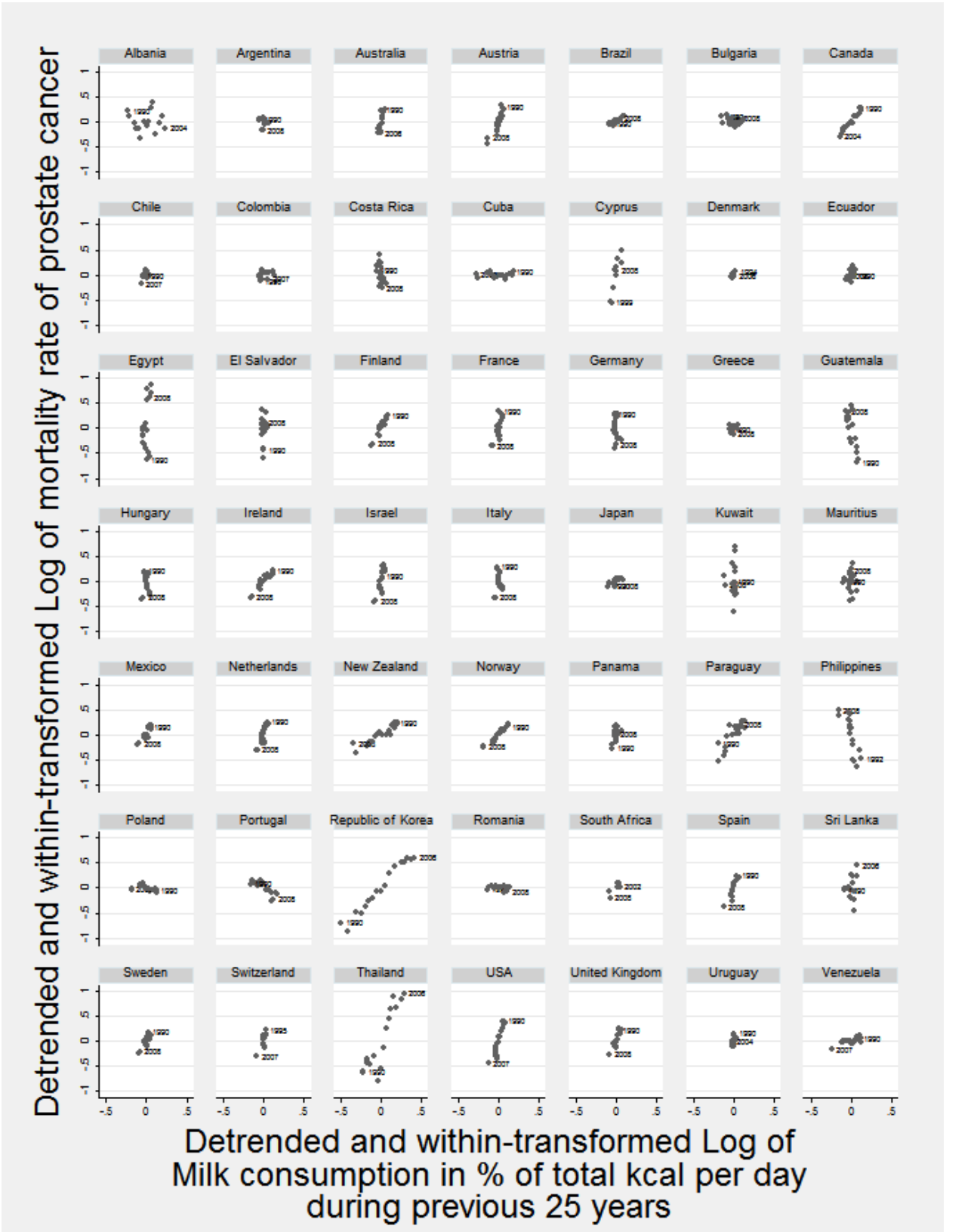


FIGURE 12: TIME DETRENDED WITHIN-TRANSFORMED LOG OF AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER AND THE TIME DETRENDED WITHIN-TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE DURING THE PREVIOUS 25 YEARS

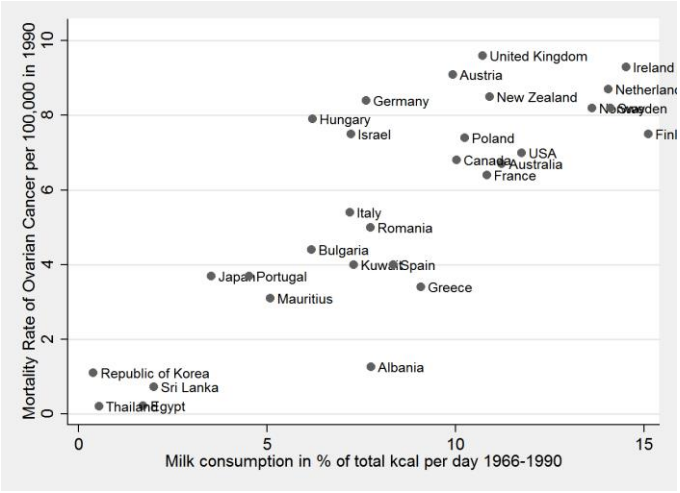
Notes: Number of countries statistically significant positive correlation coefficient: 30 out of 49
Number of countries with statistically significant positive correlation coefficient: 4 out of 49

3.3 Mortality Rate of Ovarian Cancer and Milk Consumption

With following graphs, we repeat our analyses of the last section. Please refer to last section for methodological details. With Figure 13, Figure 14, and Figure 15 simple cross sectional correlations are shown again. In contrast to the mortality of prostate cancer, the correlation does not change much over time. In 2006 (Figure 15) the correlation is still 0.74 and statistically significant at the 1% level.

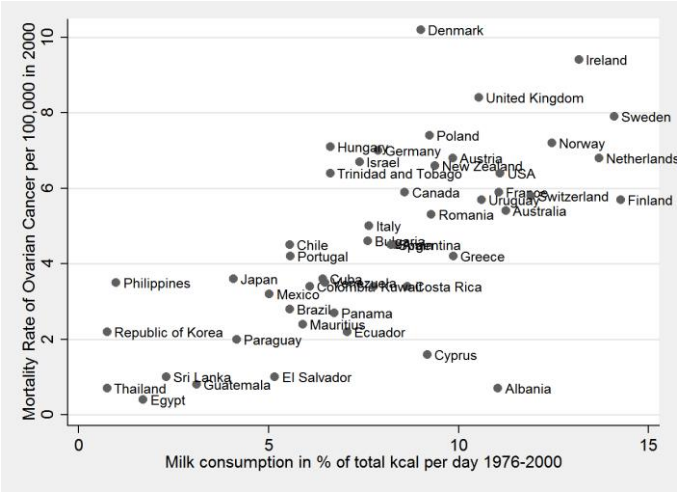
In the following Figures, we perform the same data transformation (natural logarithm, within-transformation, de-trending) as described in the last section:

- The correlation coefficient between the mortality rate of ovarian cancer and the proportion of milk consumption in the preceding 25 years is 0.73 (Figure 16). The number of countries with a statistically significant positive correlation coefficient is 24 out of 50. For 8 out of 50 the correlation coefficient is statistically significant negative (Figure 19).
- The correlation coefficient between the within-transformed log of mortality rate of ovarian cancer and the within-transformed log of proportion of milk consumption in the preceding 25 years is 0.37 (Figure 17).
- The correlation coefficient between the within-transformed and de-trended log of mortality rate of ovarian cancer and the within-transformed and de-trended log of proportion of milk consumption in the preceding 25 years is 0.38 (Figure 18). The number of countries with a statistically significant positive correlation coefficient is 18 out of 50. For 3 out of 50 the correlation coefficient is statistically significant negative (Figure 21).



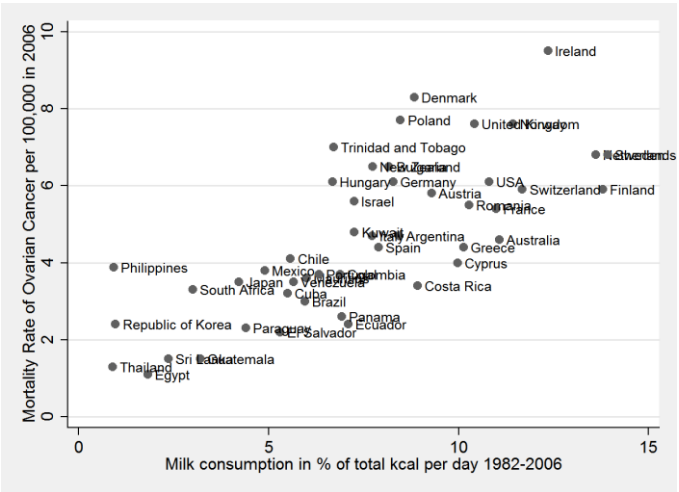
Correlation Coefficient (*p*-value): 0.8137 (0.000), N=30

FIGURE 13: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF OVARIAN CANCER 1990 AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE 1966-1990



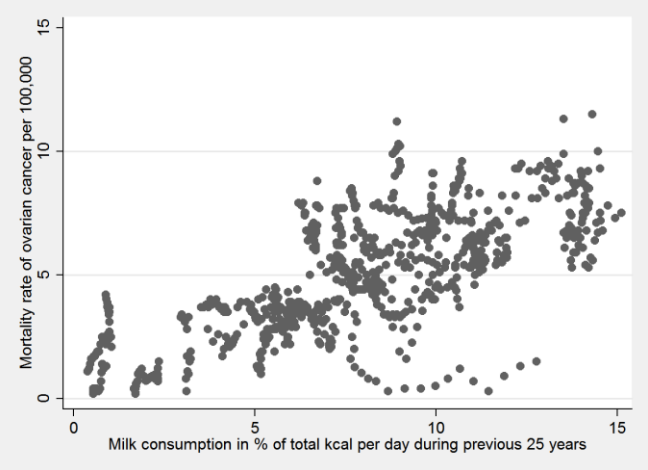
Correlation coefficient (*p*-value): 0.6679 (0.000), N=49

FIGURE 14: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF OVARIAN CANCER 2000 AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE 1976-2000



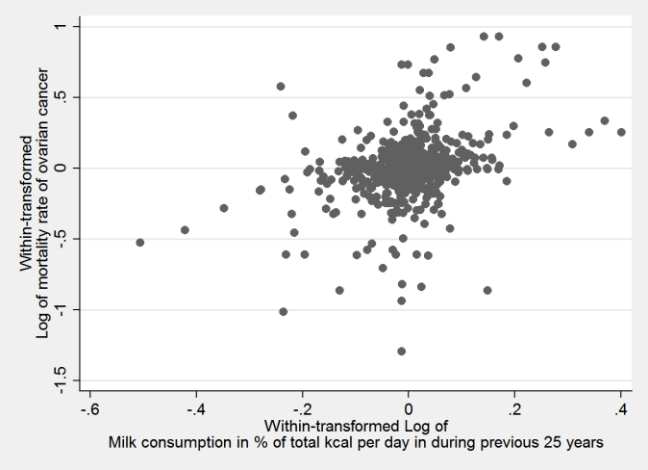
Correlation Coefficient (*p*-value): 0.7361 (0.000), N=47

FIGURE 15: CORRELATION BETWEEN THE AGE-STANDARDIZED MORTALITY RATES OF OVARIAN CANCER 2006 AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE 1982-2006



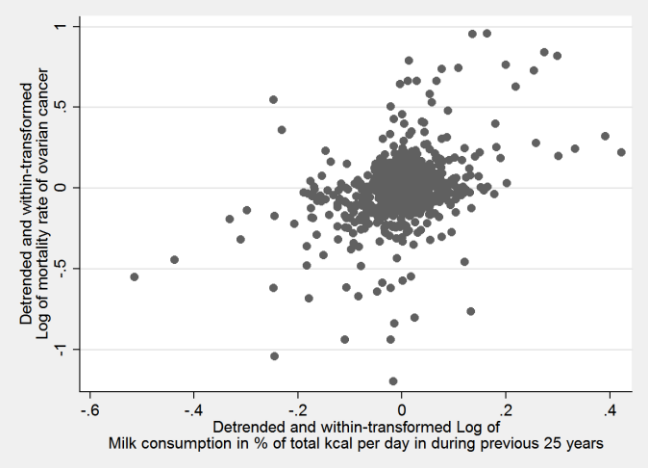
Correlation Coefficient (*p*-value): 0.7259 (0.000), Number of observations: 777

FIGURE 16: AGE-STANDARDIZED MORTALITY RATES OF OVARIAN CANCER AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE



Correlation Coefficient (*p*-value): 0.3722 (0.000), Number of observations: 777

FIGURE 17: WITHIN-TRANSFORMED LOG OF MORTALITY RATES OF OVARIAN CANCER AND THE WITHIN-TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE



Correlation Coefficient (*p*-value): 0.3834 (0.000), Number of observations: 777

FIGURE 18: DETRENDED WITHIN-TRANSFORMED LOG OF MORTALITY RATES OF OVARIAN CANCER AND DETRENDED WITHIN-TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK

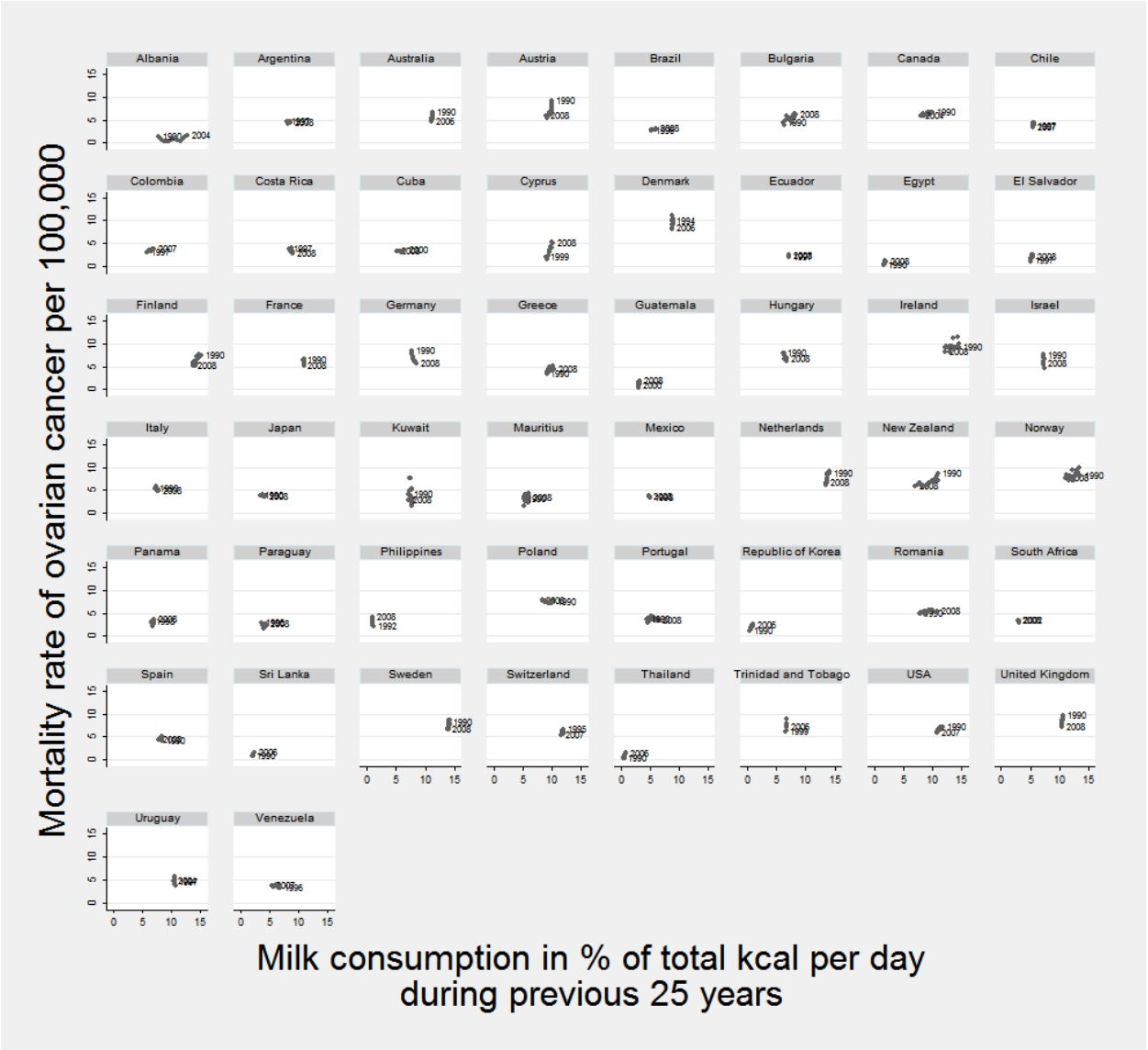


FIGURE 19: AGE-STANDARDIZED MORTALITY RATES OF OVARIAN CANCER AND THE PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE DURING THE PREVIOUS 25 YEARS

Notes: Number of countries statistically significant positive correlation coefficient: 24 out of 50
Number of countries with statistically significant negative correlation coefficient: 8 out of 50

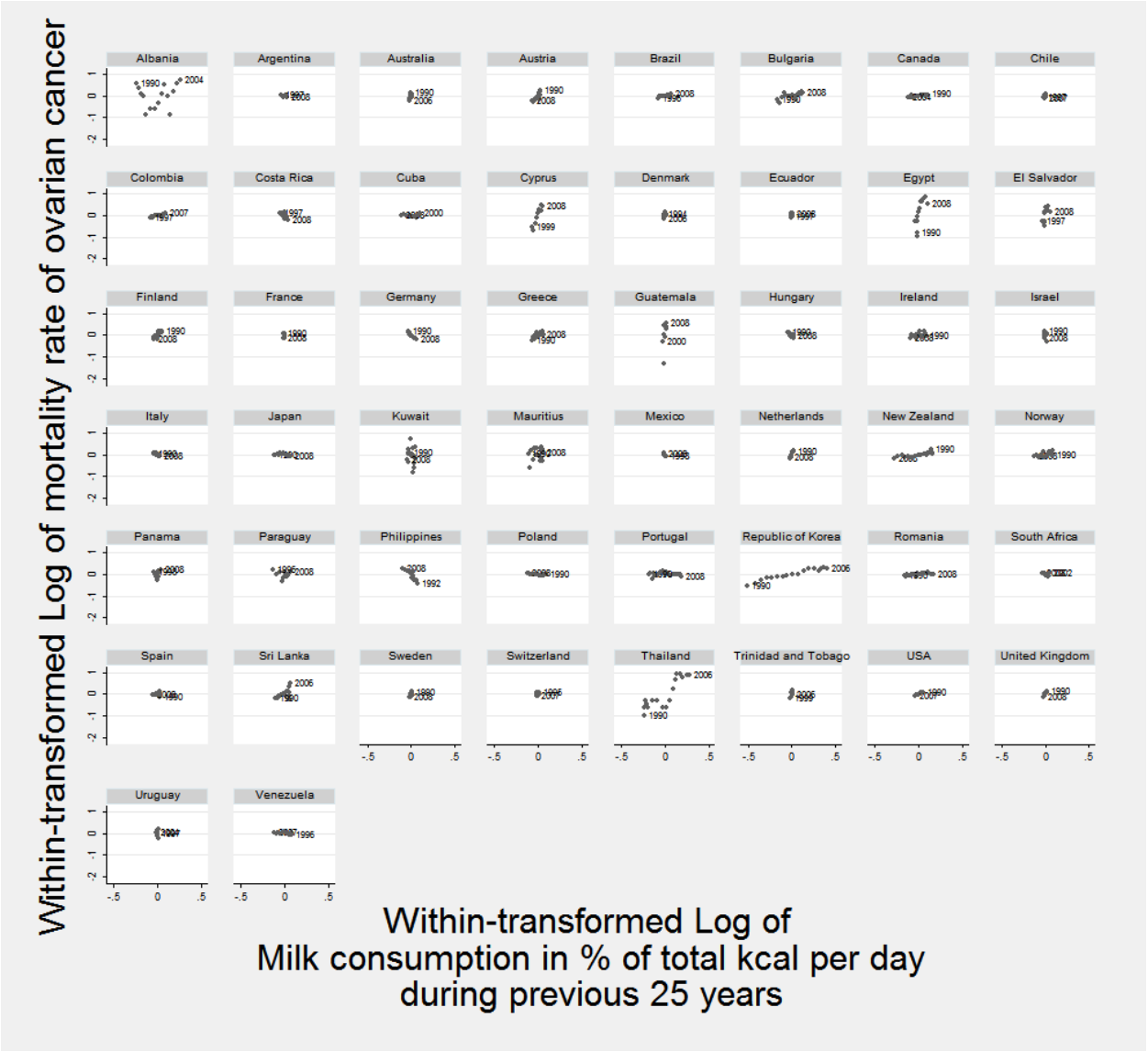


FIGURE 20: WITHIN TRANSFORMED LOG OF AGE-STANDARDIZED MORTALITY RATES OF OVARIAN CANCER AND THE WITHIN TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE DURING THE PREVIOUS 25 YEARS

Notes: Number of countries statistically significant positive correlation coefficient: 24 out of 50
Number of countries with statistically significant negative correlation coefficient: 8 out of 50

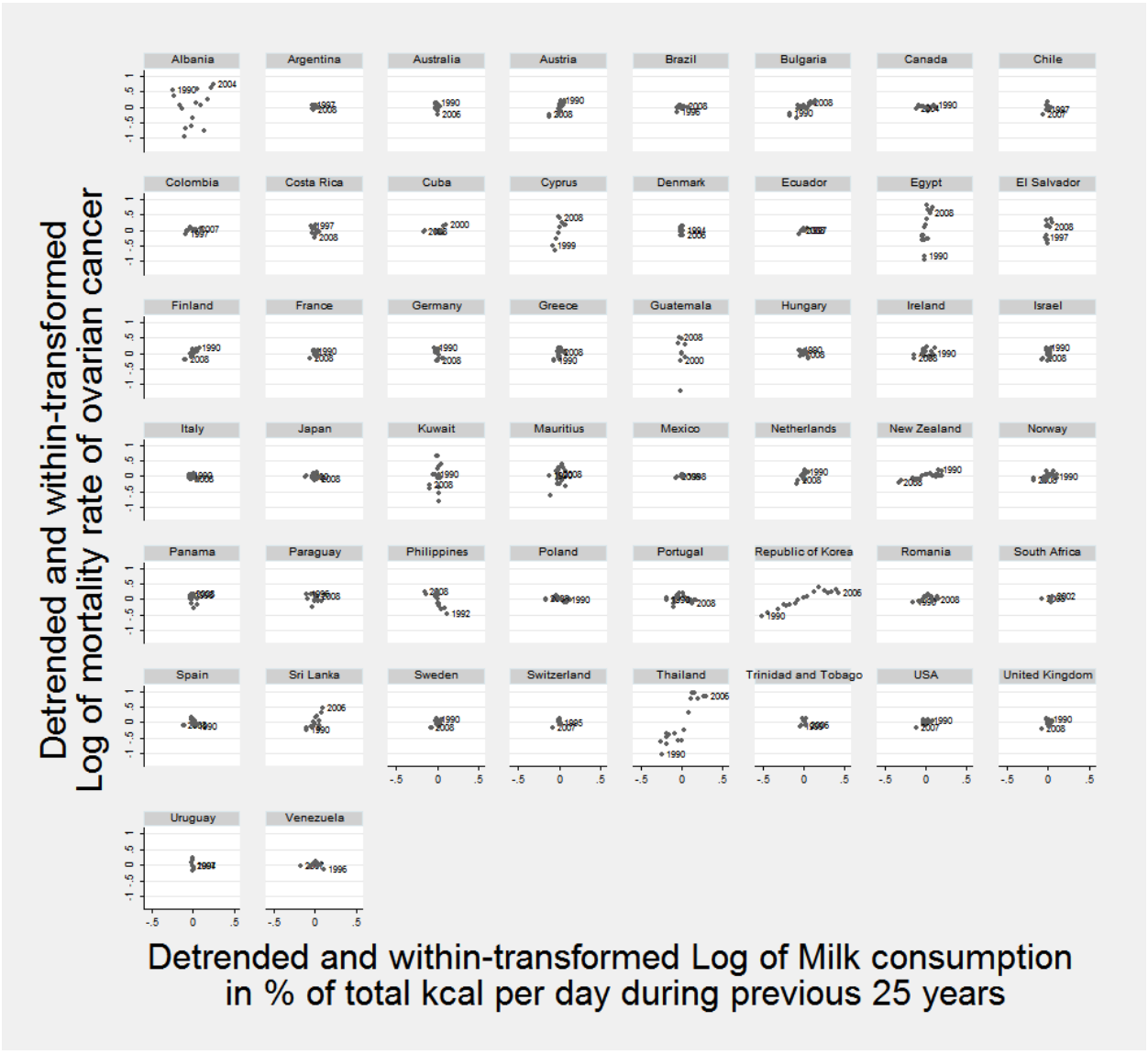


FIGURE 21: TIME DETRENDED WITHIN TRANSFORMED LOG OF AGE-STANDARDIZED MORTALITY RATES OF PROSTATE CANCER AND THE TIME DETRENDED WITHIN TRANSFORMED LOG OF PROPORTION OF CALORIES FROM MILK IN TOTAL CALORIES INTAKE DURING THE PREVIOUS 25 YEARS

Notes: Number of countries statistically significant positive correlation coefficient: 18 out of 50
Number of countries with statistically significant negative correlation coefficient: 3 out of 50

4. Econometric Methods

The aim is to explain the mortality rate of prostate cancer as well as mortality rate of ovarian cancer in an unbalanced annual panel of up to 50 countries (49 countries in most cases) covering the time period 1990 to 2008 by a set of variables for dietary practices covering the period 1966 to 2008.[7] In order to render the empirical results as robust as possible, we use different samples and methodological approaches.

All variables are transformed into natural logarithms since this leads to a better fit of the model[8] and has the advantage that the estimated coefficients can be interpreted as elasticities. That means, they express the percentage change of the dependent variable (mortality rate) if the explanatory variable (e.g. food consumption) increases – ceteris paribus – by one percent. However, it is important to understand that a change in food consumption triggers two possible effects:

1. *Calories effect*: An increase in milk consumption (for example by one percent) increases *total calories intake.* The mortality may be affected simply by calories intake independent from the composition of the dietary.
2. *Composition effect*: An increase in milk consumption changes the *composition* of the total calories intake, which may affect cancer.

In order to be able to distinguish the calories effect from the composition effect we specify the following basic model:

$$\ln(m_{it}) = \alpha_1 \ln(GDP_{it-1}) + \alpha_2 \ln(\overline{total}_{it}) + \beta_1 \ln(\overline{pmilk}_{it}) + \beta_2 \ln(\overline{psugar}_{it}) + \lambda_t + c_i + u_{it} \qquad (1)$$

for a panel of i=1…N (49 or 50) countries and t=1… T_i years (1990 up to 2008). The dependent variable m_{it} is the age-standardized mortality rate of (prostate or ovarian) cancer. u_{it} is an error term with $E[u_{it}] = 0$ and further properties depending on the estimator used.

The variable $\overline{total}_{it}$ indicates the average total calories intake per person over a period of 25 years (year t up to t–24):

$$\overline{total}_{it} = \frac{1}{25} \sum_{j=0}^{24} total_{i,t-j} \qquad \text{for } t = 1990,...,2008. \qquad (2)$$

pmilk is the proportion of calories due to milk consumption in total calories intake:

---

[7] The number of countries, years and observations depends on the explanatory variables used (see Section 3)

[8] Almost a doubling of the within R^2 (from 0.21 to 0.40 in our preferred static fixed effects for prostate cancer specification; see below).

$$\overline{pmilk}_{it} = \frac{1}{25}\sum_{j=0}^{24} pmilk_{i,t-j} = \frac{1}{25}\sum_{j=0}^{24} \frac{milk_{i,t-j}}{total_{i,t-j}} \qquad \text{for } t = 1990,...,2008. \tag{3}$$

Hence, the food variables included into the model are moving averages of the length of 25 years.

In equation (1) $\hat{\beta}_1$ gives the estimated effect of the proportion of milk in total calories intake on the mortality rate *at a given level of total calories intake*, that means, this is the pure *composition effect*. $\hat{\alpha}_2$ is an estimate of the effect total calories intake on the mortality rate. That means, by conditioning on total calories intake and by expressing the food items as calories in percent of total calories intake, it is possible to distinguish the composition effect from the calories effect.

In equation (1) the mortality rate in year *t* is explained by the average food consumption of the current and the previous 24 years. Hence, it is assumed that 25 years of a dietary practice affect mortality. As pointed out by Grant (2014), diet may affect the risk of cancer 10 to 30 years before the cancer develops. Furthermore, it is implicitly assumed that all previous years matter equally for mortality. These two assumptions are arbitrary. One could relax the restriction of equal weights for every year by including 25 variables ($pmilk_{it}$, $pmilk_{i,t-1}$…, $pmilk_{i,t-24}$). This would, however, inflate the number of explanatory variables: instead of one for milk consumption up to 25 variables. Furthermore, one could change the number of years which are included into the moving average in Eq. (2) and (3) above. For example, Ganmaa *et al.* (2002) use up to 30 years for their cross-sectional analyses. Here is a trade-off: the longer the time period covered by the moving averages, the lower the variance of the explanatory variables over time which can be used for the estimation. For this reason we also estimate models with the food variables covering a time period of 20 years only.

Besides the proportion of milk (including cheese) in total calories intake (*pmilk*) we will include also other food items which have been associated with cancer such as sugar (*psugar*) as well as animal fats (including butter) and meat (*pmeat*). As all food items (*pmilk*, *psugar*, *pmeat* etc.) sum up to 100%, at least one food item must be defined as base category and this variable must be omitted. Since, furthermore, we have the problem of low variation and multicollinearity (see Section 3.1) we define all foods of plant origin (fruits, vegetables, cereals, pulses, tree nuts) as base category. Moreover, in order to avoid multicollinearity and due to the fact that we are primarily interested in the effect of milk, we aggregate meat, animals fats (including butter), eggs and fish into one category in the analysis of prostate cancer.

The explanatory variable *GDP* is the gross domestic product per capita in purchasing power parity and in constant international Dollars from the World Bank Open Database. This variable serves as a proxy for the average income of the countries and, hence, controls for private and public health expenditure. This GDP variable is highly correlated with health expenditures per capita[9], which is only available for 1995 to 2008.[10] As a sensitivity analysis in section we will include health expenditures per capita as a control variable instead of GDP per capita. In either case it seems of great importance in cross country studies to control for average income.

The country fixed effects (c_i) are an important part of our estimation strategy. These capture time constant difference between the mortality rates of cancer between the countries which are not already included into the model. c_i may be arbitrarily correlated with each explanatory variable for every t (Wooldridge, 2010) and especially the food variables without leading to an inconsistent estimate of the coefficients. By using these fixed effects (c_i) it is possible to absorb time constant unobserved variables and to avoid that they bias the estimates, called "omitted-variable bias" in econometrics. In epidemiologic research these unobserved variables are termed "confounding factors". In microeconometrics this phenomenon leading to biased results is called "selection on unobservables". Hausman tests will indicate in Section 7 and 8 that random effects models which require c_i to be uncorrelated with the explanatory variables are inconsistent. Hence, all "between estimation strategies" are likely to be inconsistent. Such time constant effects may be, for example, differences in the health system as well as genetic differences affecting the mortality rate. Note that "time constant" does not necessarily mean "forever", but during the period of investigation. Another example for country fixed effects is the geographical location of a country determining the sunlight level which has been related to prostate cancer (see Colli and Colli, 2006).

Another important aspect of our estimation strategy are the fixed (year) time effects (λ_t), specified as a set of up to 18 dummy variables. They may capture medical progress which is common to all countries. Whether and to what extent medical progress leads to a drop in the mortality rates of the countries depends also on the unobserved type of the health system (which is hopefully captured by the country fixed effects c_i) as well as average income captured by GDP per capita.

[9] Health expenditures per capita are available from the world bank open database.
[10] The correlation coefficient of $\ln(GDP_{it-1})$ and $\ln(health_{it-1})$ in the estimation sample is 0.95.

Concerning the econometric approaches, we start the estimation using a simple OLS **fixed effects estimator**. OLS estimation of equation (1) is based on the assumption of strict exogeneity of the explanatory variables, that is, conditional on the fixed effects c_i, all future and past values of the explanatory variables are uncorrelated with the error term u_{it}. The statistical test of Arellano and Bond (1991) rejects the null hypothesis (H0) of no serial correlation of the error term[11], the H0 of homoscedasticity is rejected by a Wald test[12], and the H0 of cross-sectional independence is rejected by the test proposed by Pesaran (2004)[13]. Therefore, we report *t* ratios based on standard errors which are robust to heteroscedasticity, serial and spatial correlation, following Driscoll and Kraay (1998).[14] The error structure of u_{it} is then assumed to be heteroskedastic, autocorrelated, and possibly correlated between the countries (panels). The correlation of u_{it} between the countries may stem, for example, by medical progress in one country affecting the mortality rate in other countries.

As we cannot be sure that there are not any unobservable variables left causing a biased estimate of β_1, we additionally specify a **dynamic model**, that means, we extend equation (1) by the inclusion of a lagged dependent variable $\ln(m_{it-1})$:

$$\ln(m_{it}) = \rho \ln(m_{it-1}) + \alpha_1 \ln(GDP_{it-1}) + \alpha_2 \ln(\overline{total}_{it}) + \beta_1 \ln(\overline{pmilk}_{it}) + \beta_2 \ln(\overline{psugar}_{it}) + \ldots + \lambda_t + c_i + u_{it} \quad (4)$$

We do not think that there is "*true path dependency*" in mortality rate, but a "*spurious*" one resulting from omitted explanatory variables.

Simply applying the OLS Fixed Effects (**FE-OLS**) estimator in a dynamic setup leads to the well-known Nickell bias, which means that the estimator is inconsistent for finite *T* and large *N* (Nickell, 1981; Phillips and Sul, 2007). This not only means a downward bias of the OLS estimate of ρ, but also a biased estimate of the coefficients of the other explanatory variables. Though this bias is known to decrease with the number of time periods, our *T* (here the mean is about 15 in case of mortality of prostate cancer and 13 in case of ovarian cancer) may still be too small to avoid a substantial bias.

One way to deal with the Nickell bias is to use the first-differenced GMM estimator (**FD-GMM**) proposed by Arellano and Bond (1991). In the FD-GMM estimator the fixed effects

[11] The Stata command *abar* by Roodman (2009a) is applied.

[12] The Stata command *xttest3* by Baum (2001) is applied.

[13] We apply the Stata command *xtcsd* by Sarafidis and De Hoyos (2006) implementing the test by Pesaran (2004). However, in order to obtain a sufficient number of years, we apply this test to our preferred specification and only to those 37 countries which have at least 15 time periods.

[14] The Stata command *xtscc* implemented by Hoechle (2007) is applied.

are eliminate by first differencing the variables. The first differenced lagged dependent variable $\Delta y_{i,t-1}$ (as well as further predetermined or endogenous variables) are instrumented by their lagged levels $y_{i,t-1}, y_{i,t-2}, y_{i,t-3}\ldots$. The moment condition for the instruments of the lagged dependent (or other endogenous) variable(s) is $E\left[y_{i,t-s}\Delta u_{is}\right]=0$ for each $t\geq 3$ and $s\geq 2$ (see Roodman, 2009b). This is instrumentation is only valid in case of no second order serial correlation of the error term, which has to be tested.

Since this estimator has been found to have large finite sample bias and poor precisions when the time series are persistent or the ratio of the variance of the fixed effect c_i to the variance of error term u_{it} becomes too large, the system GMM (**SYS-GMM**) estimator by Blundell and Bond (1998) is often preferred. The SYS-GMM uses lagged differences of the variables as instruments for equations in levels, in addition to using as in the FD-GMM lagged levels of the variables as instruments for equations in first differences. This requires the additional moment condition $E\left[\Delta y_{i,t-1}\left(u_{is}+c_i\right)\right]=0$ for each $t\geq 3$. As pointed out by Roodman (2009b) this moment condition is not trivial, but, fortunately, the Hansen test should detect any violation of the assumption,

One further advantage of the GMM estimators is the possibility to treat explanatory variables as endogenous and instrument them in the same way as the lagged dependent variable. Note, that endogeneity does *not* mean correlation of the variable with the country fixed effects c_i, but with the error term u_{it}. All the models estimated in this paper do not require the explanatory variables to be uncorrelated with the fixed effects. A variable x_{it} is *strictly exogenous*, if it is never correlated with u_{it}, that means $E\left[x_{it}u_{is}\right]=0$ for all t and s. The variable is *endogenous* if $E\left[x_{it}u_{is}\right]\neq 0$ for $s\leq t$ and $E\left[x_{it}u_{is}\right]=0$ for all $s>t$. In panel econometrics endogeneity is usually explained by feedback effects: the error term in year s has some feedback on the subsequent realizations of x_{it} in year $t\geq s$. For example, this could be the case if the mortality rate in a certain year and certain country affected the current and future dietary of the whole country. Obviously, this is very unlikely. However, if there are time-varying omitted variables (confounding factors) left which are correlated with the milk variable and if this leads to a correlation of the milk variable with the error term, instrumentation of the milk (and the other food) variable(s) may be a remedy. Furthermore, instrumenting explanatory variables may solve another methodological problem: As pointed out by Bond *et al.* (2001) instrumenting the explanatory variables (including the lagged dependent variable) in the GMM-style noted above is a mean against temporary additive measurement errors in the variables. This seems

surprising since (in case of the FD-GMM) a first-differenced mismeasured variable is instrumented by lagged levels of the mismeasured variable. The measurement error is assumed to be serially uncorrelated. The moment conditions of the FD-GMM becomes $E\left[y_{i,t-s}\Delta u_{is}\right]=0$ for each $t\geq 4$ and $s\geq 3$ and the moment condition of the level equation is $E\left[\Delta y_{i,t-2}\left(u_{is}+c_i\right)\right]=0$ for each $t\geq 4$ (Bond *et al.* 2001). In practical terms, the somewhat stronger moment condition simply means, that one do *not* use the instruments $y_{i,t-2}$ as well as $\Delta y_{i,t-1}$. Apart from that, the same instrument matrix is defined. Whether there is a problem with measurement errors should be detected by the Hansen test of over-identifying restrictions when using the weaker moment condition. Note that permanent (constant) measurement errors are absorbed into the country fixed effects (see Bond *et al.* 2001).

First-differencing is (besides within-transformation) one way to remove the fixed effects c_i, and hence, to eliminate time constant omitted variables / confounding factors. Another possibility proposed by Arellano and Bover (1995) is the forward orthogonal deviation (**FOD**) transformation of variables which subtracts the average of all available future observations (see Roodman, 2009a). Since lagged observations of a variable do not enter the formula for the transformation, they remain orthogonal to the transformed errors if there is no serial correlation and, thus, they are possible instruments. The FOD transformation has the advantage of preserving the sample size in panels with gaps (see Roodman, 2009a). Note that in SYS-GMM with orthogonal deviations, the levels equation is still instrumented with differences as described above. That means, the FOD transformation only applies to the difference equation within the SYS-GMM.

The consistency of all the GMM requires large *N*, which may not be given in our application. However, Monte Carlo simulations show that, given predetermined variables in the explanatory variables *X*, the SYS-GMM estimator has a lower bias and higher efficiency than the FD-GMM or the fixed-effects estimator (Soto 2009). We apply the two-step version of the SYS-GMM. Nevertheless, for macro panels (small *N* of 20 up to 100 individuals, and *T* of 10 up to 20 periods) the Monte Carlo analysis by Judson and Owen (1999) suggest that, that the one-step **FD-GMM1** may be a good choice. Therefore we apply this estimator as well.

The relatively small *N* leads to a further problem: it is not possible to use the full set of instrumental variables since Windmeijer (2005) and Roodman (2009b) show that using too many instruments might bias the results. For this reason, we "collapse" instrument matrix as described in Roodman (2009b). Furthermore, only recent values up to 5 lags are used. A very

rough rule of thumb is to include fewer instruments than cross-sections (countries) and to check whether the results change if the number of instruments is reduced (see Roodman, 2009b).

Another recently proposed approach to deal with the problems arising from a large instrumental variable matrix in the SYS-GMM estimators is to replace instruments with their **principal components** (**PCs**) (Roodman, 2013; Kapetanios and Marcellino, 2010; Bai and Ng, 2010; Mehrhoff, 2009).[15] This seems to be a rather systematic method to reduce the number of instruments since it is a "*minimally arbitrary way to limit the instrument count while minimizing loss of identifying information. It gives the user a way to control this trade-off*."[16] The performance of PC analysis is examined by two measures: Firstly, by the proportion of the variance of the instruments explained by PCs. Secondly, by the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy (see Kaiser, 1974). Both measures take values between 0 and 1 and both should be as high as possible. The KMO measure should not be smaller than 0.60 in order to be acceptable (Kaiser, 1974).

The standard errors of the estimated coefficients of the FD-GMM and SYS-GMM are robust to serial correlation and heteroskedasticity within panels and they are corrected following Windmeijer (2005) in case of the two-step methods.[17] Note that the standard errors are not robust with regard to spatial correlation of the error term.

Another estimation strategy to deal with the Nickell bias when estimating Equation (4) is to apply the bias-corrected least square dummy variable (**LSDVc**) estimator, which has been proposed by Kiviet (1995) and extended by Bruno (2005a, 2005b) to unbalanced panel data,[18] and which turns out to have better properties in case of small N (Bruno 2005a; Judson and Owen 1999). An obvious drawback of this estimator is the assumption of strict exogeneity of all explanatory variables. Furthermore, the error term is assumed to be i.i.d. The standard errors of the LSDVc are bootstrapped.

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[15] Again we apply the Stata command *xtabond2* implemented by Roodman (2013). As explained in Roodmann (2013): "*Principal components analysis is run on the correlation, not covariance, matrix of the "GMM-style" instruments. By default xtabond2 will select all components with eigenvalues at least 1, and will select more if necessary to guarantee that instruments are at least as numerous as regressors, favoring those with largest eigenvalues*."

[16] David Roodman in STATALIST on 9 Jul 2012.
http://www.stata.com/statalist/archive/2012-07/msg00290.html

[17] The Stata command *xtabond2* implemented by Roodman (2009a) is applied.

[18] The Stata command *xtlsdvc* implemented by Bruno (2005b) is applied. The SYS-GMM estimator (Blundell and Bond 1998) is used to initialize the bias correction. The accuracy of the approximation is up to $O(1/NT^2)$.

Recently Han and Phillips (2010) proposed a GMM estimator which has advantages of being not biased in case ρ is near unity or in very small samples.[19] The estimator is not based on $N \to \infty$, but on $NT \to \infty$ which may be attractive in our case (up to 50 countries). The drawback is again the assumption of strict exogeneity of all explanatory variables. Moreover, in contrast to the Arellano and Bond (1991) as well Blundell and Bond (1995) estimators, it requires the error term to be white homoskedastic.

As mentioned, in our data we find serial correlation, heteroskedasticity, cross sectional dependence, and a low within variation. Given the nature of our data, the SYS-GMM with FOD is likely to be the method of choice for estimating the dynamic version of our model, since SYS-GMM works also with low within variation and heteroskedasticity and FOD is way to deal with highly unbalanced data.

A last sensitivity check is to apply quantile regression methods (**QR**). The approaches discussed so far seek to estimate the milk effect at the conditional mean of the distribution of the mortality rate. With QR it is not only possible to estimate the impact of the explanatory on the median of the mortality rate, but also on any quantile of the distribution of the mortality rate. Here we focus on the 0.25-, 0.50- (median), and the 0.75-quantile. A further advantage of QR is that it is less sensitive to outliers than methods estimating the conditional mean. Hence, it is a kind of outlier robust regression.

We perform the QR method with country fixed effects by applying a very simple two-step procedure recently proposed by Canay (2011):

1. Estimation of fixed effects OLS of Equation (1) and calculation of the estimated country specific fixed effects $\hat{c}_i$.
2. Running a pooled quantile regression of $\left(\ln m_{it} - \hat{c}_i\right)$ on the explanatory variables in Equation (1) excluding c_i to obtain quantile regression estimates of the coefficients.

The standard errors are bootstrapped over both steps.

Finally, there is the widely neglected issue of *model uncertainty* about the choice of explanatory variables (see Magnus *et al.* 2010). It would be clearly favorable to include all available explanatory variables into the model in order to avoid an omitted variable bias of the estimated coefficient of the milk variable. However, as explained above, the within variation of our explanatory variables is rather low and this can lead to multicollinarity problems. For this reason, it is important to include only those control variables which show statistically signifi-

[19] We apply the stata command *xtregdhp* implemented by Shehata (2012).

cant coefficients. As stressed by De Luca and Magnus (2012) standard econometric practice of using the same data for model selection (the choice of explanatory variables) and estimating – while ignoring that the resulting estimators are in fact pretest estimators – leads to false inference, since traditional statistical test theory is not directly applicable.

Approaches to deal with this difficulty are the "*extreme bounds analysis*" (**EBA**) (see Leamer, 2008) and the "*Bayesian model averaging*" (**BMA**) technique within a linear regression model (see Magnus *et al.* 2010, and De Luca and Magnus 2012). Here, both the EBA as well as the BMA technique are applied.

The motivation for applying EBA and BMA is threefold:

- Firstly, we want to know whether milk consumption – our variable of interest – robustly affect mortality of cancer.
- Secondly, we want to know which control variables should be included into the models in order to an avoid omitted variable bias of the coefficient of interest.
- Thirdly, at the same time, we want to reduce the number of control variables to minimum in order to avoid multicollinearity. Multicollinearity is most likely a problem since the time variation of the food variables is low due to their specification as 25-years the moving averages.

5. Prostate Cancer: Bayesian Model Averaging and Extreme Bounds Analysis for Model Selection

In this section we try to select the explanatory variables (milk and the control variables) for the model for prostate cancer in a systematic way by applying Bayesian Model Averaging (BMA) techniques as well as extended Extreme Bounds Analysis (EBA).

In BMA techniques the idea is to define two sets of explanatory variables: *focus regressors* which are included in the model on theoretical or other grounds, and *auxiliary regressors* which contain additional explanatory variables of which the researcher is less certain. The results for mortality of prostate cancer can be seen in Table 3 where besides baseline fixed-effects regression results four different specifications are shown.

- First of all, as a kind of baseline, a simple OLS two-way fixed-effects model estimate of Equation (1) with *t*-ratios based on Driscoll-Kraay standard errors is shown. By definition all explanatory variables are focus regressors. However, due to possible multicollinearity this result should not be interpreted.
- In Column (1), the fixed time effects and fixed country effects (not shown) as well as total calories intake are focus regressors. All food items and the GDP are defined as auxil-

iary regressors in order to test which variables should really be included into the model. According to Magnus *et al.* (2010) a rough guideline for the robustness of a regressor is a value of the posterior inclusion probability (*pip*) of 0.5 which corresponds approximately with an absolute t-ratio of 1. By definition, for all focus regressors the *pip* equals 1, since these regressors are included in the model with probability one (see Magnus *et al.* 2010). Most important, the absolute value of the *t*-ratios of *milk* is about 1.7 and the *pip* is near 0.8. Hence, the results in Table 4 clearly indicate that *milk* has an impact which is robust to different model specifications. Therefore, *milk* should be included into the regression model to explain the variation in the mortality rate of prostate cancer. This is in line with the statistically significant effect of milk in the fixed-effects estimate. Also meat, eggs, and sugar clearly should be part of the model. With regard to pulses, vegetables + fruits, fish and vegetable oils as well as the GDP per capita the results of the BMA indicate that these could be dropped from the model. However, as we want to have GDP per capita included into the model for theoretical reasons (mortality of cancer is clearly affected by average income) we define it as a focus regressor. Furthermore, we do not drop the fish variable but aggregate it into one variable together with meat and eggs.

- In Column (2) milk is additionally defined as focus regressor. Despite the aggregation of the meat, fish, eggs variable, the *pip* and the *t*-ratios of the variables vegetables, pulses and vegetable oils still indicate that they can be dropped.
- In Column (3) milk is again defined as auxiliary regressor in order to detect whether it is really a relevant regressor. The *pip* and the *t*-ratio clearly indicate that milk, sugar and meat+fish+eggs should be included into the model while pulses, vegetables and vegetable oils can safely be excluded.
- Finally, we include the lagged dependent variable and ignore the Nickell bias described above.[20] The most important insight from Column (4) is that the *pip* and the *t*-ratio still indicate that milk should be included.

One can conclude from the BMA analysis that the milk variable is indeed an important determinant of the mortality rate of prostate cancer. Also important are variables for sugar as well as meat, fat, fish and eggs. In contrast, all variables for foods of plant origin (fruits, vegetables, cereals, pulses, vegetable oils) seem not to be important determinants.

[20] The reason for not applying one of the "proper" models (such as the LSDVc) introduced in the last section is the computation time which would be needed.

TABLE 3: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – BMA REGRESSION RESULTS

	FE-OLS (with Driscoll-Kraay S.E.)		BMA (1)			BMA (2)			BMA (3)			BMA (4)		
	Coef.	*t*-ratio	Coef.	*t*-ratio	*pip*	Coef.	*t*-ratio	*pip*	Coef.	*t*-ratio	pip	Coef.	*t*-ratio	*pip*
Log mortality rate, *t*-1												0.710	23.56	1.00
Log total calories intake	0.999	2.62	1.130	3.13	1.00	1.020	2.83	1.00	1.020	2.83	1.00	-0.117	-0.42	1.00
Log GDP per capita, *t*-1	0.021	0.15	0.001	0.06	0.04	0.092	0.09	1.00	0.008	0.09	1.00	-.0141	-0.18	1.00
Log milk / total	0.434	2.68	0.329	1.71	0.82	0.547	4.64	1.00	0.545	4.59	1.00	0.238	1.48	0.76
Log sugar / total	1.420	6.08	1.457	9.03	1.00	1.323	9.31	1.00	1.323	9.31	1.00	0.230	1.21	0.67
Log pulses / total	-0.036	-0.42	-0.003	-0.13	0.05	-0.001	-0.08	0.04	-0.001	-0.08	0.04	0.000	-0.03	0.04
Log vegetables and fruits / total	0.068	0.38	0.001	0.04	0.04	0.006	0.16	0.05	0.006	0.16	0.05	0.065	0.55	0.28
Log eggs / total	0.311	4.02	0.359	3.05	0.97									
Log meat and fat / total	0.515	3.15	0.533	3.71	0.99									
Log fish / total	0.053	0.60	0.001	0.07	0.04									
Log meat, fat, eggs & fish / total						0.834	6.13	1.00	0.835	6.13	1.00	0.149	0.86	0.49
Log vegetable oils / total	-0.100	-1.65	-0.007	-0.20	0.07	-0.010	-0.25	0.09	-0.010	-0.25	0.09	0.003	0.14	0.05
No. of observations	797		797			797			797			731		
No. of focus regressors	77		68			70			69			70		
No. of auxiliary regressors	0		9			5			6			6		
No. of models	1		512			32			64			64		

Notes: Fixed time effects, fixed country effects and a constant are not shown (focus regressors).
The estimation results for the auxiliary regressors are marked with a grey background.

The idea of EBA is again, to fit all possible models and to find the minimum and maximum effect size of the variables of interest (Levine and Renelt, 1992). In the following (extended) EBA analysis proposed by Young *et al.* (2013) not only the extreme bounds (the minimum and maximum possible estimate) of the estimated parameter of interest (here the coefficient of the milk variable β_1 and the corresponding standard error) are analyzed, but the whole distribution of possible estimates. This is done by regressing the dependent variable on all combinations of the control variables. If there are *k* control variables (besides milk), there are 2^k possible models to be estimated. By doing this, not only the classical "sampling standard error" of β_1 is estimated, but also the so-called "modeling standard error" resulting from model uncertainty by the list of potential control variables.

The results can be seen in Table 3 and the corresponding distribution of the estimated β_1 coefficient in the following Figures.

In Column (1) we start with a parsimonious specification where we estimate Equation (1) with an OLS fixed-effects estimator. With 3 possible control variables[21], there are only 8 ($=2^3$) models to be estimated. The mean $\hat{\beta}_1$ across the 8 models is 1.388. The total standard error (consisting of the classical sampling standard error as well as the model standard error[22]) is 0.109. The total *t*-ratio of 12.76 indicates by the standards of a t-test a highly statistical significant effect of milk.[23] More important all 8 models lead to positive significant coefficient of milk – the whole distribution of estimates is positive (see Figure 22). Turning to the model influence, that is, the question what controls have the greatest impact on $\hat{\beta}_1$ we find the fixed time effects λ_t to be most import. When the fixed time effects are included into the model, the estimated effect of the milk variable on the mortality rate of prostate cancer is on average 4.8% lower. This means, on the other hand, that the fixed time effects should be included in order to avoid a bias due omitted variables. The analogous argumentation is true for GDP as well as total calories intake.

In Column (2) the whole exercise is repeated with two additional explanatory variables which turned out to be important regressors in the BMA. The mean $\hat{\beta}_1$ shrinks to about 1.

[21] Here, the fixed time effects are counted as one variable; the fixed country effects are no regressors since they are eliminated by within transformation.

[22] The total standard error $\sqrt{V_t}$ is calculated as $\sqrt{V_m + V_s}$ with V_m indicating the modeling variance and V_s the sampling variance.

[23] However, as stressed Young *et al.* (2013) this does not mean that the distribution of estimates follows a *t* distribution. This is a robustness rather than a significant test.

However, the total *t*-ratio of 2.18 indicates by the standards of a t-test a highly statistical significant effect of milk. In 75% out of 32 models the effect of milk is positive and statistically significant. In no single model the effect of milk is negative. Both results are documented with Figure 22. With regard to the model influence it can be seen that especially sugar and the further food of animal sources (meat, fat, eggs, and fish) have an important (negative) effect on the estimate of the milk coefficient. Therefore, both variables should be included into the model.

Column (3) shows the results if regressors which have turned out to be unimportant in terms of the BMA analysis are included, and meat, fish and eggs are included as separate explanatory variables. Again, not a single out of 1,024 models indicate a negative coefficient on the milk variable. However, the total 95% robustness interval overlaps the zero now.

Finally, in Column (4) we include the lagged dependent variable and ignore the Nickell bias described above. The milk variable show in 80% out of 64 models a statistically significant positive coefficient. The 95%-robustness interval does not overlap zero.

TABLE 4: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – EBA RESULTS

	EBA (1)	EBA (2)	EBA (3)	EBA (4)
Model Robustness Statistics:				
Mean $\hat{\beta}_1$	1.388	0.991	0.923	0.690
Sampling SE	0.097	0.345	0.387	0.259
Modeling SE	0.049	0.308	0.330	0.436
Total SE	0.109	0.455	0.512	0.502
Total *t*-ratio:	12.76	2.18	1.82	1.37
Total 95%-Robustness interval	[0.744, 2.113]	[0.099 , 1.880]	[-0.091 , 1.868]	[0.057, 1.797]
Significance Testing $\hat{\beta}_1$:				
Positive	100%	100%	100%	100%
Positive and Sig	100%	75%	62%	80%
Negative	0%	0%	0%	0%
Negative and Sig	0%	0%	0%	0%
Model Influence: Percent Change from Mean $\hat{\beta}_1$				
Log mortality rate, *t*-1				-111.7%
Log total calories intake	-1.9%	-2.3%	-3.4%	-1.3%
Log GDP per capita, *t*-1	-3.4%	-2.8%	-2.3%	-0.6%
Log sugar / total		-48.8%	-61.5%	-35.7%
Fixed time effects	-4.8%	-5.6%	-5.1%	-3.2%
Log meat, fat, eggs & fish / total		-35.6%		-30.8%
Log pulses / total			-1.6%	
Log vegetables and fruits / total			0.7%	
Log eggs / total			-20.7%	
Log meat and fat / total			-24.8%	
Log fish / total			6.9%	
Log vegetable oils / total			0.4%	
No. of observations	797	797	797	731
Possible control terms	3	5	10	6
Number of models	8	32	1,024	64

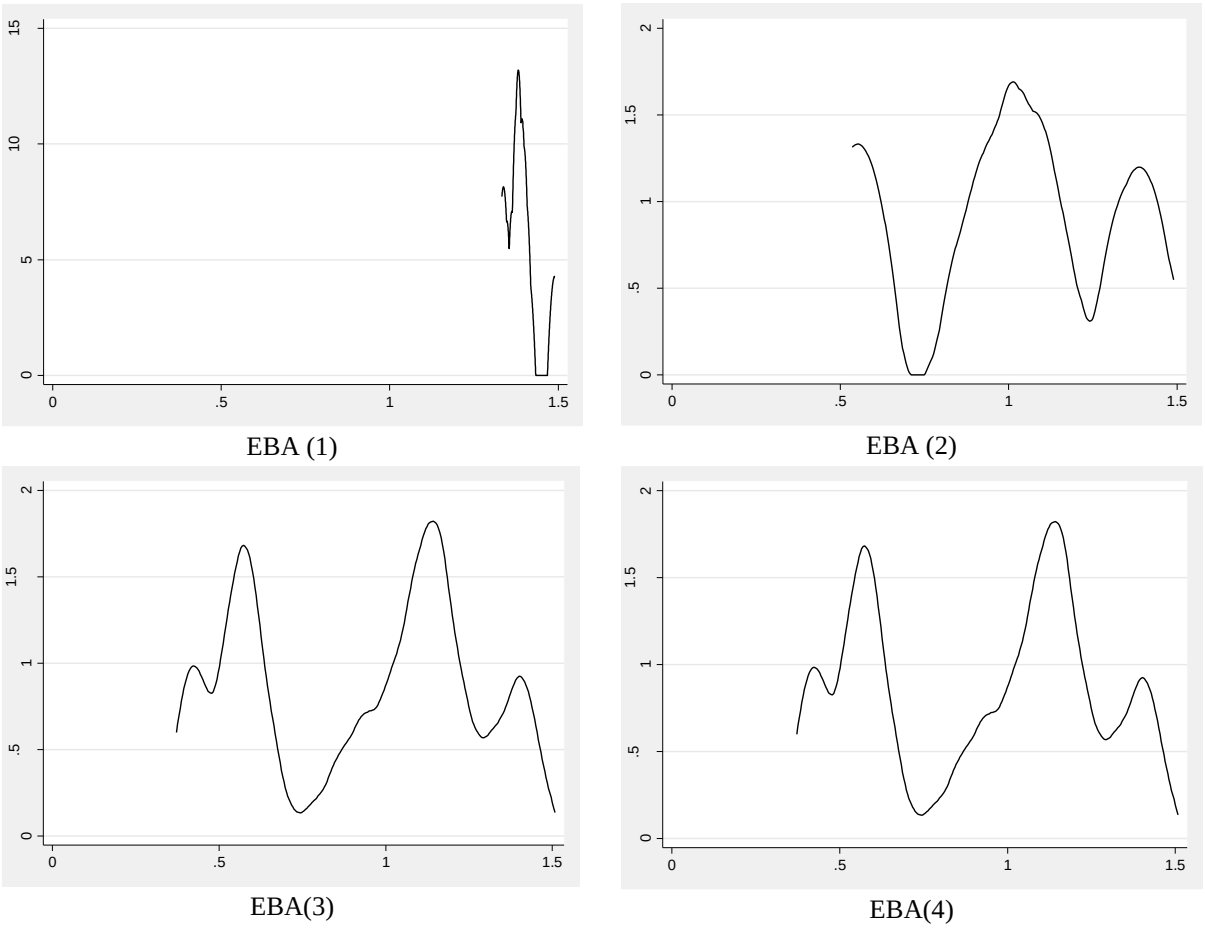


FIGURE 22: MODELLING DISTRIBUTION OF THE ESTIMATED COEFFICIENT FOR MILK IN THE EBA FOR PROSTATE CANCER

We draw the following three conclusions from the analyses (BMA and EBA) of this section:

Firstly, all results indicate that *milk* is a robust regressor for explaining the mortality rate of prostate cancer. Secondly, even if not always statistically significant, the estimated coefficient of milk is in all models positive.

Thirdly, we define the following specification as our preferred one:

$$\ln(m_{it}) = \rho \ln(m_{it-1}) + \alpha_1 \ln(GDP_{it-1}) + \alpha_2 \ln(\overline{total}_{it}) + \beta_1 \ln(\overline{pmilk}_{it}) + \beta_2 \ln(\overline{psugar}_{it}) + \beta_3 \ln(\overline{pmeat + pfish + peggs}_{it}) + \lambda_t + c_i + u_{it} \quad (7)$$

where ρ is restricted to zero in static models. Though *GDP* and *total* calories intake seem not to be robust and important regressors we keep them in the model for the theoretical reasons discussed in Section 4.

6. Ovarian Cancer: Bayesian Model Averaging and Extreme Bounds Analysis for Model Selection

We repeat the whole analysis of the last Section for the determinants of the mortality rate of ovarian cancer. The results can be seen in Table 5 where besides baseline fixed-effects regression results of four different specifications are shown.

First of all, as a kind of baseline, a simple OLS two-way fixed-effects model estimate of Equation (1) with *t*-ratios based on Driscoll-Kraay standard errors is shown. By definition, all explanatory variables are focus regressors. However, due to possible multicollinearity this result should not be interpreted.

In Column (1), the fixed time effects and fixed country effects (not shown) as well as total calories intake are focus regressors. All food variables as well as the GDP are defined as auxiliary regressors in order to test which item should really be included into the model. First of all, the absolute value of the *t*-ratios of the coefficient of the milk variable is 3.69 and the *pip* is 0.99. Hence, the results in Table 5 clearly indicate that milk has a robust impact. Also the variable for eggs should be part of the model. All other variables could be dropped from the model. However, as we want again GDP per capita to be included into to model for theoretical reasons (mortality of cancer is clearly affected by average income) we define it as a focus regressor. Furthermore, we aggregate fish into one variable together with meat and eggs. The *pip* as well as the *t*-ratio of vegetable oils indicate that the variable can be dropped although both measures are near their threshold values. In Column (2) the milk variable is additionally defined as a focus regressor. Sugar is clearly a variable which should not be dropped from the model. Eggs and vegetable oils are near the threshold of being important regressors. In Column (3) milk is again defined as auxiliary regressor in order to detect the whether it is really a relevant for the model. The *pip* and the *t*-ratio clearly indicate that milk and sugar should be included into the model, while vegetable oils and eggs are again near the threshold. Finally, we include the lagged dependent variable and ignore the Nickell bias described above. The most important insight from Column 4 is that the *pip* and the *t*-ratio still indicate that milk should be included.

One can conclude from the BMA analysis that the milk variable is indeed an important determinant of the mortality rate of ovarian cancer.

TABLE 5: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – BMA REGRESSION RESULTS

	FE-OLS (with Driscoll-Kraay S.E.)		BMA (1)			BMA (2)			BMA (3)			BMA (4)		
	Coef.	*t*-ratio	Coef.	*t*-ratio	*pip*	Coef.	*t*-ratio	*pip*	Coef.	*t*-ratio	*pip*	Coef.	*t*-ratio	*pip*
Log mortality rate, *t*-1												0.390	10.77	1.00
Log total calories intake	0.132	0.68	2.035	4.65	1.00	2.022	4.64	1.00	2.023	4.64	1.00	0.736	1.87	1.00
Log GDP per capita, *t*-1	1.855	2.94	0.011	0.23	0.08	0.136	1.28	1.00	0.136	1.29	1.00	0.131	1.25	1.00
Log milk / total	0.581	3.59	0.647	3.69	0.99	0.652	3.94	1.00	0.646	3.74	0.99	0.633	4.90	0.99
Log sugar / total	1.155	7.40	1.102	5.95	1.00	1.070	5.78	1.00	1.072	5.74	1.00	0.043	0.34	0.14
Log pulses / total	0.022	0.27	0.000	-0.01	0.04							0.002	0.10	0.04
Log vegetables and fruits / total	0.010	0.04	0.005	0.12	0.05							0.003	0.07	0.04
Log eggs / total	0.249	1.96	0.161	0.96	0.55	0.147	0.90	0.52	0.150	0.91	0.52	0.033	0.38	0.17
Log meat and fat / total	0.017	0.09	0.004	0.10	0.05							0.002	0.05	0.04
Log fish / total	0.014	0.09	-0.001	-0.03	0.04							0.002	0.10	0.05
Log vegetable oils / total	-0.226	-2.50	-0.100	-0.77	0.43	-0.111	-0.83	0.47	-0.111	-0.83	0.47	0.002	0.11	0.05
No. of observations	708		708			708			708			647		
No. of focus regressors	77		68			70			69			69		
No. of auxiliary regressors	0		9			3			4			8		
No. of models	1		512			8			16			256		

Notes: Fixed time effects and fixed country effects are not shown (focus regressors).
The estimation results for the auxiliary regressors are marked with a grey background.

The results of the extended EBA can be seen in Table 6 and the corresponding distribution of the estimated β_1 in Figure 23.

TABLE 6: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – EBA RESULTS

	EBA (1)	EBA (2)	EBA (3)	EBA (4)
Model Robustness Statistics:				
Mean $\hat{\beta}_1$	1.127	0.871	0.877	0.729
Sampling SE	0.306	0.358	0.318	0.249
Modeling SE	0.031	0.253	0.233	0.228
Total SE	0.318	0.440	0.387	0.333
Total *t*-ratio:	3.54	1.98	2.26	2.19
Total 95%-Robustness interval	[0.489 , 1.748]	[0.063 , 1.781]	[0.142 , 1.663]	[0.183 , 1.475]
Significance Testing $\hat{\beta}_1$				
Positive	100%	100%	100%	100%
Positive and Sig	100%	76%	83%	100%
Negative	0%	0%	0%	0%
Negative and Sig	0%	0%	0%	0%
Model Influence: Percent Change from Mean $\hat{\beta}_1$				
Log mortality rate, *t*-1				-50.2%
Log total calories intake	-1.2%	-3.7%	-5.5%	-2.6%
Log GDP per capita, *t*-1	-1.9%	-1.6%	-2.5%	-0.8%
Log sugar / total		-53.3%	-48.1%	-25.0%
Log pulses / total		1.5%		
Log vegetables and fruits / total		-0.6%		
Log eggs / total		-13.9%	-18.5%	-22.6%
Log meat and fat / total		-7.4%		
Log fish / total		5.7%		
Log vegetable oils / total		8.2%	3.9%	0.4%
Fixed time effects	3.6%	4.4%	3.9%	5.5%
No. of observations	708	708	708	647
Possible control terms	3	10	6	7
Number of models	8	1024	64	128

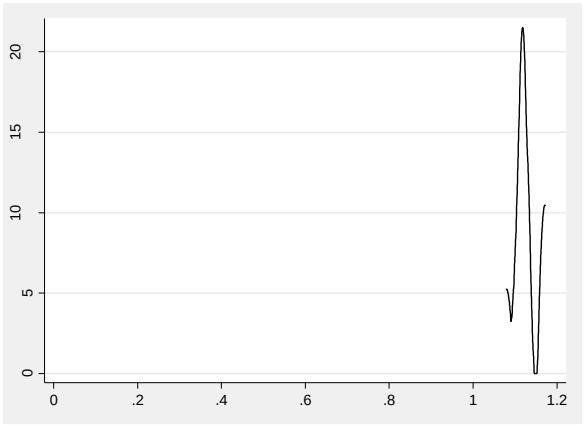
In Column (1) we start with a parsimonious specification where we estimate Equation (1) with an OLS fixed-effects estimator. With 3 possible control variables[24], there are only 8 (=2^3) models to be estimated. The mean $\hat{\beta}_1$ across the 8 models is 1.127. The total standard error is 0.318. The total *t*-ratio of 3.54 indicates by the standards of a t-test a highly statistical significant effect of milk. All 8 models lead to positive significant coefficient of milk – the whole distribution of estimates is positive (Figure 22). Turning to the model influence, that is, the question what controls have the greatest impact on $\hat{\beta}_1$ we find the fixed time effects to be most important.

Column (2) shows a specification where all explanatory food variables are included. Still 76% out of 1,024 models find a statistically significantly positive coefficient of milk. None of

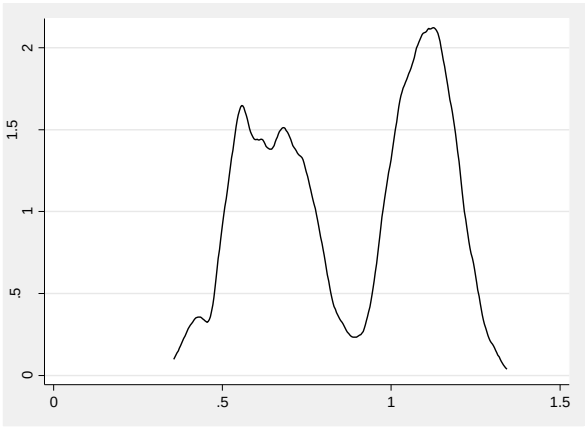
[24] Here, the fixed time effects are counted as one variable; the fixed country effects are no regressors since they are eliminated by within transformation.

the models reveal a negative coefficient. In line with the results of the BMA in Table 5 the most influential regressors (with regard to parameter of interest $\hat{\beta}_1$) are sugar and eggs, followed by vegetable oils as well as meat and fat.

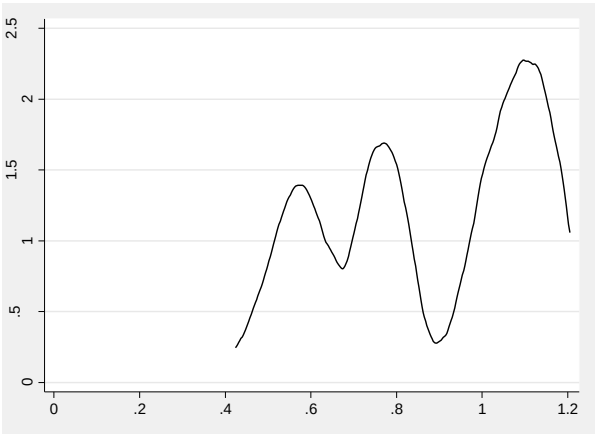
Column (3) shows a specification which will be mostly applied in the econometric analyses. The most important model influence on our parameter of interest comes from the variables sugar, eggs, and total calories intake. Finally Column (4) shows this "preferred specification" extended with a lagged dependent variable. Here the coefficient of interest is positive and statistically significant in 100% out of 128 models.



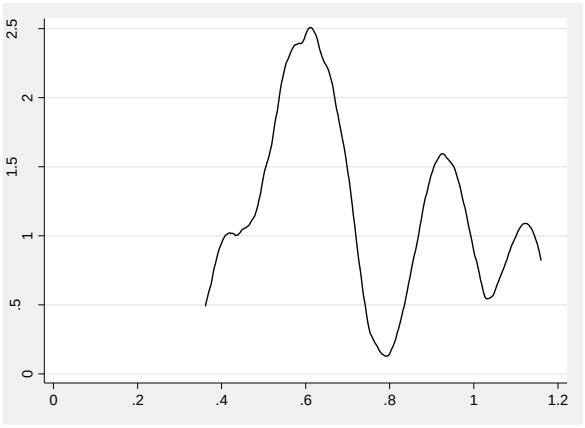
EBA (1)



EBA (2)



EBA(3)



EBA(4)

FIGURE 23: MODELLING DISTRIBUTION OF THE ESTIMATED COEFFICIENT FOR MILK IN THE EBA FOR OVARIAN CANCER

Summarizing, we draw the following four conclusions from the analyses (BMA and EBA) of this section for the mortality of ovarian cancer:

Firstly, all results indicate that *milk* is a robust regressor for explaining the mortality rate of ovarian cancer. Secondly, even if not always statistically significant, the estimated coefficient of milk is in all models positive.

Thirdly, we define the following specification as our preferred one:

$$\ln(m_{it}) = \rho \ln(m_{it-1}) + \alpha_1 \ln(GDP_{it-1}) + \alpha_2 \ln(\overline{total}_{it}) + \beta_1 \ln(\overline{pmilk}_{it}) + \beta_2 \ln(\overline{psugar}_{it}) + \beta_3 \ln(\overline{peggs}_{it}) + \beta_4 \ln(\overline{poil}_{it}) + \lambda_t + c_i + u_{it} \quad (8)$$

where ρ is restricted to zero in static models. Although *GDP* seem not to be a robust and important regressor, we keep it in the model for the theoretical reasons discussed in Section 4.

Fourthly, as both variables – the proportion of eggs and proportion of vegetable oils – are not robust regressors in all cases, β_3 and β_4 are restricted to zero in some specifications.

7. Results for Models Explaining the Mortality Rate of Prostate Cancer

Table 7 presents the estimation results of the static two-way fixed effects models derived in Section 4 and Section 5. Static means that ρ is restricted to zero. Estimation is based on OLS; the *t* statistics are based on Driscoll and Kraay (1998) standard errors. The Driscoll and Kraay (1998) standard errors are larger than the "usual" FE standard errors. However, surprisingly they are smaller than the White (1980)-robust standard errors being only robust against serial correlation and heteroskedasticity, but not robust cross sectional dependence.[25] The Driscoll-Kraay standard errors are based on large *T*; the White standard errors are based on large *N*. Since furthermore the tests on cross-sectional dependence are ambiguous, estimates of the usual standard errors and the White (1980)-robust standard errors are shown in Table A 4 in the Appendix. Recently, Vogelsang (2012) developed a asymptotic theory for the Driscoll-Kraay standard errors. As his suggested procedure lead to smaller standard errors here, and since we want to be as conservative as possible with our inference, we show the *p*-values of the "usual" Driscoll and Kraay (1998) standard errors. Alternative results are shown in Table A 4 in the Appendix.

All specifications include fixed time affects (year dummies), which are always jointly significant at the 1% level. At the bottom of Table 7 we show some further relevant information: the sample mean of the mortality rate, the sample mean of the proportion of milk, the proportion of total calories which is covered by the included food items (proportion of milk, sugar, meat etc.), the within R^2, as well as the correlation coefficient of the country fixed effects with the explanatory variables. The latter already indicate that a random effects specification is likely to be inconsistent as it assumes a zero correlation. Moreover, we report the Arellano and Bond (1991) test of serial correlation (AR(1) and AR(2)).

[25] As pointed out by Daniel Hoechle "*Such a situation can arise if the residuals between two cross-sectional units are on average negatively correlated*." http://www.stata.com/statalist/archive/2007-05/msg00181.html

We start in Column (1) with a specification, where the (log of the age standardized) mortality rate of prostate cancer is only explained by the (log of) GDP per capita in the previous year. Surprisingly the estimated coefficient is positive. This may be explained by dietary practices being correlated with the GDP. This can be seen in Columns (3) and (4): After including the proportion of milk and the proportion of sugar in total calories intake the estimated coefficient becomes small and statistically insignificant. Both milk and sugar show statistically significant positive effects. That means: the higher the proportion of milk (or sugar) in total calories intake (at a given total calories intake level), the higher is the mortality rate. The estimated coefficient of the total calories intake variable is around 1.0 indicating that an x% increase in total calories intake (at a given GDP, dietary composition, year and country) increase the mortality rate of prostate cancer by x%.

Column (5) shows our preferred static specification based on the analyses in Section 5. The additionally included variable for the proportion of meat, fat, fish and eggs in total calories intake has a statistically significant coefficient. Most important the coefficient for milk is still highly statistically significant. With the three food item variables 36.7% of calories intake is covered.

In order to demonstrate that the efficient random effects Generalized Least Squares (GLS-RE) model is inconsistent we show in Column (6) the results of a GLS-RE model using the same explanatory variables as in Column (5). Though the estimated coefficients are comparable a Hausman test indicates that der RE-GLS is inconsistent. The Hausman test is a χ^2-test of the null hypothesis that the difference in coefficients are not systematic.[26] As expected, the null hypothesis is rejected (*p*-value: 0.0066) and hence the RE method is inconsistent and will not be considered further.

In Column (7) and (8) further food variables are included. The estimated coefficient for the milk variable hardly changes.

[26] While FE is consistent under H0 and Ha, RE is inconsistent under Ha and efficient under H0.

TABLE 7: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – STATIC FIXED EFFECTS RESULTS (*p*-values based on robust Standard Errors)

	(1) FE-OLS	(2) FE-OLS	(3) FE-OLS	(4) FE-OLS	(5) FE-OLS	(6) RE-GLS	(7) FE-OLS	(8) FE-OLS
Log GDP per capita, *t*-1	0.319***	0.429***	0.179*	-0.0218	0.0097	-0.160**	-0.0233	-0.0369
	(0.000)	(0.002)	(0.097)	(0.805)	(0.919)	(0.020)	(0.864)	(0.799)
Log total calories intake		1.279***	1.115***	1.530***	1.029**	0.819***	1.055**	0.916**
		(0.000)	(0.009)	(0.001)	(0.024)	(0.009)	(0.031)	(0.047)
Log milk / total			**1.332*** **	**0.891*** **	**0.545*** **	**0.483*** **	**0.540*** **	**0.562*** **
			(0.000)	(0.000)	(0.001)	(0.000)	(0.003)	(0.001)
Log sugar / total				1.272***	1.319***	1.374***	1.351***	1.388***
				(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Log meat, fat, fish & eggs / total					0.837***	0.711***	0.786***	0.793***
					(0.000)	(0.000)	(0.000)	(0.000)
Log vegetables and fruits / total							0.141	0.158
							(0.425)	(0.360)
Log pulses / total							-0.0589	-0.0295
							(0.494)	(-0.706)
Log vegetable oils / total								-0.125*
								(0.067)
Numb. of obs.	1133	797	797	797	797	797	797	797
Numb. of countries	71	49	49	49	49	49	49	49
Av. numb. of years	16.0	16.3	16.3	16.3	16.3	16.3	16.3	16.3
Min. numb. of years	5	7	7	7	7	7	7	7
Max. numb. of years	19	19	19	19	19	19	19	19
AR(1)-test (*p*-value)	0.0001	0.0007	0.0006	0.0003	0.0002		0.0001	0.0001
AR(2)-test (*p*-value)	0.0004	0.0029	0.0048	0.0035	0.0016		0.0012	0.0011
Mean *mrate*	15.85	17.34	17.34	17.34	17.34	17.34	17.34	17.34
Mean *milk / total*	7.7%	7.7%	7.7%	7.7%	7.7%	7.7%	7.7%	7.7%
Prop. of kcal in *X*	0%	0%	7.7%	20.7%	36.7%	36.7%	44.1%	54.2%
within R^2	0.1284	0.1175	0.2948	0.3634	0.3952	0.3905	0.3963	0.3980
Corr. coef. (c_i, X_{it})	0.2697	-0.2213	-0.7165	-0.5129	-0.5839	0	-0.5624	-0.5260

Notes: Fixed time effects and fixed country effects are not shown
p-values based on Driscoll-Kraay standard errors in parentheses, Exception: the *p-values* statistics of the random effects estimator are based on conventional standard errors.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

In the following we will turn to the dynamic analysis, that is, we allow ρ to be different from zero. Doing so we only present results for our preferred specification in Eq. (7) (a dynamic version of Column (5) of Table 7), with the mortality being explained by the lagged mortality rate, GDP, total calories intake, sugar, meat+fat+fish+eggs as well as, of course, fixed country effects and fixed time effects. In Table 8 all explanatory variables *X* (except the lagged dependent) are assumed to be strictly exogenous.

In order to obtain the effects from the dynamic models which are comparable to those of the static models, the estimated coefficients $\hat{\beta}$ have to be divided by $(1-\hat{\rho})$. The resulting long-term elasticities (LT elast.) and the corresponding *p*-values of these LT elast. are shown in the middle part of Table 8. We start with a simple FE-OLS estimator (again with Driscoll-Kraay standard errors). Although the time dimension is with an average of 14.9 years not "small",

the estimates may still be affected by the Nickell bias described in Section 4. All food composition variables are statistically significant. The long-term elasticities are statistically significant and show a comparable magnitude as the coefficients from the static models in Table 7.

In Column (2) the results of the LSDVc estimator are shown.[27] All but the lagged dependent variable are statistically significant.[28] Correspondingly, all estimated long-term elasticities are far from being significant. It is, however, striking that the magnitudes of the (statistically insignificant) long-term elastiticies are again comparable to the uncorrected dymamic FE-OLS as well as the static FE-OLS in Table 7. The LSDVc may not be suitable as it assumes an i.i.d. error term.

The Columns (3) to (6) show the results of the Arellano and Bond (1991) / Blundell and Bond (1995) GMM estimators. Two tests are relevant for all of them: the Hansen test of over-identifying restrictions, with the null hypothesis that the instruments are uncorrelated with the error term, and the Arellano and Bond (1991) test of second order serial correlation (AR(2)) are reported. The *p*-values of the Hansen test indicate that the models' over-identifying restrictions cannot be rejected. The second order serial correlation test statistics of the residuals do not reject the specification of the error term. The number of instruments is chosen in a way to prevent that the number of instruments exceeds the number of countries (49). This is realized by using the "*laglimit*" as well as the "*collapse*" option of the stata command *xtabond2* by Roodman (2013).

In Column (3) the one-step GMM with forward orthogonal deviation of the variables (FOD-GMM1) is shown. The estimated coefficients change significantly in comparison to the inconsistent dynamic fixed-effects estimator in Column (1). However, the resulting long-term elasticities hardly change.

Column (4) shows the results of a SYS-GMM and Column (5) the results of a SYS-GMM with FOD (see Section 4). Both show a statistically significant long-term effect of milk. In Column (6) the results of SYS-GMM estimator are presented, where the instruments are replaced with their principal components (PCs) in order to reduce the number of instruments. The 20 components with the largest *eigenvalues* are chosen as instruments. In Column (6) for the very first time the long term elasticity of the mortality rate with regard to the GDP is statistically significant at the 10% level and is -0.19. The remainder of the long-term elasticities

[27] Bias correction up to order O(1/NT^2). Bias correction initialized by SYS-GMM estimator. 1,000 Bootstrap replications.

[28] This may be explained by the fact that the standard errors are based on a parametric bootstrap method, which assumes normality and homoscedasticity of the error term.

hardly changes. As a "visual" test of the goodness of fit in Figure A 1 in the Appendix the observed values of the dependent variable and the predicted values based on the model in Column (5) are shown. The fit seems to be excellent.

Finally, the results of the Han and Philips (2010) estimator in Column (7) turn out to be inconsistent here, since all tests reject homoscedasticity. [29]

In Table 9 the food variables are treated as endogenous and they are instrumented in the same way as the lagged dependent variable. As explained in Section 4 instrumenting is a way of dealing with a correlation of explanatory variables with the error term due to time-varying omitted variables as well as a method to handle time-varying measurement errors.

As impressively demonstrated by Roodman (2009b), using too many instruments can lead to several statistical problems, especially a so-called overfitting bias of the coefficients of the endogenous variables and a weak Hansen test of instrument validity. As a solution Roodman (2009b, p. 156) suggests "*[r]esults should be aggressively tested for sensitivity to reductions in the number of instruments.*" As pointed out in Section 4, a systematic way to do this is to apply principal component (PC) analysis for the instrument matrix.

In Table 9 we start in Column (1) with an obviously "overfitted" model with 89 instruments which leads to the misleading results of a *p*-value of 1.000 of the Hansen Test. Apart from the lagged dependent variable the milk variable is the only regressor which shows a statistically significant coefficient. The same is true for the LT elasticities. In Column (2) the number of instruments is cut in half. The point estimates hardly change and with increasing standard errors all estimated coefficients and long-term elasticities become insignificant. In Columns (3) to (8) we use PCs instead of the instruments and reduce the number of PCs step by step from 30 (which means 49 instruments here) to 10 (29 instruments). A further reduction leads to an insignificant coefficients on all (including the lagged dependent) variable. In summary, almost all variables, but the lagged dependent, and almost all long-term elasticties are statistically insignificant. If there is a coefficient at all, that is significantly positive, than it is the coefficient of the milk variable. The point estimates of the long-term elasticities of the milk variable are (in case of "smaller" *p*-values in Column(1), (2), (3) and (8)) near the magnitude found in the specifications without instrumentation. We interpret the latter as an indication for the plausibility of our assumption that the milk variable is strictly exogenous.[30]

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[29] H0: Panel Homoscedasticity. Lagrange Multiplier Test: *p*-value = 0.0000, Likelihood Ratio Test: *p*-value = 0.0000; Wald Test: *p*-value= 0.0000

[30] This, of course, is not a rigorous statistical test. But the statement is within the logic of the Wu-Hausman test of endogeneity in the case of IV-two-stage least squares. If there is no endogeneity, both OLS and IV are

TABLE 8 : DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – DYNAMIC PANEL MODEL RESULTS (*p*-values) ; FOOD VARIABLES ARE TREATED AS STRICTLY EXOGENOUS

	(1) FE-OLS	(2) LSDVc	(3) FOD-GMM1	(4) SYS-GMM	(5) SYS-GMM FOD	(6) SYS-GMM 20 PCs[31]	(7) Han-Philips (2010)
Log mortality rate, *t*-1	0.690***	0.828***	0.309**	0.594***	0.671***	0.702***	0.548***
	(0.000)	(0.000)	(0.028)	(0.000)	(0.000)	(0.000)	(0.000)
Log GDP per capita, *t*-1	-0.003	0.011	0.0233	-0.0202	-0.050	-0.056	0.066
	(0.959)	(0.910)	(0.874)	(0.684)	(0.312)	(0.163)	(0.604)
Log total calories intake	-0.136	-0.361	0.145	-0.147	0.054	-0.0088	0.241***
	(0.584)	(0.356)	(0.855)	(0.615)	(0.797)	(0.964)	(0.690)
Log milk / total	0.218***	0.145	0.465**	0.174**	0.148	0.128**	0.768***
	(0.014)	(0.250)	(0.042)	(0.018)	(0.101)	(0.039)	(0.000)
Log sugar / total	0.329**	0.180	0.787**	0.238**	0.293**	0.210*	0.984***
	(0.037)	(0.189)	(0.022)	(0.018)	(0.030)	(0.064)	(0.000)
Log meat, fat, fish & eggs / total	0.267*	0.170	0.616*	0.169**	0.181**	0.141*	0.906***
	(0.021)	(0.203)	(0.081)	(0.035)	(0.028)	(0.080)	(0.000)
LT elast. (*p*-value)							
Log total calories intake	-0.43	-2.10	0.21	-0.36	0.16	-0.03	0.53
	(0.585)	(0.359)	(0.854)	(0.597)	(0.797)	(0.964)	(0.692)
Log milk / total	**0.70*** **	**0.85**	**0.68***	**0.43*** **	**0.45*** **	**0.43*** **	**1.70*** **
	(0.002)	(0.247)	(0.052)	(0.003)	(0.002)	(0.001)	(0.005)
Log sugar / total	1.06**	1.05	1.14**	0.58***	0.89***	0.71***	2.18***
	(0.016)	(0.207)	(0.013)	(0.006)	(0.002)	(0.000)	(0.004)
Log meat, fat, fish & eggs / total	0.86***	0.99	0.89**	0.42***	0.55**	0.47**	2.01***
	(0.002)	(0.185)	(0.045)	(0.003)	(0.011)	(0.001)	(0.005)
Numb. of obs.	731	731	682	731	731	731	731
Numb. of countries	49	49	49	49	49	49	49
Numb. of instruments			**38**	**41**	**40**	**43**	
Av. numb. of years	14.9	14.9	13.9	14.9	14.9	14.9	14.9
Min. numb. of years	5	5	4	5	5	5	5
Max. numb. of years	18	18	17	18	18	18	18
within R^2	0.6740						
AR(1)-test (*p*-value)							
AR(2)-test (*p*-value)	0.3867	-	0.257	0.578	0.441	0.489	
Hansen test joint validity of instr. (p-value)			0.822	0.374	0.416	0.589	
Difference-in-Hansen tests of exogeneity of instrument subsets							
GMM instruments for levels							
Hansen test excluding group (*p*-value)				0.351	0.358		
Difference (H0 = exogenous) (*p*-value)				0.405	0.681		

Notes: Fixed time effects (and fixed country effects) are not shown. The *p-values* in (1) are based on Driscoll-Kraay standard errors. The *p-values* in (2) are boostrapped (1000 replications) assuming homoscedastic residuals being uncorrelated over time and between countries. In (3) the *p-values* are based on standard errors being robust to heteroskedasticity and autocorrelation. The *p-values* in (4) to (6) are based Windmeijer's (2005) finite-sample correction for the two-step covariance matrix being robust to heteroskedasticity and autocorrelation within panels. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

consistent, but IV is inefficient. The Idea of the Wu-Hausman test is to see if the estimates from OLS and IV are different.

[31] Extracted 20 principal components from GMM-style instruments; Portion of variance explained by the components = 0.564; Kaiser-Meyer-Olkin measure of sampling adequacy =0.786.

TABLE 9: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – DYNAMIC PANEL MODEL RESULTS (*p*-values) – FOOD VARIABLES ARE TREATED AS ENDOGENOUS AND ARE INSTRUMENTED

	(1) SYS-GMM FOD	(2) SYS-GMM FOD	(3) SYS-GMM FOD 30 PCs	(4) SYS-GMM FOD 28 PCs	(5) SYS-GMM FOD 25 PCs	(6) SYS-GMM FOD 20 PCs	(7) SYS-GMM FOD 15 PCs	(8) SYS-GMM FOD 10 PCs
Log mortality rate, *t*-1	0.808***	0.808***	0.770***	0.808***	0.866***	0.892***	0.965**	-0.111
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.017)	(0.931)
Log GDP per capita, *t*-1	-0.00966	0.0105	-0.0717	-0.0551	-0.0635	-0.0690	-0.0401	0.101
	(0.854)	(0.803)	(0.443)	(0.412)	(0.355)	(0.497)	(0.681)	(0.836)
Log total calories intake	-0.247	-0.208	0.745	0.471	0.553	0.464	0.956	-0.948
	(0.658)	(0.691)	(0.428)	(0.513)	(0.378)	(0.383)	(0.686)	(0.738)
Log milk / total	0.154*	0.130	0.136	0.129	0.0965	0.0882	-0.00583	0.898
	(0.067)	(0.139)	(0.244)	(0.259)	(0.168)	(0.264)	(0.987)	(0.108)
Log sugar / total	0.0696	0.063	0.223	0.206	0.126	0.135	0.214	-1.418
	(0.649)	(0.668)	(0.533)	(0.536)	(0.648)	(0.618)	(0.461)	(0.603)
Log meat, fat, fish & eggs / total	0.0393	0.0180	-0.0431	-0.0424	-0.0596	-0.0481	-0.225	0.150
	(0.700)	(0.865)	(0.764)	(0.778)	(0.722)	(0.786)	(0.786)	(0.907)
LT elasticities (*p*-value)								
Log total calories intake	-1.29	-1.09	3.242	2.448	4.126	4.294	27.24	-0.853
	(0.6556)	(0.6859)	(0.399)	(0.505)	(0.434)	(0.332)	(0.942)	(0.704)
Log milk / total	**0.801***	**0.68**	**0.590**	**0.672***	**0.720**	**0.817**	**-0.166**	**0.808**
	(0.0513)	(0.1428)	(0.104)	(0.093)	(0.182)	(0.165)	(0.989)	(0.104)
Log sugar / total	0.36	0.33	0.970	1.070	0.943	1.249	6.096	-1.276
	(0.6331)	(0.6500)	(0.428)	(0.449)	(0.612)	(0.582)	(0.930)	(0.734)
Log meat, fat, fish & eggs / total	0.204	0.09	-0.187	-0.221	-0.445	-0.445	-6.397	0.135
	(0.6854)	(0.8927)	(0.783)	(0.795)	(0.752)	(0.800)	(0.947)	(0.895)
Numb. of obs.	731	731	731	731	731	731	731	731
Numb. of countries	**49**	**49**	**49**	**49**	**49**	**49**	**49**	**49**
No. of instruments	**89**	**44**	**49**	**47**	**44**	**39**	**34**	**29**
Av. numb. of years	14.9	14.9	14.9	14.9	14.9	14.9	14.9	14.9
Min. numb. of years	5	5	5	5	5	5	5	5
Max. numb. of years	18	18	18	18	18	18	18	18
AR(1)-test (*p*-value)	0.004	0.006	0.010	0.008	0.005	0.005	0.051	0.949
AR(2)-test (*p*-value)	**0.415**	**0.444**	**0.326**	**0.335**	**0.336**	**0.332**	**0.425**	**0.803**
Hansen test joint validity of instr. (*p*-value)	**1.000**	**0.381**	**0.373**	**0.340**	**0.472**	**0.356**	**0.541**	**0.907**

Difference-in-Hansen tests of exogeneity of instrument subsets								
GMM instruments for levels:								
Hansen test excluding group (*p*-value)	0.999	0.263						
Difference (H0 = exogenous) (*p*-value)	1.000	0.656						
Numb. of PCs from GMM-style instruments			30	28	25	20	15	10
Portion of variance explained by the PCs			0.482	0.462	0.431	0.374	0.306	0.219
Kaiser-Meyer-Olkin measure of sampling adequacy			0.743	0.743	0.743	0.743	0.743	0.743

Notes: Fixed time effects (and fixed country effects) are not shown

p-values are in parentheses. The *p-values* are based Windmeijer's (2005) finite-sample correction for the two-step covariance matrix being robust to heteroskedasticity and autocorrelation within panels.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 10 shows the result of the twostep quantile regression (QR) with fixed effects proposed by Canay (2011). We present the results for the 0.50-quantile, the 0.75-quantile and the 0.25-quantile. Below the estimated coefficients the corresponding 95% confidence intervals are revealed. In summary, there are three main conclusions from Table 10. Firstly, the estimated coefficients of the food composition variables are significantly positive since the 95% confidence intervals do not overlap zero. Secondly, the estimated coefficients do not vary strongly between the quantiles. Thirdly, the magnitudes of the estimated coefficients are very similar to the static FE-OLS estimates in Column (5) in Table 7. Hence, the results of all mean regressions (Table 7, Table 8, Table 9 etc.) are probably not driven by outliers.

TABLE 10: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – TWO-STEP QUANTILE REGRESSIONS WITH FIXED EFFECTS
[95% Bias Corrected Confidence Interval]

	(1) Median QR	(2) 0.75 QR	(3) 0.25 QR
Log GDP per capita, *t*-1	0.019 [-0.158; 0.198]	0.013 [-0.149; 0.203]	0.013 [-0.167; 0.192]
Log total calories intake	0.915 [-0.361; 1.951]	0.932 [-0.237; 2.143]	1.003 [-0.250; 2.187]
Log milk / total	**0.555** [0.288; 0.823]	**0.510** [0.229; 0.802]	**0.581** [0.300; 0.872]
Log sugar / total	1.296 [0.953; 1.700]	1.319 [0.973; 1.758]	1.308 [0.942; 1.724]
Log meat, fat, fish & eggs / total	0.830 [0.487; 1.163]	0.822 [0.457; 1.131]	0.852 [0.514; 1.218]
Number of Observations	797	797	797
Pseudo-R2 of the QR	0.8462	0.8170	0.8524

Notes: The results of the two-step fixed- effects quantile regression estimator proposed by Canay (2011). The first step is the estimate in Column (5) in Table 7.
Fixed time effects are not shown.
The 95% bias corrected confidence interval is based on a boostrap procedure with 1.000 replications.

In Table 11 the results for different time periods are compared. The Columns (1)-(3) show the results for 1990 to 1999; the Columns (4)-(6) the results for 2000-2008. Dividing the time period into two is based on the following considerations. First of all, it is of obvious interest whether the effects found are driven by only one specific time period. This seems to be not the case. Although the estimated effect of milk is statistically insignificant in 1 out of 6 specifications, the overall impression is, that milk has a positive impact. Secondly, the assumption that unobserved differences (time constant omitted variables) are captured by fixed effects is the more plausible the shorter the time period of investigation. At least in the dynamic specifications in Columns (2), (3), (5), and (6) milk has always a statistically significant impact.

In Columns (7), (8), (9) the shortening of the time period is achieved by dropping every second year. We chose this approach as a way to increase the time variation of the food variables.[32] The long-term effect of milk is statistically significant in 2 out of 3 specifications.

[32] However, the coefficient of variation of the proportion of milk variable (without log) based exclusively on the within standard deviation increases only slightly from 0.0565 to 0.0590 if every second year is dropped

TABLE 11: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER (*p-values*) – COMPARING TIME PERIODS

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
		1990 – 1999			**2000 – 2008**			**1990, 1992, 1994, … , 2006, 2008**	
	FE-OLS	SYS-GMM	SYS-GMM FOD	FE-OLS	SYS-GMM	SYS-GMM FOD	FE-OLS	SYS-GMM	SYS-GMM FOD
Log mortality rate, *t*-1		0.648***	0.755***		0.628**	0.522**		0.555***	0.696***
		(0.000)	(0.000)		(0.029)	(0.011)		(0.000)	(0.000)
Log GDP per capita, *t*-1	0.131**	-0.0108	-0.00514	0.356	-0.0143	-0.0935	0.0524	-0.0302	-0.0470
	(0.052)	(0.828)	(0.861)	(0.185)	(0.852)	(0.195)	(0.568)	(0.547)	(0.376)
Log total calories intake	0.980**	0.0648	-0.0314	1.193*	-0.122	-0.122	1.053**	-0.216	-0.262
	(0.091)	(0.824)	(0.852)	(0.073)	(0.679)	(0.667)	(0.029)	(0.509)	(0.541)
Log milk / total	0.0545	0.178**	0.149	0.613**	0.150	0.177	0.392**	0.166	0.0918
	(0.691)	(0.032)	(0.116)	(0.044)	(0.366)	(0.128)	(0.010)	(2.10)	(0.340)
Log sugar / total	1.508***	0.309**	0.150	0.538	0.268	0.348*	1.338***	0.329**	0.219
	(0.000)	(0.043)	(0.158)	(0.115)	(0.227)	(0.064)	(0.000)	(0.044)	(0.133)
Log meat, fat, fish & eggs / total	0.539**	0.0901	0.0506	0.953***	0.175	0.320**	0.878***	0.198**	0.161
	(0.048)	(0.348)	(0.504)	(0.001)	(0.122)	(0.011)	(0.000)	(0.049)	(0.306)
LT elasticities (*p-value*)									
total calories intake	0.98*	0.18	-0.13	1.19*	-0.33	-0.26	1.05**	-0.48	-0.86
	(0.091)	(0.8211)	(0.8535)	(0.073)	(0.6926)	(0.6703)	(0.029)	(0.5046)	(0.467)
milk / total	**0.055**	**0.51*** **	**0.61*** **	**0.61** **	**0.40** **	**0.37*** **	**0.39** **	**0.37***	**0.30**
	(0.691)	(0.0014)	(0.0011)	(0.044)	0.0323	(0.0098)	(0.010)	(0.0782)	(0.2482)
sugar / total	1.51***	0.88**	0.61***	0.54	0.72**	0.73**	1.34***	0.74**	0.72
	(0.000)	(0.0119)	(0.0085)	(0.115)	(0.0167)	(0.0106)	(0.000)	(0.0112)	(0.2056)
meat, fat, fish & eggs / total	0.54**	0.26	0.21	0.95***	0.47	0.67***	0.88***	0.44**	0.53
	(0.048)	(0.2471)	(0.4016)	(0.001)	(0.1385)	(0.0069)	(0.000)	(0.0169)	(0.1370)
Numb. of obs.	411	359	359	386	372	372	422	365	365
Numb. of countries	**47**	**46**	**46**	**49**	**49**	**49**	**49**	**49**	**49**
Av. numb. of years	8.7	7.8	7.8	7.9	7.6	7.6	8.6	7.5	7.5
Min. numb. of years	1	1	1	3	2	2	4	2	2
Max. numb. of years	10	9	9	9	9	9	10	9	9
AR(1)-test (*p*-value)	0.0036**	0.026**	0.350	0.0985*	0.047	0.035**	0.0011***	0.006***	0.011**
AR(2)-test (*p*-value)	**0.9973**	**0.098** **	**0.858**	**0.8425**	**0.527**	**0.628**	**0.1051**	**0.340**	**0.291**
Hansen test (*p*-value)	**-**	**0.239**	**0.172**	**-**	**0.285**	**0.279**	**-**	**0.186**	**0.112**
No. of instruments	-	**23**	**31**	**-**	**23**	**31**	**-**	**23**	**22**
Diff.in-Hansen tests of exogeneity of instrument subsets; GMM instruments for levels									
Hansen test excluding group (*p*-value)		0.267	0.120		0.205	0.259		0.164	0.123
Difference (H0 = exogenous) (*p*-value)		0.208	0.734		0.766	0.391		0.361	0.200

p-values in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

As mentioned in Section 4, there is trade-off in choosing the number of years for the moving averages of the food variables. On the one hand, it is plausible (and epidemiological research indicates it) that the dietary of a longer time period is relevant for cancer. On the other hand, calculating moving averages is a smoothing technique, that is, a method to eliminate time variation. As a further sensitivity analysis we estimate some models with food variables including only the **20 previous years**.[33] In this case, the food variables cover the time span 1970 to 2008.

TABLE 12: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER (*p*-values) – FOOD VARIABLES AS 20 YEARS MOVING AVERAGES

	(1)	(2)	(3)	(4)
	Food variables strictly exogenous			**Food variables endogenous**
	FE-OLS	SYS-GMM	SYS-GMM FOD	SYS-GMM FOD
Log mortality rate, t-1		0.598***	0.675***	0.843***
		(0.000)	(0.000)	(0.000)
Log GDP per capita, *t*-1	0.0362	-0.0213	-0.0511	0.0222
	(0.741)	(0.669)	(0.312)	(0.497)
Log total calories intake	-0.0555	-0.192	-0.000510	-0.373
	(0.867)	(0.515)	(0.998)	(0.412)
Log milk / total	0.802***	0.193**	0.164*	0.110
	(0.000)	(0.012)	(0.096)	(0.139)
Log sugar / total	0.849***	0.244**	0.269**	-0.00115
	(0.000)	(0.020)	(0.047)	(0.994)
Log meat, fat, fish & eggs / total	0.879***	0.176**	0.194**	0.0321
	(0.000)	(0.034)	(0.029)	(0.850)
LT elasticities (*p*-value)				
total calories intake	0.04	-0.48	-0.00	-2.4
	(0.741)	(0.4808)	(0.9981)	(0.2831)
milk / total	**-0.06**	**0.48*** **	**0.50*** **	**0.71***
	(0.867)	(0.0004)	(0.0006)	(0.0866)
sugar / total	0.85***	0.61***	0.83***	-0.01
	(0.000)	(0.0048)	(0.0033)	(0.9935)
meat, fat, fish & eggs / total	0.88***	0.44***	0.60**	0.21
	(0.000)	(0.0019)	(0.0105)	(0.8380)
Numb. of obs.	797	731	731	731
Numb. of countries	49	49	49	49
Av. numb. of years	16.3	14.9	14.9	14.9
Min. numb. of years	7	5	5	5
Max. numb. of years	9	18	18	18
AR(1)-test (*p*-value)	0.0000	0.013	0.008	0.005
AR(2)-test (*p*-value)	0.0000	0.586	0.446	0.400
Hansen test (*p*-value)		0.358	0.378	0.174
No. of instruments		**41**	**40**	**39**
Diff.in-Hansen tests of exogeneity of instrument subsets;				
GMM instruments for levels				
Hansen test excluding group (*p*-value)		0.359	0.317	0.133
Difference (H0 = exogenous) (*p*-value)		0.298	0.768	0.421

p-values in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

[33] $\overline{pmilk}_{it} = \frac{1}{20}\sum_{j=0}^{19} pmilk_{i,t-j} = \frac{1}{20}\sum_{j=0}^{19} \frac{milk_{i,t-j}}{total_{i,t-j}}$ for $t = 1990,...,2008$

The results can be seen in Table 12. In comparison with the results of the 25 year-version, the LT elasticities hardly change. The statistical significance of the coefficients even increases. We conclude that our results are not sensitive with regard to the 25-years-definition of the food variables.

TABLE 13: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER (*p-value*) – HEALTH CARE EXPENDITURES INSTEAD OF GDP

	(1)	(2)	(3)	(4)	(5)
	Food variables strictly exogenous			**Food variables endogenous**	
	FE-OLS	SYS-GMM	SYS-GMM FOD	SYS-GMM	SYS-GMM FOD
Log mortality rate, *t*-1		0.556***	0.573***	0.830***	0.806***
		(0.001)	(0.000)	(0.000)	(0.000)
Log GDP per capita, *t*-1	0.0611	-0.0475	-0.0418	-0.0335	-0.000792
	(0.416)	(0.129)	(0.257)	(0.423)	(0.985)
Log total calories intake	1.056**	-0.0501	0.00202	0.347	-0.460
	(0.029)	(0.824)	(0.994)	(0.466)	(0.375)
Log milk / total	0.724***	0.176	0.121	0.183	0.183**
	(0.001)	(0.109)	(0.188)	(0.105)	(0.027)
Log sugar / total	1.120***	0.341**	0.339***	0.0943	0.0563
	(0.000)	(0.011)	(0.003)	(0.551)	(0.824)
Log meat, fat, fish & eggs / total	0.547***	0.187*	0.237**	-0.128	0.00652
	(0.000)	(0.011)	(0.003)	(0.551)	(0.824)
LT elasticities (*p*-value)					
total calories intake	1.06**	-0.11	0.004	2.04	-2.36
	(0.029)	(0.8204)	(0.9942)	(0.5060)	(0.2894)
milk / total	**0.72*** **	**0.40** **	**0.28***	**1.08***	**0.94** **
	(0.001)	(0.0115)	(0.0762)	(0.0856)	(0.0313)
sugar / total	1.12***	0.77***	0.79***	0.55	0.29
	(0.000)	(0.0000)	(0.0015)	(0.5433)	(0.8207)
meat, fat, fish & eggs / total	0.55***	0.42**	0.55**	-0.75	0.03
	(0.000)	(0.0190)	(0.0114)	(0.4232)	(0.9654)
Numb. of obs.	584	565	565	565	565
Numb. of countries		50	50	50	50
Av. numb. of years		11.30	11.30	11.30	11.30
Min. numb. of years		5	5	5	5
Max. numb. of years		13	13	13	13
AR(1)-test (*p*-value)		0.018	0.020	0.004	0.004
AR(2)-test (*p*-value)		**0.419**	**0.415**	**0.471**	**0.447**
Hansen test (*p*-value)		**0.382**	**0.516**	**0.304**	**0.229**
No. of instruments		**33**	**30**	**44**	**34**
Diff.in-Hansen tests of exogeneity of instrument subsets; GMM instruments for levels					
Hansen test excluding group (*p*-value)		0.311	0.429	0.124	0.231
Difference (H0 = exogenous) (*p*-value)		0.966	0.882	0.985	0.325

p-values in parentheses
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

In the following we include total (=public + private) **health care expenditures per capita** instead of GDP per capita. Since both variables are highly correlated it does not make any

sense to include them simultaneously.[34] This restricts the sample to the years 1996-2008, but increases the number of countries from 49 to 50 since now Cuba can be included.

Another possibility is to carry out the analyses separately for "**richer**" countries versus "**poorer**" countries in terms of GDP per capita and check whether the results stay stable. We first calculate the mean GDP per capita over the estimation period for every country. Secondly, we divide all countries into two groups, the 24 "poorer" countries and the 25 "richer" countries (see Table A1 in the Appendix). Thirdly, we perform the analysis separately for both groups. Since now "large *N*" is not given for sure, we stick to FE-OLS and ignore the Nickell bias in case of the dynamic specification. The results in Table 14 suggest that the results are mainly driven by the rich countries. However, the *p*-value of the estimated long-term elasticity in Column (2) is only slightly above 0.1

TABLE 14: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER (*p-value*) – POOR VERSUS RICH COUNTRIES

	(1)	(2)	(3)	(4)
	Pour Countries		**Rich Countries**	
	FE-OLS	FE-OLS	FE-OLS	FE-OLS
Log mortality rate, *t*-1		0.666***		0.524***
		(0.000)		(0.000)
Log GDP per capita, *t*-1	-0.144**	-0.0936	0.449***	0.285*
	(0.010)	(0.129)	(0.003)	(0.088)
Log total calories intake	2.384***	0.227	-1.976***	-1.063
	(0.000)	(0.504)	(0.005)	(0.242)
Log milk / total	0.0623	0.185	0.569***	0.212**
	(0.759)	(0.111)	(0.000)	(0.022)
Log sugar / total	1.521***	0.395**	0.868***	0.415**
	(0.000)	(0.047)	(0.000)	(0.010)
Log meat, fat, fish & eggs / total	0.167	0.0990	0.756***	0.414***
	(0.346)	(0.407)	(0.000)	(0.003)
LT elasticities (*p-value*)				
total calories intake	2.38***	0.68	-1.98***	-2.23
	(0.000)	(0.4244)	(0.005)	(0.1936)
milk / total	**0.062**	**0.55**	**0.57*** **	**0.45** **
	(0.759)	(0.1156)	(0.000)	(0.0158)
sugar / total	1.52***	1.18**	0.87***	0.87**
	(0.000)	(0.0218)	(0.000)	(0.0126)
meat, fat, fish & eggs / total	0.17	0.30	0.76***	0.87***
	(0.346)	(0.4148)	(0.000)	(0.0000)
Numb. of obs.	378	339	419	392
Numb. of countries	24	24	25	25
Av. numb. of years	15.8	14.1	16.8	15.7
Min. numb. of years	7	6	7	5
Max. numb. of years	19	18	19	18
Mean *mrate*	16.2	16.4	18.3	18.3
Mean *milk / total*	6.0	6.0	9.4	9.4

p-values based on Driscoll-Kraay standard errors in parentheses.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

[34] The correlation coefficient (*p-value*) is 0.9448 (0.0000).

8. Results for Models Explaining the Mortality Rate of Ovarian Cancer

The analyses for the determinants of the mortality rate of ovarian cancer in this section proceed in the same way as the analyses of the determinants of the mortality rate of prostate cancer in the previous section.

Table 15 presents the estimation results of the static two-way fixed effects models derived in Section 6 by BMA analysis and EBA. All specifications include fixed time affects (year dummies), which are always jointly significant at the 1% percent. Alternative estimates of the standard errors are shown in Table A 5 in the Appendix. See Section 5 and Section 7 for further details.

We start in Column (1) with a specification, where the (log of the age standardized) mortality rate of ovarian cancer is only explained by the (log of) GDP per capita in the previous year. Again, the estimated coefficient is positive. Again, this may be explained by dietary practices being correlated with the GDP. This can be seen in Columns (3) and (4): After including the proportion of milk and the proportion of sugar in total calories intake the estimated coefficient for GDP becomes small and statistically insignificant. Both milk and sugar show statistically significant positive effects. That means: the higher the proportion of milk (and sugar) in total calories intake (at a given total calories intake level), the higher is the mortality rate. The estimated coefficient of the total calories intake variable is now around 2.0 indicating that an x% increase in total calories intake (at a given GDP, dietary composition, year and country) increase the mortality rate of prostate cancer by 2·x%.

Column (6) shows our preferred static specification based on the analyses in Section 6. The additionally included variable for the proportion of eggs in total calories intake shows a statistically significant coefficient as well. The proportion of vegetable oils has a negative impact. Most important, the coefficient for milk is still highly statistically significant. With these three food variables 36.7% of total calories intake is covered. A comparison with a RE-GLS model via a Hausman test in Column (7) indicates again that a random effects specification would be inconsistent due to the correlation of explanatory variables with the country fixed effects. [35]

Finally Column (8) shows that the coefficient for milk stays statistically significant and positive after other (not relevant) food variables are included.

[35] The H0 is rejected with a *p-value* of 0.0300.

TABLE 15: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – STATIC FIXED EFFECTS RESULTS (*p*-VALUES BASED ON ROBUST STANDARD ERRORS)

	(1) FE-OLS	(2) FE-OLS	(3) FE-OLS	(4) FE-OLS	(5) FE-OLS	(6) FE-OLS	(7) RE-GLS	(8) FE-OLS
Log GDP per capita, *t*-1	0.166	0.510**	0.244	0.135	0.124	0.137	0.116*	0.132
	(0.314)	(0.041)	(0.189)	(0.450)	(0.469)	(0.431)	(0.096)	(0.504)
Log total calories intake		1.976***	1.939***	2.143***	2.190***	1.907***	1.917***	1.855***
		(0.000)	(0.002)	(0.000)	(0.000)	(0.000)	(0.000)	(0.009)
Log milk / total			**1.120*** **	**0.765*** **	**0.541*** **	**0.566*** **	**0.328*** **	**0.581*** **
			(0.000)	(0.000)	(0.004)	(0.003)	(0.000)	(0.002)
Log sugar / total				0.951***	1.038***	1.174***	1.096***	1.155***
				(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Log eggs / total					0.300**	0.258*	0.333***	0.249*
					(0.027)	(0.058)	(0.000)	(0.066)
Log vegetable oils / total						-0.213**	-0.131*	-0.226**
						(0.026)	(0.084)	(0.023)
Log meat & fat / total								0.0173
								(0.927)
Log fish / total								0.0141
								(0.927)
Log vegetables & fruits / total								0.0101
								(0.969)
Log pulses/ total								0.0224
								(0.791)
Numb. of obs.	933	708	708	708	708	708	708	708
Numb. of countries	71	49	49	49	49	49	49	49
Av. numb. of years	13.1	14.4	14.4	14.4	14.4	14.4	14.4	14.4
Min. numb. of years	2	6	6	6	6	6	6	6
Max. numb. of years	19	19	19	19	19	19	19	19
AR(1)-test (*p*-value)	0.0167	0.0138	0.0069	0.0031	0.0045	0.0043		0.0044
AR(2)-test (*p*-value)	0.0788	0.0918	0.1843	0.1671	0.1795	0.1787		0.1745
Mean *mrate*	5.29	4.97	4.97	4.97	4.97	4.97	4.97	4.97
Mean *milk / total*	7.97	7.97	7.97	7.97	7.97	7.97	7.97	7.97
Prop. of kcal in *X*			7.97	20.53	21.83	31.10	31.10	54.63
within R^2	0.0295	0.0927	0.2433	0.2820	0.2906	0.2965	0.2965	0.2966
Corr. coef. (c_i, X_{it})	0.4131	-0.2291	-0.8107	-0.6898	-0.6147	-0.5577	0	-0.5426

Notes: Fixed time effects and fixed country effects are not shown

p-values based on Driscoll-Kraay standard errors in parentheses, Exception: the *p-values* statistics of the random effects estimator are based on conventional standard errors.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

In Table 16 the results of the dynamic specifications assuming strictly exogenous food variables and the resulting LT elasticities are shown. The order is again the same as in the last Section. All the estimated coefficients of the LSDVc[36] in Column (2) except the coefficients of the lagged dependent variable as well as the milk variable are statistically significant. In case of the GMM estimators in Columns (3), (4), (5) and (6) the AR(2)-test as well as the Hansen Tests do not rejects the necessary assumptions of the estimators (see Section 4). As a visual test of the goodness of fit Figure A 2 in the Appendix shows besides the observed val-

[36] Bias correction up to order O(1/NT^2). Bias correction initialized by SYS-GMM estimator. 1000 Bootstrap replications.

ues of our dependent variables also the predicted value by the model in Column (5). The fit seems satisfactory. But it is clearly worse than in case of the model for prostate cancer (Figure A 1). The Han and Philips (2010) estimator in Column (7) is again inconsistent due to heteroscedasticity.[37]

The estimated long-term elasticities of milk is in all specifications statistically significant and positive. In contrast to the mortality rate of prostate cancer, the total calories intake is highly relevant too. The implications of this result are as follows (Column (5)). If a society increases milk consumption by 1 percent without reducing calories intake from other sources (such as cereals) over a time period of 25 years on average, the number of deaths (per 100,000) due to ovarian cancer increase by almost 1.8% (=0.335+1.426), with 0.335% resulting from a dietary composition which includes more milk and 1.426% being caused by the higher total calories intake.

[37] H0: Panel Homoscedasticity. Lagrange Multiplier Test: p-value = 0.0000, Likelihood Ratio Test: p-value = 0.0000; Wald Test: p-value= 0.0000

TABLE 16: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – DYNAMIC PANEL MODEL RESULTS (*p*-values) ; FOOD VARIABLES ARE TREATED AS STRICTLY EXOGENOUS

	(1) FE-OLS	(2) LSDVc	(3) FOD-GMM1	(4) SYS-GMM	(5) SYS-GMM FOD	(6) SYS-GMM 20 PCs[38]	(7) Han-Philips (2010)
Log mortality rate, *t*-1	0.374***	0.509***	0.0438	0.333*	0.328	0.667***	0.223*
	(0.001)	(0.000)	(0.800)	(0.059)	(0.178)	(0.000)	(0.099)
Log GDP per capita, *t*-1	0.0977	0.0542	0.172	0.0601	0.105*	0.0252	0.179
	(0.614)	(0.684)	(0.294)	(0.314)	(0.092)	(0.524)	(0.179)
Log total calories intake	0.891*	0.692	1.467	0.947**	0.958**	0.561	1.470***
	(0.056)	(0.197)	(0.145)	(0.031)	(0.011)	(0.179)	(0.005)
Log milk / total	0.435**	0.367**	0.641***	0.153*	0.225**	0.0866**	0.706***
	(0.017)	(0.038)	(0.005)	(0.086)	(0.015)	(0.024)	(0.000)
Log sugar / total	0.325**	0.155	0.727*	0.341**	0.267	0.172	0.643***
	(0.025)	(0.496)	(0.067)	(0.033)	(0.194)	(0.369)	(0.005)
Log eggs / total	0.223**	0.200	0.293	0.255***	0.158	0.0735	0.313**
	(0.044)	(0.129)	(0.124)	(0.002)	(0.190)	(0.242)	(0.020)
Log vegetable oils / total	0.0206	0.0457	-0.0594	-0.0475	-0.0735	-0.0161	-0.0248
	(0.795)	(0.705)	(0.773)	(0.383)	(0.329)	(0.624)	(0.841)
LT elast. (*p*-value)							
total calories intake	1.423**	1.409	1.534	1.421***	1.426***	1.683***	1.892**
	(0.034)	(0.199)	(0.156)	(0.005)	(0.004)	(0.004)	(0.012)
milk / total	**0.695*****	**0.746****	**0.670****	**0.229****	**0.335*****	**0.260****	**0.909*****
	(0.002)	**(0.035)**	**(0.015)**	**(0.031)**	**(0.005)**	**(0.046)**	**(0.001)**
sugar / total	0.518***	0.316	0.760**	0.512**	0.397	0.515	0.828**
	(0.009)	(0.494)	(0.049)	(0.015)	(0.113)	(0.163)	(0.012)
eggs / total	0.357*	0.407	0.307*	0.383***	0.235*	0.220**	0.402**
	(0.067)	(0.130)	(0.093)	(0.001)	(0.053)	(0.047)	(0.031)
vegetable oils / total	0.0329	0.0929	-0.0621	-0.0713	-0.109	-0.0483	-0.0320
	(0.800)	(0.705)	(0.770)	(0.390)	(0.227)	(0.617)	(0.842)
Numb. of obs.	647	647	598	647	647	647	647
Numb. of countries	49	49	49	49	49	49	49
Numb. of instruments			**39**	**42**	**36**	**44**	
Av. numb. of years	13.2		12.20	13.2	13.2	13.2	13.2
Min. numb. of years	4		3	4	4	4	4
Max. numb. of years	18		17	18	18	18	18
within R^2	0.3719						
AR(1)-test (*p*-value)	0.0586		0.050	0.027	0.071	0.018	
AR(2)-test (*p*-value)	0.4235		0.986	0.264	0.339	0.131	
Hansen test joint validity of instr. (*p*-value)			0.211	0.582	0.225	0.442	
Difference-in-Hansen tests of exogeneity of instrument subsets							
GMM instruments for levels							
Hansen test excluding group (*p*-value)				0.513	0.200		
Difference (H0 = exogenous) (*p*-value)				0.898	0.398		

Notes: Fixed time effects (and fixed country effects) are not shown

The *p-values* in (1) are based on Driscoll-Kraay standard errors. The *p*-values in (2) are boostrapped (1000 replications) assuming homoscedastic residuals being uncorrelated over time and between countries. In (3) the *p*-values are based on standard errors being robust to heteroskedasticity and autocorrelation. The *p-values* in (4) to (6) are based Windmeijer's (2005) finite-sample correction for the two-step covariance matrix being robust to heteroskedasticity and autocorrelation within panels. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

[38] Extracted 20 principal components from GMM-style instruments. Portion of variance explained by the components = 0.569; Kaiser-Meyer-Olkin measure of sampling adequacy = 0.822.

In Table 17 the food variables are treated as endogenous and they are instrumented in the same way as the lagged dependent variable.

When we include variables for eggs and vegetable oils, it is practically impossible to find a specification which is not rejected by the AR(2) test and/or the Hansen Test. Since both variables (eggs and vegetable oils) were also limit cases in the BMA analysis and EBA in Section 6 – they are not unquestionable "important regressors" – we drop both variables in the following analyses. In Table 17 we start in Column (1) with an "overfitted" model with 49 instruments which leads to the misleading results of a *p*-value of 0.957 of the Hansen Test. Apart from the lagged dependent variable the total calories variable is the only regressor which shows a statistically significant coefficient. The same is true for the LT elasticities. In Column (2) the number of instruments is reduced to 39. The point estimates hardly change and with increasing standard errors all estimated coefficients and long-term elasticities become insignificant.

In the next step, we want to vary the number of instruments by using PCs instead of the instrument matrix again (see Section 7). However, the portion of variance explained by the PCs is rather low (see Column (3) as well as the Footnote 39). And the Kaiser-Meyer-Olkin measure of sampling adequacy is only slightly above 0.6. The results are completely contrary to the ones we find with other approaches, which is probably an indication for weak instruments. For this reason we do not use PCs here, but reduce the number of instruments by using only certain lags of instruments. We reduce the number of instruments from 49 to 35 in Column (4) and 31 in Column (5). A further reduction leads to an insignificant coefficients on all (including the lagged dependent) variable.

Summarizing Table 17, many coefficients and long-term elasticities are statistically insignificant. The total calories intake variable is statistically significant positive. The corresponding long-term elasticity is much larger than 1, indicating a very strong effect. The coefficient of the milk variable and the corresponding long-term elasticity are only significant in two specifications. The point estimates of the long-term elasticities of the milk variables is in case of "smaller" *p*-values in Column (2), (3) and (5) larger than, but not total different from, the magnitude found in the specifications without instrumentation. We interpret the latter as an indication for the plausibility of our assumption that the milk variable is strictly exogenous.

TABLE 17: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – DYNAMIC PANEL MODEL RESULTS (*p*-values)
– FOOD VARIABLES ARE TREATED AS ENDOGENOUS AND ARE INSTRUMENTED

	(1) SYS-GMM FOD	(2) SYS-GMM FOD	(3) SYS-GMM FOD 18 PCs[39]	(4) SYS-GMM FOD	(5) SYS-GMM FOD
Log mortality rate, *t*-1	0.477***	0.419***	0.979***	0.540***	0.617***
	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)
Log GDP per capita, *t*-1	-0.158	-0.209	0.0590	-0.166	-0.211
	(0.384)	(0.233)	(0.637)	(0.270)	(0.356)
Log total calories intake	2.415*	2.589**	-0.800	2.293***	2.521*
	(0.098)	(0.021)	(0.354)	(0.009)	(0.087)
Log milk / total	0.261	0.384*	0.123	0.338*	0.264
	(0.175)	(0.072)	(0.256)	(0.082)	(0.185)
Log sugar / total	0.305	0.304	-0.400*	0.150	0.111
	(0.556)	(0.505)	(0.092)	(0.678)	(0.760)
LT elasticities (*p*-value)					
Log total calories intake	4.62*	4.45**	-37.28	4.99**	6.59
	(0.071)	(0.017)	(0.846)	(0.029)	(0.190)
Log milk / total	**0.50**	**0.66***	**5.71**	**0.74***	**0.69**
	(0.150)	(0.057)	(0.832)	(0.053)	(0.147)
Log sugar / total	0.58	0.52	-18.63	0.32	0.29
	(0.541)	(0.494)	(0.848)	(0.667)	(0.753)
Numb. of obs.	647	647	647	647	647
Numb. of countries	**49**	**49**	**49**	**49**	**49**
No. of instruments	**49**	**39**	**37**	**35**	**31**
Av. numb. of years	13.2	13.2	13.2	13.2	13.2
Min. numb. of years	4	4	4	4	4
Max. numb. of years	18	18	18	18	18
AR(1)-test (*p*-value)	0.019	0.020	0.016	0.016	0.012
AR(2)-test (*p*-value)	**0.175**	**0.169**	**0.118**	**0.134**	**0.108**
Hansen test joint validity of instr. (*p*-value)	**0.957**	**0.351**	**0.680**	**0.420**	**0.283**
Difference-in-Hansen tests of exogeneity of instrument subsets					
GMM instruments for levels:					
Hansen test excluding group (*p*-value)	0.978	0.318		0.577	0.224
Difference (H0 = exogenous) (*p*-value)	0.250	0.431		0.224	0.224

Notes: Fixed time effects (and fixed country effects) are not shown
P-values are in parentheses. The *p-values* are based Windmeijer's (2005) finite-sample correction for the two-step covariance matrix being robust to heteroskedasticity and autocorrelation within panels.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 18 shows the result of the twostep quantile regression (QR) with fixed effects proposed by Canay (2011). Again, we present the results for the 0.50-quantile, the 0.75-quantile and the 0.25-quantile. Below the estimated coefficients the corresponding 95% confidence intervals are shown which are generated by a bootstrap procedure over both steps. In summary, we draw the same conclusions from Table 10 as in the analysis of prostate cancer. The

[39] Extracted 15 principal components from GMM-style instruments; Portion of variance explained by the components = 0.310; Kaiser-Meyer-Olkin measure of sampling adequacy = 0.630

estimated coefficients of the variables for total calories intake, milk and sugar are statistically significant and positive since the 95% confidence intervals do not overlap zero. Again, the estimated coefficients do not vary strongly between the quantiles and the magnitudes of the estimated coefficients are very similar to the static OLS-FE estimates in Column (6) in Table 15. Again we can draw the important conclusion that, the results of all mean regressions are probably not driven by outliers.

TABLE 18: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – TWO-STEP QUANTILE REGRESSIONS WITH FIXED EFFECTS
[95% Bias Corrected Confidence Interval]

	(1) Median-QR	(2) 0.75-QR	(3) 0.25-QR
Log GDP per capita, *t*-1	0.137 [-0.136; 0.419]	0.130 [-0.194; 0.367]	0.140 [-0.134; 0.421]
Log total calories intake	1.876 [0.719; 3.161]	1.674 [0.454; 2.919]	2.016 [0.975; 3.427]
Log milk / total	**0.569** [0.275; 0.886]	**0.573** [0.302; 0.864]	**0.564** [0.256; 0.843]
Log sugar / total	1.164 [0.714; 1.664]	1.137 [0.703; 1.692]	1.193 [0.727; 1.697]
Log eggs / total	0.253 [-0.005; 0.518]	0.236 [-0.006; 0.525]	0.303 [0.076; 0.603]
Log vegetable oils / total	-0.207 [-0.458; 0.028]	-0.219 [-0.467; 0.004]	-0.181 [-0.404; 0.063]
Number of Observations	708	708	708
Pseudo-R2 of the QR	0.8313	0.7922	0.8425

Notes: The results of the two-step fixed- effects quantile regression estimator proposed by Canay (2011). The first step is the estimate in Column (6) in Table 15.
Fixed time effects are not shown.
The 95% bias corrected confidence interval is based on a boostrap procedure with 1,000 replications.

In Table 19 the results for different time periods are compared. The Columns (1)-(2) show the results for 1990 to 1999[40]; the Columns (3)-(5) the results for 2000-2008. The motivation for doing this is discussed in Section 7.

At least in the dynamic specifications in Columns (2), (4), and (5) milk has always at statistically significant impact.

In Columns (6), (7), (8) the estimates are based on a sample where every second year is dropped. This approach is a way to increase the time variation of the food variables.[41] The long-term effect of milk is statistically significant in 2 out of 3 specifications.

[40] As we were not able to find a specification for the SYS-GMM with FOD which was not rejected by the Hansen Test and the AR(2) test, we do not report the results.

[41] However, the coefficient of variation of the proportion of milk variable (without log) based exclusively on the within standard deviation increases only slightly from 0.056 to 0.059 if every second year is dropped

TABLE 19: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER (*p-values*) – COMPARING TIME PERIODS

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	1990 – 1999		**2000 – 2008**			**1990, 1992, 1994, … , 2006, 2008**		
	FE-OLS	SYS-GMM	FE-OLS	SYS-GMM	SYS-GMM FOD	FE-OLS	SYS-GMM	SYS-GMM FOD
Log mortality rate, *t*-1		0.485		0.534***	0.563***		0.866***	0.0833
		(0.176)		(0.000)	(0.000)		(0.000)	(0.752)
Log GDP per capita, *t*-1	0.0216	0.106	0.579***	0.0628	0.0744	0.149	-0.0113	0.142
	(0.889)	(0.200)	(0.003)	(0.318)	(0.154)	(0.436)	(0.549)	(0.122)
Log total calories intake	0.795*	1.112	0.269	0.583**	0.674**	1.539**	0.204	1.396***
	(0.061)	(0.223)	(0.760)	(0.039)	(0.024)	(0.024)	(0.198)	(0.001)
Log milk / total	0.359	0.169	0.285	0.121	0.102*	0.623***	0.0190	0.222*
	(0.108)	(0.229)	(0.204)	(0.113)	(0.080)	(0.006)	(0.363)	(0.096)
Log sugar / total	0.846**	0.257	0.507	0.218	0.237*	1.295***	0.0296	0.567***
	(0.028)	(0.406)	(0.121)	(0.100)	(0.093)	(0.000)	(0.677)	(0.006)
Log eggs / total	0.332	0.0594	1.106***	0.113*	0.0718	0.125	0.0315	0.230*
	(0.154)	(0.632)	(0.000)	(0.076)	(0.284)	(0.305)	(0.283)	(0.062)
Log vegetable oils / total	-0.222**	-0.0546	-0.827***	-0.00324	0.00406	-0.261***	-0.00772	-0.00852
	(0.016)	(0.425)	(0.000)	(0.948)	(0.951)	(0.006)	(0.657)	(0.918)
LT elasticities (*p-value*)								
total calories intake	0.80*	2.16***	0.58***	1.25***	1.54***	1.54**	1.53**	1.52***
	(0.061)	(0.001)	(0.003)	(0.008)	(0.003)	(0.024)	(0.015)	(0.000)
milk / total	**0.36**	**0.32***	**0.27**	**0.26***	**0.23***	**0.62*****	**0.14**	**0.24****
	(0.108)	(0.063)	(0.760)	(0.071)	(0.079)	(0.006)	(0.212)	(0.021)
sugar / total	0.85**	0.50	0.29	0.47*	0.54*	1.30***	0.22	0.62***
	(0.028)	(0.203)	(0.204)	(0.072)	(0.056)	(0.000)	(0.616)	(0.004)
eggs / total	0.33	0.11	0.51	0.24*	0.16	0.13	0.24	0.25**
	(0.154)	(0.580)	(0.121)	(0.078)	(0.270)	(0.305)	(0.128)	(0.025)
vegetable oils / total	-0.22**	-0.11	1.11***	-0.00	0.00	-0.26***	-0.06	-0.00
	(0.016)	(0.380)	(0.000)	(0.948)	(0.951)	(0.006)	(0.675)	(0.918)
Numb. of obs.	322	276	386	371	371	373	319	319
Numb. of countries	**46**	**44**	**49**	**49**	**49**	**49**	**49**	**23**
Av. numb. of years	7.0	6.3	7.9	7.6	7.6	7.6	6.5	6.5
Min. numb. of years	1	1	3	2	2	3	1	1
Max. numb. of years	10	9	9	9	9	10	9	9
AR(1)-test (*p*-value)	0.530	0.103	0.2921	0.044	0.012	0.005	0.009	0.415
AR(2)-test (*p*-value)	**0.504**	**0.225**	**0.3451**	**0.461**	**0.144**	**0.868**	**0.839**	**0.840**

Hansen test joint validity of instr. (*p*-value)		**0.191**		**0.412**	**0.418**	**0.503**	**0.399**
No. of instruments		**24**		**43**	**40**	**38**	**23**
Difference-in-Hansen tests of exogeneity of instrument subsets							
GMM instruments for levels:							
Hansen test excluding group (*p*-value)		0.130		0.307	0.277	**0.588**	**0.431**
Difference (H0 = exogenous) (*p*-value)		0.968		0.578	0.633	**0.337**	**0.242**

p-values in parentheses; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

As a further sensitivity analysis we estimate some models with food variables including only the **20 previous years** (Table 20). In this case the food variables cover the time span 1970 to 2008. In Column (3) we drop $y_{i,t-2}$ and $\Delta y_{i,t-1}$ from the instrument matrix since otherwise the Hansen Test rejects the specification.

TABLE 20: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER (*p-values*) – FOOD VARIABLES AS 20 YEARS MOVING AVERAGES

	(1)	(2)	(3)	(4)
	Food variables strictly exogenous			**Food variables endogenous**
	FE-OLS	SYS-GMM	SYS-GMM FOD	SYS-GMM
Log mortality rate, *t*-1		0.171	0.360*	0.554**
		(0.545)	(0.085)	(0.012)
Log GDP per capita, *t*-1	0.144	0.106	0.0989	-0.0216
	(0.431)	(0.230)	(0.173)	(0.863)
Log total calories intake	0.869**	1.135*	0.713**	0.187
	(0.015)	(0.071)	(0.015)	(0.863)
Log milk / total	0.819***	0.216*	0.229*	0.316
	(0.000)	(0.091)	(0.064)	(0.132)
Log sugar / total	0.871***	0.495***	0.265	0.142
	(0.000)	(0.004)	(0.120)	(0.382)
Log eggs / total	0.183	0.206	0.157	0.283
	(0.109)	(0.112)	(0.132)	(0.227)
Log vegetable oils / total	-0.259**	-0.0329	-0.0435	-0.0511
	(0.011)	(0.722)	(0.496)	(0.741)
LT elasticities (*p-value*)				
total calories intake	0.87**	1.37**	1.12**	0.420
	(0.015)	(0.011)	(0.015)	(0.867)
milk / total	**0.82*** **	**0.26*** **	**0.36*** **	**0.71****
	(0.000)	(0.006)	(0.005)	(0.014)
sugar / total	0.87***	0.60**	0.41	0.32
	(0.000)	(0.021)	(0.137)	(0.334)
eggs / total	0.18	0.25	0.25**	0.63
	(0.109)	(0.169)	(0.041)	(0.106)
vegetable oils / total	-0.26**	-0.04	-0.07	-0.11
	(0.011)	(0.709)	(0.451)	(0.736)
Numb. of obs.	708	647	647	647
Numb. of countries	49	49	49	49
Av. numb. of years	14.4	13.2	13.2	13.2
Min. numb. of years	6	4	4	4
Max. numb. of years	19	18	18	18
AR(1)-test (*p*-value)	0.0036	0.124	0.049	0.033
AR(2)-test (*p*-value)	0.1701	0.556	0.260	0.159
Hansen test (*p*-value)		0.358	0.182	0.547
No. of instruments		**31**	**30**	**37**
Diff.in-Hansen tests of exogeneity of instrument subsets;				
GMM instruments for levels				
Hansen test excluding group (*p*-value)		0.263	0.180	0.656
Difference (H0 = exogenous) (*p*-value)		0.706	0.255	0.356

p-values in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

In Column (4) of Table 20 all food variables are treated as endogenous and they are instrumented. Since all specifications using FOD are rejected by the Hansen Test as well as the

AR(2) test, we use first differences (FD) instead. In all specification – even when instrumented (Column (4) – an increase in the proportion of milk in total calories intake within a period of 20 years increases the number of people dying due to ovarian cancer by 0.26% up to 0.82%.

TABLE 21: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER (*p-value*) – HEALTH CARE EXPENDITURES INSTEAD OF GDP

	(1)	(2)	(3)	(4)	(5)
	Food variables strictly exogenous			**Food variables endogenous**	
	FE-OLS	SYS-GMM	SYS-GMM FOD	SYS-GMM	SYS-GMM FOD
Log mortality rate, *t*-1		0.453	-0.00875	0.636***	0.295**
		(0.206)	(0.949)	(0.000)	(0.015)
Log GDP per capita, *t*-1	0.0127	0.0406	0.0639	-0.0816	-0.245
	(0.848)	(0.383)	(0.384)	(0.307)	(0.346)
Log total calories intake	1.758**	0.879	1.618***	1.369	3.671
	(0.024)	(0.178)	(0.001)	(0.117)	(0.139)
Log milk / total	0.736***	0.127*	0.282*	0.257	0.468**
	(0.002)	(0.055)	(0.068)	(0.107)	(0.013)
Log sugar / total	0.991***	0.264	0.474**	-0.0472	0.0347
	(0.000)	(0.318)	(0.018)	(0.838)	(0.931)
Log eggs / total	0.339	0.172	0.264		
	(0.154)	(0.235)	(0.106)		
Log vegetable oils / total	-0.0342	0.00733	0.0075		
	(0.781)	(0.877)	(0.932)		
LT elasticities (*p*-value)					
total calories intake	1.76**	1.61***	1.60***	3.76*	5.21
	(0.024)	(0.000)	(0.000)	(0.057)	(0.109)
milk / total	**0.74*****	**0.23****	**0.28****	**0.71****	**0.66*****
	(0.002)	(0.030)	(0.040)	(0.023)	(0.009)
sugar / total	0.99***	0.48**	0.47**	-0.13	0.05
	(0.000)	(0.019)	(0.030)	(0.838)	(0.931)
eggs / total	0.34	0.31***	0.26*		
	(0.154)	(0.001)	(0.074)		
vegetable oils / total	-0.03	0.01	-0.01		
	(0.781)	(0.873)	(0.932)		
Numb. of obs.	562	530	530	530	530
Numb. of countries	50	50	50	50	50
Av. numb. of years	11.2	10.6	10.6	10.6	10.6
Min. numb. of years	6	4	4	4	4
Max. numb. of years	13	13	13	13	13
AR(1)-test (*p*-value)	0.0133	0.141	0.138	0.041	0.059
AR(2)-test (*p*-value)	0.7316	**0.598**	**0.452**	**0.404**	**0.580**
Hansen test (*p*-value)		**0.916**	**0.806**	**0.330**	**0.773**
No. of instruments		**29**	**30**	**34**	**38**
Diff.in-Hansen tests of exogeneity of instrument subsets; GMM instruments for levels					
Hansen test excluding group (*p*-value)		0.871	0.766	0.427	0.772
Difference (H0 = exogenous) (*p*-value)		0.775	0.543	0.226	0.482

p-values in parentheses, * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

In the Table 21 we include total (=public + private) **health care expenditures per capita** instead of GDP per capita. Interestingly the coefficient of lagged dependent variable becomes

statistically insignificant in some specifications. However, most important, the estimated long-term elasticity of milk is always statistically significant.

Finally we carry out the analysis separately for "**rich** " countries versus "**poor**" countries in terms of GDP per capita and check whether the results stay stable (see Section 7). As discussed in Section 7, since in this case *N* is definitely not large, we cannot apply the GMM estimators. Therefore we simply ignore the Nickell bias in the dynamic specifications. The results in Table 22 show that the finding with regard to milk is robust to subsamples and that an increase in milk consumption (and total calories intake) is more fatal in poor than in rich countries – even after controlling for GDP per Capita.

TABLE 22: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER (*p-value*) – POOR VERSUS RICH COUNTRIES

	(1)	(2)	(3)	(4)
	Pour Countries		**Rich Countries**	
	FE-OLS	FE-OLS	FE-OLS	FE-OLS
Log mortality rate, *t*-1		0.307**		0.235
		(0.025)		(0.204)
Log GDP per capita, *t*-1	-0.0189	-0.00110	0.303*	0.199
	(0.923)	(0.996)	(0.068)	(0.438)
Log total calories intake	2.703***	1.418**	1.383**	1.178*
	(0.000)	(0.015)	(0.027)	(0.054)
Log milk / total	0.700*	0.702*	0.383***	0.295**
	(0.066)	(0.053)	(0.001)	(0.018)
Log sugar / total	1.404***	0.536**	0.720***	0.438**
	(0.000)	(0.030)	(0.003)	(0.023)
Log eggs / total	-0.0998	0.0898	0.0765	0.0476
	(0.654)	(0.687)	(0.300)	(0.488)
Log vegetable oils / total	-0.127	0.109	-0.0173	0.0607
	(0.582)	(0.579)	(0.790)	(0.422)
LT elasticities (*p-value*)				
total calories intake	2.70***	2.05***	1.385**	1.54*
	(0.000)	(0.001)	(0.027)	(0.055)
milk / total	**0.70***	**1.01****	**0.38*****	**0.39*****
	(0.066)	(0.029)	(0.001)	(0.002)
sugar / total	1.40***	0.77**	0.72***	0.57**
	(0.000)	(0.015)	(0.003)	(0.030)
eggs / total	-0.10	0.13	0.08	0.06
	(0.654)	(0.693)	(0.300)	(0.518)
vegetable oils / total	-0.13	0.16	-0.02	0.08
	(0.582)	(0.593)	(0.790)	(0.423)
Numb. of obs.	289	255	419	392
Numb. of countries	24	24	25	25
Av. numb. of years	12	10.6	16.8	15.7
Min. numb. of years	6	4	7	5
Max. numb. of years	19	18	19	18
Mean *mrate*	5.9	6.1	9.4	9.4
Mean *milk / total*	15.3	15.1	18.3	18.3

p-values based on Driscoll-Kraay standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

9. Robustness of the results with regard to time-varying confounding factors

The reliability of the results crucially depends on the question whether there are time-varying unobserved omitted variables (confounding factors) which bias the estimated effect of milk consumption ($\hat{\beta}_1$). Note that time-constant unobserved variables are absorbed by the country fixed effects (c_i). Time effects which are common to all countries are controlled by time fixed effects (λ_t).

Recently, Oster (2013) suggested a very intuitive approach to evaluate the bias due to confounding factors under the assumption that the selection on the observed variables is proportional to the selection on the unobserved variables (confounding factors).[42] She shows how coefficient movements, along with movement in R^2 values, can identify the effect of the confounding factors. If δ=1, observed and unobserved variables have an equally important effect on $\hat{\beta}_1$. As pointed out by Oster (2013), $\delta \in \{0, 1\}$ is a plausible bound on the degree of selection on the unobserved variables. As we want to be as conservative as possible, we assume δ=1. Furthermore, one has to assume a maximum value of R^2, under the assumption all unobservable variables would be perfectly observable and would be included into the model. Here we assume a $R^2_{\max}$ =1, which means again a conservative assumption in this approach. Given these assumptions, Oster's (2013) approach can be used to estimate the hypothetical effect of milk on the mortality rate, if all unobserved variables (confounding factors) would be included into the model (β_1^*). This may be called the *bias-adjusted effect of milk consumption on the mortality rate under the assumption of proportional selection on observable and unobservable variables*. We perform this analysis for our static fixed effects results. First, one has to estimate a "*baseline effect*", which is the estimated coefficient on the milk variable in the simple pooled regression $\ln(m_{it}) = \beta_1 \ln(\overline{pmilk}_{it}) + u_{it}$. The resulting coefficient is $\dot{\beta}_1$ and the resulting coefficient of determination is $\dot{R}^2$. Secondly, the "*controlled effect*" is the coefficient from the fixed-effects regression with all observable control variables. The resulting coefficient on the milk variable is $\hat{\beta}_1$ and the corresponding coefficient of determination is

[42] Assume the following regression model $Y = \beta X + W_1 + W_2$, with β indicating the coefficient of interest, W_1 is an observed control variable, and W_2 is an unobserved control variable (confounding factor). The proportional selection assumption means $\frac{Cov(X, W_2)}{Var(W_2)} = \delta \frac{Cov(X, W_1)}{Var(W_1)}$ with the degree of proportionality being denoted by δ (see Oster, 2013).

R^2. Here we interpret the fixed-effects as a set of 49 dummy variables within an OLS regression. The R^2 is the coefficient of determination from this OLS regression.

Now, Oster (2013) shows that – given these values and these assumptions on δ, $R^2_{\max}$, and the proportionality of observables and unobservables – the bias-adjusted effect of milk consumption on the mortality rate can be approximated by $\beta_1^* = \hat{\beta}_1 - \delta\left[\hat{\beta}_1 - \dot{\beta}_1\right]\frac{R^2_{\max} - R^2}{R^2 - \dot{R}^2}$.

The point estimates for both mortality rates and for two different specifications are presented in the following Table 23. We show two rows for prostate cancer (corresponding to two specifications in Table 7) and two rows for ovarian cancer (corresponding to two specifications in Table 15). The first column shows the baseline effects which are the results of pooled regressions only on the milk variable. The second column shows the controlled effects which are simply the results from Table 7 and Table 15. Finally, the third column shows the estimated bias-adjusted effect. We learn from these results that the bias-adjusted effect of milk is only slightly lower than the estimates from the fixed effects regressions. Hence, this may be interpreted as further evidence that our results are not mainly driven by confounding factors.

TABLE 23: SENSITIVITY OF THE FIXED-EFFECTS RESULTS TO THE PROPORTIONAL SELECTION ADJUSTMENT ASSUMPTION – POINT ESTIMATES

	Baseline Effect Coef. $\dot{\beta}_1$ ($\dot{R}^2$)	Controlled Effect Coef. $\hat{\beta}_1$ (R^2)	Bias-adjusted effect Coef. β_1^*
Estimated effect of milk on the mortality rate of **prostate cancer** Table 7, Col (5)	0.829 (0.513)	0.545 (0.954)	0.516
Estimated effect of milk on the mortality rate of **prostate cancer** Table 7, Col (8)	0.829 (0.513)	0.562 (0.953)	0.533
Estimated effect of milk on the mortality rate of **ovarian cancer** Table 15, Col (6)	0.673 (0.466)	0.566 (0.943)	0.553
Estimated effect of milk on the mortality rate of **ovarian cancer** Table 15, Col (8)	0.673 (0.466)	0.581 (0.943)	0.570

Assumptions: $R^2_{\max}$ =1 and δ=1.

10. What can we learn from the results? Some illustrative simulations.

We have found relatively stable – in the sense of statistically significant – positive effects of milk on the mortality rate of prostate cancer as well as mortality rate of ovarian cancer. However, are the estimated effects important also in quantitative terms? For answering this question we choose a rather conservative estimate of the milk effects (SYS GMM with FOD in Column (5) of Table 8 and Column (5) of Table 16) as well as a "medium" estimate of the effect (Fixed Effects OLS in Column (5) of Table 7 and Column (6) of Table 15).

Furthermore, we try to answer the following question: *What would the mortality rates have been in 1991 to 2008 if the residents of the countries in the sample had had lower milk consumptions*? "*Lower*" means here that the given total calories intake is based on less milk and more on food from plant sources, such as cereals or vegetables. The simulations are always based on a "ceteris paribus" assumption, that means, all other variables such as the GDP per capita, the total calories intake, the sugar consumption, the country fixed effects (differences in health systems, genetic differences and so on) as well as the time fixed effects (common trends in medical progress and milk consumption) are unchanged.

In Table 24 we show the results for prostate cancer for three different scenarios. In the first scenario we assume that the proportion of total calories intake covered by milk is reduced by one quarter. Based on the observed sample mean of 7.8% (calories from milk in total calories intake) a reduction in the proportion of milk in total calories by one quarter to 5.85% reduces the mean mortality rate from 17.4 to 16.0 per 100,000 persons and per year according to the SYS-GMM FOD model. This annual reduction of 1.4 losses of lives per 100,000 persons corresponds to an annual decrease of 8%. The FE-OLS model predicts only an annual decrease of 3%.

In the second scenario in Table 24 it is assumed that milk consumption is reduced by 50% and therefore milk in total calories intake is only 3.9% (=7.8% / 2). Now, the number of men dying as a result of prostate cancer is reduced by 2.4 (SYS-GMM) or 3.9 (FE-OLS) per 100,000 persons and per year. This corresponds to an annual decrease of 14% or 23%.

The third scenario in Table 24 assumes that all countries reduce their milk consumption to 1% of total calories intake. This is approximately the level in Thailand. The mean proportion of milk in the sample is 7.8%. In countries such as Netherlands, Sweden Finland and Albania the proportion of milk in total calories is more than 13% (see Table 1 in Section 3.1). In Switzerland, Ireland and Romania it is more than 11%. Table 24 reveals that the conservative estimated annual effect of such a milk reduction is 30%, and 65% in case of the FE-OLS model.

TABLE 24: SIMULATIONS OF THE ANNUAL MORTALITY RATE OF PROSTATE CANCER, 1991-2008

	Observed sample means		**Predicted sample means**					
			SYS-GMM FOD Table 8, Col(5)			**Fixed Effects OLS** Table 7, Col (5)		
	Prop. of milk	*Mortality rate per 100,000*	*Mortality rate per 100,000*	*Change absolute*	*Change relative*	*Mortality rate per 100,000*	*Change absolute*	*Change relative*
Scenario	(1)	(2)	(3)	(4) =(3)-(2)	(5) =(4)/(2)	(6)	(7) =(5)-(1)	(8) =(6)/(1)
4. One-quarter reduction in milk consumption	7.8%	17.4	16.0	*-1.4*	*-8%*	16.8	*-0.6*	*-3%*
5. Halve the milk consumption	7.8%	17.4	15.1	*-2.4*	*-14%*	13.5	*-3.9*	*-23%*
6. Only 1% percent of total calories due to milk	7.8%	17.4	12.3	*-5.2*	*-30%*	6.1	*-11.3*	*-65%*

In Table 25 the simulations are shown for the mortality rate of ovarian cancer. First of all, it is important to note that the mean proportion of milk in total calories is slightly different than in Table 24 since the samples are not identical (see Section 3). Secondly, the initial level of the mortality rate of ovarian cancer is with 4.9 per 100,000 persons and per year much lower. However, the *relative changes* in the mortality rate resulting from the decreased milk consumption are comparable with those found for prostate cancer. For example, the third scenario indicates that if calories from milk would be reduced to 1% (approximately the level of Thailand) 1.8 up to 3.4 women per 100,000 persons would be saved from dying of ovarian cancer every year. These absolute numbers correspond to an annual decrease of 37% up 68%.

TABLE 25: SIMULATIONS OF THE ANNUAL MORTALITY RATE OF OVARIAN CANCER, 1991-2008

	Observed sample means		**Predicted sample means**					
			SYS-GMM FOD , Col(5)			**Fixed Effects OLS** Table 15, Col (6)		
	Prop. of milk	*Mortality rate per 100,000*	*Mortality rate per 100,000*	*Change absolute*	*Change relative*	*Mortality rate per 100,000*	*Change absolute*	*Change relative*
Scenario	(1)	(2)	(3)	(4) =(3)-(2)	(5) =(4)/(2)	(6)	(7) =(5)-(1)	(8) =(6)/(1)
1. One-quarter reduction in milk consumption	7.9%	4.9	4.7	-0.2	*-5%*	4.5	-0.4	*-9%*
2. Halve the milk consumption	7.9%	4.9	4.3	-0.6	*-13%*	3.6	-1.4	*-27%*
3. Only 1% percent of total calories due to milk	7.9%	4.9	3.1	-1.8	*-37%*	1.6	-3.4	*-68%*

11. Discussion and Conclusions

There is some evidence from previous studies that milk consumption may increase the risk of prostate cancer and ovarian cancer. While the findings in the majority of previous studies indicate that milk is harmful with regard to prostate cancer, previous results for ovarian cancer are less clear. For obvious reasons, there are no randomized controlled trials available for this topic, but large-scale observational studies. To the best of our knowledge three of those observational studies are based on aggregated country-level data. However, we are not convinced by the method of those country-level studies. The cross-sectional approach used by those studies is not an appropriate way of identifying a causal effect since it is almost impossible to control for all confounding factors (possible variables affecting the mortality rates).

For this reason, we do not use the cross-sectional (between-country) variation of the mortality rates and the nutrition variables, but time-series (within-country) variation to identify the effect of average milk consumption in countries on their mortality rates of cancer. That means, we estimate whether a change in milk consumptions over a time period of 25 years within countries is associated with a change in the mortality rate, after controlling for other factors. Since in this approach every country serves as its own "control group", that means, every country is compared with itself, the problem of confounding factors is clearly less of an issue.

Of course, we cannot rule out that our estimation results are still biased by confounding factors? Which kind of confounding factors are possible here? *First of all*, all confounding factors, which are constant over the time span of the analysis (1990-2008) – such as genetic differences, solar radiation, differences in living standards and certain aspects of the national health systems – are absorbed by the country fixed effects in the regressions analyses. That is exactly one reason why we perform our analyses also for shorter subsamples (1990-1999; 2000-2008), since this raises the plausibility, that confounding factors are time constant. *Secondly*, time varying confounding factors which are common to all countries, such as medical progress (better diagnostics and more effective therapies), are controlled by time fixed effects (a set of year dummy variables). *Thirdly*, time-varying differences in the living-standards and certain aspects of the national health system are hopefully controlled by the inclusion of the GDP per capita variable. *Fourthly*, we use dynamic models with a lagged dependent variable in order to control for time-varying confounders (omitted variables). *Fifthly*, we control for total calories intake per person and estimate the milk effect by specifying the milk as proportion of total calories intake which should already lead to a more conservative estimate. *Sixth-*

ly, we apply the approach proposed by Oster (2013) to evaluate the relevance of time-varying confounding factors and cannot find any evidence that there may be a problem. *Finally*, if there are time-varying confounders left which are correlated with the milk variable and if these omitted variables lead to a correlation of the milk variable with error term, instrumentation of the milk (and the other food) variable(s) may be a remedy. However, even after instrumentation, we find a significantly positive effect of the milk variable on the mortality rates in many specifications.

Summarizing all models, we find a statistically significant (harmful) effect of milk products (including cheese and excluding butter) on the mortality rates of both types of cancer.

In order to illustrate the quantitative meaning of the estimated effects we perform several simulations. By doing this we want to answer the question, what the mortality rates would have been in 1991 to 2008 if the residents of the countries in the sample had had lower milk consumptions. "Lower" means here that the given total calories intake is based on less milk and more on food from plant sources, such as cereals or vegetables. The simulations are always based on a "ceteris paribus" assumption, that means, all other variables are kept unchanged. Please note, that we do not claim that our models are suitable for accurate predictions – all we want to do is to make the estimation results understandable. The simulations show the following results: (1.) A moderate reduction in the consumption of milk products by 25% (for equivalent increases of vegetable food) would reduce the deaths from both kinds of cancer to less than 10%. (2.) A halving of the consumption of dairy products (for equivalent increases of vegetable food) would reduce the deaths of both types of cancer by 10% to 30%. (3.) If all countries reduced their milk consumption from 7.8% to 1% of total calories intake, which is approximately the level in Thailand, the number of deaths from both types of cancer would drop by 1/3 to 2/3.

The usual demand of empirical researchers applies here too: more data is needed. Since the methods applied are mostly based on large *N* (number of countries), and since the assumption that omitted variables can be captured by country fixed effects is more likely for small *T*, especially including more countries into the analysis could increase the credibility of the results.

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APPENDIX

TABLE A 1: SUMMARY OF EMPIRICAL STUDIES FOR MILK AND PROSTATE CANCER

Authors	Dep. Variable	Data	Association	Summary of Results (direct quotes)
Chan *et al.* (2001)	Incidence	Individual	+	These results support the hypothesis that dairy products and calcium are associated with a greater risk of prostate cancer.
Michaud *et al.* (2001)	Incidence	Individual Level	+	Intakes of red meat and dairy products appear to be related to increased risk of metastatic prostate cancer.
Ganmaa *et al.* (2002)	Incidence Mortality	Aggregate Country	+	Among the food items examined, milk (1961–90) was most closely correlated with prostatic cancer incidence (r=0.711)…The food (1961–90) most closely correlated with mortality of prostatic cancer was milk (r = 0.766)…
Berndt *et al.* (2002)	Incidence	Individual Level	0	Dairy products, including milk, were not associated with an increased risk of prostate cancer. The adjusted odds ratio of prostate cancer was 1.26 (95% confidence interval 0.57 to 2.79; P(trend) = 0.73) for men with high dairy intakes compared with those with low dairy intakes.
Rodriguez, *et al.* (2003)	Incidence	Individual Level	0	Dairy intake was not associated with prostate cancer risk.
Tseng *et al.* (2005)	Incidence	Individual Level	+	Our findings are consistent with most studies that observed an elevated risk of prostate cancer with greater dairy or milk intake
Zhang and Kesteloot (2005)	Incidence	Aggregate Country	+	In this ecological study, we demonstrated a strong, significant, positive association between milk consumption and incidence of prostate and female breast cancers.
Colli and Colli (2006)	Mortality	Aggregate Country	+	The strongest correlation between increased prostate cancer mortality and foods are: sugar (R=0.71), total animal calories (R=0.70), total animal fat calories (R=0.67), meat (R=0.65), coffee (R=0.65), alcoholic beverages (R=0.60), milk (R=.57), animal fat (R=0.55), …
Koh *et al.* (2006)	Incidence	Individual Level	0	shows no significant relation between higher intakes of dairy products and risk of prostate cancer, whether in age- or multivariate-adjusted analyses (P trend¼0.16 and 0.23, respectively).
Torniainen *et al.* (2007)	Incidence	Individual Level	+	Analysis of different milk products showed some evidence for low-fat milk as a potential risk factor for prostate cancer.
Park *et al.* (2007)	Incidence	Individual Level	(+) (–)	… we found no association between the intakes of calcium and vitamin D and prostate cancer risk, but low-/nonfat milk consumption was moderately associated with higher risk and whole milk consumption was associated with slightly decreased risk of prostate cancer
Ahn *et al.* (2007)	Incidence	Individual Level	+	… our study provides evidence that higher dairy product and dietary calcium intakes are modestly related to increased risk for prostate cancer, particularly nonaggressive disease.
Kurahashi *et al.* (2008)	Incidence	Individual Level	+	…our results suggest that the intake of dairy products may be associated with an increased risk of prostate cancer
Allen *et al.* (2008)	Incidence	Individual Level	+	The results support the hypothesis that a high intake of protein or calcium from dairy products may increase the risk for prostate cancer

Park *et al.* (2009)	Incidence	Individual Level	+	…dairy food was …. positively associated with prostate cancer.
Torfadottir *et al.* (2011)	Incidence	Individual Level	+	These data suggest that frequent milk intake in adolescence increases risk of advanced prostate cancer
Melnik *et al.* (2012)	Incidence Progression	Individual Level	+	Epidemiological evidence points to increased dairy protein consumption as a major dietary risk factor for the development of PCa [Prostate Cancer]. … Increased cow´s milk protein-mediated mTORC1 signaling along with constant exposure to commercial cow´s milk estrogens derived from pregnant cows may explain the observed association between high dairy consumption and increased risk of PCa in Westernized societies.
Pettersson *et al.* (2012)	Incidence Mortality	Individual Level	0 +	…our results suggest that among prostate cancer patients, overall intakes of milk and dairy products are not associated with a greater risk of lethal prostate cancer. We observed decreased risk of lethal disease among men with higher intakes of post-diagnostic low-fat dairy intake, and increased risk of lethal prostate cancer among men with higher intakes of whole milk ... our finding of an inverse association between low-fat dairy intake and risk of lethal prostate cancer should be interpreted with caution..
Song *et al.* (2013)	Incidence Mortality	Individual Level	+	… total dairy product intake and calcium from dairy foods were positively associated with overall risk of PCa. …, among all the PCa cases, we conducted a survival analysis to evaluate the associations of prediagnostic dairy food intake with risk of progression to fatal PCa after initial diagnosis and found that whole milk was the only dairy food that was significantly associated with an increased risk of PCa-specific mortality

TABLE A 2: SUMMARY OF EMPIRICAL STUDIES FOR MILK AND OVARIAN CANCER

Authors	Dep. Variable	Data	Association	Summary of Results (direct quotes)
Bosetti *et al.* (2001)	Incidence	Individual Level	0	Among other protein-rich foods, fish has been found to have a protective effect on cancer of the ovary, whereas milk, dairy products and eggs did not show any relevant association with ovarian cancer.
Goodman *et al.* (2002)	Incidence	Individual Level	– 0	Consumption of all dairy products, all types of milk, and low-fat milk was significantly inversely related to risk of ovarian cancer …, but consumption of whole milk was not. These results suggest that intake of low-fat milk, calcium, or lactose may reduce the risk of ovarian cancer.
Nagle *et al.* (2003)	Incidence Mortality	Individual Level	+	… the positive associations, and hence worse survival, seen with increasing intake of lactose, dairy products and calcium, with the highest third of intake carrying about a 30% excess risk of early death compared to the lowest third.
Pan *et al.* (2004)	Incidence	Individual Level	0	… we did not observe an association of ovarian cancer risk with dietary fat intake, including saturated, monounsaturated, and polyunsaturated fatty acids, protein, carbohydrate, dietary fiber, fruit, dairy products, meat products, fish, chicken, grain products, nut products, baked desserts, margarine, butter, mayonnaise, ...

Larsson *et al.* (2004)	Incidence	Individual Level	+	… women who consumed 4 servings of total dairy products/d had a risk of serous ovarian cancer … twice that of women who consumed 2 servings/d. No significant association was found for other subtypes of ovarian cancer. Milk was the dairy product with the strongest positive association with serous ovarian cancer.
Ganmaa and Sato (2005)	Incidence	Aggregate Country	+	The simple correlation coefficient, r, showed the greatest correlation between milk and ovarian cancer (r = 0.779),
Genkinger *et al.* (2006)	Incidence	Individual Level	(+)	…no associations were observed for intakes of specific dairy foods or calcium and ovarian cancer risk. A modest elevation in the risk of ovarian cancer was seen for lactose intake at the level that was equivalent to three or more servings of milk per day.
Koralek *et al.* (2006)	Incidence	Individual Level	0	No statistically significant relations were found for consumption of specific dairy foods, lactose, or vitamin D and ovarian cancer risk.
Kiani *et al.* (2006)	Incidence	Individual Level	(+) –	There was increased risk of ovarian cancer with higher whole fat milk intake but this was not statistically significant. Intake of low fat milk 1 time/day versus never, on the other hand, was associated with about a 50% reduced risk for both all ovarian cancer (p for trend = 0.05) and postmenopausal ovarian cancer (p for trend = 0.08).
Mommers *et al.* (2006)	Incidence	Individual Level	0	No association was seen between consumption of milk, yoghurt, cheese or fermented dairy products and ovarian cancer risk.
Schulz *et al.* (2007)	Incidence	Individual Level	0	…no significant association between the major animal food groups (total meat, eggs, fish, total dairy products) and risk of OVC [Ovarian Cancer], neither with the quintile analysis nor with the linear analysis (Table 2). In addition, meat subgroups (red meat, poultry, processed meat) and dairy products (milk, yogurt, cheese) did not show any relationships with incident OVC (Table 2).
Park *et al.* (2009)	Incidence	Individual Level	0	Dairy food, dietary, supplemental, and total calcium were not related to breast, ovarian, and endometrial cancer.
Dolecek, McCarthy and Joslin (2010)	Mortality	Individual Level	+	An increased HR (Hazard Ratio) was also observed for the milk (all types) subgroup …. … consumption of animal food products including red meats, cured/processed meats, milk (all types), and 2% milk showed increasing intakes to be statistically significantly associated with greater HRs and, thus, poorer survival time
Faber *et al.* (2012)	Incidence Progression	Individual Level	+	… intake of dairy products is associated with a modest increased risk of ovarian cancer. In addition, ovarian cancer development was associated with lactose intake
Merritt *et al.* (2014)	Incidence	Individual Level	0	These findings do not support the hypothesis that higher lactose intake increases EOC risk

TABLE A 3: LIST OF COUNTRIES IN THE SAMPLE

Poor		**Rich**	
Albania	Panama	Australia	Japan
Argentina	Paraguay	Austria	Kuwait
Brazil	Philippines	Canada	Netherlands
Bulgaria	Poland	Cyprus	New Zealand
Chile	Romania	Denmark	Norway
Colombia	South Africa	Finland	Portugal
Costa Rica	Sri Lanka	France	Republic of Korea
Ecuador	Thailand	Germany	Spain
Egypt	Trinidad and Tobago	Greece	Sweden
El Salvador	Uruguay	Hungary	Switzerland
Guatemala	Venezuela	Ireland	USA
Mauritius	(Cuba)	Israel	United Kingdom
Mexico		Italy	

TABLE A 4: DETERMINANTS OF (LOG OF) MORTALITY RATE OF PROSTATE CANCER – STATIC FIXED EFFECTS RESULTS: *P*-VALUES BASED ON DIFFERENT VARIANCE-COVARIANCE MATRIX ESTIMATORS

	(1) FE-OLS	(2) FE-OLS	(3) FE-OLS	(4) FE-OLS	(5) FE-OLS	(7) FE-OLS	(8) FE-OLS
Log GDP per capita, *t*-1	**0.319**	**0.429**	**0.179**	**-0.0218**	**0.0097**	**-0.0233**	**-0.0369**
	(0.000)	(0.000)	(0.060)	(0.816)	(0.915)	(0.816)	(0.714)
	[0.000]	[0.140]	[0.388]	[0.915]	[0.958]	[0.922]	[0.880]
	{0.000}	{0.004}	{0.126}	{0.808}	{0.919}	{0.865}	{0.783}
Log total calories intake		**1.279**	**1.115**	**1.530**	**1.029**	**1.055**	**0.916**
		(0.002)	(0.003)	(0.000)	(0.004)	(0.003)	(0.014)
		[0.365]	[0.352]	[0.171]	[0.409]	[0.403]	[0.401]
		{0.004}	{0.017}	{0.001}	{0.039}	{0.048}	{0.068}
Log milk / total			**1.332**	**0.891**	**0.545**	**0.540**	**0.562**
			(0.000)	(0.000)	(0.001)	(0.003)	(0.000)
			[0.000]	[0.003]	[0.138]	[0.139]	[0.125]
			{0.000}	{0.000}	{0.003}	{0.006}	{0.003}
Log sugar / total				**1.272**	**1.319**	**1.351**	**1.388**
				(0.000)	(0.000)	(0.000)	(0.000)
				[0.020]	[0.007]	[0.005]	[0.010]
				{0.000}	{0.000}	{0.000}	{0.000}
Log meat, fat, fish & eggs / total					**0.837**	**0.786**	**0.793**
					(0.000)	(0.000)	(0.000)
					[0.077]	[0.094]	[0.088]
					{0.000}	{0.000}	{0.000}
Log vegetables and fruits / total						**0.141**	**0.158**
						(0.288)	(0.23)
						[0.702]	[0.673]
						{0.448}	{0.386}
Log pulses / total						**-0.0589**	**-0.0295**
						(0.453)	(0.716)
						[0.744]	[0.869]
						{0.512}	{0.713}
Log vegetable oils / total							**-0.125**
							(0.158)
							[0.672]
							{0.092}

Notes: Fixed time effects and fixed country effects are not shown; see Table 7 for further details.
p-values based on: (usual standard errors),
[white-robust standard errors],
{Driscoll-Kraay Standard errors adjusted with the method proposed by Vogelsang, 2012)

FIGURE A 1: GOODNESS OF FIT OF THE SYS GMM MODEL WITH FOD IN COLUMN (5) OF TABLE 8 FOR THE DETERMINANTS OF LOG OF MORTALITY OF PROSTATE CANCER.

TABLE A 5: DETERMINANTS OF (LOG OF) MORTALITY RATE OF OVARIAN CANCER – STATIC FIXED EFFECTS RESULTS: *P*-VALUES BASED ON DIFFERENT VARIANCE-COVARIANCE MATRIX ESTIMATORS

	(1) FE-OLS	(2) FE-OLS	(3) FE-OLS	(4) FE-OLS	(5) FE-OLS	(6) FE-OLS	(8) FE-OLS
Log GDP per capita, *t*-1	**0.166** (0.014) [0.221] {0.345}	**0.510** (0.000) [0.015] {0.000}	**0.244** (0.023) [0.125] {0.222}	**0.135** (0.203) [0.407] {0.472}	**0.124** (0.240) [0.429] {0.489}	**0.137** (0.192) [0.374] {0.453}	**0.132** (0.265) [0.514] {0.520}
Log total calories intake		**1.976** (0.000) [0.285] {0.000}	**1.939** (0.000) [0.170] {0.003}	**2.143** (0.000) [0.143] {0.000}	**2.190** (0.000) [0.126] {0.000}	**1.907** (0.000) [0.148] {0.000}	**1.855** (0.000) [0.183] {0.016}
Log milk / total			**1.120** (0.000) [0.001] {0.000}	**0.765** (0.000) [0.002] {0.000}	**0.541** (0.000) [0.065] {0.008}	**0.566** (0.000) [0.056] {0.006}	**0.581** (0.000) [0.082] {0.004}
Log sugar / total				**0.951** (0.000) [0.040] {0.000}	**1.038** (0.000) [0.026] {0.000}	**1.174** (0.000) [0.014] {0.000}	**1.155** (0.000) [0.028] {0.000}
Log eggs / total					**0.300** (0.000) [0.149] {0.042}	**0.258** (0.000) [0.214] {0.081}	**0.249** (0.046) [0.217] {0.089}
Log vegetable oils / total						**-0.213** (0.000) [0.386] {0.041}	**-0.226** (0.026) [0.398] {0.036}
Log meat & fat / total							**0.0173** (0.907) [0.959] {0.927}
Log fish / total							**0.0140** (0.893) [0.963] {0.927}
Log vegetables & fruits / total							**0.0101** (0.953) [0.977] {0.969}
Log pulses/ total							**0.0224** (0.801) [0.878] {0.793}

Notes: Fixed time effects and fixed country effects are not shown; see Table 15 for further details.
p-values based on: (usual standard errors),
[white-robust standard errors],
{Driscoll-Kraay Standard errors adjusted with the method proposed by Vogelsang, 2012)

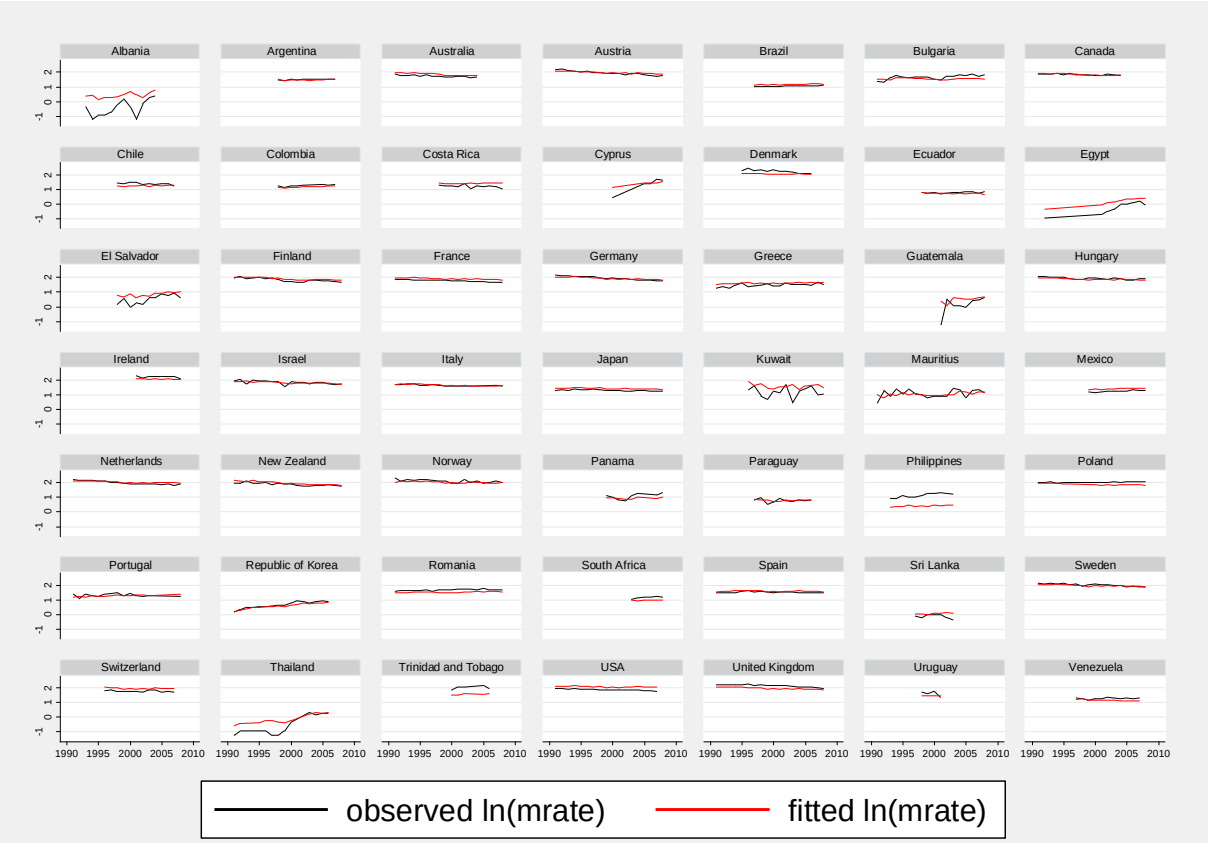

FIGURE A 2: GOODNESS OF FIT OF THE SYS-GMM MODEL WITH FOD IN COLUMN (5) OF TABLE 16 FOR THE DETERMINANTS OF LOG OF MORTALITY OF OVARIAN CANCER.