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ABSENCE OF EXCESS BODY FATNESS

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2.3. Excess body fatness in early life and subsequent cancer risk

WHO defines children as individuals younger than 19 years (WHO, 2016). The scope of this section includes children and young adults up to age 25 years, the age range collectively referred to as early life.

It is generally held that childhood obesity is strongly associated with obesity in adulthood. According to a recent systematic review (Simmonds et al., 2015), obese children are more than 5 times as likely as non-obese children to be obese as adults. However, childhood BMI is not a good predictor of the occurrence of obesity in adulthood; 80% of people older than 30 years who are obese were not obese in adolescence. Similarly, many obesity-related diseases occur in adults who had a healthy weight in childhood.

Few comprehensive reviews or meta-analyses are available on the topic of body shape and weight in early life and subsequent cancer risk. The literature review for this section identified three categories of studies: (i) prospective studies that directly measured weight and height in childhood and related these parameters with subsequent cancer occurrence; (ii) prospective cohort studies that determined body shape in early adulthood by recall and related these parameters with subsequent cancer occurrence; and (iii) studies that determined trajectories of body shape (from repeated determinations) from childhood to late adulthood and related these parameters with subsequent cancer occurrence.

[The Working Group considered that the relationship between weight at birth and subsequent cancer risk was beyond the scope of this *Handbook*.]

2.3.1 Weight and height measured in childhood

The few prospective studies that directly measured weight and height in childhood and related these parameters with subsequent cancer occurrence have been reviewed recently (Simmonds et al., 2015). These include data from the Helsinki Birth Cohort Study (ages 7 years and 15 years; Hilakivi-Clarke et al., 2001), the 1946 United Kingdom Medical Research Council National Survey of Health and Development (ages 2–15 years; De Stavola et al., 2004), the Copenhagen School Health Records Register (ages 7–15 years; Ahlgren et al., 2006; Aarestrup et al., 2014; Berentzen et al., 2014; Kitahara et al., 2014a, 2014b; Cook et al., 2015), the Norwegian health surveys (ages 14–19 years; Engeland et al., 2003; Bjørge et al., 2004, 2008), the Israeli army (ages 16–19 years; Levi et al., 2011; Leiba et al., 2013), and the Harvard Alumni Health Study (ages 18–21 years; Gray et al., 2012). [These cohorts have the advantage that height and weight were directly measured, but they have relatively small sample sizes. Because the baseline data were collected more than half a century ago, extrapolation to the current childhood and adolescent population may not apply, and it is not always clear whether these cohorts were representative of the general population.] The relationship between weight and height in childhood and subsequent cancer occurrence is presented separately for cancer of the breast (Table 2.3a) and for other cancers (Table 2.3b).

Table 2.3a lists study characteristics and breast cancer risk estimates from three studies (Hilakivi-Clarke et al., 2001; De Stavola et al., 2004; Ahlgren et al., 2006), which included a total of 3576 breast cancer cases. There was no evidence that excess weight directly measured in childhood is associated with subsequent breast cancer risk. Indeed, there is some evidence of an inverse association.

Table 2.3b lists, for boys and/or girls, study characteristics and risk estimates of mortality and incidence for the following types of cancer: colon cancer (Bjørge et al., 2008; Levi et al., 2011), rectal cancer (Levi et al., 2011), oesophageal adenocarcinoma (Cook et al., 2015), gastric non-cardia cancer (Levi et al., 2013), hepatocellular carcinoma (Berentzen et al., 2014), pancreatic cancer (Levi et al., 2012), ovarian cancer (Engeland et al., 2003), prostate cancer (Gray et al., 2012; Aarestrup et al., 2014; Batty et al., 2015), renal cancer (Bjørge et al., 2004; Leiba et al., 2013), urothelial cancer (Leiba et al., 2012), glioma (Kitahara et al., 2014a), and thyroid cancer (Farfel et al., 2014; Kitahara et al., 2014b). Although the number of studies per cancer type is small, for boys, excess weight in childhood and adolescence (generally expressed per increase of 1 or 2 standard deviations in BMI) was generally associated with increased risk of colon cancer (but not rectal cancer), oesophageal adenocarcinoma, hepatocellular carcinoma, pancreatic cancer, renal cancer, or urothelial cancer. There was no association with subsequent prostate cancer occurrence. For girls, there was evidence that excess weight in childhood and adolescence (generally expressed per increase of 1 or 2 standard deviations in BMI) was associated with increased risk of colon cancer (but not rectal cancer), oesophageal adenocarcinoma, hepatocellular carcinoma, and ovarian cancer. The association with renal cancer was uncertain [because of a large confidence interval]. No associations were seen for glioma or thyroid cancer in either sex.

2.3.2 Body shape in early adulthood determined by recall

A larger number of prospective cohort studies have determined body shape in early adulthood (ages 18–25 years) by recall, typically using the Sørensen scale (silhouette drawings), and converting the results to BMI values. [There is a risk of recall bias, but distributions of recalled BMI have been tested against BMI distributions from population data contemporaneous with the respective age strata and were found to be similar (Renehan et al., 2012). It is worth remembering that the mean values of BMI distributions of a cohort at ages 18–25 years are considerably lower than those in later adulthood. For example, in the NIH-AARP cohort, the mean BMI at age 18 years was 21.5 kg/m^2 in men and 20.8 kg/m^2 in women (Renehan et al., 2012). In addition, there is a survival bias, in that individuals have had to survive to baseline age (typically > 50) to participate in the cohort study. Finally, in these studies, risk estimates from multivariate analyses are commonly expressed as those from separate models adjusted for several potential confounders and as those from models adjusted for several potential confounders plus baseline (current-age) BMI. The latter models are of mechanistic relevance; for the purpose of a public health message in this *Handbook*, risk estimates from the former models are reported.]

These studies are dealt with in the individual cancer site-specific sections. Here, specific note is made in relation to breast cancer.

Prospective cohort studies of recalled BMI at ages 18–25 years and subsequent postmenopausal or premenopausal breast cancer risk are presented in Table 2.3c and Table 2.3d, respectively.

For BMI at ages 18–25 years determined by recall, several cohort studies showed no association (van den Brandt et al., 1997; Suzuki et al., 2011; Fagherazzi et al., 2013; Krishnan et al., 2013; Catsburg et al., 2014) or inverse associations (Ahn et al., 2007; Palmer et al., 2007; Baer et al., 2010; Kawai et al., 2010; White et al., 2012) with subsequent breast cancer risk. The same level of association was observed for postmenopausal (Table 2.3c) and premenopausal (Table 2.3d) women.

Some studies additionally evaluated BMI or weight at ages younger than 18 years determined

by recall: age at menarche in the French cohort (Fagherazzi et al., 2013), at age 12 years in the Iowa Women's Health Study (Bardia et al., 2008), and at ages 5 years and 10 years in the Nurses' Health Study (1988–2004) and the Nurses' Health Study II (1989–2005) cohorts (Baer et al., 2010) (data not shown in tables). These studies are consistent in showing that body fatness at ages 5–12 years or age at menarche is independently and inversely associated with subsequent premenopausal (Baer et al., 2010) and postmenopausal breast cancer (Bardia et al., 2008; Baer et al., 2010; Fagherazzi et al., 2013).

2.3.3 *Trajectories of body shape determined from early life*

Additional information may be gained by exploring weight changes with time and cancer risk. Recently, Song et al. (2016) reported combined analyses from the Nurses' Health Study (73 581 women) and the Health Professionals Follow-up Study (32 632 men) for several cancer sites (Table 2.3e). Using a data-driven latent class approach, they identified five distinct trajectories of body shape from age 5 years to age 60 years: maintained a lean body shape (lean-stable), started lean and experienced a moderate increase in body shape (lean-moderate increase), started lean and gained a substantial amount of weight (lean-marked increase), maintained a medium body shape (medium-stable), and started heavy and maintained or gained weight (heavy-stable/increase). Compared with women with the lean-stable trajectory, women with the lean-marked increase and the heavy-stable/increase trajectories had higher risks of colorectal, oesophageal, pancreatic, renal, and endometrial cancers. For postmenopausal breast cancer risk, early-life adiposity with no loss in later life (heavy-stable/increase trajectory) showed no association, whereas late-life adiposity (lean-marked increase trajectory) was positively associated. In men, excess body fatness during any life period was associated with a higher risk of colorectal cancer and oesophageal adenocarcinoma; in addition, the heavy-stable/increase trajectory was associated with a higher risk of pancreatic cancer and a lower risk of advanced prostate cancer.

In the French E3N cohort, Fagherazzi et al. (2013) evaluated the risk of breast cancer associated with body shape (using the Sørensen scale) at ages 8 years, age at menarche, 20–25 years, and 35–40 years. Six lifetime trajectories of body shape were derived, using a finite mixture modelling approach (Jones & Nagin, 2007). In this analysis, from age 8 years and/or at menarche, a constantly elevated body size was associated with a significantly decreased risk of ER-positive and PR-positive postmenopausal breast cancer (approximately 80% of breast cancers). No significant association with other body shape trajectories was found.

Table 2.3a Prospective studies of childhood cohorts where weight and height were directly measured and subsequent risk of cancer of the breast

Reference Cohort Period of study	Number at baseline (Birth cohort)	Number at follow-up	Number of breast cancers	Adult age at final follow-up (years)	Childhood age at measurement (years)	Relative risk (95% CI) per SD or unit increase in BMI
Hilakivi-Clarke et al. (2001) Helsinki Birth Cohort 1971–1995	3447 (1924–1933)	3447	177	Minimum, 38 (76% > 50)	7 15	0.91 (0.73–1.05) 0.85 (0.70–1.00)
De Stavola et al. (2004) United Kingdom Medical Research Council National Survey of Health and Development 1946–1999	2547 (March 1946)	2187	59	47–53	2 4 7 11 15	1.02 (0.78–1.33) 0.88 (0.67–1.14) 0.87 (0.66–1.15) 0.89 (0.68–1.18) 0.86 (0.65–1.14)
Ahlgren et al. (2006) Girls in Copenhagen, Denmark (Copenhagen School Health Records Register) Until 2001	161 063 (1930–1975)	117 415	3340	NR	14	0.97 (0.96–0.98)

BMI, body mass index (in kg/m^2); CI, confidence interval; NR, not reported; SD, standard deviation

Table 2.3b Prospective studies of childhood cohorts where weight and height were directly measured and subsequent risk of other cancers, by sex and by organ site

Reference Cohort	Number at baseline Period of recruitment	Number at follow-up	Number of cancers	Adult age at final follow-up (years)	Childhood age at measurement (years)	Relative risk (95% CI) per SD or unit increase in BMI
Boys						
Colon cancer: mortality						
Bjørge et al. (2008) Norwegian Cancer Registry	114 977 (1963–1975)	NR	97	Mean, 40	14–19	≥ 85th percentile vs 25th–75th percentile: 2.1 (1.1–4.1)
Colon cancer: incidence						
Levi et al. (2011) Israeli military cohort	1 109 864 (1947–1966)	NR	445	19–57	16–19	1.21 (1.07–1.38)[b]
Rectal cancer: incidence						
Levi et al. (2011) Israeli military cohort	1 109 864 (1947–1966)	NR	193	19–57	16–19	0.96 (0.88–1.10)[b]
Oesophageal adenocarcinoma: incidence						
Cook et al. (2015) Boys in Copenhagen, Denmark (Copenhagen School Health Records Register)	188 360 (1930–1989)	128 330	216	> 40	7 8 9 10 11 12 13	1.11 (0.95–1.30) 1.10 (0.94–1.29) 1.15 (0.98–1.35) 1.18 (1.00–1.38) 1.21 (1.03–1.42) 1.25 (1.07–1.47) 1.25 (1.06–1.46)
Gastric non-cardia: incidence						
Levi et al. (2013) Israeli military cohort	1 088 530 (1967/2005–2006)	NR	130	19–57	16–19	vs BMI 18.5–24.9: BMI 25–29.9: 0.98 (0.51–1.89) BMI ≥ 30: 2.62 (0.96–7.15)
Hepatocellular carcinoma: incidence						
Berentzen et al. (2014) Boys in Copenhagen, Denmark (Copenhagen School Health Records Register)	188 360 (1930–1980)	144 417	229	Median, 59	7 8 9 10 11 12 13	1.18 (1.01–1.37) 1.17 (1.00–1.37) 1.25 (1.07–1.47) 1.29 (1.10–1.51) 1.31 (1.12–1.53) 1.36 (1.16–1.59) 1.36 (1.17–1.60)
Pancreatic cancer: incidence						
Levi et al. (2012) Israeli military cohort	720 927 (1967–1995)	NR	98	29–56	16–19	1.17 (0.96–1.52)[b]

Table 2.3b (continued)

Reference Cohort	Number at baseline Period of recruitment	Number at follow-up	Number of cancers	Adult age at final follow-up (years)	Childhood age at measurement (years)	Relative risk (95% CI) per SD or unit increase in BMI
Prostate cancer: mortality						
Gray et al. (2012) Harvard Alumni Health Study	19 593 (1914–1952)	NR	NR	NR	Mean, 18.4	1.04 (0.93–1.16)
Prostate cancer: incidence						
Aarestrup et al. (2014) Boys in Copenhagen, Denmark (Copenhagen School Health Records Register)	188 360 (1930–1969)	133 647	3355	Median, 66.5 (range, 40–81)	7 8 9 10 11 12 13	1.04 (0.98–1.10) 1.04 (0.98–1.11) 1.02 (0.96–1.09) 1.03 (0.97–1.09) 1.02 (0.96–1.08) 1.02 (0.96–1.08) 1.02 (0.96–1.09)
Batty et al. (2015) Scottish Mental Health Survey Scotland, United Kingdom	2332 1947–2014	2332	109	Maximum, 77	11	0.97 (0.80–1.18)
Renal cancer: incidence						
Bjørge et al. (2004) Norwegian Cancer Registry	115 267 (1963–2001)	NR	109	Mean, 45	14–19	≥ 85th percentile vs 25th–75th percentile: 2.64 (1.48–4.70)
Leiba et al. (2013) Israeli military cohort	1 110 835 (1967–2005)	NR	274	Mean, 44	16–19	1.19 (1.04–1.37)[b]
Urothelial cancer:[a] *incidence*						
Leiba et al. (2012) Israeli military cohort	1 110 835 (1967–2005)	NR	661	Mean, 35	16–19	1.21 (1.06–1.38)[b]
Glioma: incidence						
Kitahara et al. (2014a) Boys in Copenhagen, Denmark (Copenhagen School Health Records Register)	188 360	162 295	355	> 40	7 8 9 10 11 12 13	1.01 (0.86–1.17) 1.04 (0.89–1.22) 1.03 (0.88–1.21) 1.02 (0.87–1.19) 1.02 (0.87–1.19) 1.00 (0.86–1.17) 1.04 (0.89–1.21)

Table 2.3b (continued)

Reference Cohort	Number at baseline Period of recruitment	Number at follow-up	Number of cancers	Adult age at final follow-up (years)	Childhood age at measurement (years)	Relative risk (95% CI) per SD or unit increase in BMI
Thyroid cancer: incidence						
Farfel et al. (2014) Israeli military cohort	1 145 865 (1967–2005)	NR	425	19–57	16–19	BMI, Q5 vs Q1: 1.19 (0.87–1.63)
Kitahara et al. (2014b) Boys in Copenhagen, Denmark (Copenhagen School Health Records Register)	165 978	162 632	64	> 40	7 8 9 10 11 12 13	1.22 (0.93–1.60) 1.24 (0.94–1.63) 1.23 (0.93–1.63) 1.21 (0.91–1.60) 1.24 (0.94–1.65) 1.25 (0.94–1.66) 1.25 (0.93–1.66)
Girls						
Colon cancer: mortality						
Bjørge et al. (2008) Norwegian Cancer Registry	111 701 (1963–1975)	NR	108	Mean, 43	14–19	≥ 85th percentile vs 25th–75th percentile: 2.0 (1.2–3.5)
Oesophageal adenocarcinoma: incidence						
Cook et al. (2015) Girls in Copenhagen, Denmark (Copenhagen School Health Records Register)	184 276 (1931–1971)	126 723	38	> 40	7 8 9 10 11 12 13	1.30 (0.90–1.87) 1.41 (0.97–2.06) 1.49 (1.02–2.16) 1.44 (0.99–2.11) 1.63 (1.12–2.36) 1.55 (1.07–2.26) 1.68 (1.15–2.44)
Hepatocellular carcinoma: incidence						
Berentzen et al. (2014) Girls in Copenhagen, Denmark (Copenhagen School Health Records Register)	184 276 (1930–1980)	141 467	62	Median, 60.2	7 8 9 10 11 12 13	1.20 (0.90–1.60) 1.12 (0.84–1.50) 1.12 (0.83–1.51) 1.03 (0.77–1.39) 1.05 (0.78–1.40) 1.15 (0.85–1.54) 1.23 (0.93–1.65)
Ovarian cancer						
Engeland et al. (2003) Norwegian Cancer Registry	NR (1963–1999)	111 883	7882	Mean, 41	14–19	1.22 (1.01–1.49)[b]

Table 2.3b (continued)

Reference Cohort	Number at baseline Period of recruitment	Number at follow-up	Number of cancers	Adult age at final follow-up (years)	Childhood age at measurement (years)	Relative risk (95% CI) per SD or unit increase in BMI
Renal cancer: incidence						
Bjørge et al. (2004) Norwegian Cancer Registry	111 954 (1963–2001)	NR	45	Mean, 45	14–19	≥ 85th percentile vs 25th–75th percentile: 1.48 (0.57–3.85)
Glioma: incidence						
Kitahara et al. (2014a) Girls in Copenhagen, Denmark (Copenhagen School Health Records Register)	184 276	158 130	253	> 40	7 8 9 10 11 12 13	0.96 (0.79–1.16) 0.95 (0.79–1.16) 0.95 (0.79–1.16) 0.87 (0.72–1.06) 0.93 (0.76–1.13) 0.91 (0.75–1.10) 1.01 (0.83–1.22)
Thyroid cancer: incidence						
Farfel et al. (2014) Israeli military cohort	478 445 (1989–2005)	NR	323	19–57	16–19	BMI, Q5 vs Q1: 1.14 (0.81–1.60)
Kitahara et al. (2014b) Girls in Copenhagen, Denmark (Copenhagen School Health Records Register)	161 262	158 453	171	> 40	7 8 9 10 11 12 13	1.13 (0.96–1.33) 1.12 (0.95–1.32) 1.18 (1.00–1.39) 1.14 (0.96–1.35) 1.11 (0.94–1.31) 1.09 (0.92–1.29) 1.13 (0.96–1.34)

BMI, body mass index (in kg/m^2); CI, confidence interval; NR, not reported; SD, standard deviation.

[a] Bladder, ureter, and renal pelvis.

[b] Taken from the systematic review and meta-analysis by Simmonds et al. (2015).

Table 2.3c Prospective cohort studies of BMI at ages 18–25 years determined by recall and subsequent risk of cancer of the breast in postmenopausal women

Reference Cohort Country	Total number in cohort	Follow-up period (years)	Baseline age (years)	Recall age (years)	Number of cases	Relative risk (95% CI)
van den Brandt et al. (1997) Netherlands Cohort Study The Netherlands	62 573	4.3	55–69	20	626	Per 8 kg/m²: 0.79 (0.58–1.08)
Ahn et al. (2007) NIH-AARP Diet and Health Study USA	99 039	3.9	50–71 All postmenopausal	18	2111	BMI ≥ 30.0 vs 18.5–22.4: HRT non-users 0.48 (0.27–0.86) HRT current users 0.65 (0.35–1.23)
Palmer et al. (2007) Black Women's Health Study USA	9542	10	21–69	18	442	BMI ≥ 25.0 vs < 20.0: 0.55 (0.37–0.82)
Baer et al. (2010) Nurses' Health Study (NHS) and NHS II USA	188 860	16	NHS, 30–55 NHS II, 25–42	20	4974	Per 1 kg/m²: 0.93 (0.90–0.95)
Kawai et al. (2010) Miyagi Cohort Study Japan	10 106	12.8	40–64	20	108	BMI ≥ 23.8 vs < 20.5: 0.44 (0.24–0.81)
Suzuki et al. (2011) Japan Public Health Cohort Study Japan	41 594	10	40–59	20	232	Per 5 kg/m²: 0.77 (0.59–1.02)
White et al. (2012) Multiethnic Cohort USA	82 971	NR	45–75	21	3030	BMI ≥ 30.0 vs < 20.0–24.9: 0.63 (0.43–0.91)
Fagherazzi et al. (2013) French E3N cohort France	81 089	NR	40–64	20–25	2828	Level ≥ 4 vs level 1:[a] 0.86 (0.74–1.00)
Krishnan et al. (2013) Melbourne Collaborative Cohort Study Australia	14 441	16.5	27–76 (99% 40–69)	18–21	668	Per 5 kg/m²: 0.90 (0.79–1.04)

Table 2.3c (continued)

Reference Cohort Country	Total number in cohort	Follow-up period (years)	Baseline age (years)	Recall age (years)	Number of cases	Relative risk (95% CI)
Catsburg et al. (2014) Canadian Study of Diet, Lifestyle and Health Canada	2210	12	67	20	541	BMI ≥ 30.0 vs 18.5–24.9: 0.21 (0.03–1.59)

BMI, body mass index (in kg/m^2); CI, confidence interval; HRT, hormone replacement therapy; NR, not reported

[a] Participants were asked to recall their body fatness by using a 9-level figure drawing, where level 1 represents the most lean and level 9 represents the most overweight.

Table 2.3d Prospective cohort studies of BMI at ages 18–25 years determined by recall and subsequent risk of cancer of the breast in premenopausal women

Reference Cohort Country	Total number in cohort	Follow-up period (years)	Baseline age (years)	Recall age (years)	Number of cancers	Relative risk (95% CI)
Palmer et al. (2007) Black Women's Health Study USA	42 538	10	21–69	18	491	BMI ≥ 25.0 vs < 20.0: 0.63 (0.46–0.87)
Baer et al. (2010); Michels et al. (2012) Nurses' Health Study (NHS) and NHS II USA	188 860	16	NHS, 30–55 NHS II, 25–42	20	2188	Per 1 kg/m^2: 0.89 (0.86–0.93)
Suzuki et al. (2011) Japan Public Health Cohort Study Japan	41 594	10	40–59	20	220	Per 5 kg/m^2: 0.78 (0.57–1.06)
Fagherazzi et al. (2013) French E3N cohort France	81 089	NR	40–64	20–25	745	Level ≥ 4 vs level 1:[a] 1.22 (0.88–1.69)
Catsburg et al. (2014) Canadian Study of Diet, Lifestyle and Health Canada	1110	14	45	20	556	BMI ≥ 30.0 vs 18.5–24.9: 0.96 (0.33–2.81)

BMI, body mass index (in kg/m^2); CI, confidence interval; NR, not reported

[a] Participants were asked to recall their body fatness by using a 9-level figure drawing, where level 1 represents the most lean and level 9 represents the most overweight.

Table 2.3e Relative risk of selected cancers according to trajectories of body shape from age 5 years to age 60 years in women and in men

Cancer type	Category of body shape trajectory[a]				
	Lean-stable	Lean-moderate increase	Lean-marked increase	Medium-stable	Heavy-stable/increase
Women					
Number of participants	13 183	18 405	18 217	23 288	11 699
Colorectal cancer	1.00	0.97 (0.80–1.17)	1.22 (1.00–1.49)	1.02 (0.85–1.22)	1.40 (1.13–1.74)
Oesophageal adenocarcinoma	1.00	1.02 (0.29–3.63)	2.56 (0.82–8.03)	1.04 (0.30–3.57)	2.19 (0.63–7.70)
Pancreatic cancer	1.00	1.18 (0.82–1.69)	1.36 (0.93–1.98)	1.15 (0.81–1.63)	1.39 (0.91–2.12)
Kidney cancer	1.00	1.26 (0.78–2.04)	1.89 (1.19–3.03)	1.05 (0.65–1.69)	1.92 (1.15–3.21)
Postmenopausal breast cancer	1.00	1.30 (1.17–1.45)	1.41 (1.26–1.58)	1.05 (0.94–1.17)	1.11 (0.97–1.28)
Endometrial cancer	1.00	0.99 (0.75–1.29)	1.57 (1.21–2.03)	0.94 (0.73–1.22)	2.08 (1.59–2.73)
Ovarian cancer	1.00	0.88 (0.66–1.16)	0.93 (0.70–1.25)	0.88 (0.67–1.15)	0.84 (0.59–1.19)
Men					
Number of participants	5946	6881	14 225	5725	4929
Colorectal cancer	1.00	1.36 (1.03–1.80)	1.23 (0.95–1.60)	1.26 (0.92–1.72)	1.47 (1.05–2.05)
Oesophageal adenocarcinoma	1.00	1.90 (0.67–5.34)	2.09 (0.80–5.48)	1.53 (0.48–4.84)	3.01 (1.04–9.13)
Pancreatic cancer	1.00	0.85 (0.54–1.35)	1.20 (0.81–1.78)	1.12 (0.70–1.80)	1.50 (0.92–2.46)
Kidney cancer	1.00	1.05 (0.67–1.64)	0.94 (0.63–1.43)	1.07 (0.66–1.74)	0.93 (0.53–1.64)
Advanced prostate cancer	1.00	1.16 (0.91–1.47)	0.97 (0.78–1.21)	1.00 (0.76–1.32)	0.67 (0.47–0.95)

[a] Trajectories of body shape: maintained a lean body shape (lean-stable); started lean and experienced a moderate increase in body shape (lean-moderate increase); started lean and gained a substantial amount of weight (lean-marked increase); maintained a medium body shape (medium-stable); started heavy and maintained or gained weight (heavy-stable/increase).

Source: Song et al. (2016). Data for women are from the Nurses' Health Study, and data for men are from the Health Professionals Follow-up Study.

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