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Radiation Dose and Cancer Risk Estimates in 16-Slice Computed Tomography Coronary Angiography

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Abstract

Background—Recent advances have led to a rapid increase in the number of computed tomography coronary angiography (CTCA) studies performed. While several studies have reported effective dose (E), there is no data available on cancer risk for current CTCA protocols.

Methods and Results—E and organ doses were estimated, using scanner-derived parameters and Monte Carlo methods, for 50 patients having 16-slice CTCA performed for clinical indications. Lifetime attributable risks (LARs) were estimated with models developed in the National Academies' Biological Effects of Ionizing Radiation VII report. E of a complete CTCA averaged 9.5 mSv, while that of a complete study, including calcium scoring when indicated, averaged 11.7 mSv. Calcium scoring increased E by 25%, while tube current modulation reduced it by 34% and was more effective at lower heart rates. Organ doses were highest to the lungs and female breast. LAR of cancer incidence from CTCA averaged approximately 1 in 1600, but varied widely between patients, being highest in younger women. For all patients, the greatest risk was from lung cancer.

Conclusions—CTCA is associated with non-negligible risk of malignancy. Doses can be reduced by careful attention to scanning protocol.

Over the past five years, technological advances in computed tomography (CT), most notably the introduction of multislice scanners with faster gantry rotation times, have made possible a rapid, accurate, and noninvasive assessment of the cardiovascular system.^{1, 2} Concomitant with the technological advances has been a rapid growth in the number of CT coronary angiography (CTCA) scans performed.

The convenience and outstanding image quality of CTCA is potentially offset by its attendant radiation exposure.³ It has been reported that CT scans currently contribute 75% to the collective radiation dose given to patients in a radiology department.⁴ While several estimates

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of typical doses encountered with CTCA have been reported⁵, including two recent reports estimating radiation dose in populations of patients under clinical practice conditions^{6, 7}, there is little data addressing organ dose^{8, 9} and no data on the relationship between radiation dose and cancer risk in actual patients undergoing clinical CTCA examinations.

In this study, we report on estimated radiation doses from a series of patients receiving clinically indicated CTCA and study the effect of scan protocol parameters on dosing. Effective dose (E) is determined using both data available from the scanner console and doses determined from a computer simulation model of radiation dose from CT using Monte Carlo methods. Equivalent doses to individual organs are determined. Finally, the attributable risks of fatal and nonfatal malignancy are estimated based on methodology recently developed by the National Research Council.

METHODS

Patients

Fifty consecutive patients having CTCA performed at the Mount Sinai Hospital for whom scan data were available were considered for the analysis. The study was approved by the Mount Sinai institutional review board.

CT Scan Protocol

All examinations were performed on a 16-slice multidetector-row CT scanner (Somatom Sensation 16 equipped with VB10 software, Siemens AG, Munich) with spiral technique. Intravenous beta blockers were given to lower patients' heart rates to a target rate of less than 60 beats per minute. Calcium scoring was performed if requested by the referring physician, or otherwise at the discretion of the performing physician. Five image data sets were collected: a low-dose topogram for localization, pre-contrast images for the detection of coronary calcium (denoted *calcium scan*), premonitoring images, monitoring images, and CTCA scan images. For a grouped description of protocol components, the following terms are employed in this paper: *bolus tracking* (referring to the combination of premonitoring and monitoring images), *complete CTCA* (denoting the premonitoring, monitoring, and CTCA scan images), and *complete study* (denoting the complete CTCA in addition to the calcium scan, if performed).

Calcium scan images were acquired using a gantry rotation time of 0.42 seconds, collimation of 16×1.5 mm, table feed of 6.8 mm/rotation, effective mAs (tube current, in mA, multiplied by gantry rotation time, in seconds, divided by pitch) typically 150 mAs, voltage of 120 kV, retrospective electrocardiographic gating, and electrocardiographically-controlled tube current modulation (ECTCM).

CTCA was performed using a bolus tracking approach. One or more premonitoring images were obtained to identify the location of the ascending aorta, using an effective mAs of 50 mAs and a voltage of 120 kV. Patients received an intravenous infusion of 80 cc of iodinated contrast, followed by 50 cc of saline, at a rate of 3.5–4.0 cc/sec. The arrival of the bolus to the ascending aorta was monitored in a region of interest placed in the tubular ascending aorta. Monitoring images were acquired each second, using an effective mAs of 50 mAs and a voltage of 120 kV, until the mean attenuation reached 100 Hounsfield Units. Four or five seconds after this threshold was reached, acquisition of the CTCA was automatically started, using a rotation time of 0.42 seconds, collimation of 16×0.75 mm, table feed of 3.4 mm/rotation, voltage of 120 kV, and retrospective electrocardiographic gating. An effective mAs of 500 mAs was adjusted by the performing physician on the basis of patient weight and habitus. ECTCM was employed at the physician's discretion, typically for all patients unless high heart rate or irregular rhythm suggested a likely role for systolic reconstruction images in the accurate

assessment of the patient's coronary anatomy.¹⁰ For patients with low baseline heart rates, a modified protocol with a table feed of 2.6 mm/rotation could be selected.

Scanner Data

Various parameters are used to quantify the radiation output of the CT scanner and the biological effects of ionizing radiation.^{11, 12} Scan time, beginning and end table positions, patient heart rate, tube voltage, maximum and mean effective mAs (the latter is lower when ECTCM is employed), volume CT dose index ($CTDI_{vol}$), and dose-length product (DLP)^{13, 14} were recorded from the CT scanner console.

Methods for Effective Dose and Organ Dose Estimation

For premonitoring and monitoring images E was determined from the scanner-derived DLP. E can be estimated from a DLP by means of the formula

$$E = E_{DLP} \cdot DLP \quad (1)$$

where E_{DLP} is a conversion factor, depending on region of the body, relating DLP to E ; here we used the European Guidelines on Quality Criteria for CT estimate for thorax E_{DLP} of $0.017 \text{ mSv} \cdot \text{mGy}^{-1} \cdot \text{cm}^{-1}$.¹⁴ For the calcium scan and CTCA, E was determined both from a scanner-derived DLP and equation 1 above, and also by performing Monte Carlo simulation, implemented using ImpactDose (VAMP GmbH, Erlangen, Germany). ImpactDose estimates radiation dose to organs and the whole body using a mathematical phantom, representing a male or female adult patient. It yields dosimetric estimates of primary radiation from measurements and manufacturer specifications, and of scatter using Monte Carlo calculations based on the approach of the Gesellschaft für Strahlen- und Umweltforschung.¹⁵ In the ImpactDose models, the bottom of the scan was placed just below the base of the heart, and the top of the scan was adjusted for each patient so that the ImpactDose scan length matched that in the actual scan.

Organ doses can be characterized both in terms of equivalent doses and weighted equivalent doses.¹⁶ The equivalent dose to a particular organ corresponds to the E of a hypothetical scan in which each organ received the same dose as did the particular organ in the original scan. Weighted equivalent dose corresponds to the contribution to the E of the radiation absorbed by the particular organ, and is calculated by multiplying the equivalent dose by a tissue weighting factor. Here, tissue weighting factors used were those of the International Commission on Radiological Protection (ICRP) in its 1990 recommendations, i.e. ICRP Publication 60.¹⁶ Weighted equivalent doses were determined for the 12 organs for which individual weighting factors are assigned in ICRP 60. ImpactDose estimates of E were determined by summing these 12 weighted equivalent doses, as well as a weighted equivalent dose reflecting the remainder of the organs.

Estimates of Attributable Risk of Cancer

Lifetime attributable risk (LAR) of cancer incidence and mortality was estimated for CTCA and calcium scoring, using models recently developed by the Nuclear and Radiation Studies Board of the National Academies, as reported in the Biological Effects of Ionizing Radiation VII (BEIR VII)–Phase 2 report.¹⁷ Organ equivalent doses were determined using ImpactDose as described above. A linear no-threshold model of cancer risk is assumed,^{18, 19} and thus LARs of cancer incidence and mortality were determined by multiplying the BEIR VII age- and gender-specific rates of cancer incidence and mortality from a 100 mSv organ exposure, by the ratio of the organ equivalent dose to the specified 100 mSv dose. Age-specific risks were determined by linear interpolation from risks at the two ages closest to the patient's age at the time of the scan. Organ-specific cancer rates were determined from equivalent doses for those organs specified in BEIR VII and ICRP 60. All-cancer LARs were estimated with the

above methods, by summing site-specific LARs for all organs, using a composite equivalent dose for “other” malignancies, relatively weighting each component by its ICRP 60 tissue weighting factor. E was related to LAR of cancer incidence by performing linear regression through the origin, i.e., using a regression model with y-intercept forced to the value of zero, so that an E of 0 would correspond to an LAR of cancer incidence of 0.

Statistical Analysis

Statistical analysis was performed using STATA 9.2 (StataCorp LP, College Station, Texas) and Excel 2003 (Microsoft, Redmond, WA). Continuous data is presented as mean $\pm$ standard deviation (range). Tests for normality were performed using the Shapiro-Wilk W test. Correlations between two variables were determined using the Pearson product moment correlation coefficient, or in the event of non-normality, using the Spearman rank order correlation coefficient. Comparisons between groups were performed using independent samples t-tests, paired samples t-tests, or Wilcoxon rank-sum or signed-rank tests as appropriate. All tests of significance were two-tailed; a P value of less than 0.05 was considered to indicate significance.

RESULTS

Patients

CTCAs were evaluated from 50 patients (30 males and 20 females). The mean age was 61 ± 12 (34–82) years. Nine patients who were status-post coronary artery bypass surgery, and two additional patients, had a scan including the heart, ascending aorta, and aortic arch. The low heart rate protocol was used in two patients. The average heart rate during the CTCA, after beta blockade, was 62.9 ± 8.6 (49–88) bpm.

Scan Parameters

The average maximum effective mAs for CTCA was 500 ± 37 (400–590) mAs. For the 33 subjects in which ECTCM was used, the average mean effective mAs was 387 ± 92 (250–550) mAs. For these patients, the average reduction in effective mAs, which approximately parallels the reduction in dose, was $34.5 \pm 4.8\%$ (20.4–44.4), and there was a significant inverse correlation between heart rate and this reduction ($r = -0.77$, $P < 0.001$).

Calcium scoring was performed according to protocol in 39 patients. ECTCM was used in all of these scans, with an average effective mAs reduction of $33 \pm 17\%$ (–3–64). Again, there was a significant inverse correlation between heart rate and the reduction ($r = -0.33$, $P = 0.04$).

Effective Doses

Scanner-derived DLPs for bolus tracking were not available for six patients. For the remaining 44 patients, Es for the various components of a standard scan are summarized in Table 1. Using ImpactDose to estimate E, the mean E was 8.8 ± 2.9 (3.4–15.9) mSv for CTCA and 2.7 ± 0.8 (1.4–4.2) mSv for calcium scores performed. Mean E for a complete study was 11.7 ± 2.7 (7.2–17.1) mSv. The E from the premonitoring scan(s) was small, with a maximum dose of 0.26 mSv. The E from the monitoring scan, however, varied widely, averaging 0.58 mSv but ranging up to 1.3 mSv, depending on the number of images acquired until the 100 Hounsfield Unit threshold was reached.

Mean Es were similar when estimated from scanner-derived DLPs: 8.7 ± 2.9 (3.9–16.6) mSv for CTCA and 2.7 ± 0.7 (1.5–4.5) mSv for calcium scores performed. There was close correlation between the two methods of determining E, with correlation coefficients of 0.84 for CTCA and 0.86 for calcium scoring both significant with $P < 0.001$. Nevertheless, mean E varied between male and female patients, depending on the method employed. While using

scanner-derived estimates the mean E for CTCA was greater in males than in females (9.2 vs. 7.9 mSv, $P = 0.07$), reflecting the higher tube currents (mean effective mAs after ECTCM of 393 vs. 373 mAs) employed for male patients who typically have larger habitus, using ImpactDose the mean E for CTCA was greater in females (9.6 vs. 8.2 mSv, $P = 0.06$), reflecting also the higher organ dose to the breast. Mean Es using ImpactDose for a complete CTCA and complete study, respectively, were 9.2 and 11.0 mSv in males and 10.1 and 12.7 mSv females.

The mean E was significantly greater in patients receiving a study including the ascending aorta and aortic arch, e.g. a bypass graft evaluation, than in a study in which only the heart was scanned. The mean ImpactDose estimates for a complete CTCA in these subjects were 12.5 mSv and 8.7 mSv, respectively ($P = 0.001$). This difference in E was due primarily to a significant difference in scan length (20.6 vs. 14.4 cm, $P < 0.001$), rather than a difference in $CTDI_{vol}$ (33.2 vs. 29.3 mGy, $P = 0.053$).

Organ Doses

Equivalent doses and weighted equivalent doses to individual organs from CTCA are illustrated in Figure 1. The highest weighted equivalent doses, reflecting the organ contributions to E, were those to the lungs (mean weighted equivalent dose 4.2 ± 1.3 (1.5–7.2) mSv) and, in women, breast (mean 1.9 ± 0.4 (1.3–2.8) mSv). These were followed by the esophagus (1.0 ± 0.3 (0.3–1.9) mSv), bone marrow (1.0 ± 0.4 (0.3–2.0) mSv), and stomach (0.7 ± 0.3 (0.3–1.2) mSv). In women, though equivalent dose to the breast was greater than that to the lung (mean 38 vs. 34 mSv, $P < 0.001$), the higher ICRP 60 tissue weighting factor for lung than breast led to a greater weighted equivalent dose for the lung. For the calcium scan, the greatest weighted equivalent doses were those to the lungs (1.2 ± 0.3 (0.7–1.7) mSv), female breast (0.5 ± 0.14 (0.3–0.7) mSv), esophagus (0.3 ± 0.08 (0.2–0.5) mSv), and bone marrow (0.3 ± 0.08 (0.2–0.5) mSv).

Estimates of Attributable Risk of Cancer

Estimates of LARs of cancer incidence and mortality from CTCA are summarized in Table 2. The average risk of developing cancer from a single CTCA was approximately 1 in 1600, and the average risk of dying from cancer from the CTCA was approximately 1 in 1900. These estimates varied widely from patient to patient, with maximum risks of about 1 in 500 and 1 in 700, respectively, in the sample studied. Risks for female patients were roughly twice those from male patients, particular for younger women, as is illustrated in Figure 2. There was a significant inverse correlation between LAR of cancer incidence and age for female patients ($r = -0.79$, $P < 0.001$), corresponding to lower cancer risk for older patients, and the same trend was observed for male patients, although without reaching statistical significance ($r = -0.32$, $P = 0.086$). The primary contributor to cancer incidence and mortality risk from the CTCA scans was lung cancer, which accounted for two thirds of all cases and three quarters of all deaths attributable to the CTCA. For all patients, the greatest risk of cancer was from lung cancer. There was a strong correlation between the E of the CTCA and the LAR of cancer incidence, as shown in Figure 3. LARs of cancer incidence and mortality from calcium scans were very small, averaging 1 in 5364 (range 1 in 1926 to 37092) and 1 in 6407 (range 1 in 2712 to 31620), respectively.

DISCUSSION

The main findings in this study are: (i) mean E for a complete CTCA was 9.2 mSv in males and 10.1 mSv in females, while the inclusion of calcium scoring increased this value by 12.25% to 11.0 and 12.7 mSv, respectively, (ii) weighted equivalent doses were highest to the lungs (4.2 mSv) and female breast (1.9 mSv), (iii) risks of developing cancer from CTCA averaged one in 1600 and risk of developing fatal cancer averaged one in 1900, and (iv) the

primary contributor to cancer risk from CTCA is lung cancer, due to its high weighted organ equivalent dose.

Radiation Dose in CTCA

The contrast-enhanced CTCA scan is generally regarded as the primary source of radiation exposure to the patient undergoing a complete cardiac study. Our data suggests that the other components of the scan can contribute sizably to the total E. Bolus tracking can add up to 25% of the E of the angiogram. Calcium scoring together with bolus tracking typically adds an additional radiation burden of 37% of the angiogram dose; in a worse-case scenario this can double the E. These results underscore the importance of optimizing every aspect of a scan protocol, not just the CTCA scan, so as to minimize radiation dose to the patient. For example, the topogram can be performed with a tube voltage of 90 kVp and low current; the calcium scan should utilize prospective gating when available and be limited to the region of the heart even in studies for which the CTCA scan incorporates the aorta; and monitoring images should be performed with low tube current and not be obtained earlier than approximately 10 seconds after contrast injection. Particularly significant for the CTCA scan is the use of ECG gated tube current modulation, which here resulted in an average dose reduction of roughly 34%. We found this method to be more effective at lower heart rates, as has been previously described²⁰, underscoring the importance of beta blockade. Alternative approaches for reducing radiation dose during CTCA include low-voltage protocols, particularly in thin patients²¹, or algorithms based on pre-contrast image noise.²²

We determined E from CTCA using two different methods, one using Monte Carlo methods and the other calculated from a scanner-derived DLP. While there was close correlation between the two estimates of E, and means for the entire population were virtually identical, calculations based on DLP underestimated E to female patients and overestimated E to male patients in comparison to ImpactDose estimates. This reflects the fact that the European Guidelines on Quality Criteria for CT thoracic E_{DLP} estimate of 0.017 is a composite value for males and females, thereby underestimating breast dose in females but overestimating it in males. Validated gender-specific conversion coefficients, while desirable, are not currently available. Thus, the ImpactDose estimates of E should be regarded as the more accurate ones here.

The dose estimates reported in this study are those of reasonably typical 16-slice CTCA exams, but dose will vary depending on the scan protocol used. Baseline tube voltage, tube current, scan area, and scan time, as well as the employment of tissue attenuation- and/or electrocardiographically-controlled tube current modulation, can markedly affect organ doses, effective dose⁷, and LARs of cancer. Consistent with this, Figure 1 demonstrates a marked variability between patients in the doses to individual organs. Es here were lower than those in another recent report using 16-slice CTCA by Cole et al.⁶, despite the fact that the two studies employed similar tube current, tube voltage, and pitch. A notable difference between the two studies was the employment of ECTCM in 66% of patients in this study. Had this technique been employed in a comparable proportion of patients by Cole et al., mean E would be reduced from 14.7 to 11.3 mSv, assuming a comparable dose reduction. Another difference between the two studies was that this study employed Monte Carlo methods using the approach of the Gesellschaft für Strahlen- und Umweltforschung, while Cole et al. used the equally respected approach of the National Radiological Protection Board.

Cancer Risk Modeling

Despite the numerous sources of uncertainty in cancer incidence and mortality rate estimates, the BEIR VII models adopted in this study provide the most comprehensive and updated assessment of risk currently available. Using this model in conjunction with the Monte Carlo

estimates of organ equivalent doses provided by ImpactDose constitutes the best feasible estimate of cancer risk from CTCA. The E for a complete study is on average 39 ± 5 (5–110)% greater than that for CTCA alone, and thus the LARs of cancer incidence and mortality would be expected to be correspondingly higher.

The BEIR VII model is based primarily on epidemiologic studies of survivors of the atomic bombings in Hiroshima and Nagasaki, and also on studies of individuals with occupational and medical exposures to radiation. Numerous assumptions underlie these models, and their applicability to estimation of cancer risk from CTCA. The assumption of a linear radiation dose-response relationship for solid tumors appears to best fit the existing evidence¹⁹, and is supported by several expert panels than have recently reviewed this data^{23–26} although this is sometimes contentious.^{27, 28} Confidence intervals on the LAR of malignancy estimated in the BEIR VII report reflect several important sources of uncertainty including statistical variability in model parameter estimation, uncertainty in transporting data from a Japanese to an American population with different baseline cancer rates, and uncertainty in adjusting risk from the atomic bomb survivor population to a population with low dose and low dose rate exposure. Additional confounding factors include errors in cancer detection and diagnosis, uncertainty in the optimal choice of mathematical model, secular trends in Japanese baseline cancer rates, accounting for differences in relative biological effectiveness between the γ rays and fast neutrons to which atomic bomb survivors were exposed and x-rays, and the extrapolation of atomic bomb survivor data to exposure scenarios, such as CTCA, where organs receive substantially different doses.¹⁷

The risk to an individual patient of malignancy from diagnostic x-rays is small but real. In a study encompassing 15 developed nations, the percentage of cancers attributed to diagnostic x-rays ranged from 0.6 to 4.4% and paralleled x-ray frequency, with the highest rate occurring in Japan, the country with the highest frequency of diagnostic x-rays.²⁹ The primary contributor to cancer risk in CTCA is lung cancer, due to its high weighted organ equivalent dose. In the worst case, we found the probability of developing cancer from a single CTCA study to be nearly one in 500, including a one in 800 chance of lung cancer. A test such as CTCA with a nontrivial risk of malignancy should be used judiciously, with appropriately selected patients and vigilance in the selection of scan settings and imaging protocols.

Study Limitations

In addition to the assumptions inherent in the cancer risk models, other factors limit the generalizability of our results. Radiation doses were determined from CTCA studies performed using a single 16-slice scanner at a single institution. Guidelines for CTCA protocols have not yet been published, and scan parameters vary from site to site for a given scanner. More significantly, radiation dosimetry, in particular the approach to radiation dose reduction, varies markedly among manufacturers and scanners.³⁰ The specific scanner in this study was set to use tube current modulation based on ECG gating, but not on tissue attenuation. Calcium score doses, while low in this study, could potentially be lowered further using a prospectively gated protocol. Sixteen-slice scanners, as studied here, currently represent the largest installation base of scanners capable of being used for CTCA, but the subsequent generation of multidetector-row scanners with 64 or more slices is rapidly gaining ground. Further investigation is required to characterize the radiation dosimetry and cancer risks of these new scanners. Towards this goal, a recent Monte Carlo study evaluated the effect of age, sex, and scan protocol on cancer incidence attributed to 64-slice CTCA and noted the potential for higher cancer risks than were observed here.³¹

The concept of effective dose was defined by the ICRP to be applied to populations¹⁶, and not for specific individuals. While the existence of conversion factors, such as those offered by the European Guidelines on Quality Criteria for CT, makes it easy to estimate an effective dose to

a particular patient from a DLP reported on the scanner console, such use is “off label.” Similarly, even Monte Carlo simulations, which can incorporate more patient-specific information into organ dose estimates, should not strictly speaking be used to estimate effective dose for an individual patient, since the tissue weighting factors used to calculate E, which reflect the relative stochastic risks to different organs, are gender-averaged and not patient-specific. More properly, stochastic risk from a particular imaging study to a patient should be characterized in terms of absorbed or equivalent doses to critical organs.

Nevertheless, many physicians understandably seek a single number to simply characterize radiation risk from an individual patient study, and effective dose is increasingly being used for this purpose. While for comparisons here we report Es for individual studies, in actual clinical practice this should be done with great caution and an understanding of its limitations.

A limitation in dose estimation in general is that current methods are based on standardized patient phantoms, and as such accurately estimate the radiation dose to a patient only insofar as the phantom is reflective of the patient’s habitus and anatomy.¹² Thus, while numerous factors in the dosimetry calculations in this study were matched to those in individual patient scans, including the scanner model, tube current and voltage, scan range, gantry rotation time, and pitch, it is not currently possible to precisely simulate all aspects of a scan, most notably patient anatomy, thereby necessitating the assumption of standardized anatomic phantoms. While accurate patient-specific dosimetry would be desirable, this would require Monte Carlo simulations utilizing phantoms based on retrospective three-dimensional organ segmentation. The effect of habitus and anatomy on organ doses in CT is an area requiring further investigation.

CONCLUSIONS

CTCA is associated with a non-negligible risk of malignancy, which is greater in younger women. Calcium scoring and bolus tracking algorithms add to radiation doses, while ECG modulation results in a substantial reduction in dose. The risk of cancer attributed to CTCA mandates careful patient selection and imaging protocol optimization.

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Figure 1.
The range of estimated Equivalent Doses and Weighted Equivalent Doses from CTCA

Figure 2.
BEIR VII estimate of lifetime attributable risk of cancer incidence as a function of age

Figure 3.
BEIR VII estimate of lifetime attributable risk (LAR) of cancer incidence versus effective dose (E) of CTCA determined with ImpactDose

Effective doses of scan components

		Effective Doses (mSv)				% of CTCA Dose Added by Calcium Score + Bolus Tracking		
		CaSc	Premonitoring	Monitoring	CTCA	Complete CTCA	Complete Study	
Impact Dose	Mean	2.7	0.12	0.58	8.8	9.5	11.7	37.2
	S.D.	0.8	0.05	0.18	2.9	2.9	2.7	19.9
	Minimum	1.4	0.09	0.24	3.4	4.3	7.2	4.1
	Maximum	4.2	0.26	1.28	15.9	16.5	17.1	111.3
Scanner-Derived	Mean	2.7	0.12	0.58	8.7	9.4	11.6	38.9
	S.D.	0.7	0.05	0.18	2.9	3.0	2.7	20.5
	Minimum	1.5	0.24	0.09	3.9	4.8	7.3	4.8
	Maximum	4.5	0.26	1.28	16.6	17.5	17.5	109.5

Top: E for CaSc and CTCA determined using ImpactDose

Bottom: E for CaSc and CTCA determined from scanner-derived DLPs.

CaSc = calcium scan, S.D. = Standard deviation

Table 2
Lifetime attributable risks (LARs) and odds of cancer incidence and mortality from CTCA

	Incidence			Odds			Mortality			Odds	
	LAR (per million)						LAR (per million)				
	Mean	Maximum	Mean	Maximum	Mean	Maximum	Mean	Maximum	Mean	Maximum	Maximum
All Cancers											
All	627	1952	1:1595	1:511	523	1365	1:1911	1:732			
Males	427*	754	1:2343	1:1326	406*	699	1:2463	1:1430			
Females	927*	1952	1:1078	1:511	699*	1365	1:1430	1:732			
All Solid Cancers											
All	571	1885	1:1749	1:529	473	1289	1:2115	1:775			
Males	363*	645	1:2756	1:1550	349*	604	1:2867	1:1653			
Females	884*	1885	1:1130	1:529	659*	1289	1:1517	1:775			
Lung Cancer											
All	412	1184	1:2427	1:844	403	1060	1:2480	1:942			
Males	288*	508	1:3471	1:1967	305*	527	1:3279	1:1897			
Females	598*	1184	1:1672	1:844	550*	1060	1:1816	1:942			
Breast Cancer											
Female	143	471	1:6972	1:2123	41	122	1:24337	1:8196			
Leukemia											
All	55	127	1:18111	1:7884	50	118	1:19871	1:8501			
Males	64*	127	1:15649	1:7884	57*	118	1:17496	1:8501			
Females	42*	84	1:23704	1:11905	40*	76	1:24954	1:13130			

* P≤0.01 for males vs. female