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対象の内訳		ヒト	動物	地 域	欧米	研究の種類	縦断研究
	対象	一般健常者	空白		( )		コホート研究
	性別	男性	( )		( )		( )
	年齢	30-79歳	[redacted]		( )		前向きコホート
	対象数	5000～10000			( )		( )
調査の方法	実測	( )					
アウトカム	予 防	なし	糖尿病予防	な し	なし		( )
	維持・改善	な し	な し	な し	なし	(	( )

図 表

Table 3. Adjusted Odds Ratios for Incidence of Impaired Fasting Glucose and Type 2 Diabetes by Cardiorespiratory Fitness Level and Other Potential Risk Factors Estimated by Multiple Logistic Regression

Variable	Odds Ratio (95% CI)	*P* Value
Impaired fasting glucose*		
Cardiorespiratory fitness levels†		
High	1.0	
Moderate	1.4 (1.1–1.7)	0.002
Low	1.7 (1.3–2.1)	<0.001
Age (every 10 years)	1.5 (1.3–1.7)	<0.001
Body mass index ≥ 27 kg/m^2	1.5 (1.2–1.8)	0.002
High blood pressure (>140/90 mm Hg or history of hypertension)	1.1 (0.9–1.4)	>0.2
Triglyceride level ≥ 1.69 mmol/L	1.2 (0.9–1.8)	0.16
Parental diabetes	1.2 (1.0–1.4)	0.12
Type 2 diabetes*		
Cardiorespiratory fitness level†		
High	1.0	
Moderate	1.4 (0.9–2.2)	0.11
Low	2.6 (1.6–4.2)	<0.001
Age (per 10 years)	1.6 (1.3–2.0)	<0.001
Body mass index ≥ 27 kg/m^2	2.0 (1.4–2.9)	<0.001
High blood pressure (>140/90 mm Hg or history of hypertension)	1.5 (1.0–2.2)	0.045
Triglyceride level ≥ 1.69 mmol/L	2.0 (1.4–2.7)	<0.001
Parental diabetes	1.9 (1.4–2.7)	<0.001

図表掲載箇所	P92, Table3
概 要 (800字まで)	30－79歳の8633名を対象に、体力が2型糖尿病の発症に関係しているかを6年間の追跡調査による検討した論文である。体力は、Balkeプロトコールに従い、トレッドミルの最大運動負荷テストにより評価され、(メッツで示された。心肺体力が13.7メッツの集団は、11.3メッツ、9.3メッツの集団と比較して、2型糖尿病を発症するリスクが、1.4(0.9-2.2)、2.6(1.6-4.2)と有意に高かった。
結 論 (200字まで)	体力を高く保つことで、糖尿病になるリスクを下げることが示された。
エキスパートによるコメント (200字まで)	体力を高めることは、様々な疾患予防に重要であることは、多くの研究により示されているところである。今後、国民全体において体力を高めることが出来るような方策へつなげていくことが強く必要とされている。

担当者　村上晴香

日本人男性における有酸素能力と生命予後に関する縦断的研究

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目的　低い有酸素能力が生活習慣病のリスクファクターであることが多くの研究により確認されており，生活習慣病の予防に有酸素運動が寄与することが知られている。しかしながら，有酸素能力と生命予後に関する縦断的研究は少なく，なかでも日本人を対象とした研究はみあたらない。本研究は，日本人における低い有酸素能力が全死因死亡のリスクファクターとなるか調査することを目的に，縦断的研究を実施した。

方法　T社において1982年から1984年の間に最大下運動負荷テストおよび定期健康診断を受診した男性9,986人（平均36.7歳：19–59歳）を対象とした。これらの対象者を平均14年間追跡し，死亡情報を把握した（総観察人年：139,836人年）。観察期間中の死亡者数は247人であった。対象者を観察開始時の推定最大酸素摂取量で5分位に分類し，Cox比例ハザードモデルを用いて最も低い有酸素能力群（Q_1）を基準として各群の年齢調整ハザード比と年齢，BMI，高血圧の有無，および尿蛋白陽性の有無を調整した多変量調整ハザード比を求めた。

結果　Q_1に対する各群の年齢調整ハザード比（95%CI）は0.54（0.39–0.77），0.66（0.47–0.94），0.58（0.39–0.86），0.46（0.27–0.78）であり，いずれも有意に低いハザード比であった。また，多変量調整ハザード比は0.52（0.37–0.73），0.60（0.42–0.87），0.50（0.33–0.75），0.39（0.22–0.67）であり，年齢以外の交絡因子を調整した後も有意に低いハザード比を示した。

以上の結果は，日本人においても低い有酸素能力は全死因死亡のリスクファクターであることを示唆している。

Key words：生命予後，有酸素能力，身体活動，最大酸素摂取量，比例ハザード，縦断的研究

I　はじめに

厚生省は1996年，日本における主要死因（悪性新生物・脳血管疾患・心疾患）の一次予防を重視する観点から，これまでの「成人病」に代わる「生活習慣病」という新たな概念を導入した[1)]。生活習慣病は「食生活，運動習慣，休養，喫煙，飲酒等の生活習慣が，その発症・進行に関与する疾患群」と定義されており[1)]，生活習慣を改善することにより，その疾病の発症や進行を予防できると考えられる疾患である。運動習慣に関しては縦断的調査により，高い有酸素能力または持久的な身体活動が，悪性新生物[2～5)]・脳血管疾患[6,7)]あるいは心疾患[8～18)]による死亡率を低下させることが報告されている。また，持久的な身体活動が有酸素能力を高めることが知られている[19)]。

これらのことから，高い有酸素能力を維持することは主要死因を予防することにつながると考えられる。いいかえれば，高い有酸素能力の維持が生命予後によい影響を及ぼすことが推測される。しかしながら，有酸素能力と生命予後に関する縦断的研究は少なく，また対象者は欧米人に限られており日本人を対象とした研究はみあたらない。そこで本研究は，日本人における低い有酸素能力が全死因死亡のリスクファクターとなるか調査することを目的に，日本人男性労働者を対象とした縦断的研究を実施した。

II　研究方法

1.　対象者

対象職域であるT社は，首都圏でガスの供給・販売を主な業務とするガス会社である。社員数

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は調査を開始した1982年3月末時点で12,899人（男性：11,978人・女性：921人），平均年齢は男性37.9歳，女性26.1歳であった。

本研究の対象者は，T社において1982年度から1984年度の3年間に定期健康診断および運動負荷テストを受診した男性9,986人であった。女性社員については人数が少ないことから本研究の対象から除いた。

2. 追跡調査

対象者が3年間のなかで最初に運動負荷テストと定期健康診断を同じ年に受けた際のデータを調査開始時時点のデータとして採用した。1996年3月31日を調査終了とし，この間の生存・死亡情報を調査した。平均観察期間は14年（範囲：4–178カ月）であった（総観察人年：139,836人年）。

退職後の死亡については，T社退職者で組織された会の事務局による家族に対する電話聞き取り調査により死亡状況を把握した。退職者で組織された会では，毎年会員の在籍を確認しており追跡期間中の全死亡を把握している。また，在職中の死亡については，人事情報をもとにT社医療スタッフが死亡者の所属する職場総務担当者に対する電話聞き取り調査により死亡状況を把握した。追跡期間中の全死亡者について，死亡状況より死因をICD–10（International Statistical Classification of Diseases and Related Health Problems, Tenth Revision）の死因分類表[20]に従って分類した。死因が特定できなかった4人については，全死因死亡の解析のみに含めた。

観察終了まで生存していた例は観察終了時点で観察打ち切りとした。また，T社退職者で組織された会に入会しなかった526人（5.3%）については，死亡の記録を把握できないことから，退職時点で観察打ち切りとした。

3. 検査項目および測定方法

労働安全衛生法に基づく健康診断内容から身長，体重，血圧，および尿検査の測定結果を解析に用いた。身長，体重の測定から，Body Mass Index（BMI：体重（kg）/身長（m）^{2}）を求め，体格の指数とした。安静血圧の測定は椅子座位で水銀血圧計を用いて測定した。尿蛋白および尿糖は試験紙法により測定した。

運動負荷は，モナーク社製自転車エルゴメータを用いて各段階4分間の最大下負荷を2段階あるいは3段階かけた。心拍数は心電図のR–R間隔から算出した。年齢と性別から推定した最大心拍数（220−年齢）の85%を目標心拍数に設定し，目標心拍数に到達した者はその時点で運動負荷テストを中止した。最終段階の最後の1分間から得られた仕事量と最後の10秒間から得られた心拍数からÅstrandとRyhmingのノモグラム[21]とÅstrandの年齢補正係数[22]を用いて最大酸素摂取量（maximal oxygen uptake: $\dot{V}O_2max$）を推定した。運動負荷テストは，実施前に対象者から循環器疾患者あるいは糖尿病患者を事前に除いていおり，それぞれの疾患や薬剤の服用が$\dot{V}O_2max$測定果へ及ぼす影響を取り除いた。また，測定当日も治療の有無，最近の健康状態を確認して，運動負荷試験の実施が困難と判断された者は対象から除いた。

また，運動負荷テストの実施直前に自記式アンケートにより，日常生活での運動実施状況を4段階で把握し，各段階をポイント化して集計した（何もしない：1ポイント・職場体操程度の運動をする：2ポイント・職場体操以外にも時々運動する：3ポイント・職場体操以外にも毎日運動する：4ポイント）。

4. 統計手法

BMIは日本肥満学会の分類基準[23]に基づいて4群に分類した。BMIが19.8未満を「やせ」，19.8以上24.2未満を「普通」，24.2以上26.4未満を「過体重」，26.4以上を「肥満」とした。血圧については，収縮期血圧が160 mmHg以上あるいは拡張期血圧が95 mmHg以上を高血圧群とし，非高血圧群と2群に分類した。また，$\dot{V}O_2max$は5分位に分類し，$\dot{V}O_2max$が30.9 ml/kg·min未満を「Q_1」，30.9以上34.5未満を「Q_2」，34.5以上38.1未満を「Q_3」，38.1以上41.9未満を「Q_4」，そして41.9以上を「Q_5」とした。

有酸素能力と生命予後に関する量的な評価および交絡因子の調整のためにCox比例ハザードモデル[24]を用いた。共変量として年齢（連続数），身長（連続数），体重（連続数），BMI（4群），高血圧（有無），尿糖陽性（有無），尿蛋白陽性（有無），運動実施状況（4群）を投入し，ステップワイズ法により比例ハザードモデルの当てはまりがもっともよくなる共変量を選択し，年齢，BMI，高血圧，尿蛋白陽性を採用した。そして，$\dot{V}O_2max$の最も低い群（Q_1）を基準として各分

位の全死因死亡のハザード比を求めた。ハザードの比例性の検討は，ログマイナスログ・プロットを用いて視覚的に検討し，プロットから比例性が成立していることを確認した。統計解析には，SPSS 統計パッケージ（Statistical Package for Social Science）を使用した。有意水準は p 値を0.05として，p 値がこれより小さければ統計的に有意とした。

Ⅲ 研究結果

調査開始時点での平均年齢（±SD）は36.7±9.6歳（範囲：19–59歳）であった。対象者の調査開始時点における BMI は，「普通」が全体の61.1%を占め，「肥満」は8.5%であった。血圧については94.9%が正常であった。また，運動実施状況については「何もしない」あるいは「職場体操程度」と答えた対象者が全体の55.3%であり，ほぼ半数の対象者が非活動的な生活を送っていると考えられた。生存群と死亡群の調査開始時点における身体的特徴を表1に示した。死亡群は247人（2.5%）であった。生存群と比較して死亡群は年齢，収縮期血圧，拡張期血圧が有意に高く，$\dot{V}O_2max$ は有意に低かった。また，運動実施状況は死亡群においてやや低いポイントを示した。

表2に死因，死亡年齢別にみた死亡者数および調査開始時点における年齢別対象者数を示した。死因別に分類すると，循環器疾患（ICD：9000–9500）72人（29.2%），全がん（ICD：2000–2202）112人（45.3%），その他63人（25.5%）であった。

表3に有酸素能力別にみた対象者の調査開始時点における身体的特徴を示した。有酸素能力の高い群ほど平均年齢が若くなる傾向にあった。BMI，収縮期血圧，および拡張期血圧は有酸素能力の高い群ほど低くなる傾向にあった。また，自記式アンケートから得られた運動実施状況は有酸素能力の高い群ほどポイントが高くなる傾向にあった。

最も低い有酸素能力群（Q_1）に対する各分位

表1　生存群および死亡群の調査開始時点における身体的特徴

	生存群	死亡群	p 値
人　数（人）	9,739	247	
年　齢（歳）	36.4±9.5*	45.8±9.0	<0.001²*
BMI	22.7±2.6	22.6±2.7	0.45²*
$\dot{V}O_2max$ (ml/kg·min)	36.8±7.0	33.3±6.4	<0.001²*
収縮期血圧 (mmHg)	125.7±13.3	127.5±15.0	0.03²*
拡張期血圧 (mmHg)	74.8±12.1	79.2±12.0	<0.001²*
運動実施状況 (ポイント)	1.43±0.78	1.34±0.72	0.05³*

：平均±SD　²：t 検定　³*：U 検定

表2　死因，死亡年齢別にみた死亡者数および調査開始時点における年齢別対象者数

	39歳以下	40–49歳	50–59歳	60–69歳	70歳以上	計
全死因	20	47	88	80	12	247
循環器疾患	3	13	22	29	5	72
全がん	5	20	51	32	4	112
その他	12	14	15	19	3	63
対象者数	6,153	2,564	1,269	—	—	9,986

表3　有酸素能力別にみた対象者の調査開始時点における身体的特徴

	Q_1	Q_2	Q_3	Q_4	Q_5
$\dot{V}O_2max$ (ml/kg·min)	27.5±2.4*	32.3±1.0	35.9±1.0	39.9±1.3	47.3±4.8
人　数	1,793	2,038	2,123	2,143	1,889
年齢（歳）	42.6±8.6	39.6±8.9	36.6±8.9	34.3±8.8	30.7±8.2
BMI	24.3±2.7	23.4±2.4	22.7±2.4	22.0±2.2	21.4±2.1
収縮期血圧（mmHg）	129.7±13.9	126.9±13.3	125.7±13.2	123.9±13.1	122.7±12.1
拡張期血圧（mmHg）	80.6±11.3	77.6±11.6	75.0±11.7	72.2±11.4	69.7±11.3
運動実施状況（ポイント）	1.29±0.73	1.38±0.74	1.36±0.78	1.48±0.76	1.62±0.82

*：平均±SD

表4 最も低い有酸素能力群（Q_1）に対する各分位の年齢調整ハザード比および多変量調整ハザード比（全死因）

	人数	死亡者数	年齢調整ハザード	95%Cl	p値	多変量調整*ハザード比	95%Cl	p値
Q_1	1,793	96	1.0	—	—	1.0	—	—
Q_2	2,038	50	0.54	0.39–0.77	<0.001	0.52	0.37–0.73	<0.001
Q_3	2,123	49	0.66	0.47–0.94	0.022	0.60	0.42–0.87	0.006
Q_4	2,143	35	0.58	0.39–0.86	0.006	0.50	0.33–0.75	<0.001
Q_5	1,889	17	0.46	0.27–0.78	0.004	0.39	0.22–0.67	<0.001
			p<0.01^2*			p<0.001^2*		

*：共変量：年齢，BMI，高血圧の有無，尿蛋白陽性の有無
2*：トレンド検定

表5 最も低い有酸素能力群（Q_1）に対する各分位の多変量調整ハザード比（循環器疾患，全がん）

	循環器疾患死亡者数	多変量調整*ハザード比	95%Cl	p値	全がん死亡者数	多変量調整*ハザード比	95%Cl	p値
Q_1	28	1.0	—	—	46	1.0	—	—
Q_2	14	0.52	0.27–1.00	0.051	27	0.60	0.37–0.98	0.039
Q_3	14	0.68	0.35–1.33	0.259	19	0.51	0.29–0.89	0.018
Q_4	9	0.53	0.24–1.16	0.111	14	0.45	0.24–0.85	0.013
Q_5	7	0.74	0.30–1.80	0.509	6	0.31	0.13–0.75	0.010
		p=0.25^2*				p<0.01^2*		

*：共変量：年齢，BMI，高血圧の有無，尿蛋白陽性の有無
2*：トレンド検定

の全死因死亡に関連する年齢調整ハザード比と，交絡因子として年齢，BMI，高血圧の有無，および尿蛋白陽性の有無を調整した多変量調整ハザード比を表4に示した。Q_1 に対する各分位の年齢調整ハザード比はいずれも有意に低く，有酸素能力が高くなるに従ってハザード比も低くなる傾向（$p<0.01$）を示した。多変量調整ハザード比は年齢調整ハザード比と比較してさらに低い値を示した。

循環器疾患死亡あるいは全がん死亡の多変量調整ハザード比を表5に示した。循環器疾患死亡においては多変量調整ハザード比が有意ではなかったが，Q_1 に対してすべての分位で低い傾向にあった。全がん死亡についてはすべての分位が Q_1 に対して有意に低く，有酸素能力が高くなるに従って多変量調整ハザード比も低くなる量反応関係（$p<0.01$）を示した。

Ⅳ 考 察

1. 対象者について

本研究の対象者は首都圏に住居を持つ男性労働者であり，対象者の有酸素能力が日本人男性を代表するか，小林の作成した日本人成人の Aerobic Power に関する5段階の評価区分[25]を利用して検討した。この評価区分では35歳から39歳の「普通」にあたる値は34.6～42.5 ml/kg·min であった。本研究の対象者における $\dot{V}O_2max$ の平均値（±SD）は36.7±7.0 ml/kg·min であり「普通」の範囲内であった。以上のことより本研究の対象者は有酸素能力に関して，おおむね日本人男性を代表していると考えられた。

2. データの妥当性

1） 有酸素能力測定の妥当性

本研究で有酸素能力の指標として用いた $\dot{V}O_2max$ の推定式である Åstrand と Ryhming のノモグラムの推定精度に関してはその妥当性を評価する多くの報告がある[21,26~28]。例えば，

Shephard[26]は10人の男性を対象にÅstrandとRyhmingのノモグラムを用いて推定した$\dot{V}O_2max$と，直接法にて測定した$\dot{V}O_2max$と比較して r=0.87の相関があったと報告している。これらのことから，本研究において推定した$\dot{V}O_2max$の値は対象者の有酸素能力をほぼ反映していると考えられる。

2）死因の妥当性

本研究では死因に関する把握は社内医療スタッフおよび退職者で組織された会の事務局の聞き取り調査により死因を把握しており，死亡小票の確認は行っていない。しかしながら，本研究においては，電話による確認であることや，在職死亡者（160人：64.8%）については社内医療スタッフが死亡者についての在職中の健康診断結果あるいは社内診療所における医療情報等をあらかじめ持ったうえでの，死亡者の所属する職場総務担当者に対する電話聞き取り調査であったことから，ある程度の妥当性を確保していると考えられる。

死因において，本研究では全がん死亡が死亡者全体の45.3%という高い割合を占めている。本研究は，19歳から59歳を対象とした平均14年間の追跡調査であり追跡終了時点では70歳以上の死亡者は死亡者全体の4.9%（12人）しか観察できていない。このため，50～69歳の死亡者が67.2%（166人）を占めている。一方，日本人における50～69歳の死因の第1位は悪性新生物であり，5歳きざみで見た死亡割合（%）はそれぞれ，39.4，43.0，44.5，41.9と本研究と同様に高い割合を占めている[29]。これらのことから，本研究の高い全がん死亡割合は対象者の死亡年齢に起因していると考えられる。また，在職死亡者が死亡者全体の64.8%と高い割合を占める理由については，本研究では追跡終了時点で退職が対象者全体の18.2%（1,816人）しか発生しておらず，このために退職後の死亡数と比較して相対的に高い在職死亡者数を示していると考えられる。

3）解析の妥当性

本研究には，死亡の情報を把握できなかった562人においても，生存が確認されている期間の情報を生かすために，途中打ち切り例（censored case）を扱える比例ハザードモデルを採用し解析の精度を高めた。

観察開始初期の死亡例は，その死亡を引き起こした潜在的疾患によって低い有酸素能力をもたらした可能性がある。そこで本研究の対象者から5年以降の打ち切りデータのみを利用して多変量調整ハザード比を求めた（対象：9,720人）。その結果は全データを用いた解析結果とほぼ同じであった。加えて，30歳未満の若年層を除いた解析も実施したが，全データを用いた解析結果と同様の結果であった。

先行研究と比較した本研究の弱点は，生命予後と関係の深いと考えられる喫煙習慣や飲酒習慣あるいは血液生化学検査結果が比例ハザード算出時に多変量調整されていないことである。これらの交絡因子のなかで，とりわけ喫煙習慣については生命予後に影響を与える重要な因子と考えられ，多くの研究で交絡因子として調整されている[3～5,10,12～18]。Slatteryら[14]は，有酸素能力と全死因死亡について，喫煙調整前後のデータを示し，喫煙調整後も同様な結果であった事を報告している。しかしながら，男性における日本人の喫煙率は，欧米と比較して高いことから[30]，今後，日本人について喫煙習慣も考慮した研究が必要であると思われる。

3. 有酸素能力と生命予後

1）有酸素能力と全死因死亡

これまでに8つの有酸素能力と全死因死亡の関係に関する縦断的研究が報告されている[3,4,13～18]。これら8つの報告すべて，有酸素能力が全死因死亡に有意に関係していると報告している。また，6つの研究[3,4,14～16,18]は有酸素能力と全死因死亡の関係に量反応関係があることを報告している。

8つの研究のうち最も総観察人年が多い研究は，Kampertら[4]の研究である。彼らは米国人男性25,341人を対象にトレッドミルによる最大運動負荷テストを行い，テストから得られた有酸素能力を5分位に分けた。そして，全死因死亡について，Q_1に対する各分位の年齢，測定年，喫煙習慣の有無，慢性疾患の有無，異常心電図の有無を調整した多変量調整ハザード比（95%CI）が，それぞれ，0.55（0.44–0.70），0.61（0.48–0.78），0.52（0.41–0.66），0.49（0.37–0.64）であったと報告している。本研究の結果（表5）は彼らの結果と非常に似通っており，Q_1に対する各分位の多変量調整ハザード比はいずれも有意に低く，有酸素能力が高くなるに従って多変量調整ハザード比

も低くなる量反応関係を示している。これらのことは，日本人においても欧米人同様，低い有酸素能力が全死因死亡のリスクファクターであることを示唆している。

低い有酸素能力，または持久的な身体活動の不足が循環器疾患死亡[8～18]，高血圧[31～33]，がん[2～5]，およびインシュリン非依存性糖尿病[34]への罹患率や，それが原因となる死亡率を増加させることが報告されており，これら単一あるいは複合的な機序が低い有酸素能力と全死因死亡の関係を説明すると思われる。

2)　有酸素能力と循環器疾患死亡

有酸素能力と循環器疾患死亡との関係を縦断的に調査した研究はこれまでに16報告されている[3,8～18,35～38]。これら16の報告のうち12[3,8～18]は有酸素能力が循環器疾患死亡と有意に関係していると報告している。本研究では表5に示すように，循環器疾患死亡におけるハザード比はQ_1に対して各分位とも低い値を示したがいずれも有意ではなかった。これは，本研究では死因を特定する方法が死亡小票の確認ではなく聞き取り調査であったことから，疾患の終末期の状態としての心不全が循環器疾患死亡に加わってしまったことによる交絡が原因かも知れない。あるいは，日本人が欧米人と比較して循環器疾患死亡が少ない事にともなう統計学的検出力（Statistical Power）の低さが，本研究において有酸素能力と循環器疾患死亡とに有意な関係がみいだせなかった原因かも知れない。

3)　有酸素能力と全がん死亡

有酸素能力と全がん死亡との関係を縦断的に調査した研究は少なく，これまでに3つの報告が存在する[3～5]。そのうち2つは全がん[3,4]を，残りの1つは前立腺がん[5]を対象に調査している。Arraizら[3]は，カナダホームフィットネステストというステップテスト（最大下自転車運動負荷テストとの相関：r＝0.72）を受けたカナダ人2,267人を7年間追跡し，有酸素能力と全がん死亡の関係を調査した。彼らの考える望ましい有酸素能力を持つ群を基準とした最低限の有酸素能力を持つ群および望ましくない有酸素能力を持つ群の年齢，性別，喫煙状況，飲酒量を調整した多変量調整オッズ比（95%CI）は，それぞれ1.6（0.4–5.4），1.9（0.8–4.5）であったと報告している。Arraizらは量反応関係が認められながらも各オッズ比が有意でなかった点について，データ数が少なかったことによる検出力の低さが原因であると考察している。彼らの観察した約16,000人年という総観察人年は，本研究の139,836人年と比較しても明らかに少なく，検出力の低さが不明瞭な結果をもたらしたと考えられる。一方，Kampertら[4]は有酸素能力と全がん死亡に有意な関係があったと報告している。彼らは，米国人男性25,341人を対象にトレッドミルによる最大運動負荷テストを行い，有酸素能力を5分位に分け，全がん死亡との関係を調査している。そして，Q_1に対する各分位の年齢，測定年，喫煙習慣の有無，慢性疾患の有無，異常心電図の有無を調整した多変量調整ハザード比（95%CI）は，それぞれ，0.54（0.35–0.84），0.56（0.36–0.87），0.59（0.38–0.90），0.36（0.21–0.61）であったと報告し，彼らはこれらの知見について強く興味を持ち，有酸素能力とがん死亡との関係について更なる研究を展開すると述べている。また，Oliveriaら[5]は，有酸素能力と前立腺がん罹患との間に有意な関係があったと報告している。彼らは，米国人男性12,975人を対象にトレッドミルによる最大運動負荷テストを実施し対象者を4分位に分類した。前立腺がんへの罹患をエンドポイントとして，Q_1に対する各分位の年齢，BMI，喫煙状況を調整した多変量調整ハザード比（95%CI）は，それぞれ，1.10（0.63–1.77），0.73（0.41–1.29），0.26（0.10–0.63）であったと報告している。そして，運動がもたらす低いtestosterone値が前立腺がんの予防に寄与しているのではないかと考察している。

本研究においても，全がん死亡における多変量調整ハザード比はQ_1に対して各分位とも有意に低い値を示し，量反応関係も認められた（表5）。これらの知見は，高い有酸素能力あるいは持久的な身体活動が，全がん死亡もしくは部位特異的ながんによる死亡の予防に寄与していることを示唆している。しかしながら，有酸素能力とがん死亡あるいはがん罹患に関してはその機序を含め不明な点が多く，有酸素能力と部位特異的ながんそれぞれについて更なる研究が必要であると思われる。

4.　おわりに

以上，日本人男性における有酸素能力と生命予

後の関係について検討した。運動負荷テストから得られた有酸素能力と生命予後の関係を調査したこれまでの研究は，すべて欧米人を対象としており，日本人を対象とした研究はみあたらない。欧米人と日本人では，遺伝的要素の違いとともに，生活環境あるいは生活習慣に違いがみられる。したがって，有酸素能力と生命予後に関して日本人を対象として調査を行ったことは意義あることと考えられる。

本研究の結果は，日本人においても低い有酸素能力は，全死因死亡および全がん死亡のリスクファクターであることを示唆している。

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PROSPECTIVE STUDY ON THE RELATIONSHIP BETWEEN PHYSICAL FITNESS AND ALL-CAUSE MORTALITY IN JAPANESE MEN

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Key words: Mortality, Physical fitness, Physical activity, Maximal oxygen uptake, Proportional hazard, Prospective study

This study was conducted to examine the relationship between physical fitness and all-cause mortality in Japanese men. We evaluated the physical fitness and risk for all-cause mortality of 9,986 Japanese men who were given a submaximal exercise test and a medical examination between 1982 and 1984. Physical fitness was measured using a bicycle ergometer test, and maximal oxygen uptake was estimated. The average follow-up time was 14 years, for total of 139,836 person-years of observation. There were 247 deaths during the observation period. The relative risk and 95% confidence intervals (95% CI) for all-cause mortality were obtained using the Cox proportional hazards model. Following age adjustment, and using the lowest physical fitness (quintile I) group as a reference, the hazard ratios for quintiles II through V were, 0.54 (0.39–0.77), 0.66 (0.47–0.94), 0.58 (0.39–0.86), and 0.46 (0.27–0.78), respectively. After being adjusted for age, body mass index, hypertension, and urinary protein, the hazard ratios were, 0.52 (0.37–0.73), 0.60 (0.42–0.87), 0.50 (0.33–0.75), and 0.39 (0.22–0.67), respectively.

The results presented here support the hypothesis that a low level of physical fitness in an important risk factor for all-cause mortality in Japanese men.

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論文名	日本人男性における有酸素能力と生命予後に関する縦断的研究
著 者	澤田亨，武藤孝司
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対象の内訳

	ヒト	動物	地 域	国内	研究の種類	縦断研究
対象	一般健常者	空白		(　　)		コホート研究
性別	男性	(　　)		(　　)		(　　)
年齢	平均37歳	[redacted]		(　　)		前向き研究
対象数	5000～10000	空白		(　　)		(　　)

調査の方法	実測	(有酸素能力)				

アウトカム

予 防	心疾患予防	なし	ガン予防	なし	(総死亡)	(　　)
維持・改善	なし	なし	なし	なし	(　　)	(　　)

図 表

表5　最も低い有酸素能力群（Q_1）に対する各分位の多変量調整ハザード比（循環器疾患，全がん）

	循環器疾患死亡者数	多変量調整*ハザード比	95%CI	p値	全がん死亡者数	多変量調整*ハザード比	95%CI	p値
Q_1	28	1.0	―	―	46	1.0	―	―
Q_2	14	0.52	0.27–1.00	0.051	27	0.60	0.37–0.98	0.039
Q_3	14	0.68	0.35–1.33	0.259	19	0.51	0.29–0.89	0.018
Q_4	9	0.53	0.24–1.16	0.111	14	0.45	0.24–0.85	0.013
Q_5	7	0.74	0.30–1.80	0.509	6	0.31	0.13–0.75	0.010
		p=0.25**				p<0.01**		

*：共変量：年齢，BMI，高血圧の有無，尿蛋白陽性の有無
**：トレンド検定

図表掲載箇所　P116　表5

抄録和訳

目的：低い有酸素能力が生活習慣病のリスクファクターであることが多くの研究により確認されており，生活習慣病の予防に有酸素運動が寄与することが知られている．しかしながら，有酸素能力と生命予後に関する縦断的研究は少なく，なかでも日本人を対象とした研究はみあたらない．本研究は，日本人における低い有酸素能力が全死因死亡のリスクファクターとなるか調査することを目的に，縦断的研究を実施した．
方法：T社において1982年から1984年の間に最大下運動負荷テストおよび定期健康診断を受診した男性9,986人（平均36.7歳：19-59歳）を対象とした．これらの対象者を平均14年間追跡し，死亡情報を把握した（総観察人年：139,836人年）．観察期間中の死亡者数は247人であった．対象者を観察開始時の推定最大酸素摂取量で5分位に分類し，Cox比例ハザードモデルを用いて最も低い有酸素能力群（Q1）を基準として各群の年齢調整ハザード比と年齢，BMI，高血圧の有無，および尿蛋白陽性の有無を調整した多変量調整ハザード比を求めた．
結果：Q1に対する各群の年齢調整ハザード比（95%CI）は0.54（0.39-0.77），0.66（0.47-0.94），0.58（0.39-0.86），0.46（0.27-0.78）であり，いずれも有意に低いハザード比であった．また，多変量調整ハザード比は0.52（0.37-0. 73），0.60（0.42-0.87），0.50（0.33-0.75），0.39（0.22-0.67）であり，年齢以外の交絡因子を調整した後も有意に低いハザード比を示した．
　以上の結果は，日本人においても低い有酸素能力は全死因死亡のリスクファクターであることを示唆している．

概 要（800字まで）

　1989年に米国・テキサス州ダラスのエアロビクスセンターに設置されたクーパー研究所から，有酸素能力と総死亡に関するコホート研究が発表された．米国人を対象にした研究で，有酸素能力と死亡には「負の量反応関係（有酸素能力が高い群ほど，死亡率が低くなる）」が示された．本研究は，それから10年後の1999年に発表されたコホート研究の結果である．本研究は，日本人においても米国人と同様に「有酸素能力と死亡には負の関係がある」ことを報告している．
　本研究は，通称「東京ガス・スタディ」という名称で実施されている東京ガスの社員を対象にした運動疫学研究である．本研究は東京ガスに勤務する男性労働者9,986人を対象に実施されている．本研究はコホート研究（追跡研究）である．追跡開始前に実施する調査（ベースライン調査）として，法定の定期健康診断が利用され，追跡対象者が各種の疾病（循環器疾患，糖尿病，結核等）に罹患していないことが確認されている．さらに，ベースライン調査として最大下運動負荷テストが実施され，有酸素能力（最大酸素摂取量）が測定されている．追跡対象者は有酸素能力によって5群（五分位）に分けられた後，14年間追跡され，各群における追跡期間中の死亡数（総死亡，循環器疾患による死亡，がん死亡）が確認されている．追跡開始時点における各群の年齢，BMI，高血圧罹患率，尿蛋白陽性率の違いを「比例ハザードモデル（多変量解析手法）」を用いて調整し，「最も低い有酸素能力群」を基準にして他の群の多変量調整ハザード比（相対危険度）を求めている．そして，有酸素能力と死亡の関係を統計的に検定する「トレンド検定」を実施している．その結果，有酸素能力と「総死亡」や「がん死亡」については統計的に有意な「負の量反応関係（有酸素能力が高い群ほど，総死亡やがん死亡の相対危険度が低い）」があったと報告している．

結 論（200字まで）

日本人男性において，低い有酸素能力は総死亡（全死因死亡）の危険因子であった．
身体活動の実施による有酸素能力の維持・向上は，早世を予防する可能性がある．

エキスパートによるコメント（200字まで）

日本人を対象に，有酸素能力と死亡の関係を調査した初めてのコホート研究である．
1989年に発表された米国人を対象に実施されたコホート研究の結果と同様に，低い有酸素能力は総死亡やがん死亡の危険因子であることが示されている．
本研究の結果は，有酸素運動の奨励に関する重要なエビデンスであると考えられる．

担当者　澤田亨

5. 座位時間およびテレビ観賞時間の参照値算出に用いた文献

Television Viewing Time and Mortality

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)

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Background—Television viewing time, the predominant leisure-time sedentary behavior, is associated with biomarkers of cardiometabolic risk, but its relationship with mortality has not been studied. We examined the associations of prolonged television viewing time with all-cause, cardiovascular disease (CVD), cancer, and non-CVD/noncancer mortality in Australian adults.

Methods and Results—Television viewing time in relation to subsequent all-cause, CVD, and cancer mortality (median follow-up, 6.6 years) was examined among 8800 adults ≥25 years of age in the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). During 58 087 person-years of follow-up, there were 284 deaths (87 CVD deaths, 125 cancer deaths). After adjustment for age, sex, waist circumference, and exercise, the hazard ratios for each 1-hour increment in television viewing time per day were 1.11 (95% confidence interval [CI], 1.03 to 1.20) for all-cause mortality, 1.18 (95% CI, 1.03 to 1.35) for CVD mortality, and 1.09 (95% CI, 0.96 to 1.23) for cancer mortality. Compared with a television viewing time of <2 h/d, the fully adjusted hazard ratios for all-cause mortality were 1.13 (95% CI, 0.87 to 1.36) for ≥2 to <4 h/d and 1.46 (95% CI, 1.04 to 2.05) for ≥4 h/d. For CVD mortality, corresponding hazard ratios were 1.19 (95% CI, 0.72 to 1.99) and 1.80 (95% CI, 1.00 to 3.25). The associations with both cancer mortality and non-CVD/noncancer mortality were not significant.

Conclusions—Television viewing time was associated with increased risk of all-cause and CVD mortality. In addition to the promotion of exercise, chronic disease prevention strategies could focus on reducing sitting time, particularly prolonged television viewing. ***(Circulation.* 2010;121:384-391.)**

Key Words: epidemiology ■ exercise ■ lifestyle ■ mortality ■ obesity ■ risk factors

Moderate- to vigorous-intensity exercise has been shown to be consistently associated with reduced risk of premature mortality.[1] However, less is known about the relationships of sedentary behavior (ie, too much sitting, as distinct from too little exercise) with mortality risk. A recent study of Canadian adults found a progressively greater risk of all-cause and cardiovascular, but not cancer, mortality across increasing levels of reported overall sitting time.[2] Another study in Japanese men and women showed all-cause mortality to be elevated in men who reported sitting for ≥8 h/d relative to those reporting sitting for <3 h/d; less prolonged sitting durations did not predict mortality risk.[3] High volumes of sitting time (≥16 h/d) have also been shown to be positively associated with cardiovascular events (both fatal and nonfatal) in postmenopausal women.[4]

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However, in these studies, sitting time has broadly encompassed the sum of time spent in several sedentary behaviors in different domains (work, leisure, and transportation). The particular relationship of television viewing time, the predominant leisure-time sedentary behavior in many developed countries,[5–8] with mortality risk has not been examined. Several studies have reported television viewing time to be detrimentally associated with weight gain, type 2 diabetes mellitus, some cancers, abnormal glucose metabolism, the metabolic syndrome, and other cardiovascular risk factors[9–24]; detrimental associations with television viewing have been observed even in those adults who met exercise guidelines.[18] Dose-response relationships have been reported, with moderate associations for at least 2 h/d[9,15,16] and stronger associations for ≥4 h/d.[9,14] Thus, it is plausible that prolonged television viewing time may be associated with risk of premature mortality. We examined the relationships of prolonged television viewing time with total, cardiovascular disease (CVD), cancer, and non-CVD/noncancer mortality in a national population-based cohort of men and women from the Australian Diabetes, Obesity and Lifestyle Study (AusDiab).

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Methods

Study Design and Population

The baseline AusDiab was conducted during 1999 to 2000.[15,25] Briefly, all eligible adults were recruited within 42 randomly selected urban and nonurban areas based on Census Collector Districts, 6 in each of the Australian states and in the Northern Territory of Australia. In total, 28 033 households were approached in the selected clusters. In the 19 215 households where contact was made, 2086 households were considered ineligible. Of the 17 129 eligible households, 5178 households refused to participate in the household survey, and the occupants of an additional 472 households were away from the residence during the survey period; thus, the number of eligible adults living in these 5650 households could not be ascertained. Of the 11 249 households that participated in the household interview, 20 347 adults (≥25 years of age) completed the household interview, and 11 247 (55.3%) had a biomedical examination after an overnight fast (minimum, 9 hours), giving an estimated overall response rate of 37%. Measurement procedures have been described previously.[15,25] We excluded those who reported that they had a previous history of CVD (coronary heart disease or stroke; n=634). We further excluded those who were pregnant at baseline (n=60), did not fast for ≥9 hours (n=25), had missing data on television viewing time (n=30), had missing data for exercise time (n=73), overreported or underreported total energy intake (n=322), had missing data for the variables under consideration (n=1296), or could not be matched to the Australian National Death Index (NDI; n=7); 8800 remained in the analysis (3846 men, 4954 women). Comparisons of those included with those excluded showed no marked differences in age (50 versus 55 years) or sex (44% versus 49% men). The Ethics Committee of the International Diabetes Institute approved the study, and permission to link the AusDiab cohort to the NDI was provided by the Australian Institute of Health and Welfare Ethics Committee. Written informed consent was obtained from all participants.

Television Viewing Time

Total time spent watching television or videos in the previous 7 days was reported.[15] This did not include time when the television was switched on but other activities (such as preparing a meal or doing other household chores) were being undertaken concurrently. This measure has been shown to provide a reliable (intraclass correlation=0.82; 95% CI 0.75 to 0.87) and valid (criterion validity=0.3) estimate of television viewing time among adults.[26] Three categories of television viewing time (<2, ≥2 to <4, and ≥4 h/d) were created based on previously identified associations with biomarkers of cardiometabolic risk.[14,20,23]

Other Measures

Demographic attributes, parental history of diabetes mellitus, smoking, highest level of educational attainment, previous history of CVD (self-reported angina, myocardial infarction, or stroke), and lipid medication use were assessed with interviewer-administered questionnaires. Exercise time was measured by the Active Australia questionnaire, which asks respondents about their participation in predominantly leisure-time exercise.[27] This measure has been shown to provide a reliable (intraclass correlation=0.59; 0.52 to 0.65) and valid (criterion validity=0.3) estimate of exercise among adults.[28,29] Dietary intake (usual eating habits over the past 12 months), total energy intake, and energy intake from alcohol were assessed with a self-administered validated food frequency questionnaire.[30] Data were considered valid and included in the analysis if total energy intake was between 500 and 3500 kcal/d for women and 800 and 4000 kcal/d for men.[31] Diet quality was assessed with the Diet Quality Index–Revised dietary assessment tool modified for Australian dietary recommendations.[32,33] Diet quality was reported on a scale of 1 to 100, with 100 being high diet quality.

Oral glucose tolerance tests were performed following World Health Organization specifications.[34] Fasting and 2-hour plasma glucose levels, fasting serum triglycerides, total cholesterol, and high-density-lipoprotein cholesterol (HDL-C) levels were obtained by enzymatic methods and measured on an Olympus AU600 analyzer (Olympus Optical, Tokyo, Japan). All specimens were analyzed at a central laboratory. Categories of abnormal glucose metabolism were determined according to the 1999 World Health Organization criteria.[35] Waist circumference and triplicate resting blood pressures were measured by trained personnel as reported previously.[25] Hypertension was defined as treatment with blood pressure–lowering medication or blood pressure ≥140/90 mm Hg.

Ascertainment of Mortality

Follow-up for mortality was to the date of death or November 16, 2006, whichever occurred first. Mortality status and underlying and contributory causes of death (*International Classification of Diseases*, 10th revision) were determined by linking the AusDiab cohort to the NDI using methods previously described.[36] The accuracy of the NDI has been established.[37] Those who were not matched to the NDI were assumed to be alive. Deaths were attributed to CVD if the underlying cause of death was coded I10-I25, I46.1, I48, I50-I99, or R96 and cancer if coded C00 to D48. In cases when uncomplicated diabetes mellitus (E109, E119, or E149) or unspecified hyperlipidemia (E785) was the underlying cause of death (n=6) and the contributory causes of death were coded as I10-I25, I48, or I50-I99 in the first position on the death certificate, CVD was considered the cause of death.

Statistical Analyses

Analyses were conducted with SPSS version 14.0 (SPSS, Chicago, Ill) and Stata Statistical Software version 10.0 (Stata Corp, College Station, Tex). A bivariate correlation (Spearman *r*) assessed the relationship of leisure-time exercise with television viewing time. For baseline characteristics, age- and sex-adjusted linear and logistic regression models were used to test differences in continuous and dichotomous variables, respectively, according to television viewing time category (<2, ≥2 to <4, and ≥4 h/d). Cox proportional-hazards models were used to estimate the hazard ratios (HRs) and 95% confidence intervals (CIs) of all-cause, CVD, cancer, and non-CVD/noncancer mortality according to television viewing time, considered a continuous variable (average hours per day) and as a categorical variable. The assumptions required for proportional hazards were met. They were assessed with graphs of log-log plots of the relative hazards by time and scaled Schoenfeld residuals. For models considering television viewing time as a continuous measure, outliers were identified through the use of plots of the deviance residuals by television viewing time (average hours per day), and the corresponding individuals were excluded (n=2). To test whether a linear relationship existed between television viewing time and the mortality outcomes, we used plots of martingale residuals by television viewing time and likelihood ratio tests of a model containing the linear television viewing time term nested within a model also containing the quadratic term for television viewing time that was adjusted for age and sex. Furthermore, unadjusted mortality rates (95% CI) per 1000 person-years according to increments of television viewing time (0, 1, 2, 3, 4, 5, and ≥6 h/d) were plotted, along with a regression line representing the linear relationship between these increments of television viewing and all-cause, CVD, and non-CVD mortality rates.

Models of the continuous television viewing time measure were initially adjusted for age, sex, leisure-time exercise, and waist circumference, a widely used indirect measure of central adiposity that has previously been shown to be an independent predictor of mortality risk.[38] Thereafter, further adjustment for smoking, education, total energy intake, alcohol intake, Diet Quality Index, hypertension, total cholesterol, HDL-C, serum triglycerides, lipid-lowering medication use, previously reported CVD, and glucose tolerance status was made. Additionally, models of categories of television viewing time were initially adjusted for age and sex, with subsequent models adjusted for all covariates listed above, with and without leisure-time exercise.

We also evaluated whether the effect of television viewing (<2, ≥2 to <4, and ≥4 h/d) on all-cause, CVD, cancer, or non-CVD/noncancer mortality was modified by age (<65 or ≥65 years), sex,

Table 1. Baseline Characteristics According to Average Hours per Day Spent Watching Television: AusDiab

	Television Viewing Time, h/d			
	<2 (n=4970)	≥2 to <4 (n=3158)	≥4 (n=672)	*P* for Linear Trend*
Men, n (%)	2049 (41)	1478 (47)	319 (47)	<0.001
Age, y	48.5 (12.7)	52.2 (14.4)	56.9 (15.3)	<0.001
Education ≥12 y, n (%)	3248 (65)	1612 (51)	251 (37)	<0.001
Lifestyle variables				
Current or ex-smoker, n (%)	2023 (41)	1509 (48)	380 (57)	<0.001
Energy intake (total), kJ/d	8245 (2729)	8396 (2833)	8311 (2840)	<0.01
Energy intake (alcohol), kJ/d	443 (590)	463 (632)	365 (617)	0.09
Diet Quality Index, %	63.8 (13.2)	62.5 (13.2)	60.3 (14.1)	<0.001
Television viewing time, h/d	0.93 (0.5)	2.6 (0.5)	5.0 (1.4)	<0.001
Exercise time, h/d	0.67 (0.8)	0.65 (0.8)	0.54 (0.71)	<0.01
Medical history/conditions, n (%)				
Hypertension†	1233 (25)	1096 (35)	292 (43)	<0.01
Lipid medication use	234 (5)	277 (9)	86 (13)	<0.001
Diagnosed diabetes mellitus‡	109 (2)	128 (4)	47 (7)	<0.001
Diagnosed diabetes mellitus >10 y‡	28 (1)	26 (1)	17 (3)	0.02
Cardiometabolic variables				
Body mass index, kg/m^2	26.4 (4.8)	27.2 (4.9)	28.3 (5.7)	<0.001
Waist circumference, cm	88.6 (13.6)	92.0 (13.4)	95.8 (14.6)	<0.001
Systolic blood pressure, mm Hg	126.4 (17.5)	130.8 (18.7)	133.8 (19.7)	<0.001
Diastolic blood pressure, mm Hg	69.5 (11.6)	70.4 (11.7)	71.1 (12.0)	0.94
Total cholesterol, mmol/L	5.6 (1.0)	5.8 (1.1)	5.9 (1.1)	<0.001
HDL-C, mmol/L	1.5 (0.4)	1.4 (0.4)	1.4 (0.4)	<0.001
Triglycerides, mmol/L§‖	1.2 (0.8–1.7)	1.3 (0.9–2.0)	1.5 (1.1–2.3)	<0.001
Fasting plasma glucose, mmol/L§	5.5 (1.0)	5.6 (1.2)	5.8 (1.7)	<0.001
2-h Plasma glucose, mmol/L§‖	5.6 (4.8–6.8)	6.0 (5.0–7.2)	6.5 (5.3–8.0)	<0.001

Data are mean (SD) when appropriate.

*Adjusted for age and sex.

†Hypertension defined as blood pressure ≥140/90 mm Hg or taking antihypertensive medication.

‡Diagnosed diabetes mellitus based on self-reported hypoglycemic medication use, a fasting plasma glucose ≥7.0 mmol/L, or a 2-hour plasma glucose level of ≥11.1 mmol/L.

§Data are median (25th to 75th percentiles).

‖*P* for trend based on logarithmic transformation of predicted variables in a regression model.

education (<12 or ≥12 years), smoking (current/ex-smoker or nonsmoker), hypertension (blood pressure <140/90 mm Hg or ≥140/90 mm Hg and taking antihypertensive medication), waist circumference (women: <80, 80 to <88, ≥88 cm; men: <94, 94 to <102, ≥102 cm), body mass index (<25, 25 to 29.9 or ≥30 kg/m^2), glucose tolerance status categories (normal glucose tolerance compared with impaired fasting glucose, impaired glucose tolerance, or diabetes mellitus), or leisure-time exercise (0, >0 to 2.49, ≥2.5 h/wk) by using log-likelihood ratio tests of models containing the variables as single terms nested within models also including the first-order interactions. To account for multiple testing, a stringent significance level of $P<0.01$ was used to test the addition of the interaction terms to the models.

Results

Participant characteristics by the 3 television viewing time categories (<2, ≥2 to <4, and ≥4 h/d) are shown in Table 1. Those who spent more time watching television had a more adverse health profile and were less likely to have completed at least 12 years of education. There was a weak but statistically significant correlation between leisure-time exercise and television viewing time (Spearman $r=-0.03$, $P<0.01$).

Over a median follow-up of 6.6 years, 284 deaths occurred. Of these, 87 (31%) were due to CVD, 125 (44%) were due to cancer, and 72 (25%) were non-CVD/noncancer deaths. For all-cause and CVD mortality, there was evidence of a steady progressive rise in the unadjusted mortality rates with each additional hour of television viewing, particularly for television viewing between 0 and 4 h/d. There was a weak relationship between television viewing time and cancer and noncancer/non-CVD mortality (the Figure). Adding a quadratic term for television viewing time to a model with television viewing time, age, and sex did not significantly improve the prediction of all-cause mortality ($P=0.64$), CVD mortality ($P=0.78$), cancer mortality ($P=0.67$), or non-CVD/noncancer mortality ($P=0.32$), thus indicating that the relationship between television viewing time and mortality outcomes was linear. Television viewing time remained significantly associated with both all-cause (HR per 1 h/d,

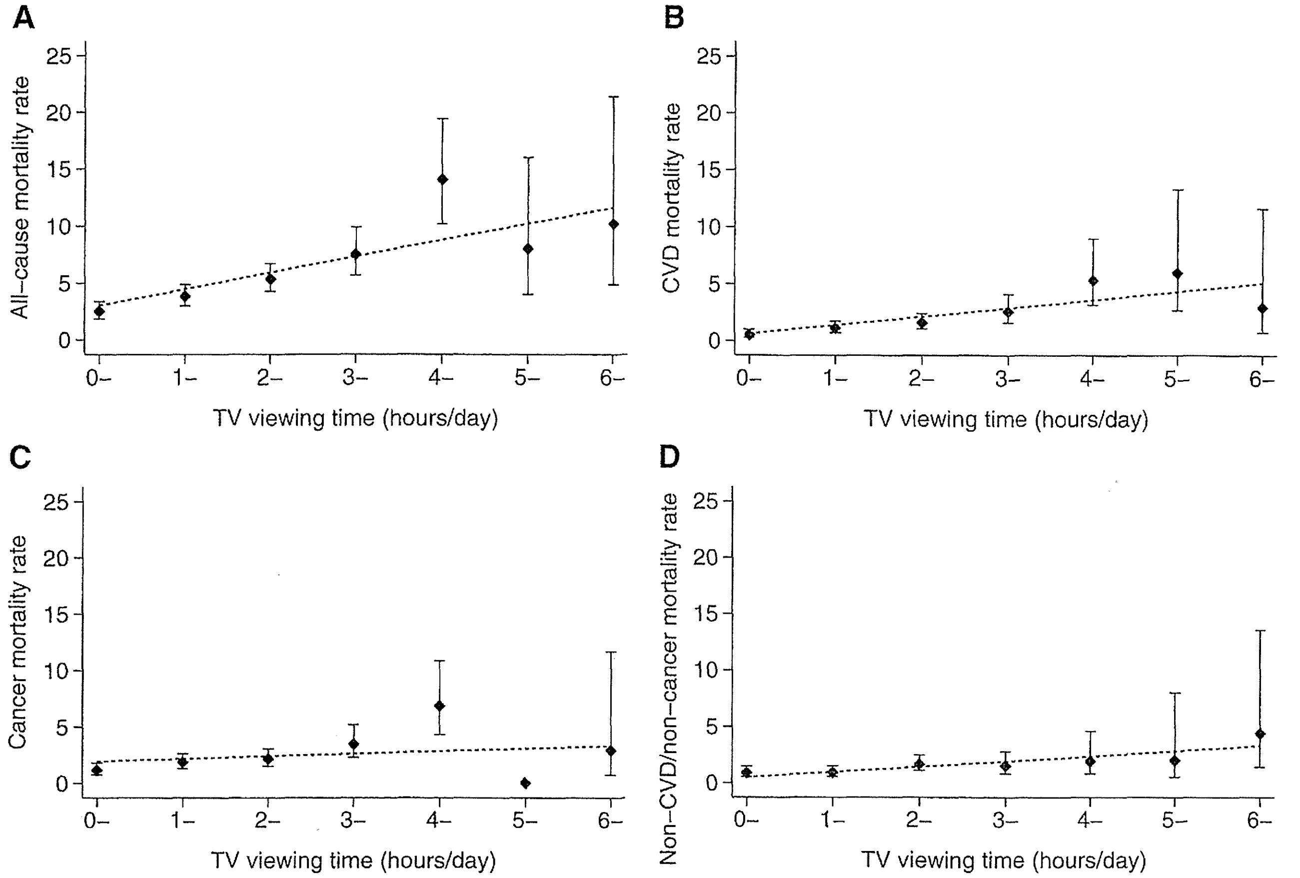

Figure. Unadjusted all-cause (A), CVD (B), cancer (C), and non-CVD/noncancer (D) mortality rates per 1000 person-years according to television (TV) viewing time (h/d). Dashed line presents the linear relationship between increments of television viewing time and all-cause, CVD, cancer, and non-CVD/noncancer mortality rates. Number of people in each television viewing category was as follows: 0 h/d, 2442; ≥1 h/d, 2528; ≥2 h/d, 2138; ≥3 h/d, 1020; ≥4 h/d, 407; ≥5 h/d, 155; and ≥6 h/d, 108.

1.11; 95% CI, 1.03 to 1.20) and CVD mortality (HR per 1 h/d, 1.18; 95% CI, 1.03 to 1.35) after adjustment for age, sex, leisure-time exercise, and waist circumference but not with cancer mortality (HR, 1.09; 95% CI, 0.96 to 1.23) or non-CVD/noncancer mortality (HR, 1.08; 95% CI, 0.92 to 1.27). Further adjustment for all other covariates attenuated the relationship between television viewing time and all-cause mortality (HR, 1.08; 95% CI, 1.00 to 1.17), CVD mortality (HR, 1.14; 95% CI, 0.99 to 1.30), cancer mortality (HR, 1.06; 95% CI, 0.93 to 1.20), and non-CVD/noncancer mortality (HR, 1.04; 95% CI, 0.89 to 1.22); however, the association with all-cause mortality remained, albeit at a borderline level of significance (P=0.048).

Compared with 0 to <2.0 h/d of television viewing, the age- and sex-adjusted HRs for ≥2 to <4 h/d and for ≥4 h/d were 1.20 and 1.67 for all-cause mortality, 1.24 and 2.12 for CVD mortality, and 1.18 and 1.68 for cancer mortality (Table 2). Except for cancer mortality, these associations remained significant for all-cause mortality (P=0.03) and showed borderline significance for CVD mortality (P=0.05) for the highest television viewing time category (≥4 h/d) after adjustments for other covariates, including exercise and waist circumference. Similar results were found when this model was reanalyzed with body mass index instead of waist circumference (data not shown).

To minimize potential bias caused by the influence of subclinical disease on the television viewing level, we further examined the associations after excluding individuals who died within the first, second, third, and fourth years of follow-up. In general, the strength and direction of the association of television viewing time with all-cause and CVD mortality were comparable to the original associations shown in Table 2. In age- and sex-adjusted models, the HRs for all-cause and CVD mortality for the highest television viewing category (≥4 h/d) after exclusion of the 26 people who died during the first year of follow-up were 1.84 (95% CI, 1.30 to 2.61) and 2.09 (95% CI, 1.13 to 3.80), respectively; 1.80 (95% CI, 1.24 to 2.64) and 1.98 (95% CI, 1.00 to 3.95) after exclusion of the 69 who died before the end of the second year of follow-up; 1.49 (95% CI, 0.97 to 2.28) and 1.94 (95% CI, 0.94 to 4.02) after exclusion of the 103 who died before the end of the third year of follow-up; and 1.69 (95% CI, 1.07 to 2.68) and 2.53 (95% CI, 1.13 to 5.67) after exclusion of the 134 people who died before the end of the fourth year of follow-up. Interaction tests showed that age, sex, education, smoking, hypertension, waist circumference, body mass index, glucose tolerance status, and leisure-time exercise did not significantly (P>0.01 for all factors) modify the associations between television viewing and all-cause, CVD, cancer, or non-CVD/noncancer mortality.

Table 2. Risk of All-Cause, Cardiovascular, Cancer, and Non-Cardiovascular/Noncancer Mortality According to Categories of Television Viewing Time: AusDiab

	Television Viewing Time, h/d		
	<2	≥2 to <4	≥4
Person-y	33 024	20 737	4326
Mortality from any cause			
Deaths, n	105	125	54
Age- and sex-adjusted HR (95% CI)	1 (Reference)	1.20 (0.92–1.56)	1.67 (1.20–2.33)
HR (95% CI) adjusted for age, sex, education, smoking, alcohol, and diet quality	1 (Reference)	1.17 (0.90–1.52)	1.49 (1.06–2.08)
Multivariate-adjusted HR (95% CI)*	1 (Reference)	1.14 (0.87–1.48)	1.49 (1.06–2.09)
Multivariate- and exercise time–adjusted HR (95% CI)*	1 (Reference)	1.13 (0.87–1.36)	1.46 (1.04–2.05)
Mortality from cardiovascular disease			
Deaths, n	26	39	22
Age- and sex-adjusted HR (95% CI)	1 (Reference)	1.24 (0.75–2.05)	2.12 (1.20–3.77)
HR (95% CI) adjusted for age, sex, education, smoking, alcohol, and diet quality	1 (Reference)	1.22 (0.74–2.03)	1.78 (1.01–3.22)
Multivariate-adjusted HR (95% CI)*	1 (Reference)	1.20 (0.72–2.00)	1.85 (1.03–3.33)
Multivariate- and exercise time–adjusted HR (95% CI)*	1 (Reference)	1.19 (0.72 –1.99)	1.80 (1.00–3.25)
Mortality from cancer causes			
Deaths, n	50	53	22
Age- and sex-adjusted HR (95% CI)	1 (Reference)	1.18 (0.80–1.76)	1.68 (1.00–2.80)
HR (95% CI) adjusted for age, sex, education, smoking, alcohol, and diet quality	1 (Reference)	1.15 (0.77–1.69)	1.53 (0.91–2.56)
Multivariate-adjusted HR (95% CI)*	1 (Reference)	1.12 (0.75–1.66)	1.50 (0.89–2.52)
Multivariate- and exercise time–adjusted HR (95% CI)*	1 (Reference)	1.12 (0.75–1.66)	1.48 (0.88–2.49)
Mortality from noncardiovascular and noncancer causes			
Deaths, n	29	33	10
Age- and sex-adjusted HR (95% CI)	1 (Reference)	1.18 (0.71–1.96)	1.18 (0.57–2.45)
HR (95% CI) adjusted for age, sex, education, smoking, alcohol, and diet quality	1 (Reference)	1.15 (0.70–1.91)	1.04 (0.50–2.17)
Multivariate-adjusted HR (95% CI)*	1 (Reference)	1.13 (0.68–1.88)	1.03 (0.49–2.16)
Multivariate- and exercise time–adjusted HR (95% CI)*	1 (Reference)	1.12 (0.67–1.87)	1.03 (0.49–2.15)

*Multivariate models are adjusted for age, sex, smoking (current or ex-smoker), education (≥12 years), total energy intake, alcohol intake, Diet Quality Index, waist circumference, hypertension (blood pressure ≥140/90 mm Hg or antihypertensive medication use), total plasma cholesterol (mmol/L), HDL-C (mmol/L), serum triglycerides (mmol/L, log), lipid-lowering medication use, and glucose tolerance status (impaired fasting glucose, impaired glucose tolerance, undiagnosed diabetes mellitus, known diabetes mellitus according to 1999 World Health Organization criteria[34]).

Discussion

These novel findings from a large population-based cohort of Australian men and women indicate that prolonged television viewing time is associated with an increased risk of all-cause and CVD mortality. Each 1-hour increment in television viewing time was found to be associated with an 11% and an 18% increased risk of all-cause and CVD mortality, respectively. Furthermore, relative to those watching less television (<2 h/d), there was a 46% increased risk of all-cause and an 80% increased risk of CVD mortality in those watching ≥4 hours of television per day, which were independent of traditional risk factors such as smoking, blood pressure, cholesterol, and diet, as well as leisure-time exercise and waist circumference.

Insufficient moderate- to vigorous-intensity exercise has long been recognized as a predictor of chronic disease and premature death.[1] However, until recently, the relationship between too much sitting and mortality had not been investigated. The HR observed for all-cause mortality with high television viewing (>4 h/d) in our study (1.46) is similar in magnitude to that reported for the highest category of sitting ("almost all the time") in a Canadian population (HR, 1.54).[2] Furthermore, the HR observed for high television viewing time in our study for CVD mortality (1.80) is comparable to that reported in Canadian adults (HR, 1.42) and concurs with findings from a cohort study of postmenopausal women in the United States in which a high level of sitting (≥16 h/d) was a predictor of fatal and nonfatal CVD.[4] The nonsignificant association with high television viewing time and cancer mortality in multivariate-adjusted models observed in our cohort is consistent with the findings observed for sitting time in Canadian adults.[2]

Television viewing time is one of several common behaviors that involve prolonged sitting.[26] Recent time-use surveys from Australia, the Unites States, and the United Kingdom indicate that, aside from sleeping, watching television is the behavior that occupies the most time in the domestic setting.[5,7,8] Our findings indicate that, regardless of leisure-time exercise levels and adiposity status, there is a progressive rise in mortality risk for each 1-hour increment in television viewing. From a public health perspective, the increased risk of all-cause and CVD mortality associated with watching television ≥4 h/d observed in this Australian cohort may have important implications in Australia and elsewhere, because recent estimates indicate that the average television viewing time is ≈3 hours in both Australia and the United Kingdom and is up to 8 hours in the United States.[39] Furthermore, a recent large population-based study of Scottish adults reported that the average television viewing and other screen-based entertainment time was 3.6 h/d in men and 3.2 h/d in women, with a strong social gradient; on average, those in the lowest socioeconomic position spent an additional 1.8 h/d on screen-based entertainment compared with those in the highest socioeconomic position.[40]

For exercise, the physiological mechanisms underlying the risk of premature mortality are suspected to involve biological, structural, and systemic effects on glucose homeostasis and other metabolic pathways of CVD risk.[41] Less is known about the mechanisms that might underlie the cardiometabolic correlates of sedentary behavior that we[12,14–16,23,42] and others[9,13,17,19–22,24] have identified. Observational studies with objective measures of sedentary time have reported significant associations of total sedentary time with blood glucose, blood lipids, and adiposity that are independent of moderate to vigorous exercise.[42,43] Animal studies have found enforced sedentary time to be related to lipoprotein lipase activity.[44,45] Our findings broadly support these hypothesized physiological links; we found that television viewing time was a significant predictor of CVD rather than non-CVD mortality.

Increased caloric intake and reduced energy expenditure are the most commonly proposed mechanisms for explaining the relationship between television viewing time and health outcomes. Increased snacking has been associated with high levels of television viewing time and increased adiposity.[46] Although in this cohort those with high volumes of television viewing time had poorer dietary profiles, the association between television viewing time and mortality was independent of diet quality and energy intake. Television viewing time could displace exercise time and thus contribute to reductions in overall daily energy expenditure. However, we[15,18] and others[20] have previously shown that television viewing time and moderate- to vigorous-intensity leisure-time exercise are only weakly correlated. It is possible, however, that television viewing time significantly displaces light-intensity physical activity, which has been shown to be beneficially associated with cardiometabolic risk markers, including 2-hour postchallenge blood glucose.[43]

Strengths of our study include the recruitment of a national sample of participants, the large size and wide age range of the cohort, and the objective measurement of key CVD risk factors. Limitations include the assessment of a single sedentary behavior (television viewing time), although this has been shown to be a reasonable proxy measure of an overall sedentary behavior pattern.[47] The television viewing measure was based on self-report; this may have led to some misclassification and regression dilution bias. However, any imprecision in the measurement is likely to have resulted in an underestimation of the strength of associations. Additionally, having only a baseline assessment of television viewing time and exercise time precluded the assessment of any changes in these behaviors during the follow-up period that could have influenced the relationships with mortality. Although we adjusted for several potential confounding variables, it is possible that other unmeasured or unknown confounding factors may have accounted for the associations that we have reported. Reverse causality, whereby diagnosed or undiagnosed illness at study induction may have been responsible for elevated television viewing time, cannot be ruled out. However, we excluded individuals who reported a previous history of CVD and adjusted for baseline health status in our models. Moreover, the findings were comparable after the exclusion of deaths occurring within the first, second, third, and fourth years of follow-up.

Conclusions

These findings indicate that television viewing time is associated with an increased risk of all-cause and CVD mortality. Although continued emphasis on current public health guidelines on the importance of moderate- to vigorous-intensity exercise should remain, our findings suggest that reducing time spent watching television (and possibly other prolonged sedentary behaviors) may also be of benefit in preventing CVD and premature death.

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Disclosures

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