The Table summarizes demographic details, comparison, and the 95% levels of agreement of steps walked and distance covered. Because there was no statistically significant difference (P=.89) between the mean age in both the age groups, no separate analysis was performed for both age groups. Multivariate analysis found statistically significant (P<.05) differences for the mean number of steps walked and the mean distance covered after controlling for height and weight. Both the Fitbit and Runtastic demonstrated statistically significant differences for the mean difference of steps walked and mean difference of distance covered. Mean percentage errors were lesser steps walked and distance covered with the Fitbit (−15.5% and −14.1%) than with Runtastic (−42.6% and −46.5%). Univariate analysis revealed a statistically significant difference between the 3 methods for both steps walked and distance covered, which remained significant on post hoc analysis.

A recent study found high accuracy with Fitbit monitors for steps walked on a treadmill.[7] However, a previous model of the Fitbit, Fitbit One, was found to overestimate the number of steps walked for moderate-vigorous PA when compared with accelerometry.[9] However, because none of these was compared against direct visualization, it could be why most studies have reported overestimation rather than underestimation of steps walked and distance covered. The Runtastic has been reported to be an accurate approach to tracking PA.[10] However, our findings suggest that Runtastic grossly underestimates the steps walked and distance covered, similar to previous studies.[3,4] Therefore, better accuracy in measuring both steps taken, and distance covered for both the Fitbit and Runtastic, is required if they are to be used in PA research.

To conclude, considerable discrepancies exist between methods of evaluation for both steps walked and distance covered. However, the Fitbit appears to have lesser deviation and percentage error from the direct measurement when compared with the Runtastic. Thus, the use of the Fitbit Charge 2 may be considered as a more valid device for promoting PA. If Runtastic is being used, it should be kept in mind that it may underestimate by approximately 45%.

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Association Between Pancreatic Cancer and Dipeptidyl Peptidase-4 Inhibitors Use in a Case-Control Study

Dipeptidyl peptidase-4 inhibitor is an incretin-based agent used to treat type 2 diabetes mellitus, which has been available in the Taiwan market since 2009.[1] Pancreatic cancer was the eighth leading cause of cancer death in Taiwan in 2017.[2] At present, no definite evidence is available on the association between pancreatic cancer and dipeptidyl peptidase-4 inhibitors in Taiwan. To evaluate this issue, a population-based case-control study was conducted using the database of the Taiwan National Health Insurance Program with 23 million citizens living in the independent country of Taiwan.[3] We identified type 2 diabetic participants aged 20 to 84 years with newly diagnosed pancreatic cancer between January 1,

TABLE. Characteristics of Pancreatic Cancer Cases and Matched Controls[a]

Variable	Matched controls (N=1222), n (%)	Cases (N=1222), n (%)	*P* value[b]
Sex			.37
Male	659 (53.9)	681 (55.7)	
Female	563 (46.1)	541 (44.3)	
Age group (y)			.97
20-39	64 (5.2)	64 (5.2)	
40-64	391 (32.0)	385 (31.5)	
65-84	767 (62.8)	773 (63.3)	
Age (y), mean ± SD[c]	68.0±10.2	67.7±10.0	.44
Ever use of dipeptidyl peptidase-4 inhibitors	34 (2. 8)	58 (4.8)	.01
Ever use of other antidiabetic drugs	982 (80.4)	1107 (90.6)	<.001
Comorbidities before the index date			
Alcohol-related disease	98 (8.0)	109 (8.9)	.42
Biliary stone	110 (9.0)	144 (11.8)	.02
Cardiovascular disease	647 (53.0)	663 (54.3)	.52
Chronic kidney disease	145 (11.9)	145 (11.9)	>.99
Chronic liver disease	409 (33.5)	415 (34.0)	.80
Chronic obstructive pulmonary disease	292 (23.9)	297 (24.3)	.81
Hyperlipidemia	696 (57.0)	708 (57.9)	.62
Hypertension	903 (73.9)	915 (74.9)	.58
Pancreatitis (acute and chronic)	14 (1.2)	15 (1.2)	.85

[a]Data are presented as the number of subjects in each group, with percentages given in parentheses.
[b]χ^2 test comparing pancreatic cancer cases and matched controls.
[c]*t* test comparing pancreatic cancer cases and matched controls.

2009, and December 31, 2013, as the cases (based on *International Classification of Diseases 9th Revision-Clinical Modification*, 157). Sex-matched and age-matched (5-year interval) participants with type 2 diabetes without pancreatic cancer were identified as matched controls. The date of cases being diagnosed with pancreatic cancer was defined as the index date. To reduce the latency bias, participants whose first-time prescriptions for dipeptidyl peptidase-4 inhibitors were prescribed within 12 months before the index date were excluded from the study. Thus, only those participants whose first-time prescriptions for dipeptidyl peptidase-4 inhibitors were prescribed more than 12 months before the index date were included. In addition, participants with any other cancer before the index date were excluded.

Insurance reimbursement claims data used in this study were available for public access. Patient identification numbers were scrambled to ensure confidentiality. Patient informed consent was not required. This study was approved by the Research Ethics Committee of China Medical University and Hospital in Taiwan (CMUH-104-REC2-115).

We identified 1222 cases with newly diagnosed pancreatic cancer and 1222 matched controls without pancreatic cancer (Table). The cases and the matched controls had similar distributions of sex and age. Nearly 54% to 56% of the study participants were male. The mean ages were 67.7±10.0 years in cases and 68.0±10.2 years in matched controls, without statistical significance (*t* test; *P*=.44). The cases had a higher proportion of dipeptidyl peptidase-4 inhibitors use than the matched controls (4.8% vs 2.8%; χ^2 test; *P* =.01). Variables that were statistically significant in a univariable logistic regression model were further included in a multivariate logistic regression model. Thus, other antidiabetic drug use and biliary stones were included for adjustment. The multivariable-adjusted logistic regression model disclosed that the adjusted odds ratio of pancreatic cancer was 1.53 (95% CI, 0.99-2.35; *P* =.06) for participants with previous use of dipeptidyl peptidase-4 inhibitors, compared with those who never used them.

The estimated period from the initiating mutation of the pancreatic cell to the detection of pancreatic cancer is long.[4] In our study, participants whose first-time prescriptions for dipeptidyl peptidase-4 inhibitors were prescribed within 12 months before the index date were excluded from the study. Although not completely, the latency bias was partially decreased in our study. Our study was a case-control study. The immortal time bias frequently found in a cohort study could be minimized. On the basis of these stringent study criteria in our study, we noted that use of dipeptidyl peptidase-4 inhibitors was not statistically associated with the risk of pancreatic cancer. This finding was partially compatible with 2 cohort studies reporting no statistical association between pancreatic cancer and use of dipeptidyl peptidase-4 inhibitors.[5,6] These other 2 cohort studies reported that an increased risk of pancreatic cancer associated with incretin-based therapy may be confounded by occult pancreatic cancer.[7] That is, people with undiagnosed pancreatic cancer might initially present with diabetes mellitus, and incretin-based therapy may be initiated. Consequently, pancreatic cancer may be detected later.[7] Incretin-based therapy is not a causal effect.

A recent comment reported that in view of the complex relationship between diabetes mellitus and pancreatic cancer, currently no sufficient evidence supports a causal relationship between pancreatic cancer and use of dipeptidyl peptidase-4 inhibitors in diabetic

patients.[8] Given that the latent period of pancreatic cancer is long, and the follow-up time of use of dipeptidyl peptidase-4 inhibitors was not long, such as in our study only covering 4 years, a long-term study is needed to observe any significant difference in pancreatic cancer risk associated with use of dipeptidyl peptidase-4 inhibitors. In addition, our study only had 92 (34+58) participants using dipeptidyl peptidase-4 inhibitors. The sample size of a future study should be large enough to detect a sufficient number of cancer cases to provide meaningful results. We conclude that no significant association can be detected between pancreatic cancer and use of dipeptidyl peptidase-4 inhibitors.

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CORRECTION

In the September 2018 issue of *Mayo Clinic Proceedings* in a Commentary entitled "**Hypothyroidism in Clinical Practice**" by Drake (Mayo Clin Proc 2018; 93:1169-1172), an error needs correction.

Page 1171, left-hand column

The third sentence of the first full paragraph should have read as follows: Thyroxine undergoes natural deiodination in the peripheral tissues to the active form of thyroid hormone, T3, with circulating concentrations of free T3 approximately 3 to 4-fold lower than those of circulating free T4 levels.

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