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In vitro screening of α-glucosidase, DPP-IV, and pancreatic lipase inhibitory activity of extracts from 181 coastal island plants of Korea

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Abstract

Obesity, hyperglycemia, and dyslipidemia are key components of metabolic syndrome (MetS), which increases the risk of cardiovascular disease and type 2 diabetes. Although lifestyle changes are essential, they are often insufficient, leading to increased use of drug therapies. However, concerns over side effects and poor compliance have driven interest in natural products with high biocompatibility and low toxicity. Coastal plants, especially those from island environments, are promising sources of bioactive compounds due to their adaptation to environmental stresses such as salinity, UV exposure, and nutrient scarcity. Among the key enzymatic targets for managing MetS, α-glucosidase is involved in carbohydrate digestion and glucose absorption, dipeptidyl peptidase-4 (DPP-IV) degrades the incretin hormones glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), which stimulate insulin secretion, and pancreatic lipase facilitates lipid digestion and absorption. Inhibition of these enzymes is considered an effective strategy for controlling blood glucose and body fat accumulation. This study evaluated the in vitro inhibitory activities of 181 plant extracts collected from coastal islands in Korea against α-glucosidase, DPP-IV, and pancreatic lipase. Several extracts demonstrated strong inhibitory effects, with some showing activity levels comparable to or higher than those of existing therapeutic agents. These findings may suggest that plant extracts collected from coastal island, which exhibited biological activities, may be strong inhibitors of α-glucosidase, DPP-IV, and pancreatic lipase and could be valuable for application in replacing synthetic drug.

Keywords: α-Glucosidase, Dipeptidyl peptidase-IV, Metabolic syndrome, Pancreatic lipase, Coastal plant

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Introduction

Metabolic syndrome (MetS) is a complex health condition characterized by the simultaneous occurrence of multiple metabolic disorders, including obesity, hypertension, hyperglycemia, and dyslipidemia, which are known as major causes of cardiovascular disease and type 2 diabetes (Heindel et al., 2017). The prevalence of MetS has been steadily increasing in recent years due to rapid industrialization and urbanization, which have led to reduced physical activity and the widespread adoption of high-calorie diets and Westernized eating habits (Asghari et al., 2015; Dziegielewska-Gesiak, 2021).

According to the 2021 Metabolic Syndrome Fact Sheet from the Korean Society of Cardiometabolic Syndrome (KSCMS), the prevalence of MetS among Korean adults increased from 22.2% in 2007 to 29.7% in 2018, reflecting a continuous upward trend since 2015, which has become a significant economic and social concern (Kim et al., 2022).

Although lifestyle modifications, such as increased physical activity, dietary adjustments, and weight loss, are essential for preventing and managing MetS, these approaches alone are often insufficient (Nam et al., 1999; Oh, 2015). Moreover, conventional pharmacological treatments for MetS are associated with high costs, side effects, and the potential for drug resistance (Casacchia et al., 2019).

As a result, there has been increasing interest in naturally derived substances as promising alternatives to conventional drugs, particularly in terms of efficacy and long-term sustainability (Graf et al., 2010). These substances are considered advantageous for therapeutic applications due to their high biocompatibility, reduced likelihood of adverse effects, and compatibility with prolonged use, which makes them appealing candidates for MetS treatment (Waltenberger et al., 2016).

Among these, coastal plants have drawn particular attention as valuable sources of such substances, owing to their adaptation to extreme environments with high salinity, ultraviolet radiation, and nutrient-poor conditions, as these stress factors are known to enhance the biosynthesis of diverse secondary metabolites with potent bioactivities (Saba Nazir et al., 2018; Sadeghi et al., 2024). These environmental stresses promote the production of diverse secondary metabolites with potent biological activities (Saba Nazir et al., 2018; Sadeghi et al., 2024). Therefore, coastal plants have significant potential as sources of natural therapeutics for the prevention and treatment of MetS (Pungin et al., 2023).

Among the various therapeutic targets for managing MetS, α-glucosidase inhibition, dipeptidyl peptidase-IV (DPP-IV) inhibition, and pancreatic lipase inhibition have received particular attention due to their roles in type 2 diabetes and obesity management (Hossain et al., 2020; Kumar & Chauhan, 2021; Lunagariya et al., 2014). α-Glucosidase, an enzyme present in the small intestine, facilitates carbohydrate digestion by breaking down disaccharides into monosaccharides, and its inhibition can slow carbohydrate digestion and reduce postprandial glucose spikes (Kumar et al., 2011). DPP-IV is an enzyme that degrades incretin hormones glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), which stimulate insulin secretion, and its inhibition can enhance insulin secretion and improve glycemic control (Barnett, 2006). Pancreatic lipase, a key enzyme in fat digestion, can be inhibited to reduce fat absorption, thereby preventing fat accumulation and promoting weight loss (Lunagariya et al., 2014).

Therefore, this study aimed to evaluate the *in vitro* inhibitory activities of 181 plant extracts collected from coastal islands in the Republic of Korea on α-glucosidase, DPP-IV, and pancreatic lipase. Through this approach, the study seeks to explore the potential of coastal plants as natural therapeutics for the prevention and treatment of MetS, providing a foundation for the development of high-efficacy, low-toxicity natural compounds that could overcome the limitations of conventional synthetic drugs (Table 1).

Materials and Methods

Chemicals and reagents

All plant extracts derived from coastal island plants were obtained from the Honam National Institute of Biological Resources (Mokpo, Korea). The reagents used in this study included Bovine Serum Albumin (BSA), sodium azide (NaN_3), α-glucosidase (from Saccharomyces cerevisiae), p-nitrophenyl-α-D-glucopyranoside (pNPG), acarbose, dipeptidyl peptidase-IV (DPP-IV; human recombinant), Gly-Pro-p-nitroanilide hydrochloride (Gly-Pro-pNA), Ile-Pro-Ile (Diprotin A), sodium acetate, lipase (from porcine pancreas), and p-nitrophenyl butyrate, all of which were purchased from Sigma-Aldrich (St. Louis, MO, USA). 10X MOPS buffer was obtained from Bioneer (Daejeon, Korea).

α-Glucosidase inhibitory activity

The α-glucosidase inhibitory activity was determined using a

Table 1. The scientific names, plant parts used, and collection sites to conduct this study

Sample no.	Scientific name	Part	Collection sites	Latitude	Longitude
1.	*Euscaphis japonica* (Thunb.) Kanitz	Stem	Geogeumdo	34.46664	127.10417
2.	*Solidago altissima* L.	Flower	Gohado	34.77303	126.36878
3.	*Solidago altissima* L.	Root	Gohado	34.77303	126.36878
4.	*Camellia japonica* L.	Leaf	Jindo	34.38439	126.21278
5.	*Sorbus alnifolia* (Siebold & Zucc.) K. Koch	Stem	Gohado	34.76917	126.36056
6.	*Pourthiaea villosa* (Thunb.) *Decne.*	Leaf	Jindo	34.39512	126.23931
7.	*Carpinus turczaninowii* Hance	Stem	Jindo	34.39549	126.23506
8.	*Amorpha fruticosa* L.	Leaf	Jindo	34.53465	126.32204
9.	*Pteridium aquilinum* var. *latiusculum*	Plant	Jindo	34.39489	126.23408
10.	*Equisetum arvense* L.	Plant	Jindo	34.53465	126.32204
11.	*Cornus macrophylla* Wall.	Leaf	Ulleungdo	37.46842	130.84261
12.	*Maianthemum dilatatum* (A. W. Wood).	Plant	Ulleungdo	37.48997	130.82846
13.	*Artemisia montana* (Nakai) Pamp.	Plant	Ulleungdo	37.46268	130.87579
14.	*Fallopia sachalinensis* (F. Schmidt) Ronse	Plant	Ulleungdo	37.46268	130.87579
15.	*Torilis japonica* (Houtt.) DC.	Plant	Suncheon	34.84712	127.47195
16.	*Litsea japonica* (Thunb.) Juss.	Leaf	Chujado	33.96584	126.28859
17.	*Litsea japonica* (Thunb.) Juss.	Stem	Chujado	36.8309	126.70734
18.	*Sageretia theezans* (Osbeck) M. C. Johnst.	Leaf	Chujado	33.95678	126.29338
19.	*Sageretia theezans* (Osbeck) M. C. Johnst.	Stem	Chujado	33.95678	126.29338
20.	*Hypochaeris radicata*	Plant	Chujado	33.93921	126.32391
21.	*Suaeda japonica* Makino	Plant	Ganghwado	36.8313	126.36477
22.	*Farfugium japonicum* (L.) Kitam.	Plant	Ulleungdo	37.47694	130.87639
23.	*Hydrangea petiolaris* Siebold & Zucc.	Leaf	Ulleungdo	37.46833	130.84306
24.	*Peucedanum japonicum* Thunb.	Plant	Ulleungdo	37.48412	130.91713
25.	*Aster spathulifolius* Maxim.	Plant	Ulleungdo	37.54294	130.90907
26.	*Sorbus ulleungensis* Chin. S. Chang	Fruit	Ulleungdo	37.47694	130.87639
27.	*Sedum takesimense* Nakai	Plant	Ulleungdo	37.51235	130.80718
28.	*Allium ochotense* Prokh.	Plant	Ulleungdo	37.48426	130.87954
29.	*Impatiens nolitangere* L.	Plant	Ulleungdo	37.5032	130.81944
30.	*Lilium lancifolium* Thunb.	Plant	Ulleungdo	37.54313	130.90877
31.	*Dystaenia takesimana* (Nakai) Kitag.	Plant	Ulleungdo	37.47694	130.87639
32.	*Artemisia littoricola* Kitam.	Plant	Ulleungdo	37.54427	130.9082
33.	*Zingiber mioga* (Thunb.) Roscoe	Plant	Haenam	34.43551	126.61835
34.	*Viburnum japonicum* (Thunb.) C. K. Spreng.	Leaf	Gageodo	34.06302	125.12193
35.	*Amaranthus powellii* S. Watson	Plant	Pyeongchang	37.71376	128.57453
36.	*Eragrostis ferruginea* (Thunb.)	Plant	Geojedo	34.79595	128.73152
37.	*Barnardia japonica* (Thunb.)	Plant	Jeju	33.21111	126.26194
38.	*Sageretia theezans* (Osbeck) M. C. Johnst.	Leaf	Damyang	35.28611	127.00083
39.	*Quercus acuta* Thunb.	Leaf	Jindo	34.46944	126.32444
40.	*Elaeagnus macrophylla* Thunb.	Leaf	Goha	34.77852	126.3557
41.	*Farfugium japonicum* (L.) Kitam.	Plant	Jindo	34.47028	126.30778
42.	*Bidens pilosa* L.	Plant	Goha	34.76667	126.36839
43.	*Hedera rhombea* (Miq.) Bean	Leaf	Jeopdo	34.37639	126.28972
44.	*Elaeagnus glabra* Thunb.	Leaf	Jindo	34.39111	126.23639

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Table 1. Continued

Sample no.	Scientific name	Part	Collection sites	Latitude	Longitude
45.	*Vaccinium bracteatum* Thunb.	Leaf	Jeopdo	34.36997	126.2827
46.	*Vaccinium bracteatum* Thunb.	Stem	Jeopdo	34.36997	126.2827
47.	*Dystaenia takesimana* (Nakai) Kitag.	Stem	Ulleungdo	37.49452	130.82752
48.	*Acer okamotoanum* Nakai	Leaf	Ulleungdo	37.5155	130.86932
49.	*Sorbus ulleungensis* Chin. S. Chang	Leaf	Ulleungdo	37.51064	130.86272
50.	*Tsuga sieblodii* Carriere	Leaf (Stem)	Ulleungdo	37.5032	130.81943
51.	*Hepatica maxima* NAKAI	Leaf	Ulleungdo	37.5155	130.86932
52.	*Lonicera insularis* Nakai	Leaf (Branch)	Ulleungdo	37.48458	130.91314
53.	*Illicium anisatum* L.	Leaf	Jindo	34.55671	126.29863
54.	*Illicium anisatum* L.	Stem	Jindo	34.55671	126.29863
55.	*Trachelospermum asiaticum* (Siebold & Zucc.) Nakai	Leaf	Bigeumdo	34.75437	125.90396
56.	*Trachelospermum asiaticum* (Siebold & Zucc.) Nakai	Stem	Bigeumdo	34.75437	125.90396
57.	*Fatsia japonica* (Thunb.) Decne. & Planch.	Leaf	Bigeumdo	34.75427	125.9211
58.	*Erythronium japonicum* Decne.	Plant	Changwon	35.17487	128.37999
59.	*Stauntonia hexaphylla* Decne.	Stem	Joyakdo	34.37465	126.94347
60.	*Staphylea bumalda* DC.	Leaf	Haman	35.22789	128.4551
61.	*Acer tataricum* subsp. *ginnala* (Maxim.) Wesm.	Stem	Haman	35.22613	128.45341
62.	*Neolitsea sericea* (Blume) Koidz.	Leaf	Dangmyosan	34.47219	127.46478
63.	*Machilus thunbergii* Siebold & Zucc. ex Meisn.	Leaf	Dangmyosan	34.4723	127.46455
64.	*Zanthoxylum piperitum* DC.	Leaf	Geoje	34.78814	128.73779
65.	*Sedum kamtschaticum* Fisch. & C. A. Mey.	Plant	Geoje	34.78814	128.73779
66.	*Ligularia taquetii* (H. Lév. & Vaniot) Nakai	Plant	Geoje	34.78794	128.73796
67.	*Cirsium japonicum* var. maackii (Regel) Kitam.	Plant	Geoje	34.78793	128.738
68.	*Machilus thunbergii* Siebold & Zucc.	Leaf	Jindo	34.40152	126.22537
69.	*Neolitsea sericea* (Blume) Koidz.	Leaf	Jindo	34.40152	126.22537
70.	*Lindera erythrocarpa* Makino	Leaf	Jindo	34.40152	126.22537
71.	*Mallotus japonicus* (L. f.) Müll.	Leaf	Jindo	34.40152	126.22537
72.	*Euscaphis japonica* (Thunb.) Kanitz	Leaf	Jindo	34.40152	126.22537
73.	*Ligustrum obtusifolium* Siebold & Zucc.	Leaf	Jindo	34.40035	126.22111
74.	*Boehmeria tricuspis* (Hance) Makino	Plant	Jindo	34.40035	126.22111
75.	*Platycarya strobilacea* Siebold & Zucc.	Leaf	Jindo	34.40035	126.22111
76.	*Rubus corchorifolius* L. f.	Leaf	Jindo	34.40007	126.22129
77.	*Toxicodendron sylvestre* (Siebold & Zucc.) Kuntze	Leaf	Jindo	34.40035	126.22111
78.	*Neolitsea sericea* (Blume) Koidz.	Leaf	Geoje	34.78816	128.73783
79.	*Litsea japonica* (Thunb.) Juss.	Leaf	Geoje	34.78823	128.73781
80.	*Ficus erecta* Thunb.	Leaf	Geoje	34.78823	128.73781
81.	*Eurya japonica* Thunb.	Leaf	Wando	34.36263	126.70973
82.	*Euscaphis japonica* (Thunb.) Kanitz	Leaf	Wando	34.36258	126.7096
83.	*Zanthoxylum piperitum* DC.	Leaf	Wando	34.36248	126.70955
84.	*Zanthoxylum ailanthoides* Siebold & Zucc.	Leaf	Wando	34.3625	126.70952
85.	*Lindera erythrocarpa* Makino	Leaf	Wando	34.36181	126.7067
86.	*Mallotus japonicus* (L. f.) Müll.	Leaf	Wando	34.36179	126.70668
87.	*Daphniphyllum macropodum* Miq.	Leaf	Wando	34.36183	126.70662
88.	*Styrax japonicus* Siebold & Zucc.	Leaf	Wando	34.36199	126.70806

Table 1. Continued

Sample no.	Scientific name	Part	Collection sites	Latitude	Longitude
89.	*Celtis sinensis* Pers.	Leaf	Wando	34.36417	126.71195
90.	*Cornus kousa* F. Buerger ex Miq.	Leaf	Wando	34.36582	126.71452
91.	*Toxicodendron sylvestre* (Siebold & Zucc.) Kuntze	Leaf	Wando	34.36601	126.71506
92.	*Cornus kousa* F. Buerger ex Miq.	Leaf	Wando	35.82067	126.45847
93.	*Mallotus japonicus* (L. f.) Müll.	Leaf	Wando	35.82069	126.45843
94.	*Toxicodendron sylvestre* (Siebold & Zucc.) Kuntze	Leaf	Sinshido	35.82062	126.45834
95.	*Eurya japonica* Thunb.	Leaf	Sinshido	35.82058	126.4583
96.	*Styrax japonicus* Siebold & Zucc.	Leaf	Sinshido	35.82064	126.45848
97.	*Euscaphis japonica* (Thunb.) Kanitz	Leaf	Sinshido	35.82069	126.45862
98.	*Platycarya strobilacea* Siebold & Zucc.	Leaf	Sinshido	35.82056	126.46062
99.	*Celtis biondii* var. *heterophylla* (H. Lév.) C. K. Schneid.	Leaf	Sinshido	35.81894	126.45331
100.	*Rubus takesimensis* Nakai	Leaf	Ulleungdo	37.52433	130.87146
101.	*Hovenia dulcis* Thunb.	Leaf	Ulleungdo	37.52433	130.87146
102.	*Maianthemum dilatatum* (A. W. Wood)	Plant	Ulleungdo	37.5241	130.86518
103.	*Prunus takesimensis* Nakai	Leaf	Ulleungdo	37.52356	130.86425
104.	*Sorbus ulleungensis* Chin. S. Chang	Leaf	Ulleungdo	37.52279	130.86356
105.	*Ulmus laciniata* (Trautv.) Mayr	Leaf	Ulleungdo	37.52279	130.86362
106.	*Kalopanax septemlobus* (Thunb.) Koidz.	Leaf	Ulleungdo	37.524	130.86421
107.	*Neolitsea sericea* (Blume) Koidz.	Leaf	Ulleungdo	37.48109	130.81373
108.	*Sambucus racemosa* subsp. *pendula* (Nakai)	Leaf	Ulleungdo	37.48202	130.81428
109.	*Ligustrum foliosum* Nakai	Leaf	Ulleungdo	37.48345	130.81937
110.	*Urtica laetevirens* Maxim.	Plant	Ulleungdo	37.49034	130.82702
111.	*Tsuga sieboldii* Carrière	Leaf	Ulleungdo	37.48994	130.82667
112.	*Alnus maximowiczii* Callier ex C. K. Schneid.	Leaf	Ulleungdo	37.49672	130.8255
113.	*Glehnia littoralis* F. Schmidt ex Miq.	Plant	Ulleungdo	37.48472	130.91792
114.	*Daphniphyllum macropodum* Miq.	Leaf	Ulleungdo	37.47877	130.89355
115.	*Zanthoxylum simulans* Hance	Leaf	Jeju	33.30121	126.25519
116.	*Cinnamomum japonicum* Siebold	Leaf	Jeju	33.25765	126.3529
117.	*Actinodaphne lancifolia* (Siebold & Zucc.) Meisn.	Leaf	Jeju	33.29577	126.26771
118.	*Sophora flavescens* Aiton	Plant	Jeju	33.29456	126.26815
119.	*Ligustrum obtusifolium* Siebold & Zucc.	Leaf	Geoje	34.81052	128.71651
120.	*Albizia julibrissin* Durazz.	Leaf	Geoje	34.78826	128.7378
121.	*Rhus javanica* L.	Leaf	Geoje	34.81047	128.71656
122.	*Osmanthus heterophyllus* (G. Don) P. S. Green	Leaf	Tongyeong	34.79593	128.42831
123.	*Lindera erythrocarpa* Makino	Leaf	Tongyeong	34.79589	128.42835
124.	*Aralia elata* (Miq.) Seem.	Plant	Tongyeong	34.79617	128.42826
125.	*Viburnum odoratissimum* var. *awabuki*	Leaf	Tongyeong	34.81075	128.43383
126.	*Albizia julibrissin* Durazz.	Leaf	Jindo	34.40046	126.21431
127.	*Callicarpa mollis* Siebold & Zucc.	Leaf	Jindo	34.40031	126.2147
128.	*Clerodendrum trichotomum* Thunb.	Leaf	Jindo	34.39929	126.21827
129.	*Cinnamomum japonicum* Siebold	Leaf	Jindo	34.39868	126.21812
130.	*Flueggea suffruticosa* (Pall.) Baill.	Leaf	Jindo	34.39701	126.2197
131.	*Styrax japonicus* Siebold & Zucc.	Leaf	Jindo	34.39627	126.22131
132.	*Zanthoxylum schinifolium* Siebold & Zucc.	Leaf	Jindo	34.39718	126.22655

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Table 1. Continued

Sample no.	Scientific name	Part	Collection sites	Latitude	Longitude
133.	*Lysimachia clethroides* Duby	Plant	Jindo	34.39662	126.22628
134.	*Eupatorium lindleyanum* DC.	Plant	Jindo	34.39591	126.22646
135.	*Eurya japonica* Thunb.	Leaf	Nangdo	34.60939	127.54828
136.	*Toxicodendron sylvestre* (Siebold & Zucc.) Kuntze	Leaf	Nangdo	34.60931	127.54833
137.	*Callicarpa japonica* Thunb.	Leaf	Nangdo	34.60894	127.54867
138.	*Lespedeza maximowiczii* C. K. Schneid.	Leaf	Nangdo	34.60877	127.54884
139.	*Weigela subsessilis* (Nakai) L. H. Bailey	Leaf	Nangdo	34.60879	127.54889
140.	*Rhus javanica* L.	Leaf	Nangdo	34.60983	127.5481
141.	*Sageretia theezans* (Osbeck) M. C. Johnst.	Leaf	Nangdo	34.61081	127.5471
142.	*Pittosporum tobira* (Thunb.) W. T. Aiton	Leaf	Nangdo	34.61536	127.5535
143.	*Ligustrum japonicum* Thunb.	Leaf	Nangdo	34.61539	127.55378
144.	*Ficus erecta* Thunb.	Leaf	Nangdo	34.61544	127.55379
145.	*Cornus macrophylla* Wall.	Leaf	Nangdo	34.61323	127.54443
146.	*Glochidion chodoense* J. S. Lee & H. T. Im	Leaf	Jodo	34.33597	126.02976
147.	*Smilax china* L.	Leaf	Jodo	34.33621	126.02929
148.	*Toxicodendron sylvestre* (Siebold & Zucc.) Kuntze	Leaf	Jodo	34.33618	126.02934
149.	*Euscaphis japonica* (Thunb.) Kanitz	Leaf	Jodo	34.33604	126.02964
150.	*Rhaphiolepis indica* var. *umbellata* (Thunb.) Ohashi	Leaf	Jodo	34.33604	126.02954
151.	*Eurya japonica* Thunb.	Leaf	Jodo	34.33599	126.0296
152.	*Mallotus japonicus* (L. f.) Müll.	Leaf	Jodo	34.3355	126.03136
153.	*Albizia julibrissin* Durazz.	Leaf	Jodo	34.33576	126.03134
154.	*Hedera rhombea* (Miq.) Bean	Leaf	Jodo	34.33565	126.03184
155.	*Elaeagnus macrophylla* Thunb.	Leaf	Jodo	34.33583	126.03214
156.	*Albizia julibrissin* Durazz.	Leaf	Imjado	35.07095	126.08951
157.	*Platycarya strobilacea* Siebold & Zucc.	Leaf	Imjado	35.07096	126.0894
158.	*Chenopodium album* L.	Leaf	Imjado	35.07176	126.08852
159.	*Smilax china* L.	Leaf	Imjado	35.08762	126.04817
160.	*Clerodendrum trichotomum* Thunb.	Leaf	Imjado	35.08763	126.04815
161.	*Lespedeza maximowiczii* var. *tricolor* (Nakai) Nakai	Leaf	Imjado	35.0877	126.04822
162.	*Rhus javanica* L.	Leaf	Imjado	35.08837	126.04828
163.	*Cudrania tricuspidata* (Carrière) Bureau ex Lavallée	Leaf	Imjado	35.08948	126.04722
164.	*Eupatorium lindleyanum* DC.	Plant	Imjado	35.09265	126.05316
165.	*Zanthoxylum schinifolium* Siebold & Zucc.	Leaf	Paryeongsan	34.63625	127.32606
166.	*Angelica decursiva* (Miq.) Franch. & Sav.	Plant	Paryeongsan	34.63666	127.32737
167.	*Cudrania tricuspidata* (Carrière) Bureau ex Lavallée	Leaf	Paryeongsan	34.63656	127.32877
168.	*Clerodendrum trichotomum* Thunb.	Leaf	Paryeongsan	34.63655	127.32876
169.	*Patrinia scabiosifolia* Fisch. ex Trevir.	Plant	Paryeongsan	34.63245	127.32781
170.	*Flueggea suffruticosa* (Pall.) Baill.	Leaf	Paryeongsan	34.65266	127.35388
171.	*Lespedeza maximowiczii* var. *tricolor* (Nakai) Nakai	Leaf	Paryeongsan	34.65267	127.35386
172.	*Lespedeza bicolor* Turcz.	Leaf	Paryeongsan	34.64938	127.3567
173.	*Rubus coreanus* Miq.	Leaf	Paryeongsan	34.61854	127.46823
174.	*Ulmus davidiana* var. *japonica* (Rehder) Nakai	Leaf	Paryeongsan	34.63205	127.4106
175.	*Aphananthe aspera* (Thunb.) Planch.	Leaf	Paryeongsan	34.63213	127.41063
176.	*Zanthoxylum piperitum* DC.	Leaf	Paryeongsan	34.63216	127.41057

Table 1. Continued

Sample no.	Scientific name	Part	Collection sites	Latitude	Longitude
177.	*Pueraria lobata* (Willd.) Ohwi	Plant	Paryeongsan	34.63224	127.41048
178.	*Zanthoxylum ailanthoides* Siebold & Zucc.	Leaf	Haenam	34.46696	126.62278
179.	*Lindera erythrocarpa* Makino	Leaf	Haenam	34.46698	126.62275
180.	*Ilex macropoda* Miq.	Leaf	Haenam	34.46698	126.62279
181.	*Cornus controversa* Hemsl.	Leaf	Haenam	34.46274	126.6234

modified method from (Indrianingsih et al., 2015). 10 μL of each plant extract or positive control Acarbose was added to a 96-well plate to reach a final concentration of 100 μg/mL. Then, 50 μL of 0.7 U/mL α-glucosidase solution in phosphate buffered saline (PBS) was added, and the initial absorbance at 405 nm was measured. The plate was incubated at room temperature for 5 minutes, followed by the addition of 50 μL of 5 mM pNPG in PBS. After an additional 5 minutes of incubation, the absorbance at 405 nm was measured to determine enzyme activity.

Dipeptidyl peptidase-IV inhibitory activity

The DPP-IV inhibitory activity was determined using a modified method from (Parmar et al., 2012). 25 μL of each plant extract or positive control Ile-Pro-Ile (Diprotin A) was added to a 96-well plate to reach a final concentration of 100 μg/mL. Then, 25 μL of 0.8 mM Gly-Pro-p-NA in 0.1 M Tris-HCl buffer (pH 8.0) was added, and the plate was incubated at 37℃ for 10 minutes. After this, 50 μL of 5 mU/mL DPP-IV enzyme solution in the same tris(hydroxymethyl)aminomethane hydrochloride (Tris-HCl) buffer was added. The plate was incubated for 60 minutes at 37℃, and the reaction was stopped by adding 100 μL of 3% acetic acid. The absorbance at 405 nm was measured to determine enzyme activity.

Pancreatic lipase inhibitory activity

The pancreatic lipase inhibitory activity was determined using a modified method from (Wei et al., 2015). 20 μL of each plant extract or positive control (orlistat) was added to a 96-well plate to reach a final concentration of 100 μg/mL. Then, 169 μL of 0.1 M Tris-HCl buffer (pH 7.0) containing 5 mM $CaCl^2$ was added, followed by 6 μL of 2.5 mg/mL lipase from porcine pancreas solution prepared in 1X MOPS buffer. The plate was incubated at 37℃ for 15 minutes. After this, 5 μL of 10 mM p-nitrophenyl butyrate dissolved in dimethyl sulfoxide (DMSO) was added, and the plate was incubated for an additional 30 minutes at 37℃. The absorbance at 405 nm was then measured to determine enzyme activity.

Calculation of inhibition rate

To correct for potential color interference from plant extracts, a blank control was included for each assay. The blank was prepared under the same conditions as the test samples but without the enzyme. The inhibition rate was calculated using the following formula:

$$\text{Inhibition (\%)} = [(\text{Absorbance of control} - \text{Absorbance of sample}) / \text{Absorbance of control}] \times 100$$

Statistical analysis

All experiments were performed in triplicate and data are presented as mean ± SD. For each enzyme assay, differences between each extract (n = 181) and the control were assessed by one-way analysis of variance (ANOVA) followed by Dunnett's multiple comparisons test in GraphPad Prism. Adjusted *p*-values are reported, statistical significance is denoted as $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$, and $^{****}p < 0.0001$.

Results and Discussion

α-Glucosidase inhibitory activity

α-Glucosidase is an enzyme that breaks down disaccharides into monosaccharides in the small intestine, facilitating glucose absorption (Kumar et al., 2011). Inhibiting this enzyme can delay carbohydrate digestion and reduce postprandial blood glucose levels, making it a promising target for type 2 diabetes management (Hossain et al., 2020; Kumar et al., 2011). This inhibitory activity is known to be mediated by various bioactive compounds, including flavonoids (e.g., quercetin), polyphenols, tannins, and triterpenoids, which are commonly found in many medicinal and edible plants (Hossain et al., 2020; Martin & Montgomery, 1996).

Therefore, this study analyzed the α-glucosidase inhibitory activities of 181 extracts from coastal island plants in Korea. The 181 plant extracts were found to have different levels of α-glucosidase inhibitory activity, ranging from 0 ± 0.31% to 85 ± 0.56% (Table 2). Among the 181 plant extracts, *Acer okamotoanum* Nakai exhibited the highest α-glucosidase

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Table 2. α-Glucosidase inhibitory activity of 181 plant extracts

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
Acarbose	69 ± 1.63****	44.	84 ± 0.48[1), ****]
1.	18 ± 1.08****	45.	62 ± 1.20****
2.	18 ± 3.24****	46.	58 ± 0.90****
3.	9 ± 2.24*	47.	51 ± 0.69****
4.	3 ± 0.60	48.	85 ± 0.56[1), ****]
5.	15 ± 1.10****	49.	70 ± 1.26****
6.	23 ± 2.73****	50.	84 ± 1.31[1), ****]
7.	23 ± 1.38****	51.	60 ± 0.83****
8.	22 ± 2.61****	52.	49 ± 1.52****
9.	6 ± 0.68	53.	52 ± 2.48****
10.	6 ± 1.59	54.	69 ± 1.40****
11.	17 ± 1.55****	55.	55 ± 2.34****
12.	7 ± 1.86	56.	51 ± 2.14****
13.	6 ± 1.49	57.	50 ± 1.70****
14.	9 ± 3.89*	58.	55 ± 1.76****
15.	8 ± 0.95	59.	51 ± 0.83****
16.	2 ± 5.50	60.	75 ± 0.35[1), ****]
17.	7 ± 2.17	61.	83 ± 0.41[1), ****]
18.	17 ± 5.51****	62.	5 ± 2.38
19.	22 ± 0.34****	63.	10 ± 2.87***
20.	57 ± 5.15****	64.	8 ± 3.29*
21.	0 ± 6.20	65.	10 ± 1.22**
22.	7 ± 3.03	66.	5 ± 2.55
23.	19 ± 8.93****	67.	12 ± 1.80****
24.	29 ± 3.86****	68.	17 ± 1.85****
25.	12 ± 3.26**	69.	16 ± 0.52****
26.	6 ± 7.42	70.	19 ± 2.29****
27.	16 ± 1.57****	71.	20 ± 0.70****
28.	7 ± 6.52	72.	21 ± 1.23****
29.	12 ± 5.16**	73.	5 ± 3.54
30.	8 ± 5.48	74.	6 ± 3.36
31.	11 ± 0.25**	75.	12 ± 2.23****
32.	6 ± 3.49	76.	11 ± 0.71***
33.	8 ± 1.82	77.	13 ± 1.91****
34.	22 ± 6.12****	78.	11 ± 2.10***
35.	9 ± 1.07*	79.	16 ± 3.89****
36.	7 ± 2.50	80.	13 ± 2.19****
37.	23 ± 1.51****	81.	15 ± 0.39****
38.	31 ± 1.37****	82.	20 ± 2.42****
39.	18 ± 0.46****	83.	19 ± 2.31****
40.	16 ± 2.59****	84.	4 ± 0.96*
41.	43 ± 1.80****	85.	3 ± 1.72
42.	48 ± 1.02****	86.	5 ± 2.02**
43.	49 ± 1.53****	87.	2 ± 1.49

Table 2. Continued

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
88.	2 ± 1.27	132.	6 ± 1.43***
89.	7 ± 0.18****	133.	13 ± 1.73****
90.	7 ± 1.81****	134.	9 ± 0.97****
91.	10 ± 0.81****	135.	11 ± 1.46****
92.	10 ± 0.66****	136.	13 ± 2.30****
93.	5 ± 1.16**	137.	10 ± 0.42****
94.	10 ± 0.34****	138.	13 ± 0.96****
95.	0 ± 0.64	139.	14 ± 1.25****
96.	2 ± 3.07	140.	21 ± 0.34****
97.	0 ± 2.04	141.	1 ± 2.86
98.	0 ± 0.31	142.	2 ± 0.93
99.	9 ± 2.42****	143.	0 ± 1.81
100.	1 ± 1.53	144.	0 ± 1.60
101.	2 ± 1.02	145.	12 ± 1.67****
102.	2 ± 1.56	146.	11 ± 1.93****
103.	0 ± 1.05	147.	8 ± 1.53****
104.	2 ± 2.07	148.	13 ± .74****
105.	0 ± 1.86	149.	11 ± 2.21****
106.	1 ± 0.22	150.	9 ± 1.39****
107.	0 ± 1.36	151.	9 ± 1.73****
108.	0 ± 1.58	152.	12 ± 1.35****
109.	2 ± 1.26	153.	11 ± 0.20****
110.	0 ± 1.26	154.	0 ± 1.39
111.	3 ± 1.99	155.	5 ± 1.13**
112.	4 ± 0.50*	156.	21 ±1.98****
113.	1 ± 0.28	157.	5 ± 1.20**
114.	6 ± 1.37***	158.	4 ± 0.30*
115.	8 ± 1.12****	159.	10 ± 0.05****
116.	9 ± 1.27****	160.	3 ± 0.73
117.	16 ± 1.12****	161.	6 ± 0.77***
118.	0 ± 3.30	162.	15 ± 1.77****
119.	4 ± 1.64*	163.	4 ± 0.94**
120.	21 ± 0.49****	164.	2 ± 2.68
121.	9 ± 1.54****	165.	2 ± 0.64
122.	1 ± 1.21	166.	4 ± 2.46*
123.	2 ± 1.02	167.	3 ± 0.92
124.	3 ± 1.12	168.	3 ± 1.95
125.	4 ± 1.96**	169.	2 ± 1.72
126.	22 ± 0.85****	170.	2 ± 0.72
127.	3 ± 0.44	171.	1 ± 1.99
128.	4 ± 1.15*	172.	3 ± 0.38
129.	4 ± 1.29*	173.	6 ± 0.58****
130.	6 ± 1.13****	174.	3 ± 1.15
131.	5 ± 0.58**	175.	1 ± 1.65

Table 2. Continued

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
176.	5 ± 1.06**	179.	5 ± 0.22***
177.	3 ± 0.91*	180.	3 ± 1.43*
178.	3 ± 1.41*	181.	4 ± 1.37**

Each extract was tested at a final concentration of 100 μg/mL, and results are presented as mean ± SD from triplicate experiments.
Differences between each extract (n = 181) and the control were assessed by one-way analysis of variance (ANOVA) followed by Dunnett's multiple comparisons test.
[1)] Acarbose was used as the positive control, and the top five extracts with the highest activity.
Adjusted *p*-values are reported; statistical significance is denoted as * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$.

inhibitory activity (85 ± 0.56%). Furthermore, the next most potent α-glucosidase inhibitory capacities (> 75%) were found in the following extracts: *Tsuga sieboldii* Carriere (84 ± 1.31%), *Elaeagnus glabra* Thunb. (84 ± 0.48%), *Acer tataricum* subsp. *ginnala* (83 ± 0.41%), and *Staphylea bumalda* DC. (75 ± 0.35%). The inhibitory activity of the five extracts listed above was greater than that of the commercial α-glucosidase inhibitor acarbose (69%) (Martin & Montgomery, 1996).

These findings suggest that the identified extracts could be promising candidates for controlling postprandial hyperglycemia, supporting the development of new α-glucosidase inhibitors for the treatment of type 2 diabetes (Kumar et al., 2011).

Dipeptidyl peptidase-IV inhibitory activity

DPP-IV is an enzyme that degrades GLP-1 and GIP, which are incretin hormones that stimulate insulin secretion and help regulate blood glucose levels (Barnett, 2006). Inhibiting DPP-IV prolongs GLP-1 activity, enhances insulin release and improves glycemic control, making DPP-IV a valuable target for type 2 diabetes therapy (Kumar & Chauhan, 2021). The inhibitory activity of DPP-IV is influenced by various bioactive compounds, including polyphenols (e.g., catechins), flavonoids and alkaloids, which are found in abundance in medicinal plants (Ansari et al., 2022; Singh et al., 2021).

This study evaluated the DPP-IV inhibitory activities of various plant extracts as illustrated in Table 3. The 181 plant extracts were found to have different levels of DPP-IV inhibitory activity, ranging from 0 ± 0.41% to 56 ± 2.76% (Table 3). Of the 181 extracts tested, *Elaeagnus macrophylla* Thunb. exhibited the highest DPP-IV inhibition (56 ± 2.76%). The next most potent DPP-IV inhibitory activities were observed in *Camellia japonica* L. (55 ± 5.97%), *Farfugium japonicum* (L.) Kitam. (55 ± 1.59%), *Bidens pilosa* L. (55 ± 1.20%), and *Solidago altissima* L. (54 ± 0.92%). Although these extracts demonstrated notable

Table 3. Dipeptidyl peptidase-IV (DPP-IV) inhibitory activity of 181 plant extracts

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
Ile-Pro-Ile	86 ± 0.31****	43.	51 ± 0.53****
1.	51 ± 2.55****	44.	52 ± 0.61****
2.	54 ± 2.23****	45.	50 ± 1.46****
3.	54 ± 0.92[1)],****	46.	51 ± 0.96****
4.	55 ± 5.97[1)],****	47.	49 ± 1.07****
5.	50 ± 2.40****	48.	49 ± 1.07****
6.	48 ± 0.80****	49.	52 ± 1.86****
7.	51 ± 0.41****	50.	52 ± 1.00****
8.	52 ± 0.85****	51.	50 ± 1.61****
9.	52 ± 2.11****	52.	53 ± 1.84****
10.	49 ± 1.16****	53.	47 ± 1.96****
11.	53 ± 4.39****	54.	16 ± 1.86****
12.	53 ± 3.08****	55.	51 ± 0.46****
13.	50 ± 0.81****	56.	53 ± 1.51****
14.	50 ± 0.70****	57.	53 ± 1.92****
15.	52 ± 2.00****	58.	50 ± 0.70****
16.	50 ± 1.01****	59.	53 ± 1.33****
17.	47 ± 0.55****	60.	51 ± 0.96****
18.	49 ± 0.85****	61.	51 ± 1.61****
19.	52 ± 5.48****	62.	12 ± 1.08**
20.	52 ± 2.65****	63.	12 ± 1.08**
21.	48 ± 0.85****	64.	16 ± 1.42****
22.	55 ± 1.59[1)],****	65.	13 ± 1.42***
23.	49 ± 0.55****	66.	7 ± 0.82
24.	52 ± 2.39****	67.	8 ± 1.42
25.	48 ± 0.55****	68.	13 ± 1.48***
26.	50 ± 0.81****	69.	11 ± 2.49**
27.	53 ± 3.99****	70.	11 ± 2.69**
28.	52 ± 0.93****	71.	10 ± 2.56**
29.	50 ± 3.57****	72.	10 ± 4.16**
30.	50 ± 1.07****	73.	16 ± 2.13****
31.	49 ± 0.61****	74.	10 ± 2.05*
32.	51 ± 1.07****	75.	6 ± 1.23
33.	49 ± 2.13****	76.	7 ± 1.78
34.	52 ± 0.85****	77.	9 ± 1.42*
35.	52 ± 0.41****	78.	9 ± 1.48*
36.	52 ± 1.60****	79.	5 ± 3.64
37.	49 ± 0.41****	80.	6 ± 3.35
38.	47 ± 2.92****	81.	2 ± 2.28
39.	49 ± 3.57****	82.	11 ± 2.58*
40.	56 ± 2.76[1)],****	83.	12 ± 3.10*
41.	50 ± 0.93****	84.	18 ± 1.87****
42.	55 ± 1.20[1)],****	85.	15 ± 2.68***

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Table 3. Continued

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
86.	18 ± 1.14****	130.	3 ± 2.08
87.	16 ± 1.55***	131.	6 ± 1.91
88.	18 ± 0.74****	132.	9 ± 2.53
89.	3 ± 3.72	133.	12 ± 1.10**
90.	18 ± 2.39****	134.	5 ± 2.16
91.	12 ± 4.65**	135.	0 ± 2.53
92.	18 ± 4.49****	136.	0 ± 3.63
93.	11 ± 6.36*	137.	5 ± 3.70
94.	13 ± 5.21**	138.	0 ± 5.51
95.	11 ± 5.28*	139.	0 ± 5.41
96.	10 ± 6.70	140.	2 ± 5.07
97.	5 ± 3.72	141.	0 ± 5.51
98.	8 ± 4.23	142.	1 ± 4.73
99.	9 ± 4.23	143.	7 ± 2.73
100.	12 ± 4.49**	144.	5 ± 4.50
101.	13 ± 3.36**	145.	19 ± 4.02****
102.	8 ± 3.72	146.	4 ± 5.12
103.	4 ± 4.88	147.	1 ± 2.20
104.	3 ± 4.79	148.	0 ± 3.70
105.	2 ± 3.24	149.	0 ± 2.91
106.	12 ± 2.39*	150.	0 ± 4.41
107.	12 ± 2.39*	151.	4 ± 2.51
108.	17 ± 2.23***	152.	0 ± 1.24
109.	20 ± 1.97****	153.	0 ± 0.41
110.	21 ± 4.53****	154.	0 ± 2.30
111.	16 ± 2.23***	155.	0 ± 2.89
112.	1 ± 1.55	156.	0 ± 1.09
113.	7 ± 3.75	157.	0 ± 0.71
114.	6 ± 3.01	158.	0 ± 2.30
115.	4 ± 5.07	159.	5 ± 2.30
116.	6 ± 5.69	160.	0 ± 2.58
117.	14 ± 6.71**	161.	0 ± 4.59
118.	5 ± 6.09	162.	0 ± 3.11
119.	10 ± 5.37	163.	1 ± 3.27
120.	10 ± 6.70	164.	2 ± 3.30
121.	19 ± 6.02****	165.	1 ± 1.49
122.	19 ± 4.55****	166.	0 ± 5.71
123.	12 ± 4.10*	167.	0 ± 4.12
124.	13 ± 3.94*	168.	0 ± 3.52
125.	6 ± 5.28	169.	0 ± 2.18
126.	6 ± 3.82	170.	0 ± 4.59
127.	5 ± 4.10	171.	7 ± 9.72
128.	0 ± 2.08	172.	0 ± 0.82
129.	5 ± 2.50	173.	0 ± 3.67
174.	5 ± 5.67	178.	7 ± 3.17
175.	7 ± 1.06	179.	10 ± 2.80
176.	12 ± 5.25*	180.	8 ± 4.33
177.	12 ± 10.74*	181.	12 ± 3.67

Each extract was tested at a final concentration of 100 μg/mL, and results are presented as mean ± SD from triplicate experiments.
Differences between each extract (n = 181) and the control were assessed by one-way analysis of variance (ANOVA) followed by Dunnett's multiple comparisons test.
[1)] Ile-Pro-Ile was used as the positive control, and the top five extracts with the highest activity.
Adjusted *p*-values are reported; statistical significance is denoted as * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, and **** $p < 0.0001$.

inhibitory activity, their inhibition rates did not exceed that of the positive control (Ile-Pro-Ile, 86%) (Singh et al., 2021).

These findings suggest that the identified extracts could be used to modulate incretin activity, supporting the development of new DPP-IV inhibitors for treating type 2 diabetes (Lin et al., 2019).

Pancreatic lipase inhibitory activity

Pancreatic lipase is an enzyme that plays a crucial part in digesting and absorbing dietary fats (Subramaniyan & Hanim, 2025). Inhibiting this enzyme reduces fat absorption and promotes weight loss, making it a promising target for obesity management (Lunagariya et al., 2014). Various natural compounds have been found to exhibit this inhibitory activity, including catechins, polyphenols, saponins and triterpenoids, which are commonly found in numerous edible and medicinal plants (de La Garza et al., 2011).

Therefore, this study analyzed the pancreatic lipase inhibitory activities of 181 extracts from coastal island plants in Korea. The 181 plant extracts were found to have different levels of pancreatic lipase inhibitory activity, ranging from 12 ± 6.84% to 105 ± 1.63% (Table 4). Among the 181 plant extracts, *Ilex macropoda* Miq. exhibited the highest pancreatic lipase inhibitory activity (105 ± 1.63%). Furthermore, the next most potent pancreatic lipase inhibitory capacities (> 90%) were found in the following extracts: *Lilium lancifolium* Thunb. (93 ± 5.02%), *Mallotus japonicus* (L. f.) Müll. (93 ± 2.50%), *Viburnum japonicum* (Thunb.) C. K. Spreng. (91 ± 2.35%), and *Suaeda japonica* Makino (90 ± 1.93%). The inhibitory activity of the five extracts listed above was greater than that of the commercial pancreatic lipase inhibitor orlistat (87%) (Rajan et al., 2020).

These findings suggest that the identified extracts could be promising candidates for reducing fat absorption, supporting

Table 4. Pancreatic lipase inhibitory activity of 181 plant extracts

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
Orlistat	87 ± 0.58****	44.	72 ± 6.37****
1.	61 ± 8.19****	45.	66 ± 10.17****
2.	54 ± 0.60****	46.	81 ± 0.60****
3.	65 ± 8.51****	47.	76 ± 3.78****
4.	49 ± 4.26****	48.	68 ± 3.14****
5.	60 ± 10.77****	49.	85 ± 8.12****
6.	65 ± 12.14****	50.	76 ± 5.83****
7.	72 ± 4.66****	51.	75 ± 1.60****
8.	81 ± 5.40****	52.	78 ± 12.78****
9.	38 ± 7.55****	53.	46 ± 13.38****
10.	76 ± 5.89****	54.	85 ± 13.08****
11.	55 ± 14.40****	55.	69 ± 10.83****
12.	72 ± 6.68****	56.	77 ± 2.64****
13.	60 ± 5.57****	57.	76± 8.85****
14.	77 ± 9.87****	58.	78 ± 7.36****
15.	88 ± 2.50****	59.	42 ± 6.87****
16.	63 ± 12.99****	60.	76 ± 10.57****
17.	80 ± 11.17****	61.	65 ± 18.79****
18.	73 ± 5.54****	62.	86 ± 11.76****
19.	72 ± 4.16****	63.	53 ± 0.58****
20.	89 ± 12.65****	64.	57 ± 3.80****
21.	90 ± 1.93[1],****	65.	50 ± 6.45****
22.	87 ± 2.51****	66.	61 ± 4.18****
23.	38 ± 3.74**	67.	63 ± 2.03****
24.	83 ± 10.58****	68.	56 ± 4.19****
25.	43 ± 11.40***	69.	65 ± 1.46****
26.	59 ± 14.13****	70.	71 ± 0.33****
27.	81 ± 12.19****	71.	41 ± 3.54****
28.	68 ± 7.56****	72.	40 ± 6.46****
29.	66 ± 11.12****	73.	47 ± 1.46****
30.	93 ± 5.02[1],****	74.	57 ± 4.07****
31.	76 ± 7.00****	75.	52 ± 4.11****
32.	83 ± 7.53****	76.	56 ± 2.61****
33.	59 ± 18.60****	77.	67 ± 4.93****
34.	91 ± 2.35[1],****	78.	61 ± 3.49****
35.	81 ± 4.14****	79.	59 ± 2.09****
36.	78 ± 12.09****	80.	65 ± 3.22****
37.	15 ± 5.93	81.	57 ± 3.34****
38.	12 ± 6.84	82.	69 ± 2.01****
39.	39 ± 13.83**	83.	62 ± 6.14****
40.	68 ± 9.32****	84.	63 ± 1.52****
41.	78 ± 11.39****	85.	46 ± 5.17****
42.	89 ± 4.25****	86.	50 ± 2.94****
43.	69 ± 13.91****	87.	45 ± 1.52****

Table 4. Continued

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
88.	44 ± 5.11****	132.	62 ± 1.51****
89.	45 ± 3.44****	133.	40 ± 10.63****
90.	36 ± 3.74****	134.	59 ± 2.72****
91.	64 ± 1.46****	135.	29 ± 7.89****
92.	37 ± 3.03****	136.	49 ± 2.85****
93.	37 ± 1.46****	137.	48 ± 3.29****
94.	54 ± 8.38****	138.	56 ± 0.75****
95.	26 ± 5.10****	139.	42 ± 7.59****
96.	47 ± 8.75****	140.	62 ± 6.91****
97.	33 ± 5.47****	141.	31 ± 7.00****
98.	32 ± 2.56****	142.	42 ± 10.98****
99.	49 ± 2.91****	143.	40 ± 0.75****
100.	56 ± 2.22****	144.	46 ± 6.44****
101.	60 ± 9.10****	145.	49 ± 6.78****
102.	51 ± 10.54****	146.	81 ±1.15****
103.	47 ± 4.14****	147.	60 ± 7.70****
104.	42 ± 5.83****	148.	85 ± 4.58****
105.	58 ± 0.42****	149.	79 ± 3.56****
106.	52 ± 4.45****	150.	81 ± 5.85****
107.	60 ± 1.43****	151.	75 ± 2.30****
108.	54 ± 5.62****	152.	93 ± 9.14[1],****
109.	54 ± 2.41****	153.	64 ± 9.98****
110.	62 ± 1.59****	154.	56 ± 0.75****
111.	22 ± 8.60****	155.	76 ± 7.22****
112.	36 ± 6.60****	156.	63 ± 8.12****
113.	40 ± 4.41****	157.	55 ± 1.71****
114.	52 ± 3.52****	158.	67 ± 6.87****
115.	52 ± 0.40****	159.	55 ± 1.71****
116.	24 ± 1.82****	160.	51 ± 6.12****
117.	32 ± 6.94****	161.	49 ± 6.16****
118.	37 ± 7.40****	162.	68 ± 3.74****
119.	47 ± 3.78****	163.	56 ± 4.86****
120.	47 ± 2.86****	164.	45 ± 4.55****
121.	56 ± 4.36****	165.	49 ± 7.22****
122.	12 ± 0.40*	166.	57 ± 2.08****
123.	44 ± 2.48****	167.	45 ± 4.31****
124.	57 ± 9.64****	168.	57 ± 4.54****
125.	49 ± 3.24****	169.	63 ± 5.88****
126.	61 ± 2.60****	170.	67 ± 2.08****
127.	63 ± 3.63****	171.	71 ± 4.17****
128.	58 ± 2.10****	172.	64 ± 1.94****
129.	59 ± 3.10****	173.	78 ± 2.45****
130.	59 ± 0.75****	174.	66 ± 1.63****
131.	37 ± 5.89****	175.	59 ± 1.71****

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Table 4. Continued

Sample no.	Inhibitory activity (%)	Sample no.	Inhibitory activity (%)
176.	70 ± 4.91****	179.	67 ± 6.72****
177.	71 ± 2.32****	180.	105 ± 1.63[1],****
178.	75 ± 2.38****	181.	62 ± 4.74****

Each extract was tested at a final concentration of 100 μg/mL, and results are presented as mean ± SD from triplicate experiments.
Differences between each extract (n = 181) and the control were assessed by one-way analysis of variance (ANOVA) followed by Dunnett's multiple comparisons test.
[1] Orlistat was used as the positive control, and the top five extracts with the highest activity.
Adjusted *p*-values are reported; statistical significance is denoted as * $p < 0.05$, ** $p < 0.01$, and **** $p < 0.0001$.

the development of new anti-obesity agents (Hou et al., 2022).

MetS is a complex health condition in which multiple metabolic disorders, including obesity, hyperglycemia, and dyslipidemia, occur simultaneously, and it is recognized as a major cause of cardiovascular disease and type 2 diabetes (Heindel et al., 2017). The increasing prevalence of MetS, primarily driven by reduced physical activity, high-calorie diets, and the widespread adoption of Westernized eating habits, is considered a significant public health concern worldwide, leading to an increasing demand for effective therapeutic strategies (Asghari et al., 2015; Dziegielewska-Gesiak, 2021). Various mechanisms have been investigated for the treatment of MetS, and among them, enzyme inhibitors targeting α-glucosidase, DPP-IV, and pancreatic lipase have been demonstrated to be effective in the management of type 2 diabetes and obesity (Hossain et al., 2020; Kumar & Chauhan, 2021; Lunagariya et al., 2014).

Acarbose, a well-known α-glucosidase inhibitor, reduces glucose absorption in the intestinal lumen and enhances insulin sensitivity, thereby effectively controlling postprandial hyperglycemia and hyperinsulinemia (Altay, 2022). It is considered a safe and effective therapeutic agent for managing postprandial blood glucose levels, as it does not increase the risk of hypoglycemia or weight gain (Uuh Narvaez & Segura Campos, 2022). However, acarbose has been associated with gastrointestinal adverse effects, such as gas, bloating, and diarrhea (Hollander, 1992), and its efficacy has not been confirmed in cardiovascular outcome trials (Altay, 2022). Similarly, Ile-Pro-Ile, also known as diprotin A, is a well-known DPP-IV inhibitor that effectively prevents the degradation of GLP-1 *in vitro* (Holst et al., 1998). However, its *in vivo* efficacy remains limited, and several adverse effects, including nasopharyngitis, headache, nausea, hypersensitivity, dermatological reactions, and pancreatitis, have been reported (Juillerat-Jeanneret, 2014). Likewise, orlistat is a well-known pancreatic lipase inhibitor that reduces fat absorption by binding to the active site of the enzyme during the digestive process, thereby promoting the excretion of undigested dietary fat (Bülbül & Çokdinleyen, 2024). However, orlistat has been reported to induce gastrointestinal side effects, including bloating, loose stools, or diarrhea (Morales et al., 2016). Given these concerns, the long-term use of synthetic drugs targeting these enzymes may lead to adverse effects and reduced patient compliance (Asliddin & Gulnaz, 2025). Accordingly, there is a need for effective and sustainable alternative treatments that can overcome the limitations of current pharmacological approaches.

In this context, natural product-based therapeutics, known for their generally greater safety than synthetic drugs, are increasingly regarded as promising alternatives, owing to their structural diversity, favorable biocompatibility, and potential for multi-target activity (Meier & Lappas, 2016; Wu et al., 2025). Especially, coastal plants represent a promising resource due to their capacity to produce diverse bioactive metabolites in response to environmental stresses such as high salinity, ultraviolet radiation, and nutrient limitations (Saba Nazir et al., 2018; Sadeghi et al., 2024). To adapt to such environmental stresses, coastal plants are known to produce a wide array of secondary metabolites, including potent antioxidants such as phenolic compounds (Stanković et al., 2023). These bioactive constituents may act as valuable enzyme inhibitors, highlighting their therapeutic potential for potential in the management of MetS.

In this study, the inhibitory activities of 181 natural extracts derived from coastal island were evaluated against α-glucosidase, DPP-IV, and pancreatic lipase *in vitro*. Several extracts exhibited inhibitory activities that were comparable to or even exceeded those of commercially available positive controls. Notably, certain extracts, including *Hypochaeris radicata*, *Vaccinium bracteatum* Thunb., *Sorbus ulleungensis* Chin. S. Chang, *Lonicera insularis* Nakai, and *Trachelospermum asiaticum* (Siebold & Zucc.) Nakai, exhibited potent inhibitory activity across all three enzyme assays, highlighting their potential as multifunctional natural therapeutics for the simultaneous regulation of carbohydrate and lipid metabolism. Given that MetS is characterized by a multifactorial pathophysiology involving the dysregulation of multiple metabolic pathways, therapeutic strategies that simultaneously modulate multiple targets are increasingly recognized as being more efficacious than those focusing on a single enzyme or pathway (Lillich et al., 2021). Accordingly, the simultaneous inhibition of α-glucosidase, DPP-IV, and pancreatic lipase by these extracts suggests a broader modulatory potential on the metabolic disturbances underlying MetS,

thereby conferring therapeutic advantages over agents targeting a single enzymatic pathway. Moreover, these extracts present as high-value natural therapeutic candidates, offering potential advantages over conventional drugs, which often face challenges such as high costs, adverse side effects, and resistance.

In conclusion, this study provides robust scientific evidence supporting the potential of island-derived natural resources as sustainable and effective therapeutic agents for managing MetS. These findings provide a strong basis for the development of natural product-based alternatives to conventional pharmacological treatments. To facilitate clinical translation into pharmacotherapy, functional food development, and preventive strategies, future studies should aim to isolate the active constituents and validate their efficacy and safety through vivo and clinical trials.

Competing interests

No potential conflict of interest relevant to this article was reported.

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Availability of data and materials

Upon reasonable request, the datasets of this study can be available from the corresponding author.

Ethics approval and consent to participate

Not applicable.

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