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Antioxidant Ability of Various Flavonoids against DPPH Radicals and LDL Oxidation

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Summary Flavonoids, a group of polyphenolic compounds, exist naturally and serve as antioxidants in vegetables, fruits, and so on. The inhibition of low density lipoprotein (LDL) oxidation may be an effective way to prevent or delay the progression of atherosclerosis. In the present study, we analyzed the radical scavenging capacity of 10 flavonoids (catechin, epicatechin [EC], epigallocatechin [EGC], epicatechin gallate [ECg], epigallocatechin gallate [EGCg], myricetin, quercetiji, apigenin, kaempferol, and luteolin) toward 1,1-Biphenyl-2 picryl-hydrazyl [DPPH]. After 20min of incubation, EGCg was the most effective DPPH radi cal scavenger, luteolin being the least active of this flavonoid group. The mutual antioxidant effect of flavonoids with a-tocopherol (a-toc) on LDL oxidizability was investigated by using the lipophilic azo radical initiator 2 ,2'-azobis(4-methoxy-2,4-dimethylvaleronitrile) [AMVN-CH3O]. An inhibitory effect of flavonoids on LDL oxidation was observed in the order of luteolin>ECg>EC>quercetin>catechin>EGCg>EGC>myricetin>kaempferol> apigenin. The shortened lag time induced by higher doses of a-toc (6mg/100mL) was re stored by flavonoids. These results suggest that 1) radical trapping effects of flavonoids differ according to their structure, and 2) flavonoids act as hydrogen donors to a-toc radical; fur thermore, by interaction with a-toc, they have a greater potential to delay the oxidation of LDL.

Key Words free radical, LDL oxidation, antioxidant, tocopherol, flavonoid

Flavonoids have antioxidant properties (1) and have been reported to prevent the development of free-radi cal-induced diseases, including forms of cancer (2), coronary heart disease (CHD) (3-5), and gastric mu cosal injury (6).
Oxidized low density lipoprotein (LDL) is considered to be an important risk factor in the atherogenic pro gression of cardiovascular disease (7). Oxidized LDL can attract monocytes into the arterial wall where they can be transformed into macrophages-the precursors of foam cells, leading to the formation of early atheroscle rotic lesions (8, 9). Protection against LDL oxidation is critical in the prevention of the initiation and progres sion of atherosclerosis.

We previously reported that red wine and cocoa, which contain dietary polyphenolic components, inhib ited LDL oxidation (10, 11). These antioxidant foods are enriched in various types of flavonoids such as catechin and quercetin. In the Zutphen Elderly Study (5), quercetin, kaempferol, myricetin, apigenin, and luteolin were compared as dietary antioxidant flavonoids. It is still unclear, however, which flavonoid has the most beneficial effects in preventing LDL oxidation.
Previous studies in vitro have demonstrated that the

fl avonoids act as hydrogen-donating free radical scav engers, and their function is dependent on their struc tural properties (1, 12, 13), of which there are three key features: 1) the o-dihydroxyl (catechol) structure in the B ring, which confers higher stability to radical form;
2) a 2,3-double bond in conjunction with the 4-oxo function in the C ring, which is responsible for electron delocalization; and 3) the 3 and 5-hydroxyl groups re sponsible for maximal radical scavenging power.
The aim of the present study was to determine the antioxidant power of flavonoids against aqueous radi cals and lipophilic radicals. In this study, we examined the antioxidant capacity of 10 structurally related fl avonoids by measuring their free-radical-scavenging

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Abbreviations: LDL, low density lipoprotein; EC, epicate chin; EGC, epigallocatechin; ECg, epicatechin gallate; EGCg, epigallocatechin gallate; DPPH, 1,1-Biphenyl-2-picryl-hy drazyl; AMVN-CH3O, 2,2'-azobis(4-methoxy-2,4-dimethyl valeronitrile); a-toc, a-tocopherol; BCA, bicinchonic acid; EDTA, ethylenediaminetetraacetic acid; PBS, phosphate buffered saline; AAPH, 2,2'-azobis(2-amidinopropane)dihy drochloride.

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Fig. 1. The chemical structures of the flavonoids used in this study

capacity and inhibition of LDL oxidation. Furthermore,
the potential power of flavonoids to recycle a-toc radical
was also investigated.

MATERIALS AND METHODS

Materials. (-)-Epicatechin (EC), (-)-epigallocate
chin (EGC), (-)-epicatechin gallate (ECg), and (-)
epigallocatechin gallate (EGCg) were donated by Itoen
Ltd., Japan. (+)-Catechin, myricetin, quercetin, api
genin, kaempferol, and luteolin were purchased from
Sigma Chemical Co. (St, Louis, MO, USA), The general
formulas for flavonoids used in this study are shown
in Fig. 1.2,2'-Azobis(4-methoxy-2,4-dimethylvaleroni
trile) [AMVN-CH30] and 1,1-diphenyl-2-picryl-hy
drazyl [DPPH] were purchased from wako Pure
Chemical Industries (Osaka, Japan). A bicinchonic acid
(BCA) assay kit for protein was from Pierce Laboratories
Inc. (Rockford, IL, USA) and a-toc was a gift from Eisai
Co., Ltd. (Tokyo, Japan).
Free radical scavenging capacity of flavonoids. The free
radical scavenging capacity of flavonoids was measured
by using DPPH (14). An aliquot of the various
fl avonoids dissolved in ethanol was mixed with 2 mL of
0.1mM DPPH solution (in ethanol). Following 20min
of incubation at 37•Ž, the absorbance at 516nm was
measured with a Beckman Model DU 650 spectropho

tometer.

Isolation and preparation of LDL. LDL was isolated
from human plasma with EDTA (1mg/mL) prepared
from fasting normolipidemic volunteers, who were
someone of authors obtained informed consent. It was
separated by a single-spin density gradient ultracen
trifugation (417,000•~g, 40min, 4•Ž) by using the
TLA-100.4 rotor fixed-angle rotor (Beckman Instru
ments Inc., CA, USA) (15) and protein was determined

Fig. 2. Dose-dependent effect of luteolin on scaveng
ing the DPPH radical. The absorbance at 516nm was
determined following 20min incubation at 37•Ž after

luteolin was added to ethanolic DPPH.

by the BCA method (16). Before the start of the oxida
tion experiments, LDL samples were diluted with PBS to
give final concentrations of 3 5 or 70ƒÊg/mL LDL pro
tein as indicated in Fig. 4 and Tables.
Oxidative modification of LDL. The oxidation of LDL
was performed as previously reported (17). LDL solu
tions in the absence (control) or presence of flavonoids
and/or a-toc were incubated with freshly prepared
AMVN-CH30 (200ƒÊM), using acetonitrile as the sol
vent. The kinetics of LDL oxidation was obtained by
monitoring the absorbance of conjugated dienes forma
tion at 234nm with a Beckman Model DU 650 spec
trophotometer at 5min intervals at 37•Ž.
The lag time of lipid peroxidation, measured as previ
ously described (18), is defined as the time interval be
tween initiation and the intercept of the two tangents

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Table 1. Effects of various flavonoids on LDL oxidation
lag time and propagation rate.

An aliquot (2ƒÊM) of each flavonoid solution was added in
LDL solution (70ƒÊg/mL LDL protein).

drawn to the lag and propagation phase of the ab sorbance curve at 234nm and was expressed in min. The propagation rate of conjugated dienes was esti mated from the linear portion of the curve, using the extinction coefficient for conjugated dienes at 234nm (29,500L/mol/cm) (18).

RESULTS

Radical scavenging capacity of flavonoids
The radical scavenging capacity of various flavonoids
was tested with DPPH in aqueous phase (Figs. 2 and 3).
As shown in Fig. 2, the absorbance of DPPH at 516nm
decreased linearly by the addition of luteolin, showing
that luteolin is an effective DPPH radical scavenger. The
relative effectiveness of the flavonoids in scavenging the
DPPH radical is shown in Fig. 3. The data revealed that
EGCg, ECg, quercetin, EC, catechin, and myricetin were
most effective and sufficed at less than 3ƒÊM for scav
enging 50% of the DPPH radical in comparison with
EGC (3.99ƒÊM) and kaempferol (5.93ƒÊM), whereas lute
olin was the least effective and required 16ƒÊM to do the
same. The value of apigenin could not be estimated be
cause no DPPH radical was trapped.
Effect of flavonoids on AMVN-CH30-induced LDL oxidation
The oxidation of LDL was used as an indication of in
vestigating the lipid chain-breaking antioxidant activity
of flavonoids. AMVN-CH30 rather than copper ions was
chosen for propagating lipid peroxyl radicals to avoid
the effects of flavonoids as metal chelators (13). As
shown in Fig. 4, when LDL (70ƒÊg protein/mL) was in
cubated with 200ƒÊM AMVN-CH30 in the absence (con
trol) or presence of 1 or 2ƒÊM quercetin, quercetin pro
longed the lag time in a dose-dependent manner.
Compared with the control group (23.9min), the lag
time was prolonged 2.5-fold at 1ƒÊM and 3.4-fold at
2ƒÊM. Flavonoids examined in this study are classified
into flavanols (catechin group), flavonols (quercetin,
myricetin, and kaempferol), or flavones (luteolin and
apigenin). The inhibitory effect of each flavonoid (2ƒÊM)

Fig. 3. The relative effectiveness of the flavonoids in scavenging DPPH radical. The concentrations of fl avonoids (IC50) necessary to scavenge 50% of DPPH radical were calculated: C, catechin; Q, quercetin; M, myricetion; K, kaempferol; L, luteolin.

Fig. 4. Effect of quercetin on the oxidation of LDL by AMVN-CH30. LDL (70ƒÊg protein/mL) was incubated
with 200ƒÊM AMVN-CH30 in the presence of 1 or
2ƒÊM quercetin: •›, control; •œ, 1ƒÊM quercetin; •¡,
2ƒÊM quercetin.

for LDL oxidation was determined as shown in Table 1.

Their order of prolongation of LDL oxidation lag time was ECg (250min)>EC (197min)>catechin (182 min)>EGCg (136min)>EGC (119min) in the flavanol group, quercetin (190min)>myricetin (106mm)> kaempferol (52.4min) in flavonols, and luteolin (434 min)>apigenin (31.6min) in flavones. The propagation rates of luteolin, quercetin, EC, C, and ECg were roughly similar to one another in the range of 0.44 to 1.21 nmol diene/min/mg protein. Among them, lute olin was the most effective in breaking the lipid peroxyl radical chain.

To analyze the correlation between the trapping abili ties of flavonoids for aqueous phase and lipid peroxyl radicals, we plotted the values of IC50 scavenging DPPH radical against LDL oxidation lag time obtained in Fig. 3 and Table 1, respectively. Luteolin scavenges lipid per

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Fig. 5. Correlation of the trapping ability of flavonoids
between aqueous phase and lipid peroxyl radicals. The
IC50 values for scavenging DPPH radical were plotted
against LDL oxidation lag time obtained in Fig. 3 and
Table 1, respectively

oxyl radical effectively, but not radicals in an aqueous
phase (Fig. 5). With respect to flavonoids except lute
olin, a significant correlation was found between IC50
scavenging DPPH radicals and the propagation rate of
conjugated dienes (r=0.781; p<0.05).
Interactive effects between a-tocopherol and flavonoids
Our previous report showed that the shortened lag
time observed at higher doses of a-toc was restored
when water-soluble antioxidants such as ascorbic and
uric acids were added simultaneously (17). Tables 2
and 3 show the interactive effects of a-toc and
fl avonoids on LDL oxidation. The addition of flavonoids
reversed the lag time, which was shortened at a high
dose of a-toc. The interaction of a-toc with catechin

prolonged the lag time as compared with the control
(20.5min). The lag time (73.6min) at 6mg/100mL a
toc, prolonged to 146.3min with 10ƒÊg/100mL cate
chin and to 172.2min with 20ƒÊg/100mL catechin.
In the same experiment, the lag time with 3mg/
100mL a-toc was 100.4min and 87.2min with
6mg/100mL a-toc. This result differs from that shown
in Table 2 because LDL used in each experiment was
taken from individual subjects, and the conditions of
the experiments also differed. When a-toc and EGCg
were added together, the lag time with 10ƒÊg/100mL
EGCg was 104.6min (3mg/100mL a-toc) or 119.9
min (6mg/100mL a-toc). This effect is due to the re
generation of a-toc from a-toc radicals by flavonoids
and ascorbate, among others.

DISCUSSION

The present study demonstrated that all the flavo noids except luteolin and apigenin (flavones) can effec tively donate an electron to DPPH radical and that fl avonoids inhibit azo radical-initiated LDL oxidation

and regenerate a-toc radical to a-toc. This effectiveness

Table 2. Effect of catechin on the further extension of
lag time of conjugated diene formation in the oxidation
of LDL (35ƒÊg/mL LDL protein) by a-tocopherol.

The oxidation was initiated by the addition of 200ƒÊM
AMVN-CH30. The suspensions of LDL and a-toc were in
cubated for 10mmn at 3 7•Ž before the addition of cate
chin and AMVN-CH30. a-Toc and catechin are expressed
as mg/100mL and ƒÊg/100mL, respectively.

Table 3. Effect of EGCg on the further extension of lag
time of conjugated diene formation in the oxidation of
LDL (3 5ƒÊg/mL LDL protein) by a-tocopherol.

The oxidation was initiated by the addition of 200ƒÊM
AMVN-CH30. The suspensions of LDL and a-toc were in
cubated for 10min at 37•Ž before the addition of EGCg
and AMVN-CH3O. a-Toc and EGCg are expressed as
mg/100mL and ƒÊg/100mL, respectively.

depends on the structural feature of flavonoids.
In DPPH assay, the electron-donating capacity
was strongest in the following order: EGCg>ECg=
quercetin>EC=catechin>myricetin>EGC>kaempfer
ol>luteolin•tapigenin (Fig. 3). This result is in agree
ment with the previous study (19) comparing
quercetin, catechin, and myricetin.
EGC is structurally similar to catechin and EC with
an additional hydroxyl group adjacent to the catechol
structure in the B ring (Fig. 1). EGCg and ECg, which
are esters of EC and EGC with gallic acid, enhanced the
DPPH scavenging ability. An orthogonal A ring-car
bonyl group conformation combined with an ability to
form an intramolecular hydrogen bond is needed for
DPPH scavenging activity (20). All the flavonoids tested
here have this structure. Considering the chemical
structures of electron-donating flavonoids, the DPPH
radical scavenging ability could depend on the number
of hydroxyl substitutions in its backbone structure, as
indicated by Cao et al. (21). Notably, apigenin has only
three hydroxyl groups, resulting in a lower effectiveness
for radical scavenging.

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In the case of radicals generated in the lipophilic
phase, quercetin prolonged the oxidation lag time in a
dose-dependent manner (Fig. 4). Flavonoids sometimes
behaved as prooxidants according to the conditions
(21), but certainly at a 1 or 2ƒÊM dose they served
as antioxidants. Previous studies have compared the
antioxidant effect of flavonoids on LDL oxidation.
Miura et al. (22, 23) reported that in a Cu2+-mediated
oxidation system, the ability to prolong the lag time was
in the order of EGCg>ECg>EC>catechin>EGC, and sesaminol>quercetin>EGCg>theaflavin=myricetin>
a-toc. The inhibition of Cu2+-mediated oxidation by
fl avonoids was attributed to chelating the Cu2+ ions,
and the lag time extension was in the order of
quercetin>luteolin=rutin>kaempferol (13). The LDL
oxidation with metmyoglobin was inhibited in the order
of ECg=EGCg=EC=catechin>EGC>gallic acid (24).
Our findings on flavanols, in part, differed from the
above-mentioned results, but were in agreement with
data using an azo initiator (AAPH) of oxidation (25).
Kaempferol and apigenin having only one hydroxyl
group on the B ring responded poorly to the LDL oxida
tion system (Table 1). In this study, the catechol struc
ture of the B ring proved to be a prime determinant of
the prevention of LDL oxidation.

Belinky et al. (26) have reported that the DPPH assay and oxidation potential (Epl/2) correlated with Cu2+ -induced and macrophage-mediated LDL oxidation in the presence of flavonoids. In agreement with these data, a good correlation was found between the DPPH assay data measured as an electron-donating test and the oxidation lag time, with the exception of luteolin (Fig. 5). Luteolin failed to donate an electron to DPPH, but showed the highest antioxidant activity in the LDL oxidation system for reasons still unknown. Further studies will be needed to investigate why luteolin be haves quite differently in the two assay systems.
We have previously reported that the shortened lag time induced by a high dose of a-toc is recovered in the presence of ascorbate (17). Several reports have shown that a high dose of a-toc is prooxidantive during the au toxidation of polyunsaturated fatty acids (27). Vitamin E leads to an increase in conjugated dienes concur rently with the formation of a propagation phase and results in a shortening of the oxidation lag time in LDL. In the experiment on the interaction between a-toc and fl avonoids, we used catechin and EGCg as flavonoids be cause we are easy to ingest them with a-toc in our daily diet. As a result, flavonoids and ascorbate were both found to restore a-toc radical or to delay the consump tion of a-toc (Tables 2 and 3). Our results on the inter action between a-toc and flavonoids are supported by a report from Viana et al. (28), using flavonoid-rich ex tracts. The cooperative effects of flavonoids and ascor bate have been recognized by Mathiesen et al. (29) and Skaper et al. (30), who described that ascorbate-pro tected flavonoids from oxidation. It thus seems very likely that the interaction between lipophilic and aque ous antioxidants within the body increases LDL resist ance to oxidation.

In conclusion, the present study showed that the an tioxidant ability of flavonoids depended on key struc tural properties and cooperative interactions with a toc, effectively inhibiting lipid oxidation. Taken alto gether, the higher antioxidant effect enough to prevent free-radical-related diseases can be expected by a mod erate intake of such dietary antioxidants as flavonoids, a-toc, and ascorbate.

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