The lack of effect of isoflavones on high-density lipoprotein cholesterol concentrations in adolescent boys: a 6-week randomised trial

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Abstract

Background: A substantial fall in high-density lipoprotein cholesterol (HDL-C) during puberty in boys, but not girls, has been reported in Western populations. The fall in boys is believed to be due to hormonal changes – androgens have been shown to be associated with lower HDL-C, whereas oestrogens are associated with higher HDL-C. The fall in HDL-C during puberty was not observed, however, in a study of Moslem boys in Israel, nor in a group of Japanese boys. A diet high in phyto-oestrogens may account for the lack of a fall in HDL-C in these populations.

Objective: To examine the effect of dietary supplementation with phyto-oestrogens on the HDL-C concentration of adolescent boys from a Western population. We hypothesised that dietary supplementation of 50 mg of the isoflavones daidzein and genistein would produce a 12% higher HDL-C concentration than in controls at the end of a 6-week intervention period.

Design: A randomised controlled trial.

Setting: Hellyer College in Burnie (Tasmania, Australia).

Subjects: Adolescent boys (aged 16–18 years) were recruited through a letter sent to parents. A total of 132 eligible participants enrolled and five subjects withdrew from the trial.

Results: No significant increase in HDL-C was observed in the treatment group (–0.02 mmol l–1, standard error (SE) 0.03, \( P = 0.53 \)) or the placebo group (0.05 mmol l–1, SE 0.03, \( P = 0.11 \)).

Conclusions: Factors other than isolated dietary isoflavones may be responsible for the lack of fall in HDL-C during puberty in Japanese and Moslem boys.

The presence of certain coronary heart disease (CHD) risk factors in childhood, including high-density lipoprotein cholesterol (HDL-C), have been related to the development of fatty streaks and plaques in the coronary arteries in early life. Moreover, low HDL-C in adult life is known to be strongly related to risk of CHD. Two separate findings suggest that it is important to obtain an understanding of the factors that raise HDL-C in children.

Evidence from Western populations shows that HDL-C falls substantially during puberty in boys, but remains unchanged in girls. However, two studies show that what has been regarded as the ‘typical’ fall of HDL-C with puberty in boys is not universal. Greenland et al. found a 10–20% fall in HDL-C between the ages of 9–10 years and 16–18 years among Jewish boys in Israel, whereas among Moslem boys there was a rise in HDL-C between these ages. Our group has previously published data comparing Japanese, Australian and American boys from the same age groups and found similar outcomes. Australian and US boys experienced a 10–20% fall in HDL-C and a concomitant fall in total cholesterol (TC), while the Japanese boys showed little change in HDL-C or TC.

The fall in HDL-C in puberty in boys has been thought to be due to the associated hormonal changes – androgens having been shown to be associated with lower HDL-C, while oestrogens are associated with higher HDL-C. Other factors shown to have the potential to substantially

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alter HDL-C in adults are physical activity, alcohol and certain drugs such as HMG Co-A reductase inhibitors. In children, physical activity has been demonstrated to elevate HDL-C in some studies but not others and the effect appears less than in adults. Obesity in children is also associated with lower HDL-C.

In our previous study, differences in physical activity and obesity seem unlikely to explain the higher HDL-C concentrations in postpubertal Japanese boys. We did not have alcohol consumption but this was examined in the Israeli study and did not appear to account for the difference found there. Given that the atypical HDL-C concentration has been observed in both a developed country, Japan, and the less developed Israeli Moslem population, it seems more likely that it is due to a lifestyle factor rather than a medical intervention. A common genetic determinant would appear unlikely.

Diet is a plausible candidate and, looking at the Japanese/Australian comparison, there are a number of striking contrasts. Of the foods that the Japanese children consume, soybean and its products are most like foods that Moslem children in Israel might also consume. The Moslem population in Israel is known to consume large quantities of lentils and chick-peas which, like soybeans, are legumes. These foods, which are not commonly consumed by the majority of Australian children, might be expected to have an effect on HDL-C through their influence on hormonal status. Soybeans, lentils and chick-peas contain isoflavones – phyto-oestrogens – that could mimic the effect of endogenous oestrogens.

The small number of trials in children and adolescents suggest that soybean products affect HDL-C in this age group. Jacques et al. found a 5% rise in HDL-C in a 4-week intervention involving soy protein in 6–12-year-old hypercholesterolaemic boys. While most of the trials have involved whole soy or soy protein, there have been interventions using isoflavones. Knight et al. observed a large increase in HDL-C in postmenopausal women with isoflavone intervention, although an Australian trial of isoflavones in adult men and postmenopausal women failed to find an effect on HDL-C.

The aim of the present study was to compare the effect of 6 weeks of isoflavones supplementation to a placebo on HDL-C in adolescent boys. We conducted the trial in northern Tasmania, an area which suffers high mortality from cardiovascular disease and where HDL-C concentrations in adults are lower than the national average.

**Subjects and methods**

The study was conducted at Hellyer College in Burnie, Tasmania (latitude 41°S) in 1999. All male students who were aged between 16 and 18 years and enrolled at the college were eligible to participate. The students were invited to participate though an information letter sent to their parent or guardian, and through information distributed through the school’s daily newsletter.

The study was approved by and performed in accordance with the ethical standards of the Human Research Ethics Committee of the University of Tasmania (project number H0003116).

Our hypothesis was that the treatment group would achieve a greater increase in mean HDL-C than the control group.

The primary outcome measure was serum HDL-C concentration. Secondary outcome measures were serum TC, low-density lipoprotein cholesterol (LDL-C) and triacylglycerol (TAG) concentrations.

Fasting venous blood was collected from subjects at baseline and at 6 weeks. Subjects fasted from 22.00 hours the previous night and all samples were collected between 08.00 and 11.00 hours. Blood collected for lipid analysis was kept cool on ice after collection and centrifuged (3000 rpm for 5 min) within 3 h.

The study was powered to detect a 12% difference in mean HDL-C (equivalent to 0.15 mmol l⁻¹) between treatment and placebo groups at post-intervention.

Height, weight, waist and hip circumferences, four skinfold measurements (triceps, biceps, subscapular and suprailiac) and blood pressure were also measured at baseline and at 6 weeks. Body mass index (kg m⁻²) was calculated from weight and height; waist-to-hip ratio was calculated from the waist and hip circumferences; and percentage body fat was calculated from the four skinfold measures. During the first week of the trial, physical
activity was assessed using a pedometer (Omron HJ-003) by measuring the average number of steps taken per day. Tanner pubertal stage was self-assessed by questionnaire using drawings made from Tanner's photographs illustrating the five stages of pubertal development. LDL-C was calculated using the Friedewald formula: \[ \text{LDL-C} = \text{TC} - \text{HDL-C} - (\text{TAG}/5) \]. No dietary restrictions were imposed; however, all subjects completed a food-frequency questionnaire and were asked about their consumption of a range of foods known to contain isoflavones.

The randomisation was determined by the manufacturer using computer-generated random numbers. Tablets were contained in individually numbered zip-locked plastic bags. Subjects were allocated the next numbered bag of tablets, in the order determined by the manufacturer, at their baseline appointment.

The person conducting the field work (K.L.H.) was not aware at any stage during the trial period of the subject allocation to treatment/control. The laboratory technicians who analysed the blood and urine samples were also blinded throughout the trial. The data analysts (K.L.H., J.L.F. and C.L.B.) were not given access to the code until the trial was completed. Subjects and field assistants were blinded. A copy of the code was sent to the team by email and was not opened until the results of the lipid analysis were available.

Subject compliance was assessed by tablet counts and urinary assay of daidzein and genistein at baseline and 6 weeks by reverse-phase high-performance liquid chromatography as previously described. The intra-assay and inter-assay coefficients of variation were 3.74% and 10.23% for daidzein, and 3.28% and 7.99% for genistein. Urine samples were taken from the first void of the morning.

Statistical analysis was performed using Stata Statistical Software, release 8.0 (StataCorp, 2003). The primary analysis was based on the intent-to-treat (ITT) principle, with the last observation carried forward for subjects with missing data. All subjects randomised to receive study treatment were included in the ITT population.

Analysis of variance methods were used to estimate the mean change in lipid values and the difference in mean change between treatment and placebo groups, both without and with adjustment for baseline levels of the lipid outcome. The outcomes examined were change in HDL-C, change in TC, change in ratio of TC to HDL-C, LDL-C, change in ratio of LDL-C to HDL-C and change in TAG. Results of statistical tests were regarded as statistically significant if the associated \( P \)-values were less than 0.05. A per-protocol analysis was also performed whereby the analysis was restricted to subjects with a diary compliance of at least 80%. To examine compliance, the outcomes examined were change in daidzein and change in genistein concentrations. Because these two isoflavone measures were not normally distributed, the regression model was applied to ranks of the data rather than their actual values.

Results

Of the 297 boys eligible to participate, 136 volunteered during the recruitment period. Four were excluded because they were outside the age limits. Five subjects withdrew during the trial. A total of 127 boys completed the trial: 69 in the treatment group and 58 in the placebo group. Figure 1 provides details of the progress through the phases of the trial.

Baseline characteristics of the two groups are shown in Table 1. There were no material differences in baseline factors between those assigned to the isoflavone and those assigned to the placebo.

Results of the urinary assay of compliance are shown in Table 2. Isoflavone supplementation resulted in significant increases in both urinary daidzein and genistein concentrations compared with the placebo group (both \( P < 0.001 \)).

In a test of subject knowledge of allocation conducted at the end of the trial, 11.6% and 10.3% of subjects in the treatment and placebo group, respectively, correctly surmised which group they were assigned to. The remainder indicated that they did not know which group they were in (76.8% and 82.8%) or they guessed incorrectly (11.6% and 6.8%). There was no significant difference between the knowledge of each group (\( \chi^2 = 0.05, P = 0.82 \)).

Results of the trial are reported in Table 3. There was no evidence of an increase in HDL-C in the treatment group; instead their mean concentration of HDL-C decreased slightly by 0.02 mmol\( \text{L}^{-1} \). A small increase of 0.05 mmol\( \text{L}^{-1} \) in mean HDL-C was observed in the placebo group. After adjusting for baseline HDL-C concentrations the placebo group had a slight increase in mean HDL-C concentration relative to the treatment group.

TC concentrations decreased more in the treatment group than the control group, but the difference between the two groups was not statistically significant after adjusting for baseline TC concentrations.

The changes in TC/HDL-C ratio, LDL-C concentration, LDL-C/HDL-C ratio and TAG concentration did not reach statistical significance. Analysing the ratio of TC to HDL-C on the log scale made no difference to the results.

We found no evidence of confounding by measures of body fatness (body mass index, percentage of body fat, waist circumference), physical activity (measured by pedometer) or age.

Per-protocol analysis of subjects with a diary compliance of \( \geq 80\% \) did not differ appreciably from the ITT analysis and is not shown.

Discussion

The studies in Japanese children and Israeli Moslem children were observational studies comparing populations

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of different ethnic backgrounds. These studies did not reveal the relevant cause of the differences in HDL-C in postpubescent boys. In the present study we tested whether isoflavones might be an important cause of the difference in HDL-C, but our results do not support this hypothesis. Neither do our results show an effect of isoflavones on other lipids.

In this randomised controlled trial of supplementation with isoflavones to the diet of 16–18-year-old Australian boys, to the level of usual Japanese intakes, a greater reduction in mean concentration of TC was observed in the treatment group than the control group. Our primary outcome measure was HDL-C, however, and this increased in the placebo group relative to the treatment group. None of these changes were statistically significant.

There are few previous studies examining the effect of isoflavones, or soy protein in general, on lipids in children and adolescents. In two clinical trials examining the effect of soy protein on lipids in children with familial hypercholesterolaemia, significant increases in HDL-C and significant decreases in very-low-density lipoprotein cholesterol were reported\(^{15,21}\). Most published studies have been conducted on adults, and these also have predominantly involved subjects with raised non-HDL lipids or menopausal and perimenopausal women. These studies tend to focus on the hypocholesterolaemic effects of soy on TC and other non-HDL lipids.

Recent reviews indicate that the relationship between consumption of soy protein, isoflavones and lipoproteins is not a clear one. Significant increases in HDL-C in

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**Fig. 1** Flow diagram of progress through the phases of the trial (ITT – intention-to-treat; PP – per-protocol).
Isoflavones fail to raise HDL-C in boys

Table 1 Characteristics of study participants at baseline

<table>
<thead>
<tr>
<th>Measure</th>
<th>Treatment group (n = 71)*</th>
<th>Placebo group (n = 61)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at start of trial (years)</td>
<td>16.63 (0.68)</td>
<td>16.56 (0.62)</td>
</tr>
<tr>
<td>Daidzein (ng μmol⁻¹ creatinine)</td>
<td>19.06 (IQR 0.00, 77.21)‡</td>
<td>30.75 (IQR 0.00, 55.65)§</td>
</tr>
<tr>
<td>Genistein (ng μmol⁻¹ creatinine)</td>
<td>10.52 (IQR 0.00, 38.59)§</td>
<td>11.5 (IQR 0.00, 34.16)§</td>
</tr>
<tr>
<td>HDL-C (mmol l⁻¹)</td>
<td>1.19 (0.24)</td>
<td>1.19 (0.26)</td>
</tr>
<tr>
<td>TC (mmol l⁻¹)</td>
<td>4.35 (0.72)</td>
<td>4.18 (0.71)</td>
</tr>
<tr>
<td>TC/HDL-C ratio</td>
<td>3.77 (0.87)</td>
<td>3.68 (0.92)</td>
</tr>
<tr>
<td>TAG (mmol l⁻¹)</td>
<td>0.93 (0.37)</td>
<td>0.96 (0.46)</td>
</tr>
<tr>
<td>LDL-C (mmol l⁻¹)</td>
<td>2.98 (0.66)</td>
<td>2.82 (0.65)</td>
</tr>
<tr>
<td>LDL-C/HDL-C ratio</td>
<td>2.61 (0.81)</td>
<td>2.50 (0.83)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>177.78 (6.92)</td>
<td>177.44 (6.86)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.78 (9.75)</td>
<td>72.62 (12.99)</td>
</tr>
<tr>
<td>BMI (kg m⁻²)</td>
<td>23.00 (2.75)</td>
<td>23.07 (3.61)</td>
</tr>
<tr>
<td>%BF</td>
<td>0.78 (0.03)</td>
<td>0.78 (0.04)</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>14.57 (4.71)</td>
<td>15.10 (5.59)</td>
</tr>
<tr>
<td>WHR</td>
<td>121.31 (12.94)</td>
<td>120.82 (13.26)</td>
</tr>
<tr>
<td>Tanner pubertal stage</td>
<td>4.47 (0.60)</td>
<td>4.57 (0.48)</td>
</tr>
</tbody>
</table>

HDL-C – high-density lipoprotein cholesterol; TC – total cholesterol; TAG – triacylglycerols; LDL-C – low-density lipoprotein cholesterol; BMI – body mass index; WHR – waist-to-hip ratio; %BF – percentage body fat; SBP – systolic blood pressure; DBP – diastolic blood pressure; IQR – interquartile range.

*Mean change and standard deviation for all variables except daidzein and genistein, where median and IQR are given.
†LDL-C calculated using the Friedewald formula: LDL-C=TC–(TAG/5).
‡51/70 (40%) had non-detectable concentrations. Excludes one subject from treatment group for whom urine measures were not available.
§19/60 (32%) had non-detectable concentrations. Excludes one subject from placebo group for whom urine measures were not available.
*Excludes one subject for whom HDL-C measures were not available.

Table 2 Assessment of compliance: change in urinary isoflavone concentrations in treatment and placebo groups

<table>
<thead>
<tr>
<th>Measure</th>
<th>Treatment group (n = 71)*</th>
<th>Placebo group (n = 61)*</th>
<th>Treatment effect minus placebo effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Within-person median change (IQR)</td>
<td>Within-person median change (IQR)</td>
<td>Mean difference (SE)</td>
</tr>
<tr>
<td>Daidzein (ng μmol⁻¹ creatinine)</td>
<td>319.93 (159.68, 550.98)</td>
<td>0.00 (−14.32, 72.21)</td>
<td>436.57 (76.03)</td>
</tr>
<tr>
<td>Genistein (ng μmol⁻¹ creatinine)</td>
<td>155.34 (78.07, 298.03)</td>
<td>0.00 (−13.98, 23.94)</td>
<td>236.42 (43.17)</td>
</tr>
</tbody>
</table>

IQR – interquartile range; SE – standard error.
*Excludes one subject who did not have a pre-intervention urine sample.
†Excludes four subjects who did not have a post-intervention sample.
‡Adjusted for the initial value of the urinary isoflavone concentrations.

Hypercholesterolaemic men and women consuming soy protein have been reported in some studies.25–27 However, a meta-analysis of 38 trials of soy protein by Anderson et al.28 found only a non-significant increase in HDL-C, and Howes et al.25 found that HDL-C did not change in postmenopausal women with mild to moderate hypercholesterolaemia supplemented with isoflavones from red clover. In a recent review of 22 randomised trials using isolate soy protein with isoflavones, Sacks et al.30 failed to find any significant effects on HDL-C, although decreased LDL-C concentrations were observed.

Among adults with elevated cholesterol concentrations most trials report a lowering of TC and LDL-C following consumption of soy protein.31 In adults with normal cholesterol concentrations, though, soy protein does not appear to impact on cholesterol concentration.31 A review of studies examining the effect of isoflavones, in particular, found that while minor reductions in LDL-C and non-HDL cholesterol have been reported, studies in which no reduction was found were also identified.32 A meta-analysis of the effects of isoflavones by Yeung and Yu33 reported no significant effects on lipid concentrations.

Reasons for the lack of consistency in the results of trials may be related to the type of dietary intervention. A wide variety of soy-based interventions are reported, including whole soy protein, isolated soy protein, soy protein with and without isoflavones, isolated isoflavones and specially manufactured foods high in soy. The inconsistency could also be due to random variation associated with the small sample size of many studies.

A recent meta-analysis34 reports that soy protein with isoflavones intact resulted in significant decreases in TC, LDL-C and TAG and a significant increase in HDL-C in adults. Tablets containing extracted soy isoflavones, on the other hand, did not have a significant effect in reducing TC, although their effect on HDL-C is not stated. It is noted, however, that only trials of greater than 12 weeks’ duration showed improvements in HDL-C and that
studies with greater than 80 mg (our study had 50 mg) showed greater effects on the lipid profile.

In this study we examined the effect of isolated isoflavones on HDL-C concentration. A review by Erdman suggests that isolated isoflavones may not have the same effect on lipids as isoflavones consumed as part of soy protein. Soy protein containing isoflavones has been shown to lower cholesterol significantly more than soy protein without isoflavones. Soy protein containing isoflavones has been shown to lower cholesterol significantly more than soy protein without isoflavones. The part of the soy responsible for affecting lipids, however, has not been clearly identified. There is increasing evidence that the various components of soy protein act in synergy to bring about their effects on lipids. In addition to isoflavones, soy protein also contains trypsin inhibitors, phytic acid, saponins and fibre – all of which have been reported to be associated with lowering non-HDL cholesterol. Saponins, for example, are present in soy protein isolate at three times the concentrations of isoflavones and have been shown, among a number of bioactivities, to reduce cholesterol. Our reason for choosing isolated isoflavones was based on the hypothesis that the lack of a ‘usual’ fall in HDL-C during puberty observed in both Japanese boys and Moslem boys from Israel may be due to a diet containing phyto-oestrogens that impact on hormonal changes at this time. In the Japanese diet the source of phyto-oestrogens is soy protein and in the Moslem diet, chick-peas and lentils, however, chick-peas and lentils, like soy protein, contain saponins as well as isoflavones.

It is possible that more than one of these constituents acting together may influence lipid concentrations.

In addition to the effects of isolated isoflavones vs. soy-protein-containing isoflavones, the timing of the consumption of dietary phyto-oestrogens should also be considered. The fall in HDL-C in boys from Western populations occurs during puberty. The participants in this trial were aged between 16 and 18 years and, based on Tanner stage, were close to physical maturity. The concentration of HDL-C in these boys had already decreased from normal childhood levels. It is possible that isoflavones may influence hormone production and subsequent HDL-C concentrations at the beginning of puberty, but have less effect afterwards. A major difference between our study population and the Japanese and Moslem boys (who do not show a decrease in HDL-C following puberty) is the consumption of isoflavones prior to puberty. By conducting the trial on older adolescents we may have missed the ‘window’ for influencing hormone concentrations. Postpubertal concentrations of endogenous male hormones may have affected the responsiveness to phyto-oestrogens. We would have preferred to conduct the study on pubescent boys but were concerned about possible powerful side-effects on bone and feminisation.

Lipid concentrations are known to vary on a day-to-day basis. In our study lipids were measured only once at each time point (pre- and post-intervention) in order to reduce the burden on subjects. Had it been feasible to make multiple measurements on each subject both pre- and post-intervention, it is likely that random error would have been reduced and the effects we observed would have been larger and measured more precisely.

Our study has not provided evidence that isolated isoflavones increase HDL-C in adolescent boys, but it raises a number of questions about the role of phyto-oestrogens, soy protein and dietary factors that may be important in influencing HDL-C. Future studies could possibly test the effects of isoflavones over a longer period or in a younger group of subjects than used here, though the ethics of administering plant oestrogens to prepubescent boys in an experimental setting would be an issue to consider. Further research on the bioactive compounds of soy protein and how the various components interact may also provide a better understanding of the factors that influence HDL-C in children and adolescents.

### Acknowledgements

The study was funded by a grant from the National Health and Medical Research Council of Australia (9937770).

### Table 3 Intention-to-treat analysis: within-person change for treatment and control groups, and differences between treatment and control groups

<table>
<thead>
<tr>
<th>Measure</th>
<th>Treatment group (n = 71) within-person mean change (SE)</th>
<th>Placebo group (n = 61) within-person mean change (SE)</th>
<th>Treatment effect minus placebo effect</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HDL-C (mmol l⁻¹)</td>
<td>−0.02 (0.03)</td>
<td>0.05 (0.03)</td>
<td>−0.07 (0.04) −0.07 (0.04)</td>
<td>0.07</td>
</tr>
<tr>
<td>TC (mmol l⁻¹)</td>
<td>−0.36 (0.07)</td>
<td>−0.14 (0.07)</td>
<td>−0.22 (0.10) −0.16 (0.09)</td>
<td>0.09</td>
</tr>
<tr>
<td>TC/HDL-C ratio</td>
<td>−0.25 (0.09)</td>
<td>−0.31 (0.07)</td>
<td>0.06 (0.11) 0.10 (0.10)</td>
<td>0.30</td>
</tr>
<tr>
<td>LDL-C (mmol l⁻¹)</td>
<td>−0.35 (0.58)</td>
<td>−0.22 (0.55)</td>
<td>−0.13 (0.08) −0.08 (0.08)</td>
<td>0.27</td>
</tr>
<tr>
<td>LDL-C/HDL-C ratio</td>
<td>−0.26 (0.76)</td>
<td>−0.31 (0.06)</td>
<td>−0.05 (0.10) 0.09 (0.09)</td>
<td>0.29</td>
</tr>
<tr>
<td>TAG (mmol l⁻¹)</td>
<td>0.04 (0.05)</td>
<td>0.07 (0.06)</td>
<td>−0.03 (0.08) −0.04 (0.07)</td>
<td>0.58</td>
</tr>
</tbody>
</table>

SE – standard error; HDL-C – high-density lipoprotein cholesterol; TC – total cholesterol; LDL-C – low-density lipoprotein cholesterol; TAG – triacylglycerols.

*Adjusted for the initial value of the lipid measure or ratio.
Isoflavones fail to raise HDL-C in boys

The tablets (Novasoy) and placebos were provided by Archer Daniels Midland Company (Decatur, IL, USA). The Tasmanian Apple and Pear Growers Association, Cripps Nubake and the Tasmanian Dairy Industry Authority donated food for breakfasts at the beginning and end of the trial. Village Cinemas Hobart donated movies passes as gifts for the study participants.

There are no conflicts of interest.

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References


