Supplementary material:
Literature search, summary of literature, and updated meta-analysis

Literature search strategy

Literature searches were repeated several times from the time of drafting of the protocol and during the study, with a final, updated search on October 30, 2013. The strategy was to first identify recent published systematic reviews of randomized placebo controlled clinical trials of vitamin D supplementation and the response-variables studied in this paper (i.HbA1c or fructosamine, ii. serum cholesterol and iii. body mass index) using search terms specified below. Then, to identify original studies on HbA1c published between the period covered by the systematic reviews and October 30, 2013, we repeated the search without restriction to systematic reviews (but adding a restriction to randomised trials, see search terms below). Titles and abstracts identified were screened by LCS to identify studies of direct relevance for comparison with our results. In addition, we (AA, KVK, HE, LCS) performed additional informal searches of PubMed and other sources, and we inspected reference lists in papers of specific interest to potentially identify further publications of relevance.

The *search terms* used in the search for systematic reviews were as follows:

1. ("systematic review" OR “meta analysis” OR "meta-analysis") AND ("vitamin D" OR cholecalciferol) AND (HbA1c OR A1C OR glycaemia OR glycemia OR glucose OR “glycated haemoglobin” OR “glycated hemoglobin” OR fructosamine)
2. ("systematic review" OR “meta analysis” OR "meta-analysis") AND ("vitamin D" OR cholecalciferol) AND (lipid* OR cholesterol)
3. ("systematic review" OR “meta analysis” OR "meta-analysis") AND ("vitamin D" OR cholecalciferol) AND (BMI OR “body mass index” OR obesity OR adiposity)

Criteria for studies to be of direct relevance to ours

Studies were considered relevant for direct comparison and potential inclusion in an informal meta-analysis together with our results if they met the following criteria:

The design should be randomized controlled trials (RCTs) with a placebo group, and reporting dependent variables included in our study measured at baseline and after a follow-up of at least 12 weeks (HbA1c, fructosamine, serum total cholesterol, serum low-density lipoprotein (LDL-) cholesterol, or serum high-density lipoprotein (HDL-) cholesterol, or body mass index (BMI)).

Intervention should be oral supplementation with vitamin D (vitamin D3 or D2, *not* 1-hydroxyvitamin D or 1,25-hydroxyvitamin D) as the only intervention.

Subjects should be non-pregnant participants aged 18 or above without a diagnosis of diabetes or other serious, chronic disease (studies in subjects with obesity or “prediabetes” that are normally not considered diagnoses were included, and relevant studies in other groups were considered for discussion but not for inclusion in meta-analysis).

Methods of summarising data: Results were summarised as estimated mean differences [(mean in treatment group at follow up – treatment group at baseline –(placebo group at follow up – placebo group at baseline)] with 95% confidence intervals. Standard deviations of mean change in each group were extracted from the papers, and if not reported (specified in the text below) we imputed this by calculating the weighted mean variance from the studies with available estimates as described by Follmann *et al.*(1) These were plotted using metan in Stata, version 12. Estimates were pooled as the weighted mean difference (fixed effects meta-analysis).

HbA1c or fructosamine

Search 1 gave 11 hits on October 30, 2013, of which the following George *et al*(2), Thomas *et al*(3) and Mitri *et al*(4) were most updated and relevant. Search in the Cochrane database did not provide any additional relevant reviews. The informal search led to the identification of a systematic review by Autier et al. published online Dec 6, 2013, and finally published in the January 2014 issue of the new journal Lancet Diabetes & Endocrinology, which is not yet indexed in PubMed; http://www.thelancet.com/journals/landia/article/PIIS2213-8587(13)70165-7/fulltext (5). This systematic review searched for both observational cohort studies and randomised controlled trials of vitamin D and a variety of biomarker and health outcomes in PubMed and Embase up to Dec 31, 2012. The results of this paper were identified after our main search and updated meta-analysis, and are briefly commented on below).

George *et al*(2) searched for publications up to March 2011 and identified 4 studies reporting HbA1c and no trials with fructosamine as the endpoint. From a total of 15 studies reporting on one or more of these endpoints, the main conclusion was that "no significant improvement was seen in fasting glucose, HbA1c or insulin resistance in those treated with vitamin D compared with placebo". Many studies were done in subjects with a diagnosis of diabetes at baseline, and of the four studies with HbA1c, this was the case for three. One study reporting on HbA1c was done with 1-hydroxy-vitamin D or placebo to subjects with prediabetes. Therefore no study was strictly comparable to ours.

Thomas *et al*(3) aimed to search for "clinical studies evaluating the impact of vitamin D on aspects of hyperglycaemia in non-pregnant adults and searched PubMed for publications during 1950 - May 2011. In the abstract, the authors concluded that "No well-designed randomised, controlled trials were identified that specifically investigated the effects of vitamin D supplementation on glucose and insulin concentrations".

Mitri *et al*(4) performed a "systematic review of longitudinal observational studies of vitamin D status and trials of vitamin D supplementation on glycemic outcomes". They searched MEDLINE and the Cochrane Database of Systematic Reviews through February 2011. They reported in the abstract that "In post hoc analyses from eight trials among participants with normal glucose tolerance at baseline and in three small underpowered (n 32–62) trials of patients with established type 2 diabetes, there was no effect of vitamin D supplementation on glycemic outcomes. In two trials among patients with baseline glucose intolerance, vitamin D supplementation improved insulin resistance."

Collectively, these 3 systematic reviews identified 4 trials reporting results on HbA1c, all of which were included in George et al. and none of which were comparable to our study. No trials were identified that reported results of fructosamine as the outcome. Few of the studies reported were done in African, Asians, or ethnic minorities or other groups know to have poor vitamin D status at baseline.

Search for recent original trials of HbA1c or fructosamine

The search for recent original trials (using the terms ("vitamin D" OR cholecalciferol) AND (HbA1c OR A1C OR glycaemia OR glycemia OR glucose OR "glycated haemoglobin" OR "glycated hemoglobin" OR fructosamine) AND (random* OR intervention OR trial) AND ("2011/01/01"[Date - Publication] : "2013/11/30"[Date - Publication])), identified 129 publications. Of these, the following five were judged to be of direct relevance to our study results and reported results on HbA1c (detailed in Supplemental Table 1): (6-10).

Another two original trials reported on other measures of glycaemia such as fasting glucose, glucose 2 hours after an oral glucose tolerance test (11,12). Gepner *et al*(11) randomized 114 healthy, community-dwelling post-menopausal women from Madison, Wisconsin, with 25OHD concentrations >10 and <60 ng/mL (mean 31 ng/mL) at baseline to 2500 IU (62.5 µg) vitamin D_3 or placebo, daily for 4 months. No significant effect was observed on fasting glucose. Muldowney *et al*(12). Conducted two RCTs of 5, 10, or 15 μg/d of vitamin D vs placebo during winter time (22 week duration). Subjects were healthy, white skinned men and women in the south and north of Ireland. One study included 202 subjects aged 20–40 years and the other included 192 subjects aged ≥64 years. Except for the group who received 15 μg vitamin D/d, there was little increase in 25OHD after intervention (but a drop in the placebo group). 25(OH)D decreased from baseline to endpoint, except in the 15 μg/d group, who maintained the baseline concentration of ~70 nmol/L. No significant effect was seen on glucose or other endpoints (lipids results discussed below).

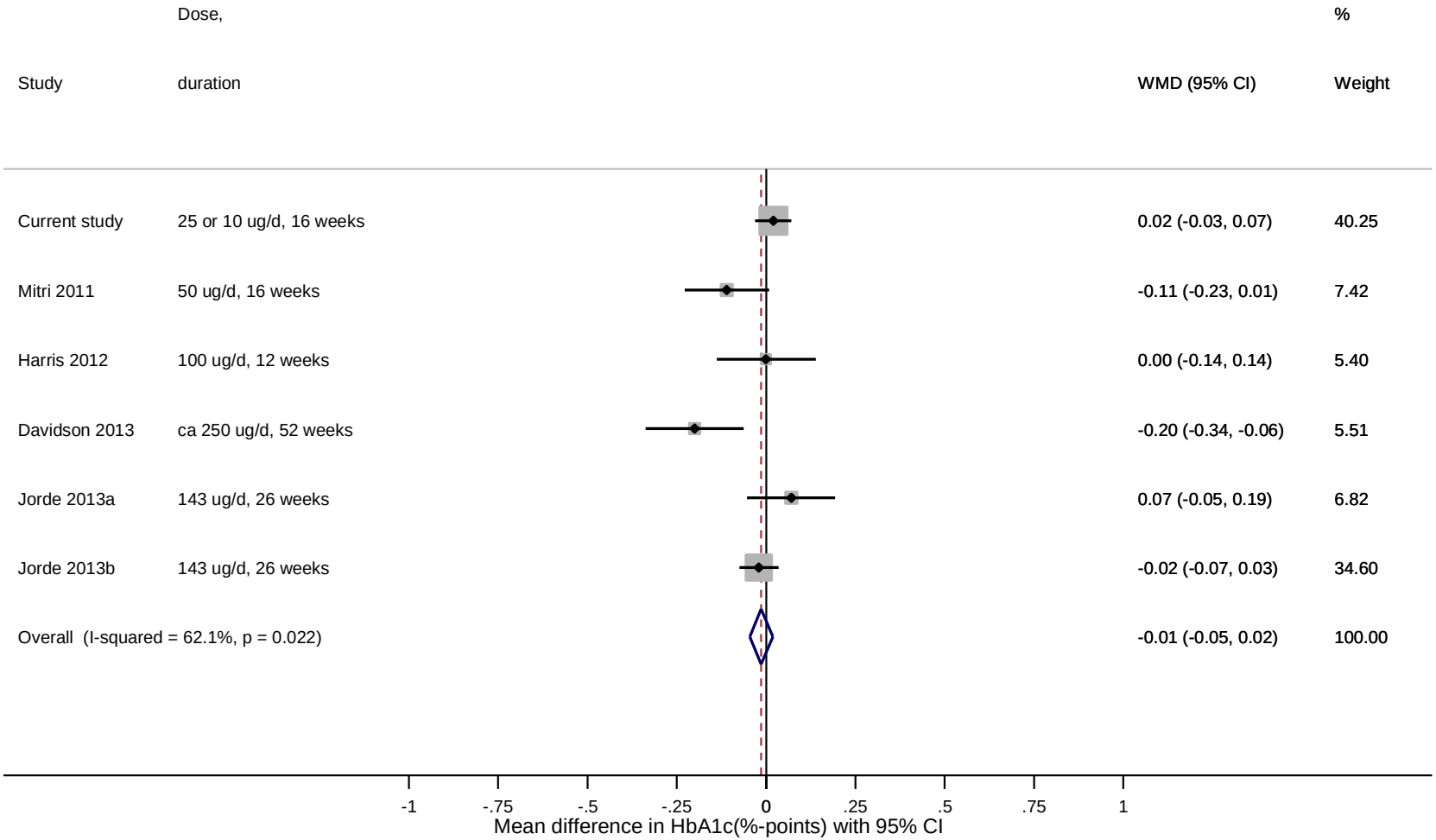
The recently published review by Autier *et al* (5) identified 16 randomised trials of vitamin D supplements reporting the effect on HbA1c, of which only six were in subject without diagnosed diabetes. Their meta-analysis showed a remarkably consistent lack of effect, even if the design and target groups were quite heterogeneous. Our search (above) and inclusion of studies considered relevant to our updated meta-analysis (below) identified all studies of relevance among those covered in Autier *et al*.

Supplemental Table 1: Recent randomised trials of oral vitamin D supplementation for at least eight weeks, reporting results on change in HbA1c in populations without a diagnosis of diabetes or other serious chronic disease.

Study	Country	Inclusion criteria	Intervention	Sample size (intervention + placebo)	Duration	Relevant endpoint reported*	Main result	Ref
Current study	Norway	Immigrants from Africa, Middle East or Asia, age 18-50y	25 or 10 µg vit D_3/d vs placebo	(75+69) + 71	16 weeks	HbA1c, fructosamine	HbA1c mean change from baseline was 0.03 %-points in the intervention group (SD:0.17) vs 0.01 (SD:0.18) for placebo; effect estimate adjusted for baseline HbA1c: 0.02 %-points (95%CI: -0.04, 0.06)	
Mitri 2011	USA	>40y, BMI 25-40 with glucose intolerance but no diabetes diagnosis (mean ~60 nM at baseline)	50 µg vit D/d vs placebo‡‡	22 + 22‡‡	16 weeks	HbA1c, FPG, 2h PG, + other measures	HbA1c changed by 0.07 in the vitamin D group and 0.18 in the placebo group (p(diff): 0.07. Significant beneficial effect on FPG, and on beta-cell function (primary outcome).	(6)
Harris 2012	USA	African Americans age >=40 y (mean ~57) BMI 25-39.9, "prediabetes"§ no diagnosis of diabetes and otherwise healthy (mean 25OHD ≈39 nM at baseline)	4,000 IU vit D_3/d (=100 µg/d) vs placebo	43+46	12 weeks	HbA1c, FPG, 2h PG	No significant effect; mean change in HbA1c in intervention group was -0.05 %-points (SEM: 0.05) vs -0.05 %-points in the placebo (SEM:0.05); effect estimate=0.00. For fasting glucose the corresponding effect estimate was +0.02 mM, and for 2h post-load glucose: +0.01 mM	(7)
Salehpour 2013	Iran	Healthy overweight or obese, premenopausal women age 18-50y (mean 25OHD 37 nM and 47 nM in the intervention and placebo groups, respectively, at baseline)	25 µg vit D_3/d vs placebo	39 + 38	12 weeks (90 d)	HbA1c, FPG, 2h PG	No statically significant effect on either outcome (HbA1c change: -1 (SD:0.5) in intervention group vs. -0.4 (SD:0.6) in placebo; effect estimate=-0.6%-points; For glucose: -0.28 – (-0.65)=0.37 mM; for 2 h post load glucose: -0.29-(-0.30)=0.01 mM. Inconsistent reporting of HbA1c results, see text.	(8)
Jorde 2013a (insulin sensitivity study) ††	Norway	Age 30-75y, 25OHD <50nM (mean 40.8 nM (SD:13.1) at baseline)	20,000 IU vit D twice per week (≈143 µg/d) vs placebo	49+45	26 weeks	HbA1c	+0.12%-points (SD: 0.32) for intervention vs. 0.05 (SD=0.29) for placebo; effect estimate (difference): 0.12-0.05=0.07%-points	(9)
Jorde 2013b ("depression study") ††	Norway	Age 30-75y, 25OHD <55nM (mean 47.5nM (SD: 15.6) at baseline)	20,000 IU twice per week (≈143 µg/d) vs placebo	119+109	26 weeks	HbA1c	-0.03% (SD: 0.20) vs. placebo: -0.01% (SD 0.22); effect estimate (difference) = =-0.02%-points	(9)
Davidson 2013	USA	Latino (~90%) or African Americans aged >=40y with prediabetes† and 25OHD <75nM (mean 25OHD was ~55nM at baseline)	Approx . 200-300 µg vit D/d **	~49 + ~49	52 weeks	HbA1c, FPG, 2h PG	HbA1c changed from 6.1 to 6.0% vs from 6.1 to 6.2% for placebo, resulting in a significant effect estimate of -0.2%-points (SD not provided, other than SD of mean at baseline: 0.4 in placebo and 0.3 in intervention); no significant effect on other measures of glycaemia	(10)

* FPG: fasting plasma glucose; 2h PG: glucose 2 hours after oral glucose tolerance test. † Complex inclusion criteria: Subjects without a diabetes diagnosis, but with at least one risk factor (increased waist circumference, family history of diabetes or history of gestational diabetes or hypertension) were screened first with HbA1c after which those with values 5.9-6.9 % underwent oral glucose tolerance test to identify subjects with prediabetes, defined as fasting plasma glucose 110–125 mg/dl or 2-h post load glucose value of 140–199 mg/dL. ‡ The numbers randomised to 5,10 and 15 µg vitamin D/d, respectively in parenthesis. § About 25% were diabetic if defined by baseline HbA1c and/or fasting glucose criteria alone. ‖ To convert glucose from mg/dl to µmol/l (mM), multiply by 0.0555. ** Complex dosing scheme calculated as (80 or 100 minus baseline serum 25OHD in ng/ml) x kg body weight x 15.7 = IU/week. For 70 kg person with baseline 25OHD of 55 nM (=22 ng/ml) this would be 63,742 or 85,722 IU/week ≈ 228-306 µg vit D/d . †† The "insulin sensitivity study" was originally published (including HbA1c) in Grimnes et al. Diabetes 2011;60:2748-2757 (including HbA1c results), while the "depression study" was originally reported by Kjærgaard et al. Br J Psychiatry 2012, but without HbA1c results. 25OHD: 25-hydroxyvitamin D. ‡‡ We excluded participants receiving calcium from our analysis to comply with our inclusion criteria.

Supplemental Figure 1. Effect of vitamin D on HbA1c in healthy adults (total n=764), data from Supplemental table 1. WMD: Weighted mean difference. Doses given are for oral vitamin D supplements, and WMD is difference from placebo group.



The results for HbA1c in Jorde *et al* (9) (two trials) and Harris et al (7) were similar to ours, while Davidson *et al*'s study (10) with extremely high doses suggested a significant beneficial effect of a magnitude that was deemed a no or little clinical significance. Mitri *et al*'s study suggested a borderline significant beneficial effect of vitamin D on HbA1c, but note that the absolute HbA1c increased in both groups (less so in the intervention group) during the 16 week intervention. Salehpour *et al* (8) reported data on HbA1c that was partially inconsistent. The point estimate of effect was -0.6%-points, which is stronger than all other studies. The authors reported a p-value of 0.06 (not significant), but this was not consistent with standard deviations and mean estimates in their table, and we decided not to include their data in the final meta-analysis. Inclusion of Salehpour *et al*'s data would lead to more marked heterogeneity between studies, but the pooled estimate was little changed (-0.02, 95%CI: -0.05, 0.02). We contacted the authors but we were unfortunately not able to obtain consistent estimates allowing us to confidently include the data in the meta-analysis. While the test for heterogeneity was statistically significant, a the magnitude of effects estimated in the different studies were judged to be of very limited clinical relevance in all cases. We also run a sensitivity analysis by employing a random effects model. This resulted in relatively smaller weights for the two large studies (the current and Jorde 2013b), but the weighted mean difference estimate was essentially unchanged (-0.03, 95% CI: -0.09, 0.03). Despite some differences in inclusion criteria between the recently published meta-analysis by Autier et al. (5) and ours, the main result was comparable except an apparently lesser degree of statistical heterogeneity in their analysis. This seems paradoxical because we made an attempt to include studies with similar design and target groups. Among 23 trials of vitamin D supplements and the effect of fasting plasma glucose identified by Autier et al (5), all but one were not statistically significant but no formal meta-analysis with forrest plot were presented. In summary, the available evidence suggest no significant effect of vitamin D on HbA1c in adults with low levels of 25OHD but otherwise without diagnoses of chronic diseases. Any potential effect is likely to very small.

Serum lipids

Search 2 gave 13 hits in PubMed on October 30, 2013, and the following two were the most updated and relevant: (13,14). A search in the Cochrane database did not provide any additional relevant reviews.

Elamin *et al* (13) systematically reviewed the literature for randomised controlled trials of vitamin D up to the end of August 2010. The review was primarily concerned with death and cardiovascular diseases as the endpoint, but included lipids and glucose as a secondary outcome. Among a total of 51 trials with moderate quality, they pooled results from 12 trials reporting on serum lipids. The weighted mean difference was 0.00 (95% CI: -0.06, 0.07) for total cholesterol (12 studies), -0.09 (95% CI: -0.24, 0.07) for LDL-cholesterol (11 studies), and 0.06 (95%CI: -0.11, 0.24) for HLD-cholesterol (12 studies). The I-squared as an estimate of between study heterogeneity was 28, 90, and 99% for total-, LDL-, and HDL-cholesterol, respectively.

Wang *et al* (14) systematically searched the literature up to the end of October 2011. They included in the meta-analysis 10-11 RCTs (7-11 studies, depending on lipid fraction), all with differences in design or inclusion criteria from our study (for instance including patients with diagnoses such as type 2 diabetes, interventions in addition to vitamin D such as weight loss or calcium supplement, and including 1-hydroxyvitamin D supplements). In their table 3 they reported a weighted mean difference in total cholesterol that significantly increased in those who received vitamin D supplements (3.23 mg/dl, 95%CI: 0.55, 5.90; or 0.08 mmol/l (95% CI: 0.01, 0.15)). However this result was referred to in the text and abstract as if it was LDL-cholesterol. Our attempt to contact the corresponding author by email to clarify this inconsistency was not successful.

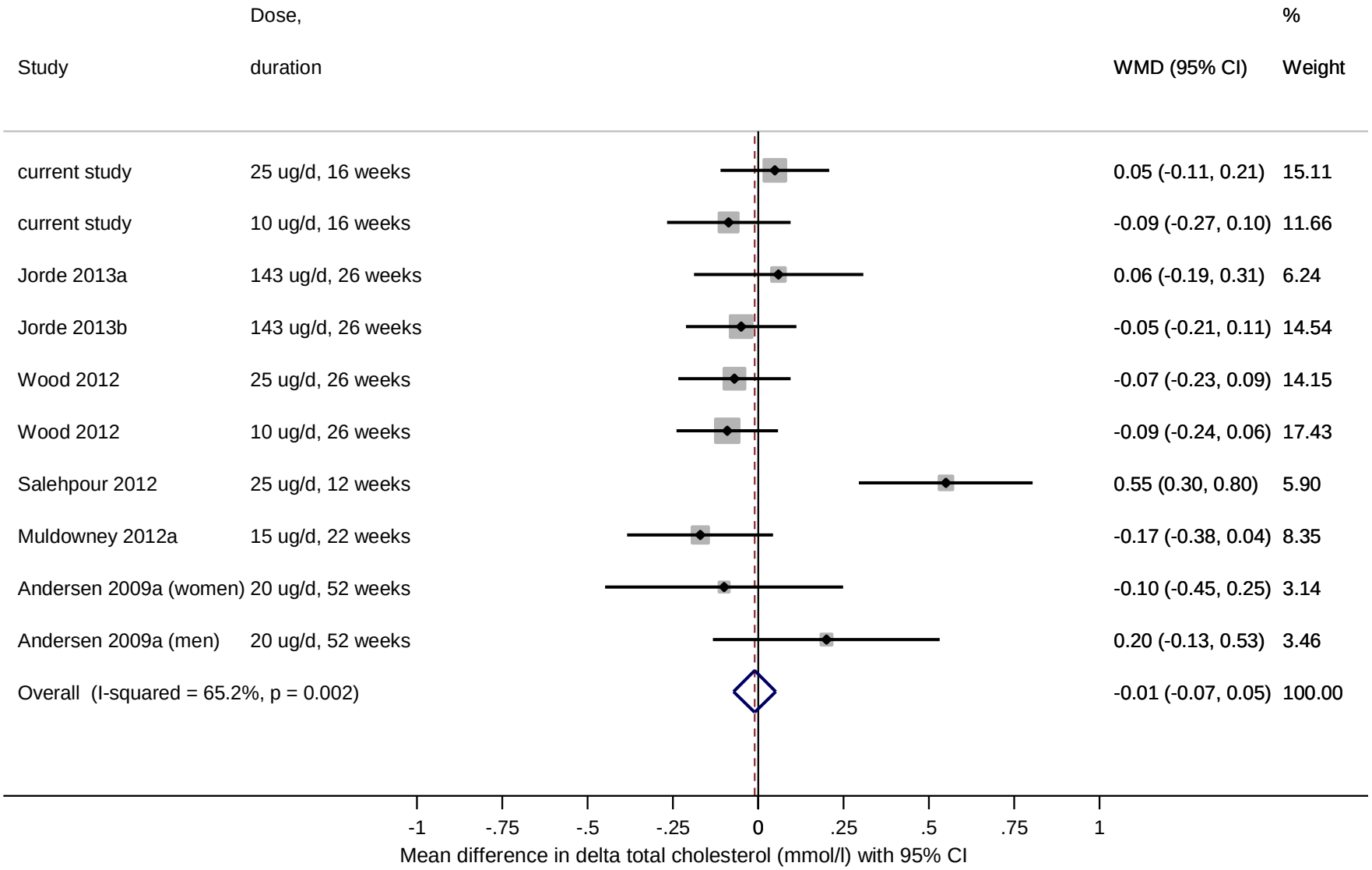
If assuming that results in the figures were correct, the pooled effect estimates showed a slightly but significantly increased total cholesterol in those who received vitamin D supplements, and no significant effect on LDL- or HDL-cholesterol.

Results from the meta-analyses conducted by Autier *et al* (5) concluded that nearly all trials, vitamin D supplementation did not affect concentrations of blood lipids (total cholesterol, LDL-cholesterol, HDL-cholesterol and triglycerides).

Several of the studies who reported on HbA1c or glucose also showed data for lipids, and we informally summarised result for total cholesterol from these and other studies(15,16) with similar design to ours, and Salehpour et al reported results from serum lipids in a separate publication in 2012 (17) (Supplemental Figure 2). Note that Andersen et al did not report mean difference between follow-up and baseline, only median levels at each point, including in the baseline data those who were lost to follow-up at 12 months (about 26%). They also

included a group randomized to 10 µg vitamin D/d not included in the meta-analysis because the data reported were not strictly comparable to those from the other studies. We contacted the authors but they were unfortunately not able to provide the comparable estimates at present. Salehpour *et al*'s results(17) were different from the other studies in that they found significantly increased total cholesterol after vitamin D intervention. In summary, the lack of effect of vitamin D on total cholesterol in our study was clearly consistent with these other studies. Any potential effect would be very weak. The other studies also reported results on LDL-cholesterol and HDL-cholesterol and similarly did not find any significant effect of vitamin D, with the exception of Salehpour *et al* (17) who found significant increase in both LDL-cholesterol and HDL-cholesterol (data not shown).

Supplemental Figure 2. Effect of vitamin D on serum total cholesterol in healthy adults (total n=1199). WMD: Weighted mean difference.



Body mass index (BMI)

Search 3 gave 34 hits in PubMed on October 30, 2013. None of the identified studies were systematic reviews of randomized trials of effect of vitamin D supplementation on BMI, but we assessed Renzaho *et al* and Saneei *et al*, both of which focussed on observational studies(18,19). They both concluded that there is a need for randomized controlled trials, and from their reference lists we identified a review of RCTs by Soares *et al*(20) as relevant for our purpose. A search in the Cochrane database did not provide any additional relevant reviews.

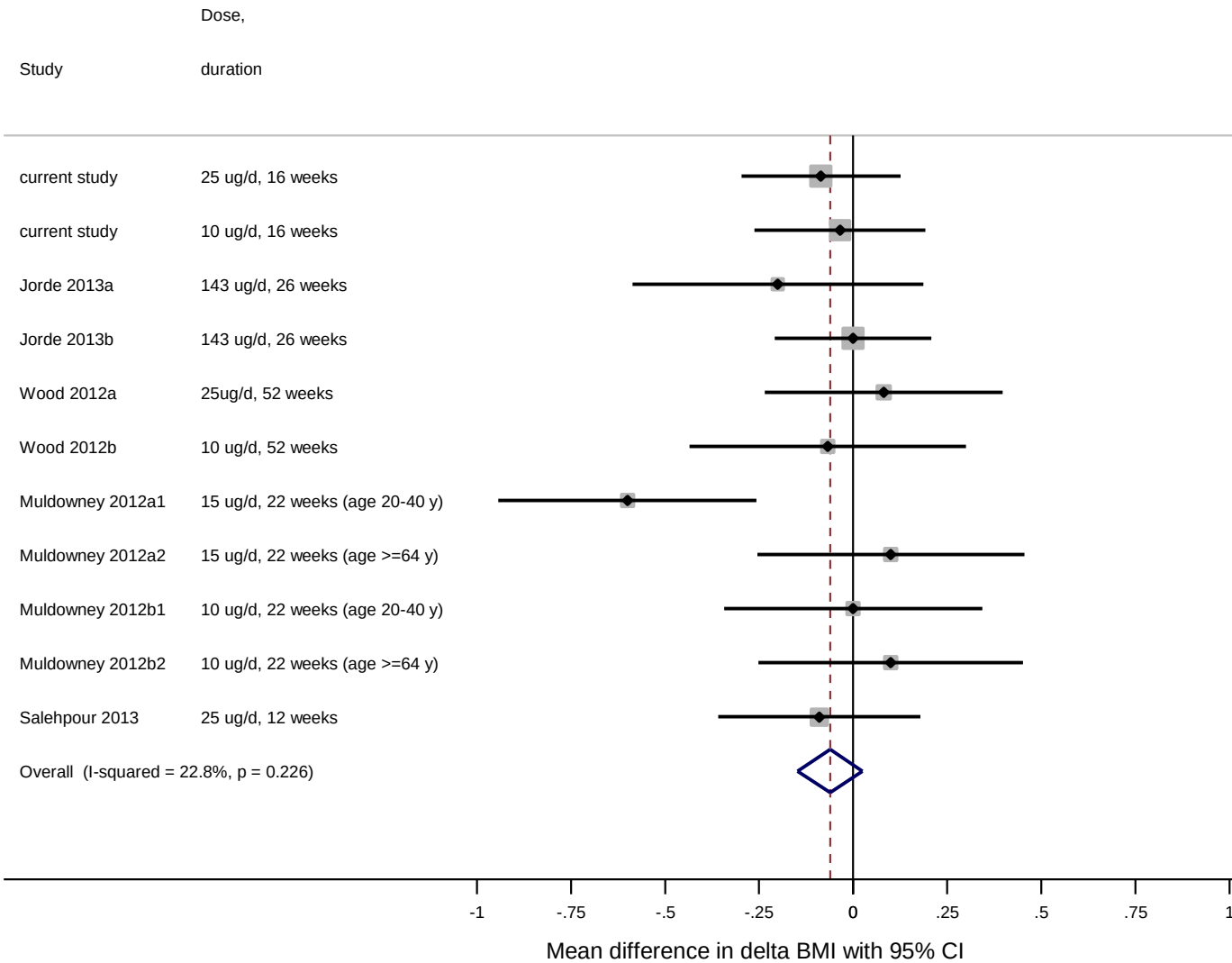
Soares *et al*(20) reviewed the literature from year 2000 to early 2011 in several databases, and focused primarily on RCTs randomizing participants to either vitamin D plus calcium or only vitamin D, and reporting effects on adiposity outcomes including BMI. The primary focus was effects during treatment of obesity (which is in contrast to our focus). Soares *et al* identified 15 trials for their analysis, of which 7 gave vitamin D alone. They concluded that "Current evidence from RCTs did not consistently support the contention that calcium and vitamin D accelerated weight or fat loss in obesity".

Among the trials reviewed by Soares *et al*, only one conformed to the strict criteria we outlined above (in the section on HbA1c) for comparability to our study, namely von Hurst *et al* (21). They randomised 81 women of South Asian origin living in New Zealand aged 23-68 years to 6 months of supplementation with 100 µg/d of vitamin D or placebo. Inclusion criteria were serum 25OHD <50nM and insulin resistance (defined as homeostasis model assessment 1 >1.93, which is approx. the upper quartile in this population) and/or triacylglycerol:HDL-cholesterol ratio >=3. Data on BMI was not shown, but the authors stated that there was no significant effect on BMI.

The 12 trials which include in systematic review by Autier *et al* (5), only results of the Women's Health Initiative (WHI) study showed significant, but small, weight loss associated with supplementation (mean loss of 0.13 kg, 95% CI 0.05–0.21).

In addition, most of the newer trials identified for HbA1c or glucose or serum lipids (discussed above) also included data on BMI. Unpublished data on BMI from Wood et al (16) were kindly obtained from H.E. MacDonald, University of Aberdeen. We informally summarised these together with our own results (Supplemental Figure 3). With the exception of one subgroup in Muldowney *et al* (12) and the overall results from other studies were quite consistent with our results suggesting no effect on BMI. Note that in Muldowney et al, the ANOVA test for variation in mean across four groups (placebo, 5 µg/d, 10 µg/d and 15 µg/d) was not significant (p=0.32) (12). It seems likely that any potential effect would be of small magnitude.

Supplemental Figure 3. Effect of vitamin D supplements on BMI in generally healthy adults. Studies with two doses were included with the same control (placebo) group twice and is therefore not strictly independent (total n=1293). Standard deviations for Muldowney were imputed as described in the text. WMD=Weighted mean difference compared to placebo.



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