Paper SD-012

The Use of SAS® in Meta-Analysis

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Abstract:

We will illustrate the dichotomy between original data and published data in the context of usability for Meta-Analysis. We will show how to use SAS® for these two cases and suggest possible solutions to overcoming the hindrances caused by the use of published data. Publication bias, Bayesian methods for Meta-Analysis, and other related topics will be discussed.

Introduction:

Meta-Analysis is a collection of techniques where the results of two or more independent studies are statistically combined to yield an overall answer to a question of interest. This method of combining data, allows us to increase the power of the analysis, when compared to the individual studies on their own. It must also be observed that the variance may also increase in the combined studies' as compared to the variance from the sample. This combination of data seems a natural process when we do not have significant sample size in our study. Bayesian methods have a natural application to meta-analysis, in that the resampling process, specifically Gibbs Resampling Algorithm, allows for a convenient method of replacing missing data, which is a common issue in meta-analysis studies.

The combining of data for a Meta-analysis is a beneficial method for looking at effects that may at times be missed in smaller data sets so that accordingly, researchers may consider small treatment differences, which are not noticeable without the power of a larger data set. According to Glass (1976) meta-analysis is "…the statistical analysis of a large collection of analysis results from individual studies for the purpose of integrating the findings. It connotes a rigorous alternative to the casual, narrative discussions of research studies which typify our attempts to make sense of a large volume of research literature." This allows us to pursue the idea that meta-analysis has a larger analytic component than just descriptive findings and as such, a primary criterion for inclusion of studies is that there is a consistency in the studies that may not be readily available to the casual reader. This consistency is that the tests essentially test the same hypothesis or compare the same treatments, but need not be literally identical. Subjectivity also enters the meta-analysis when deciding on which studies to include or exclude. This is based on the specificity of the research question being asked. Statistical benefits include high statistical power, high precision for small treatment differences, and various types of endpoints such as group means, odds ratios, and hazard ratios. (Piantadosi, 530) According to Piantadosi, (531), there are basic steps required for a formal research meta-analysis, these include: (1) formulation of a purpose and specification of an outcome for the analysis, (2) identification of relevant studies, (3) establishing inclusion and exclusion criteria for studies, (4) data abstraction and acquisition, (5) data analysis, (6) dissemination of results and conclusions. *Weaknesses in any of these steps can compromise the validity and strength of the meta-analysis.* Additional weaknesses of meta-analysis is that they are not experimental designs and thus their validity relies on

retrieving existing data and on the quality and rigor of the studies that generated the data. Publication bias may also exist due to the fact that positive trial results are more likely to appear in the published literature as compared to negative ones, thus meta-analysis can overestimate the benefits of treatment. This however is not a great concern when it comes to disease effects in a population, for example, where large benefits can occur based on small treatment effects.

To summarize thus far, we claim that the aim of a meta-analysis is to provide a combined analysis of the studies that indicates the overall strength of the evidence for a beneficial effect of the treatment of the study. Further expansion of the estimands of common interest include the differences in probabilities, $(p_{1j} - p_{0j})$, the probability or risk ratio, $\frac{p_{1j}}{p_{0j}}$, and the odds ratios, $p_j = \frac{p_{1j}/(1-p_{1j})}{p_{0j}/(1-p_{0j})}$ each of these are possible estimates for parameters for each study.

One thing to note, for example, is the fact that if the posterior distribution is close to normality even for relatively small sample sizes, we can concentrate on inference for the natural logarithm, (ln), of the odds ratio which we label, $\theta_j = \log(\rho_j)$, or more commonly called a transformation of the data. We state this to bring to the attention of the researcher that basic statistical methods are rigorous in the meta-analysis context.
It is felt at this time that the focus for our protocol methods be restated. "*Our focus is the estimation of meaningful and beneficial parameters and for this objective there appear to be three possibilities, accepting the overarching assumption that the studies in the meta-analysis, are comparable in some broad sense*".
From Gelman, et al, we see that the *first possibility* is that we view the studies as mirror images of each other, in the sense that we regard the subjects in all studies as independent samples from a common population, with the same outcome measure. The second *possibility*, is that the studies are so dissimilar that the results of any one study provides no information about the results of any of the others. The *third possibility* in a more general sense is that we regard the studies as exchangeable but not necessarily identical or dissimilar; in other words we allow differences from study to study, but such that the differences are not expected a priori to have predictable effects favoring one study over another. This leads us to establish a starting point for this type of analysis by pointing out that the first potential estimand of a meta-analysis, or a hierarchically structured problem, is 'the mean of the distribution of effect sizes, since this represents the overall 'average' effect across all studies that could be regarded as exchangeable with the observed studies and the effect size in another, comparable (exchangeable) unobserved study' (Gelman, 148). This leads us to the principles of Bayesian methodology as applied to meta-analysis.

In its basic premise, Bayesian methods comprise a philosophy that can be applicable to an infinite number of problems. *Simply stated, to be a Bayesian disciple, means to think in a manner that allows for what you knew (or proceedings of) yesterday, to be ascribed to the situations of today and, how the combination of yesterday and today, will affect tomorrow. Formally, prior information will be used to gauge the likelihood of current*

information to further the accuracy of the probability of future information. This allows for the use of prior and current data to be cohesively used in the evaluation of accurate and reliable estimates, which is clearly a desirable trait for any Meta-Analysis.

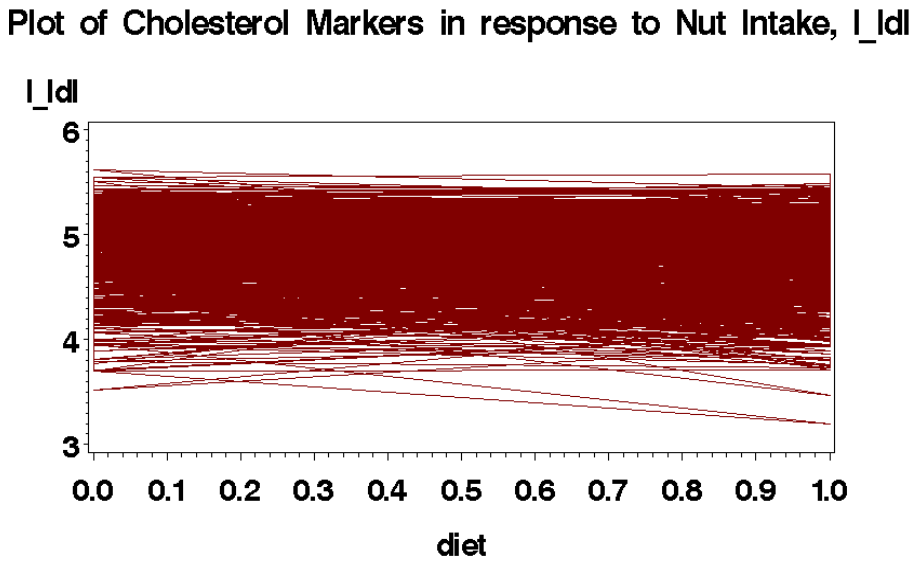
We will be using data compiled from both original and published, (secondary), sources. These take the form of, data looking at the consumption of nuts and the effects on cholesterol levels (ldl, hdl), as original data, and also the effects of exposure to passive smoking and development of Multiple Sclerosis, as secondary data. The first is discussed primarily through the body of this paper, the second may be found in Appendix C.

The Effect of Consumption of Nuts on Cholesterol Markers:

The effects on cholesterol levels from the consumption of nuts, has been examined at length by the investigators of the Adventist Health Study of Loma Linda University, Harvard Med, and Stanford Prevention Research Center, among others. Our data is a compilation of 25 different studies with a combined subject size of N=1284. We will be examining the use of frequentist and Bayesian methods for the evaluation of our meta-analysis data. Concentrating on finding an accurate method for identifying parameters for the working model, is also important here. We will go from a full model to a working one with explanation as to the role certain methods have in this process. Analysis may have a graphical and/or analytical component, yet it is often useful in Meta-Analysis to consider both.

Analysis:

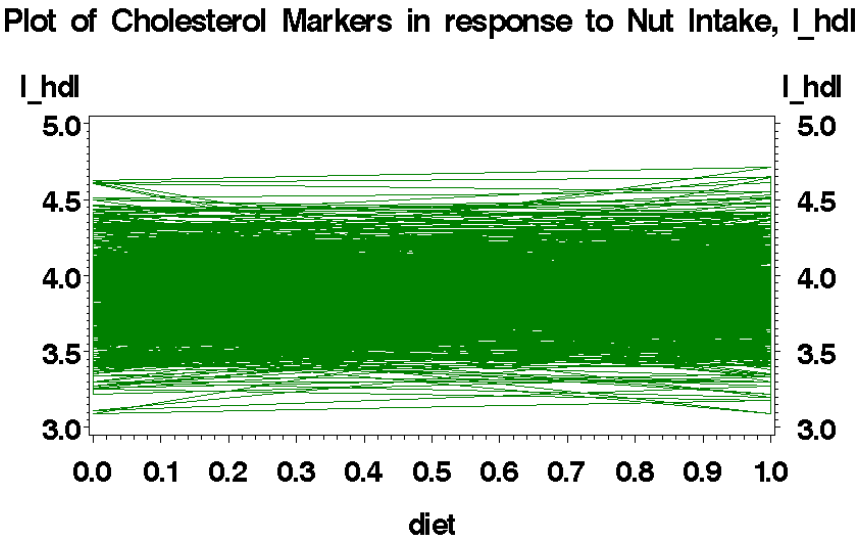
Examination of trends should be the initial stage this can be done in any number of ways, from descriptive information to graphical representation.



The two spaghetti plots, above and below, are fairly inconclusive for showing trend due to the large number of subjects (N=1284) from the combined data. It is recommended,

although not shown here, that spaghetti plots be viewed on a study to study basis so that comparison may be made between the studies.

Spaghetti Plot for l_hdl:

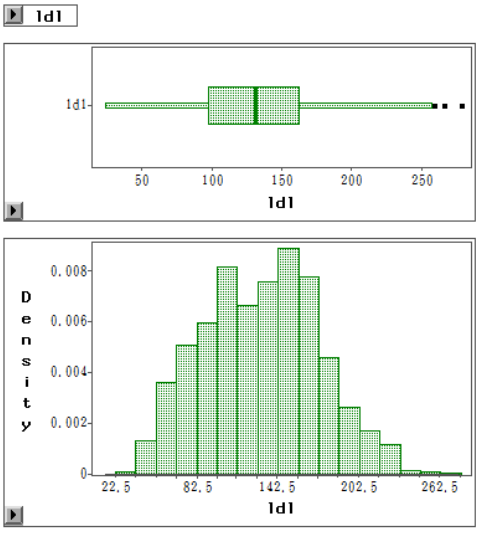


Description of Studies Included:

Study	Nut	Study design	N	Number Female	Number Male	Degree of dietary control	dx	Reference diet	Mean nut amount given	SE of nut amount	Mean age	Age SE	Mean BMI	BMI SE	Mean LDL-C	SE LDL-C
Abbey	Almond,	Consec	16	0	16	med	normal	AA	84.0	0.0	41.4	2.3	27.3	0.7	138.6	9.2
Almario	Walnut	Consec	18	13	5	med	normal	AA, Low SF	51.9	2.4	60.0	1.9	29.0	1.2	138.2	11.0
Alper	Peanu	Conse	15	7	8	med	normal	AA	89.2	3.0	32.9	2.4	23.3	0.5	99.6	6.8
Chisol	Waln	Cross	16	0	16	lo	hyper	Low SF	77.9	2.9	45.3	2.1	27.3	0.7	159.9	6.1
Colquhoun	Mac	Cross	14	7	7	med	normal	Low fat	54.0	4.9	46.4	2.9	.	.	142.7	9.1
Curb	Mac	Cross	30	15	15	hi	normal	AA	.	.	35.3	1.6	23.1	0.5	130.4	4.7
Durak	Hazel	Conse	30	12	18	lo	normal		68.6	1.7	.	.	.	.	75.7	3.1
Edwar	Pistac	Cross	10	6	4	med	hyper	AA	59.9	5.2	45.9	2.9	.	.	178.1	16.6
Garg	Mac	Conse	17	0	17	med	hyper	AA	48.4	2.5	53.9	1.6	26.2	0.8	173.6	4.3
Hyson	Almo	Conse	22	12	10	med	normal	Low SF	65.5	4.8	43.5	1.8	23.6	0.7	135.1	4.2
Iwamoto	Walnut	Cross	40	20	20	hi	normal	Low fat Asian	51.0	1.0	23.7	0.7	21.4	0.4	79.4	3.6
Jenkins	Hi Alm, Lo Alm	Cross	27	12	15	med	hyper	Low SF	55.1	3.1	63.9	1.8	25.5	0.6	163.1	4.9
KrisEt	Pearu	Cross	22	13	9	hi	normal	AA	.	.	34.0	1.8	23.5	0.4	137.1	6.5
Lovejoyl	Almond	Consec	20	10	10	med	normal	AA	100	0.0	25.1	1.5	22.9	0.5	116.7	5.2
Lovejoy2	HF Alm, LF Alm	Cross	30	17	13	hi	normal	Low SF, Med	99.9	2.0	53.9	1.9	33.1	1.0	101.2	4.5
Morg	Pecan	Para	19	15	4	lo	normal	AA	68.0	0.0	37.4	4.3	24.4	1.5	113.6	4.9
Most	Almo	Cross	24	16	8		normal	AA	87.0	3.4	46.3	1.3	30.3	1.1	122.9	6.1
Rajaram	Pecan	Cross	23	9	14	hi	normal	Low SF	85.3	1.5	38.0	1.9	25.5	1.1	117.8	4.5
Ros_n	Waln	Cross	20	12	8	med	hyper	Med	55.0	2.1	55.1	2.7	26.5	0.6	179.5	4.0
Ros_o	Alm/Wal	Cross	18	9	9	med	hyper	Med	.	.	55.2	3.2	25.7	0.5	181.5	5.4
Sabat	Waln	Cross	18	0	18	hi	normal	Low SF	79.4	3.2	30.2	1.5	23.8	0.7	112.3	3.8
SabatA	Hi Alm, Lo Alm	Cross	25	11	14	hi	normal	Low SF	64.8	3.6	40.9	2.6	25.0	0.7	144.5	8.5
Sherid	Pistac	Cross	15	4	11				.	.	59.7	2.9	27.7	0.9	163.1	9.3
Spiller	Almond	Para	45	32	13	med	hyper	AA, Med	100	0.0	51.1	2.8	25.3	0.9	175.9	5.7
Zambon	Walnut	Cross	49	23	26	med	hyper	Med	46.2	0.7	55.8	1.5	26.5	0.4	183.3	3.9

Furthering our analysis, we suggest that correlation and simple regression methods be used to identify simple effects from the data. Things such as normality and interaction should be tested at this stage. This aids in the identification of heterogeneity/homogeneity, which is of the utmost importance in Meta-Analysis. For example, we see that the cholesterol markers HDL and LDL are not normally distributed and must be transformed using a natural log transformation. This transformation is an analytical contribution that may be viewed graphically. As Follows: *using PROC Insight:*

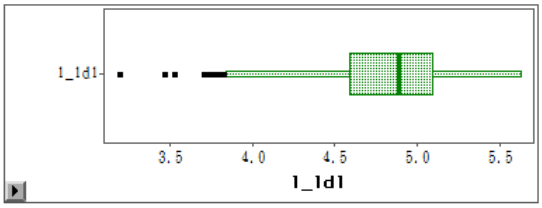
Normality: Analytical and Graphical Analysis:

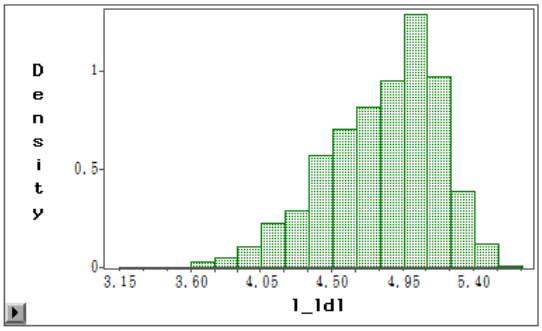


Tests for Normality		
Test Statistic	Value	p-value
Shapiro-Wilk	0.991900	0.0000
Kolmogorov-Smirnov	0.036087	<.0100
Cramer-von Mises	0.422489	<.0050
Anderson-Darling	2.585803	<.0050

95% Confidence Intervals			
Parameter	Estimate	LCL	UCL
Mean	130.4615	128.0908	132.8323
Std Dev	43.2849	41.6725	45.0280
Variance	1873.5807	1736.5966	2027.5221

l_ldl



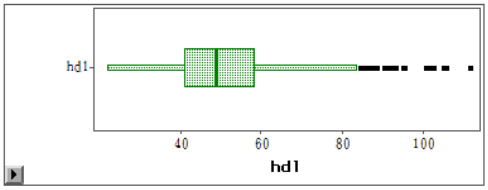


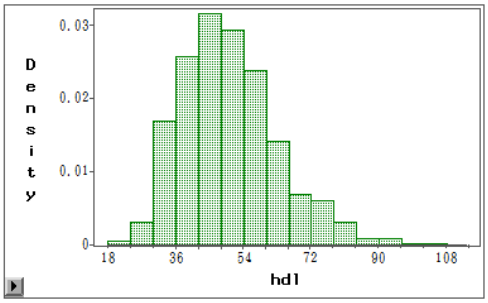
Tests for Normality		
Test Statistic	Value	p-value
Shapiro-Wilk	0.967165	0.0000
Kolmogorov-Smirnov	0.079443	<.0100
Cramer-von Mises	2.060917	<.0050
Anderson-Darling	12.01523	<.0050

95% Confidence Intervals			
Parameter	Estimate	LCL	UCL
Mean	4.8092	4.7891	4.8293
Std Dev	0.3670	0.3533	0.3817
Variance	0.1347	0.1248	0.1457

In comparing the un-transformed with the transformed ldl we see there is no change in normality, yet we keep the transformation in our model since it is useful at times, to rescale the data. We continue this by showing the difference between the untransformed and transformed data for hdl.

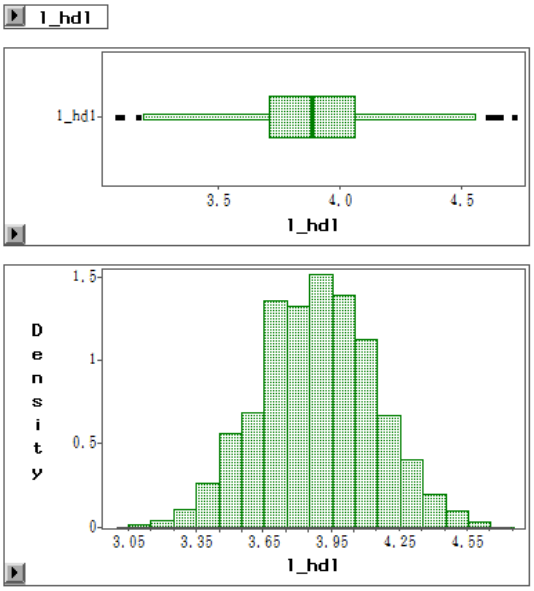
hdl





95% Confidence Intervals			
Parameter	Estimate	LCL	UCL
Mean	50.3061	49.5692	51.0429
Std Dev	13.4539	12.9527	13.9957
Variance	181.0062	167.7722	195.8785

Tests for Normality		
Test Statistic	Value	p-value
Shapiro-Wilk	0.963502	0.0000
Kolmogorov-Smirnov	0.063783	<.0100
Cramer-von Mises	1.485517	<.0050
Anderson-Darling	9.547765	<.0050



95% Confidence Intervals			
Parameter	Estimate	LCL	UCL
Mean	3.8837	3.8694	3.8981
Std Dev	0.2622	0.2524	0.2728
Variance	0.0688	0.0637	0.0744

Tests for Normality		
Test Statistic	Value	p-value
Shapiro-Wilk	0.999043	0.7568
Kolmogorov-Smirnov	0.017192	>.1500
Cramer-von Mises	0.035151	>.2500
Anderson-Darling	0.244242	>.2500

This is a good example as to how transformation is not always the best method for normalizing data. We, however, use this in the model to keep scale consistent throughout. Having looked at the effects on the model when we use the l_HDL compared to the HDL, the effects are consistent.

We now move on to the regression part of our analysis and give the following models with parameter estimation and support.

Full Model:

$$\begin{bmatrix} l\_ldl \\ l\_hdl \end{bmatrix} = \begin{bmatrix} \beta_0 \\ \alpha_0 \end{bmatrix} + \begin{bmatrix} \beta_1 \\ \alpha_1 \end{bmatrix}(diet) + \begin{bmatrix} \beta_2 \\ \alpha_2 \end{bmatrix}(age) + \begin{bmatrix} \beta_3 \\ \alpha_3 \end{bmatrix}(bmi) + \begin{bmatrix} \beta_4 \\ \alpha_4 \end{bmatrix}(nut\_amt) + \ldots + \begin{bmatrix} \beta_n \\ \alpha_n \end{bmatrix}(X_n) + \varepsilon$$

Reduced Model(s):

$$[l\_ldl] = \beta_0 + \beta_1(diet) + \beta_2(age) + \beta_3(diet \cdot bmi)$$
$$[l\_hdl] = \alpha_0 + \alpha_3(bmi) + \alpha_4(diet \cdot bmi)$$

Working Model(s):

$$[l\_ldl] = 4.3818 - 0.1876(diet) + 0.009(age) + 0.0027(diet \cdot bmi)$$
$$[l\_hdl] = 4.3747 - 0.02282(bmi) - 0.0023(diet \cdot bmi)$$

This is our suggested working model, from which true effects on the cholesterol markers may be seen. The interaction of diet and BMI is shown to be the only significant marker that with effect on the cholesterol markers. It is interesting how certain variables effect one marker positively and the other negatively. This is found to be consistent with the literature on the subject.

(For sake of example and since the procedure BGENMOD has a hard time examining interaction effects a simple model is shown below for comparison sake, this is corrected in SAS 9.2® as PROC GENMOD, is more robust in Bayesian methods than the experimental predecessor, based on our evaluation).

The following table summarizes the parameter estimations and supporting test statistics for a simple regression model of:

$$\begin{bmatrix} l\_ldl \\ l\_hdl \end{bmatrix} = \begin{bmatrix} \beta_0 \\ \alpha_0 \end{bmatrix} + \begin{bmatrix} \beta_1 \\ \alpha_1 \end{bmatrix} (diet)$$

	Model	SAS FREQ, Est.	P-Value	SAS BAYES, Est.	Credible Interval	WIN-BUGS, Est.	Credible Interval
Nut Study*		Proc Glimmix		Proc BGenMod			
	L_ldl=diet	-0.0652 (0.0475)	0.0014	-0.0654 (0.020)	(-0.1056, -0.0239)	-0.065	(-0.1048,-0.0247)
	L_hdl= diet	-0.0014 (0.0617)	0.9222	-0.0012 (0.015)	(-0.0298, 0.0280)	-0.0013	(-0.0310, 0.0276)

*Bayesian Methods such as autocorrelation and density plots can be seen in the handout provided.
**We do this more for verification of method rather than analysis of the model. The above working model and test statistics can also be seen below.

SAS® code examples and Test Statistics:

Solutions for $[l\_ldl] = 4.3818 - 0.1876(diet) + 0.009(age) + 0.0027(diet \cdot bmi)$

```
proc glimmix data=nut3;
class study sidn treatment;
model l_ldl=diet age bmi diet*age diet*bmi age*bmi /solution;
random study sidn(study) treatment(study);
run;
```

Solutions for Fixed Effects

Effect	Estimate	Standard Error	DF	t Value	Pr > |t|
Intercept	4.3818	0.2425	18	18.07	<.0001
diet	-0.1876	0.04745	514	-3.95	<.0001
AGE	0.009029	0.005063	514	1.78	0.0751
BMI	0.01316	0.009109	514	1.44	0.1492
diet*AGE	0.000685	0.000563	514	1.22	0.2247
diet*BMI	0.002716	0.001595	514	1.70	0.0892
AGE*BMI	-0.00020	0.000189	514	-1.08	0.2811

Solutions for $[l\_hdl] = 4.3747 - 0.02282(bmi) - 0.0023(diet \cdot bmi)$

```
proc glimmix data=nut3;
class study sidn treatment;
model l_hdl=diet age bmi diet*age diet*bmi age*bmi /solution;
random study sidn(study) treatment(study);
run;
```

Solutions for Fixed Effects

Effect	Estimate	Standard Error	DF	t Value	Pr > |t|
Intercept	4.3747	0.2451	18	17.85	<.0001
diet	0.05437	0.03617	514	1.50	0.1334
AGE	-0.00233	0.005216	514	-0.45	0.6548
BMI	-0.02282	0.009449	514	-2.41	0.0161
diet*AGE	0.000067	0.000431	514	0.16	0.8765
diet*BMI	-0.00228	0.001232	514	-1.85	0.0648
AGE*BMI	0.000187	0.000196	514	0.95	0.3406

Conclusion:

From the working model we do see that there is a statistically significant biological effect on cholesterol levels from consumption of nuts. (Other factors include BMI and age, as far as influencing cholesterol levels.) For the purposes of this paper, we are more interested in the overall method for combining and evaluating the data, than the scientific interpretation. To revisit what has been presented, we collected original data from unpublished sources; we combined the data based on similar variables and then proceed through evaluating overall and within effects. These effects should include, tests of homo-/heterogeneity, basic descriptive statistics, correlation between variables, and finally into regression analysis.

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8. http://www.mrc-bsu.cam.ac.uk/bugs/winbugs/winbugs-demo.pdf
9. WinBUGS. “Example Vol.1,2,3”©
10. SAS Documentation. “Bayesian”©
11. Code Notes from: Gelman, Berry, Carlin, and Ghamsary

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Note from Author:
This paper represents a compilation of projects done while at Loma Linda University and at Georgia Southern University respectively. Specifically the 'Nut Consumption and Cholesterol Effects' was done while at Loma Linda University, whereas 'Passive Smoking and Multiple Sclerosis' was done while at Georgia Southern University. This paper is not for reporting of scientific results that may be identified throughout the analysis; rather it is to serve as a working example of the statistical methodology involved in a thorough Meta-Analysis and to touch on a few of the various techniques available to the researcher. Thank you. ~RCB

Some Useful Books on the Topics Presented:
Literature Search and Book List:

Author	Title	Genre
Mullen, Brian	**Advanced BASIC Meta-Analysis**	**Meta-Analysis**
Ghamsary, Mahmood	**Bayesian Meta-Analysis via Gibbs Resampling**	**Bayesian and Meta**
Broemeling, Lyle	Bayesian Analysis of Linear Models	Bayesian/Linear Mod
Gamerman, Dani	Markov Chain Monte Carlo: Stochastic…	Bayesian/Simulation
Hunter, Douglas	Political/Military Applications of Bayesian…	Bayesian Apps
French, S.	The Practice of Bayesian Analysis	Bayesian
Dey, Dipak	Practical Nonparametric and Semi..Bayes…	Bayesian Apps
Press, S. James	**Bayesian Statistics: Principle,….**	**Bayesian Resource**
Schmitt, Samuel	Measuring Uncertainty…an elementary…	Bayesian
Denison, D.G.T.	Bayesian Methods for Nonlinear Class….	Bayesian/NonlinearRe
Bernardo, J.M.	**Bayesian Theory**	**Bayesian Resource**
Corfield, David	Foundations of Bayesianism	Bayesian Apps
SAS	SAS Language: Reference	Programming
Arthur, Bennet, etc	**Conducting Meta-Analysis Using SAS**	**Meta-Analysis/Prog.**