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Effect Modification, Confounding, Hazard Ratio, Distribution Analysis, and Probability of Non-normal Data for Head Neck Cancer

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Abstract

Interaction methods for effect modification and confounding with the O and Oc statistics that are asymptotic chi-square and a PROC IML algorithm with PROC MIXED Agravat (2011) combined with survival and probability analysis for head neck cancer are demonstrated. In support of these new interaction analysis methods are C stat, and power. A new hazard logit for survival statistics for head neck cancer due to nonsmoking by race including distribution analysis, hazard ratios, and calculations of probability are shown. Statistics based on probability, independence, and algorithms are important when data are non-normal, linearity is not present, homogeneity assumption for standard error is not met, and when no time point are given. New methods for Z scores and risks based on logits are introduced.

Keywords: Asymptotic Chi-square, logit, effect modification, confounding, non-normal, hazard ratio

1 Introduction

The interaction, probability, and hazard ratios of head and neck cancer epidemiology from the INHANCE study are analyzed for statistics such as effect modification p values, confounding p values, including and distribution analysis of no smokers vs. no drinking by race for head neck cancer. Normally, (SAS)[®] software automatically produces output intended for large samples and fixed effects data that may apply to this study. In this case, the data is non-normal for cases of head neck cancer due to non-smoking by level of race because the Shapiro-Wilks $P < 0.0003$ indicates non-normality for cases of head neck cancer. The confounding issue also is normally resolved by analyzing if there is a difference of 10 percent or more among strata for beta estimates or risk estimates, however in this analysis a new parametric technique with PROC IML and PROC MIXED Agravat (2011) Verbeke and Molenbergs (2000) with an asymptotic chi-square calculated by the new O statistics, and including the F-statistics and p value, that is utilized, for multivariate analysis, is demonstrated and the possible synergistic interaction between effect modification and confounding is demonstrated. The novel use of a survival function is implemented to obtain beta estimates.

Hazard ratios also require a proportional hazards model and assumptions require linearity and homogeneous standard error. The new hazard ratio method can produce statistics for situations when the beta estimates are obtained from a new parametric technique. The subsequent hazard ratio is also derived showing the involvement of the independence assumption and a proof Agravat (2011) as well as new formulae. This method also shows that corresponding probability distribution values are not limited by the condition when there is no probability available where values are undefined. Since errors exist, and probability distributions exist, and also the independence assumption is satisfied, one can be more confident of the related outcome survival statistic. Probability and conditional probability of event with time is derived showing that the conditional probability algorithm has significance. A new measure of heterogeneity is introduced where the negative of the hazard ratio vs probability and survival shows the difference for strata of risk.

Derivations of probability of event given time are shown with related proofs on mutually exclusive and independent events. In addition, the author proves that the probability of two mutually exclusive events equals 1 not 0. Then a method of risks for bivariate variables is shown based on derived probability proofs. Subsequently, a new method for Z scores is shown based on Agravat's distribution and probability mass function. Comments are made to include corollaries to Heisenberg's uncertainty principle because of its relation to probabilities of events given time. Plots displaying the properties using SAS and MS Excel will be given. Derivations and graphs related to special relativity corrections of the author will be rendered that includes inferences regarding atomic particles.

2 Methods

To start this procedure the interaction analysis, 2x2 by 2x3 matrices are multiplied as shown in "Formulas Calculating Risk Estimates and Testing for Effect Modification and Confounding" [Agravat \(2011\)](#). The means are also calculated in the same way for tables of observed and mean values. Next using the formulas of the \hat{O} statistic, calculate the output, through the PROC IML code, calculate the "AEM" variable for the SAS algorithm intended for evaluating confounding with PROC MIXED for effect modification of the head neck cancer data of INHANCE data. The program and algorithm for PROC IML (SAS) is from the author [Agravat \(2011\)](#), and if "AEM" is significant one may conclude that the the null of homogeneous null is rejected concluding effect modification exists. The matrix formulas are shown here in the PROC IML code as well as the O statistics. In the "New Effect Modification P Value Test Demonstrated" [Agravat \(2009\)](#), the cases variable is used in 1,0 1, 0 sequence likewise for cases or outcome in this study. This algorithm for effect modification has "fit" set to 1,1,0, and 0. In the effect modification algorithm, the technique using O statistics and matrices utilize the observed products from matrix multiplications and mean matrices and the same method of count data transformation [Agravat \(2011\)](#). (The procedure for effect modification using PROC IML and PROC MIXED and O statistics is from [Agravat \(2011\)](#)).

For confounding the procedure involves PROC IML and PROC MIXED as well as O_c statistics. The procedure is to alternate the first two entries where normally one follows the "0" for adjusting data to adjusting the alternate pairs of data entries without regards to the ca variable with the beta transformation ([Agravat \(2009\)](#) or [Agravat \(2011\)](#)). Data transformation is done for the next two pair of data and alternated with non adjusted data without regards to "ca". The "acov" (Agravat's confounding variable) variable comes from the PROC IML code for "cag". "1" is put for the first position of "acov" followed by substitution of values from the PROC IML algorithm of "cag" (confounding Agravat's) coming from the PROC IML algorithm.

2.1 Matrices for Effect Modification and Confounding with O Statistics Formula

$$\begin{aligned}
 & \begin{pmatrix} 1 & 1 \\ 0 & 1 \end{pmatrix} \\
 & \times \begin{pmatrix} 1 & 0 & 795 \\ 0 & 0 & 2586 \end{pmatrix} \\
 & = \begin{pmatrix} 2 & 0 & 1590 \\ 1 & 1 & 2586 \end{pmatrix} \\
 & \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} \\
 & \times \begin{pmatrix} 1 & 1 & 795 \\ 0 & 0 & 2586 \end{pmatrix} \\
 & = \begin{pmatrix} 2 & 2 & 1590 \\ 1 & 1 & 2586 \end{pmatrix}
 \end{aligned}$$

2.2 New Method for Confounding with O_c Statistics and Matrices

Matrix mathematics continues for this operation and all the following count values are input. The purpose of this operation is calculating the hypothetical "acov" variable after adjusting for the algorithm with PROC MIXED. The algorithm follows the pattern with cases as the first variable having 1, 0, 1, and 0. Then the hypothetical "ca" variable is alternated in 0, 1, 1, and 0's, follows next. xzy and zxy are created in the manner previously published with $observed(count) / |\beta z x * \beta z|$ and $observed(count) / |\beta y z|$ [Agravat\(2011\)](#), [Agravat \(2009\)](#), [Agravat \(2008\)](#). In the PROC IML algorithm, begin with outcome and "ca" variable. Next, pair adjusted data together with corresponding count values. Pairs of the xzy and zxy are adjusted according to the beta transformation method given, in this case irrespective of the "ca". The hypothetical "acov" variable is calculated through the matrix mathematics and O_c or use of PROC IML code provided and placed into the SAS algorithm to be analyzed with PROC MIXED. The observed values from count data is input into the PROC IML code and is followed with the calculated

```

PROC MIXED AND EFFECT MODIFICATION
data nonsmokeraem;
input cases fit zxy zxy aem count;
datalines;
1 1 1 1 2208.8 795
0 1 1 1 2586
1 0 301.1 634.2 1 763
0 0 1735.2 3655 1 4397
1 1 1 1 230.56 111
0 1 1 1 233
1 0 24.4 51.5 1 62
0 0 93.9 197.8 1 238
1 1 1 1 250.52 40
0 1 1 1 152
1 0 17.7 37.4 1 45
0 0 67.1 141.3 1 170
;
run;
proc mixed data=nonsmokeraem;
weight count;
class zxy;
model cases= zxy aem /solution ddfm=satterth covb chisq;
run;

PROC MIXED AND CONFOUNDING
data nonsmokernew;
input cases ca zxy zxy acov count;
datalines;
1 0 1 1 1 795
0 1 1 1 583.66 2586
1 1 301.1 634.2 1 763
0 0 1735.2 3655 583.66 4397
1 0 1 1 1 111
0 1 1 1 583.66 233
1 1 24.4 51.5 1 62
0 0 93.9 197.8 583.66 238
1 0 1 1 1 40
0 1 1 1 130 152
1 1 17.7 37.4 1 45
0 0 67.1 141.3 190.2 170
;
run;
proc mixed data=nonsmokernew;
class ca;
weight count;
model cases= acov zxy /solution ddfm=satterth covb chisq htype=2,3;
random int / type=un subject=cases;
run;

```

Figure 1: Agravat's Method Code for Effect Modification and Confounding

means from matrix mathematics for the mean matrix to be involved in calculations of "cag" variable renamed "acov". Matrices are multiplied by the existing code of transformed data where as demonstrated in "A New Effect Modification P Value Test Demonstrated" [Agravat \(2009\)](#). Then the observed and the means values are calculated in the matrices and substituted into the PROC IML algorithm where stated. Determinants are used and the formula for O_c statistics are followed to allow calculations necessary for the "acv" variable. The use of PROC MIXED allows for lower -2LL that implies better model fit and allows for random effects not just fixed effects data as may be the case for standard regression methods.

Effect Modification O Statistics

$$\widehat{O}=(O-\bar{O})^2 \div O \quad (1)$$

Confounding O_c Statistics

$$O_c=(\bar{O}-O)^2 \div O \quad (2)$$

2.3 SAS Algorithm with PROC IML for Confounding

An inference that can be made from the PROC IML algorithm is that differences in the final CAG values will most likely reflect in the P value resulting in significance overall if there are large differences.

```
proc iml;
  * Read data into IML ;
  use nonsmoker;
  read all ;
  * combine x0 x1 x2 into a matrix X ;
  var = zxy || xzy||n;
  var2 = cases || fit ;
  newvar=var*var2;
  varB1={
1 0,
0 1};
varB2={
1 1,
0 0};
varB3={
1 0,
0 1};
varB4={
1 1,
0 0};
varB5={
1 0,
0 1};
varB6={
1 1,
0 0};
varC1={
 1 1 795,
 1 1 2586};
varC2={
301.1 634.2 763,
1735.2 3655 4397};
varC3={
1 1 111,
1 1 233};
varC4={
24.4 51.5 62,
93.9 197.8 238};
varC5={
```

```

1 1 40,
1 1 152};
varC6={
  17.7 37.4 45,
  67.1 141.3 170};
V1={
  0.24 0.24 187.68,
  .76 .76 1978.62};
O1={
  1 1 910,
  1 1 2117};

deter1=(V1);
print deter1;
deterO1=(O1);

D1=(deter1-deterO1)#(deter1-deterO1)#1/deterO1;
print D1;
D1new={
  0.5776 0.5776 573.34745,
  0.0576 0.0576 9.0453587
};
ars=D1new[,+];
print ars;
print D1;
cag1=ars[+,];
print cag1;
cag1=
583.66321;
/*****/
print varA2;
V2={
  344 576 110,
  0 0 0
};
O2={
  602.2 1268.4 1526,
  0 0 0
};
deter2=(V2);
print deter2;
deterO2=(O2);
D2=(deter2-deterO2)#(deter2-deterO2)#1/deterO2;
print D2;
D2new={
  344 576 110,
  0 0 0};
cag2=D2new[,+];
print cag2;
print D2;
cag2=ars[+,];
print cag2;
cag2= 1030;

/*****/
print varA3;
V3={

```

```

.32 .32 35.63,
.68 .68 160.9};
O3={
  1 1 111
  1 1 233
};
deter3=(V3);
print deter3;
deterO3=(O3);
D3=(deter3-deterO3)#(deter3-deterO3)#1/deterO3;
print D3;
D3new={
  0.5776 0.5776 573.34745,
  0.0576 0.0576 9.0453587
};
ars=D3new[,+];
print ars;
print D3;

cag3=ars[+,];
print cag3;
cag3= 583.66321;
/*****/
print varA4;
V4={
48.8 103 124,
0 0 0
};
O4={
48.8 103 124,
0 0 0
};
deter4=(V4);
print deter4;
deterO4=(O4);
D4=(deter4-deterO4)#(deter4-deterO4)#1/deterO4;
print D4;
D4new={

  0.5776 0.5776 573.34745,
  0.0576 0.0576 9.0453587
};
ars=D4new[,+];
print ars;
print D4;
cag4=ars[+,];
print cag4;
cag4=583.66321;
V5={
.22 .22 8.57,
.78 .78 119.42};
O5={
  1 1 40
  1 1 152
};
deter5=(V5);
print deter5;
deterO5=(O5);

```

```

D5=(deter5-deter05)#(deter5-deter05)#1/deter05;
print D5;
D5new={
  0.22  0.22      8.57,
  0.78  0.78     119.42
};
ars=D5new[,+];
print ars;
print D5;
cag5=ars[+,];
print cag5;
cag5= 129.99;

/*****/
print varA6;
V6={
35.4  64.8  90,
0  0  0
};
O6={
35.4  64.8  90,
0  0  0
};
deter6=(V6);
print deter6;
deter06=(O6);
D6=(deter6-deter06)#(deter6-deter06)#1/deter06;
print D6;
D6new={

  35.4  64.8  90,
  0  0  0
};
ars=D6new[,+];
print ars;
print D6;
cag6=ars[+,];
print cag6;
cag6=190.2;

Ot=sum(cag1+cag2 +cag3+cag4+cag5+cag6);
print Ot;
proc iml;
P=PROBCHI( 3101.76, 8, 0)=.95 ;
print P;
P<0.0;

```

2.4 Limits and Hazard Ratio Analysis vs Probability

By definition of odds $OR1 = P1/(1 - P1)$ and odds 2 or $OR2 = P2/(1 - P2)$ so the ratio of $OR1$ and $OR2$ gives an odds ratio. However this works well for binomial covariates based on the binomial distribution. This data for head neck cancer is analyzed with the Weibull distribution to obtain the beta estimates for analysis. The proportional hazards model assumption requires homogeneous variance for model fit. If there were random effects and random variables that are not effectively analyzed or handled without assuming normality in generalized linear models and large populations, then new methods for

analysis such as by the author may be utilized. For hazard ratios, dealing with the baseline hazard and the chief covariate over time, there may be a distribution more relevant than binomial and there may not be confidence in the results due to the lack of an appropriate distribution to deal with heterogeneous covariates that are not normal for the outcome and can be chosen during the Novel R code procedure with survreg. The most appropriate distribution can be chosen any time to obtain the best results in the author's parametric method. Even the cumulative distribution function follows laws where $F(x)$ is between $[0, 1]$ for x approaching $-\infty$ and $+\infty$ for their limits. When $F(z)$ approaches 1 or $S(z)$ approaches 0 the problem of undefined denominator exists and is critical because hazard risk estimates are not easily obtained for nonnormal data. Often where low or high probability values of the denominator may be required to conclude on the effects and magnitude to of the risk statistics hence the significance.

One may want to avoid having a cumulative distribution to observe the patterns of relationships of values where multi-nomial distribution is used for risk statistics. Normally, the $Pr(X = x) = F(x_0) - F(x_0 - x)$ which is the basis of the limit as y approaches x_0 without affecting the distribution. Hence the odds ratios and hazard ratios are appropriate, but not for the case of hazard ratio when $F(x)$ is divided by $Pr(X=x)$ and x_0-x approaches 0 and is undefined because the author uses probability algorithm derived previously [Agravat \(2011\)](#). What will be appropriate for this distribution in the case of variables where probability is not binomial or extreme values of probability is the case that may often be the case for non-normal data? Agravat's probability formula, $P(z)_{new}$ can handle three level of variables plus the possibility of interaction. The probability equation of the author, for three level of confounder, is appropriate for hazard ratios and related survival calculations. In these risk statistics calculations, there are also no restrictions on prevalence being 10 percent or less or linearity. The formulae follow the independence assumption [Agravat \(2011\)](#) proved previously or the need for time points where a survival study has not been performed.

2.5 Hazard Ratios, and Distribution Analysis

The new probability density function (p .d. f.) is normally: $h(t)=d(-\log(S(t)))/dt=f(t)/S(t)$ with $S(t)$; however, with respect to the variable z , the new derivation is as follows using equations from the author [Agravat \(2011\)](#). Substituting the standard definition of probability instead of $P(z)_{new}$ in this case gives equation (6). In the formula no time point is required and the distribution functions are obtained for the calculation of the hazard function including cumulative distribution function and baseline hazard function.

HAZARD RATIO AND DISTRIBUTION ANALYSIS

$$HR(z)=\frac{1}{(P(z_{new})) * (1 - odds(y)) + odds(y)} \quad (3)$$

$$\frac{\partial}{\partial z}(-HR(z)_{new})= -HR'(z) \quad (4)$$

$$\frac{-\partial}{\partial z}((1) \div ((P_z)_{inew} * (1 - odds(y)) + odds(\hat{y}))= \quad (5)$$

$$\frac{\partial}{\partial z}(-1) \div \left(\frac{(z)}{(1+z)} * (1 - y) + y\right)= \quad (6)$$

$$\frac{\partial}{\partial z} \frac{(-1) * (1+z)}{(z) * (1 - y) + y * (1+z)} = \quad (7)$$

$$\frac{\partial}{\partial z} - \frac{(1+z)}{(z+y)} = \quad (8)$$

$$\frac{-(z+y) * (1) - (1+z) * (1+y)}{(y+z)^2} = -HR'(z) \quad (9)$$

$$-HR'(z)=\frac{(-y * z - 1)}{(y+z)^2} \quad (10)$$

$$S(z)=exp^{-HR(z)} \quad (11)$$

$$\lambda(z) = -\log(S(z)) \quad (12)$$

$$hz = -\frac{S'(z)}{S(z)} \quad (13)$$

$$hz = \frac{HRz}{\exp^{-HR(z)}} \quad (14)$$

$$\exp^{-HR(z)} = S(z) \quad (15)$$

$$\ln(\exp^{-HR(z)}) = \ln(S(z)) \quad (16)$$

$$\ln(\exp^{-HR(z)}) = \ln(S(z)) \quad (17)$$

$$I(z) = \ln(S(z)) \quad (18)$$

$$I'(z) = \frac{1}{z} \quad (19)$$

$$\lambda(z) = \frac{1}{(z * S(z))} \quad (20)$$

$$\lambda = \frac{1}{(z * S(z))} \quad (21)$$

$$S(HR_{z_{new}}) = \exp^{-HR_{z_{new}}} \quad (22)$$

is the formula for corresponding survival time or probability. This statistic derived for the hazard of z (h(z)) or hazard function is obtained by a new method the d/dz (-HR (z)new) to obtain the hazard function hz instead of the d/dt(-log(S(t)) that depends on time points. The survival time can be provided given that the probability or survival time is not undefined, or the missing time and the interaction with time is a higher power, that is quadratic or cubic. Calculating survival statistics from the hazard ratio for hazard function provides statistics using the formulae with probability for the confounder (equation 3). The new technique gives explicit answers for hazard ratios based on independence and the probability ($P(z)_{new}$) equation of the author and hazard ratio of z or HR (z)new for $-3 \leq Bz \leq 3$ for all the survival statistics demonstrated here with all combinations of possible by values which may be able to account for enormous hazard ratios (except greater than 20) that may normally not be the case with various probability methods that satisfy independence. Stratified levels are also possible by multiplying the coefficient's level in the regression equation. All possible survival distribution analysis methods are possible at all levels between these levels. This new method has the advantage of being appropriate for three level variables, beyond bivariate analysis or fixed effects or proportionate hazards model. There is no need to assume standard errors are homogeneous for proportional hazards to be constant for this approach using independence assumption using the proof of the author [Agravat \(2011\)](#) that the function and its hazard ratio exist. The non-normal data is more easily analyzed with this assumption of independence because the standard error with assumption of Beta=0 does not need to be repeated when estimating the beta for other covariates for random data.

-HAZARD RATIO-

$$HR(z) = \frac{1}{(P(z_{new})) * (1 - odds(y)) + odds(y)} \quad (23)$$

-BASELINE HAZARD FUNCTION I-

$$hz_0 = \frac{hz}{HR(z)} \quad (24)$$

$$hz = (hz_0) * HR(z) \quad (25)$$

Fit Statistics						
-2 Res Log Likelihood	21.5					
AIC (smaller is better)	23.5					
AICC (smaller is better)	25.5					
BIC (smaller is better)	22.9					

Solution for Fixed Effects						
Effect	zxy	Estimate	Standard Error	DF	t Value	Pr > t
Intercept		-0.00044	0.08109	4	-0.01	0.9959
zxy	1	0.03892	0.1262	4	0.31	0.7732
zxy	17.7	1.0000	0.8056	4	1.24	0.2823
zxy	24.4	1.0000	0.6877	4	1.45	0.2196
zxy	67.1	6.07E-17	0.4203	4	0.00	1.0000
zxy	93.9	-2.77E-19	0.3578	4	-0.00	1.0000
zxy	301	1.0000	0.2109	4	4.74	0.0090
zxy	1735.2	0
aem		0.000443	0.000097	4	4.56	0.0103

Type 3 Tests of Fixed Effects						
Effect	Num DF	Den DF	Chi-Square	F Value	Pr > ChiSq	Pr > F
zxy	6	4	26.38	4.40	0.0002	0.0866
aem	1	4	20.82	20.82	<.0001	0.0103

Power of nonsmoker count data of INHANCE							
Obs	Source	DF	ChiSq	ProbChiSq	Method	test	power
1	zxy	1	681.31	<.0001	LR	3.84146	1
2	aem	1	575.61	<.0001	LR	3.84146	1

Figure 2: Agravat's Method Output for Effect Modification

Fit Statistics						
-2 Res Log Likelihood	35.8					
AIC (smaller is better)	37.8					
AICC (smaller is better)	38.3					
BIC (smaller is better)	38.3					

Null Model Likelihood Ratio Test		
DF	Chi-Square	Pr > ChiSq
0	0.00	1.0000

Solution for Fixed Effects						
Effect	Estimate	Standard Error	DF	t Value	Pr > t	
Intercept	0.9152	0.09306	9	9.84	<.0001	
acov	-0.00160	0.000201	9	-7.97	<.0001	
zxy	0.000013	0.000056	9	0.24	0.8184	

Covariance Matrix for Fixed Effects				
Row	Effect	Col1	Col2	Col3
1	Intercept	0.008660	0.00001	-2.72E-7
2	acov	-0.000014	4.057E-8	-5.03E-9
3	zxy	-2.72E-7	-5.03E-9	93.133E-9

Type 2 Tests of Fixed Effects						
Effect	Num DF	Den DF	Chi-Square	F Value	Pr > ChiSq	Pr > F
acov	1	9	63.46	63.46	<.0001	<.0001
zxy	1	9	0.06	0.06	0.8131	0.8184

Type 3 Tests of Fixed Effects						
Effect	Num DF	Den DF	Chi-Square	F Value	Pr > ChiSq	Pr > F
acov	1	9	63.46	63.46	<.0001	<.0001
zxy	1	9	0.06	0.06	0.8131	0.8184

Power of nonsmoker count data of INHANCE							
Obs	Source	DF	ChiSq	ProbChiSq	Method	test	power
1	zxy	1	.00000	0.9998	LR	3.84146	0.05009
2	acov	1	7.6163	0.0058	LR	3.84146	0.78809

Figure 3: Agravat's Method Output for Confounding

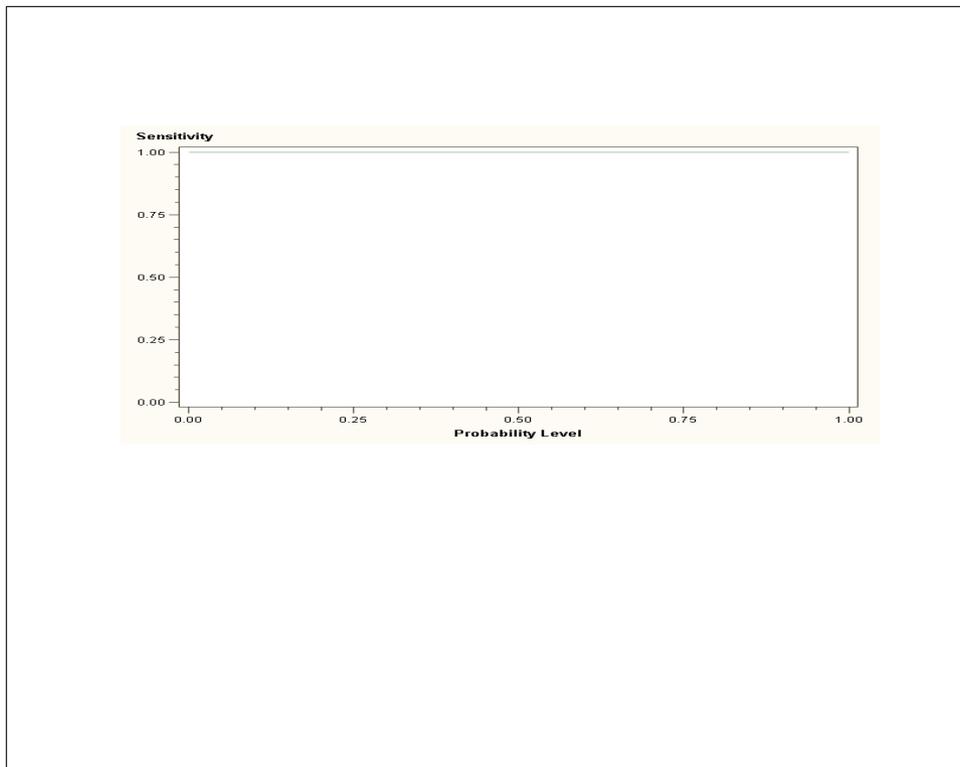


Figure 4: Agravat's Method Code for Confounding ROC curve

-BASELINE HAZARD FUNCTION II-

$$(hz_0) = \frac{\lambda(z)}{HR(z)} \quad (26)$$

-CUMULATIVE DISTRIBUTION FUNCTION-

$$F(z) = 1 - S(z) \quad (27)$$

-95 CONFIDENCE INTERVAL for HAZARD RATIOS with Agravat's Z SCORE-

$$95CIHR(z) = \exp^{(\ln(HR(z))) \pm 1.96 * \left(\sqrt{\frac{s.e.(y)}{n}} \right)} \quad (28)$$

$$-HR'(z) = \frac{-y * z - 1}{(y + z)^2} \quad (29)$$

$$-1 - y^2 = z^2 + z * y \quad (30)$$

$$-1 - y^2 = z^2 + z * y \quad (31)$$

$$z = \frac{(-1 - y^2)}{(z + y)} \quad (32)$$

$$\frac{\partial}{\partial z} = \frac{(0 * (y + z) - (-1 - y^2) * 1)}{(y + z)^2} \quad (33)$$

$$z_n = \frac{(1 + y^2)}{(y + z)^2} \quad (34)$$

$$z_n = \frac{(1)}{P(z) * (1 - y) + y} \quad (35)$$

$$z_n - 1 = \frac{(1)}{P(z) * (1 - y) + y} - 1 \quad (36)$$

$$P(z) = \frac{(1)}{(z_n) * (1 - y) + y} \quad (37)$$

$$P(z) = 1 \quad (38)$$

Normalization of Hazard Ratio, Probability and Hazard Function variant of the hazard ratio. The partial derivative of the new equation solved with quotient rule gives a new function of the hazard ratio. The inverse of that equation solved for the probability yields a value of 1 for current data of INHANCE for nonsmoking no drinking. z_n a new measure of hazards is 5.41 and $P(z_n)=1$. For no drinking no smoking, the z_n is 3.54 giving a $P(z_n)$ of .33 after inverse equations. The inverse of z_n yields a value similar to HR so that using the equation of hazard function (eq. 14), the hazard function equals the hazard ratio. The baseline hazard function is thus 1 using equation (24). This process is similar to normalization for hazard ratios and functions of nonnormal data. For head neck cancer nonsmoking no drinking and white race and the survival time is .83; the Cumulative Distribution Function is .16; the baseline hazard function is 25.11 from eq. (24) and from z_n 5.57 from eq. (34) a difference of 3 percent; hazard function is .257 from equation (14). These calculations differ from the proportional hazard models for calculations shown in table 11 for baseline hazard function by about 3 percent and do not require assumptions of proportional hazards or equal standard errors or linearity but independence assumption is required as proved by the author in [Agravat \(2011\)](#) for common conditional odds ratio being 1.

2.6 Interaction between Effect Modification and Confounding Analysis of INHANCE for Nondrinkers and Nonsmokers

The author has developed a new method to compare and measure the risks for effect modification and confounding when the exposure can be reversed using O statistics to determine if there is a cumulative effect of the two statistics. First the same measures of the transformed data are utilized. Make a table of the zero cell values from the matrix math. Sum the rows and columns. Measure the proportion of the total row value by the cell value for each row. Take the averages of the columns. Calculate the column average. Use the O statistics with the transformed data for calculating values for each level or row as in table 2. Square the answer and sum for each row as calculated. Square that value and divide by the number of zero cells squared gives a P value measure. This same procedure can be repeated for the confounding or "acov" variable as for "aem" adjusted for the exposure but the result is the same despite which O statistic formulas used. The outcome suggests that for non-drinking vs. nonsmoking the exposure has $P < 0.0107$ (see table 3). $P < 0.0107$ means that the exposure of non alcohol drinking is not significant at alpha .05 level for the possible additive interaction between effect modification and confounding. For the exposure nonsmoking the situation is different. The exposure has $P < 0.019$ for interaction due to nonsmoking vs. non-drinking. The y for non-drinking is -.9865 and z is -1.5699 yield a low and negative $P(z)$ new of -.27 for strata 1, -.5255 for strata 2, and -.5802 for strata 3 that is non-normal and may not work according to proportional hazard model requires probability being between 0 and 1. The interaction $P < 0.0107$ being significant statistically at alpha of .05 and probabilities being non-normal imply that there may be an important chance for negative effects of no drinking vs no smoking for head neck cancer because there is more heterogeneity hence one may need to analyze this effect more in details. Certainly since $P < 0.019$ for the interaction of nonsmoking vs. non-drinking requires significant analysis for the outcome head neck cancer and by different races with smoking being the primary exposure since the probability of events are greater than 1 indicating non-normal data based on probability. The F-statistics fails to reject for the exposure non-drinking vs. non-smoking for confounding by $P < 0.1081$ for nonsmoking as exposure. The new interaction p value $P < 0.0107$ supports the fact that while effect modification is significant, and confounding is significant for the exposure non-drinking and overall significance still exists.

$$O_{conf} = [(\bar{O} - O)^2 \div O^2]^2 \quad (39)$$

Table 1: Inference Statistic for Comparing Interaction of NonSmoking

Zero Cell	Col1	Col2	Col3	Col4
2	664	1138	1526	3328
4	64	92	260	316
6	40	68	90	198

Table 2: Inference Statistic for Comparing Interaction of Nonsmoking

Zero Cell	Col1	Col2	Col3
2	5.00	2.92	2.18
4	5.00	2.93	2.17
6	4.95	2.91	2.20
Average	4.98	2.92	2.18

$$\sum O_{cinf}^2 \div n^2 \quad (40)$$

$$\frac{y}{1-y} \approx P(z) - \frac{1 \div z}{1-y} \quad (41)$$

$$\frac{y}{1-y} = 5.78 - 5.90 \approx -.123 \quad (42)$$

$$\frac{y}{\exp^{1-y}} = .88 \quad (43)$$

$$\frac{y}{[\exp^{1-y}]^2} = .78 \quad (44)$$

$$\frac{y}{[\exp^{1-y}]^2 \div n} = .065 \quad (45)$$

2.7 New Algorithm for Hazard Ratios and Logits

The results of the new logits model indicate risks for the overall model are not significant based on confidence intervals containing one shown below (table 4). The risks for the overall strata for race are greater than 1.45 to x .79 where there are no statistically significant confidence intervals for the overall models reflecting nonsmoking vs. non-drinking and race as confounder for head neck cancer. The risks are actually lower slightly for Hispanic race. Y is roughly proportionate to outcome of the equation of new logits for new hazard ratio. P is the value of probability for regression variable normally called Y or the outcome estimate. After calculating the first strata the next strata's are done as shown differently. The new logit utilized for linear logits give $P < 0.065$ for strata 1 alone, the white race, for head neck cancer and the race is not enough to explain that the exposure nosmoking no drinking is not statistically significant to explain the outcome that is cancer of the head and neck [Agravat \(2011\)](#) for this nonnormal data (Shapiro-Wilks $P < 0.0003$). There is evidence that the risk from exposures and nonsmoking nondrinking does show differences of risk among races that has increased risks by hazard ratios of: 21.6 percent, 4.6 percent, and .01 percent for the races being white, black, and Hispanic for the INHANCE study because the confidence intervals are statistically significant (see section discussion).

Table 3: Inferences Statistic for Comparing NonDrinking and Nonsmoking Interaction

Statistic	Nondrinking	Nonsmoking
Additive Interaction	$P < .0107$	$P < .019$

Table 4: Probability, Hazard Ratios and New Logit Model Analysis for INHANCE and Non-smokers

Strata	P(z)	HR(p)	P	Y	95 CI (HR(p))
(1)White	5.78	1.22	.75	.88	(1.83,.81)
(2)Black	27.91	1.45	.74	.37	(1.84,.81)
(3)Hispanic	130.2	.79	1.33	.29	(1.25,.52)

$$P(z)=P(hz) \quad (46)$$

$$P(hz)=5.78 \quad (47)$$

$$\ln P(hz)=\ln(5.78)=1.754 \quad (48)$$

$$\ln\left(\frac{-2(1-y)}{y}\right)=-1.326 \quad (49)$$

$$\exp\left(\frac{2(1-y)}{y}\right)=.2654 \quad (50)$$

$$HR=.88 \quad (51)$$

$$\exp^y=.2644 \quad (52)$$

$$.2644=\left(\frac{2(1-p)}{p}\right) \quad (53)$$

$$P=.75 \quad (54)$$

$$HR(p)=\frac{1}{(P) * (1 - odds(y)) + odds(y)} \quad (55)$$

$$HR(p)=1.22 \quad (56)$$

$$3.32=\frac{2(1-y)}{y} \quad (57)$$

$$5.32y=2 \quad (58)$$

$$y=.37 \quad (59)$$

$$P(n|i)=(nC_i) * \left[\frac{\pi}{2}\right]^2 * (1-\pi)^{n-i} \quad (60)$$

```

P(10|9) = .2025
P(10|8) = .091125
P(10|7) = .0243
P(10|6) = .0042525
P(10|5) = .0005103
P(10|4) = .000042525
P(10|3) = .00000243
P(10|2) = .00000091125
P(10|1) = 2.025E-9
sum=.3227327482
1-.3227327482=.6772672519
exp*.6772672519=1.96849

```

$$P(X=t) = \binom{n}{t} \left(\frac{\sigma}{2}\right)^2 + (1 - \sigma)^{n-t}$$

Figure 5: Agravat's Method for Z Scores

```

data AgrZscore;
input z n t pi Pmi;
datalines;
1.968 10 9 .9 .2025000
1.968 10 8 .9 .091125
1.968 10 7 .9 .0243
1.968 10 6 .9 .0042525
1.968 10 5 .9 .0005103
1.968 10 4 .9 .000042525
1.968 10 3 .9 .243E-6
1.968 10 2 .9 .91125E-9
1.96 10 1 .9 .2025E-9
1.63 10 8 .8 .288
1.63 10 7 .8 .1536
1.63 10 6 .8 .08376
1.63 10 5 .8 .042902
1.63 10 4 .8 .0021804
1.63 10 3 .8 .00001543
1.63 10 2 .8 .192E-7
1.346 10 7 .7 .3969
1.346 10 6 .7 .2083725
1.346 10 5 .7 .0750141
1.346 10 4 .7 .01873325
1.346 10 3 .7 .321489E-3
1.346 10 2 .7 .361675125
1.346 10 1 .7 .24111695E-5
1.204 10 6 .6 .48384
1.204 10 5 .6 .2232432
1.204 10 4 .6 .0774144
1.204 10 3 .6 .01769472
1.204 10 2 .6 .2654208E-3
1.204 10 1 .6 .2359296E-4
;
run;

proc gsmmod data=AgrZscore descending;
title2 'Power of Agravat Z score';
model pmi = n t pi pmi z /link=log dist=normal type3;
ods output type3=tests;
run;

```

Obs	Source	DF	Power of Agravat Z score				
			Prob	ChiSq	Method	test power	
1	0	0	0.0000	<.0001	LR	3.84146	0.05000
2	0	1	103.13	<.0001	LR	3.84146	1.00000
3	04	1	15.654	<.0001	LR	3.84146	0.97766
4	04	1	199.86	<.0001	LR	3.84146	1.00000
5	z	1	5.1463	0.0233	LR	3.84146	0.62120

Figure 6: Agravat's Z Score and Power

2.8 New Z Score Method

The Agravat's distribution is capable of calculating the z table for a new distribution that is chi square and incorporated throughout this analysis. The proportion used begins with $\pi = .9$ for instance for the 90 th percentile value for $1 - .9 = .1$ divided by 2 gives .05 and a two sided test alpha will be .025. The calculations give values for the subsequent conditional probabilities calculated using Agravat's algorithm and from 10|9 to 10|1 the probabilities are calculated and summed. Then 1- sum is done finally to be exponentiated giving the z score for $alpha = .05$ of 1.968 because for $1 - \pi (= .9)$ is .1 and divided by 2 for a two sided or is the $alpha = .05$ level . The next level will be calculated by giving $\pi = .8$ and progressively for each level of alpha desired or $1 - \pi (= .8) = .2$ divided by 2 is .1 and a two sided alpha is .0516 with z score or value on algorithm and Agravat's distribution value of 1.63. The advantage of this new Z score method is that it is based on a conditional probability algorithm of the author that can be more than binomial. This new z score also functions according to the previous z score. The new Z score method has more of 97 percent for π and 100 percent for $P(n|i)$ and i. The percent of area covered under 3 standard deviations away from the mean with sample size 10 of 81.4 percent or 1/9 for the probability of the score falling in this region when $\pi = .6$ (see figures 12 and 13). The power of the new Z-score is about 100 percent (see figure 6) (and this new method is utilized throughout to calculate 95 percent confidence intervals).

2.9 Agravat's Proof of Mutually Exclusive Events and New Risks

$$P = \frac{(odds)}{(1 + odds)} \quad (61)$$

$$Q = 1 - P \quad (62)$$

$$Q = 1 - \frac{(odds)}{(1 + odds)} \quad (63)$$

$$Q = \frac{(1)}{(1 + odds)} \quad (64)$$

$$P(P \cap Q) = P(P) * P(Q) \quad (65)$$

$$P(P \cap Q) = \frac{(odds)}{(1 + odds)} * \frac{(1)}{(1 + odds)} \quad (66)$$

$$P(P \cap Q) = \frac{(odds)}{(1 + odds)^2} \quad (67)$$

$$P(z) = \frac{(odds(z))}{(1 + odds(z))^2} \quad (68)$$

$$P(P \cup Q) = P(P) + P(Q) \quad (69)$$

$$P(P \cup Q) = \frac{(odds)}{(1 + odds)} + \frac{(1)}{(1 + odds)} \quad (70)$$

$$P(P \cup Q) = 1 \quad (71)$$

$$P(y) = \frac{\exp^{(P(P)+P(Q))}}{1 + \exp^{(P(P)+P(Q))}} \quad (72)$$

$$\log P(y) = \log\left(\frac{\exp^{(P(P)+P(Q))}}{1 + \exp^{(P(P)+P(Q))}}\right) \quad (73)$$

New Probability of Mutually Exclusive, Independent Events, and New Risks The new proof of the author for probability of events according to the standard definition of probability is shown next. The standard mutually exclusive event probability is expected to be "0" but in this case it is demonstrated to be "1" that makes a significant impact to calculations involving models for regression analysis in logits because risks will not be calculated properly. Bayesian inferences may not be correct as a result that stated that the probability of two mutually exclusive events equal 0. If the probability of mutually exclusive events is made to be equal to independence of two events or one when the independence is one for the product of two events. $P(z \cap t) = P(z)$. These estimates may be used to calculate risks. The Agravat's algorithm [Agravat \(2011\)](#) is used to calculate $P(x|z)$ when combined with $P(z)$ for $P(x \cap z)$ and set equal to $P(x \cup z)$ later defined as mutually exclusive to calculate the $P(x)$. Then the log of $P(y)$ then exponentiated gives the risk for the outcome of about .87. $P(z) = .146$ using equation of logits shown by the author. If the $P(z)$ and $P(x)$ are mutually exclusive then $P(x) = P(x \cup z) - P(z)$. If $P(x \cup z) = P(x \cap z)$ then $P(x) = - (P(z)) / (1 - P(z)) = - .1709$. The exponential of $P(x)$ is .8428 with $\beta x = - 33.307$ and exponentiation of $P(z) \simeq .146$. $P(y) = .728$ and $\log(P(y)) = - .137$ so the risk of $P(y) = .87$ by ideal conditions not one or 0. The author also shows conclusively that under this assumption, the equations of logits of the author does validate the assumption of possible equality of mutually exclusive and independent events. The author suggests that one must exponentiate the resultant of $P(y)$ based on the equality proof of the author which gives for these beta estimates a value of 1.638 of risk for exposure no smoking no drinking and race based on these new logits or strata 1 or non-central European. The $\ln(P(y)) = - .702$ and $\exp^{\ln(P(y))} \simeq HR$. The natural log of $P(y)$ exponentiated gives the hazard ratio .49 from the author's method for head neck cancer in INHANCE for the exposure no-smoking vs. no drinking by race, compared to PROC PHREG (SAS) that gives $HR = .46$. The death rate according to the new method is 3 percent higher for head neck cancer for the new logit.

$$P(x \cup z) = P(x) * P(z) = (-.1709) * (.146) \approx -.025 \quad (74)$$

$$P(x \cap z) = P(x) + P(z) = (-.1709) + (.146) \approx -.025 \quad (75)$$

$$P(x) * P(z) = P(x) + P(z) \quad (76)$$

$$P(x \cup z) = P(x \cap z) \quad (77)$$

$$P(x) = - \left(\frac{P(z)}{1 - P(z)} \right) \quad (78)$$

$$\exp^{P(x)} \simeq S(z) \quad (79)$$

$$P(x) = - \left(\frac{P(z)}{1 - P(z)} \right) \quad (80)$$

$$P(z) = \frac{z}{1 + z} = \frac{1}{P(z)_{newi}} \quad (81)$$

$$\ln(P(y)) = HR(\text{Logit}(Y_{new})) \quad (82)$$

$$\exp^{\ln(P(y))} = HR \quad (83)$$

2.10 Results: Effect Modification of INHANCE for Nonsmokers

Effect Modification analysis of this study, with PROC IML, shows significant $P < 0$ with $\alpha = .05$ hence, the null of homogeneous odds is rejected and calculates values needed for the PROC MIXED algorithm in SAS. PROC MIXED for effect modification [Agravat \(2011\)](#) has $P < 0.0001$ for chi-square and $P < 0.01$ for F-statistic (a multivariate statistic) for "aem" indicating that the null is rejected and effect modification exists which is a benefit over other statistical regression methods not meant for random

effects. One may conclude that there are different risks for head neck cancer for exposure nonsmoking according to levels of race. Since $-2LL$ is 21.5 there is a good model fit with the "aem" method using O statistics and matrices [Agravat \(2011\)](#). The conclusion is that according to level of race (non-Hispanic, black, and Hispanic), the result is different risks exist for nonsmoking vs. nondrinkers by race for head neck cancer hence the homogeneous odds null is rejected and effect modification exists. In support of this new effect modification method is the power of "aem" which is 100 percent by exposure non-smoking (see figure 2).

The Breslow Day test has $P < 0.06$ and fails to reject the homogeneous odds ratio null. The conclusion is there is no effect modification compared to the author's $P < 0.0001$ at $\alpha = .05$ or reject the homogeneous null stating that there is effect modification with this study question of effect modification by non-smoking vs. non-drinking. The Hispanic race or strata 3 has chi-square $P < 0.98$ or fails to be independent an assumption of the logistic model for regression and other fixed effects methods disqualify these tests that are standard. Cancers of the oral cavity are the more serious in terms of relative risk, .19, in the never smoking never drinkers population adjusted for age, race, education, center, years of smoking cigars or pipes; followed by relative risk of .066 for oropharynx/hypopharynx (not including central Europe); relative risk of .05 for oral cavity and pharynx; and .022 relative risk for larynx cancer in a random effects model [Hashibe et. al.\(2007\)](#) all show risks greater than 10 percent variation for the different types of cancers involved in this study. Cancer rates are different for race, however race is also a factor that may compound the seriousness of race as a risk factor for head neck cancer and the exposure nonsmoking vs. nondrinking.

2.11 Confounding Analysis of INHANCE for Nonsmokers

The new formula of O_c statistics for confounding followed by the PROC IML CODE explaining the procedure in the INHANCE dataset for nonsmokers (International Head Neck Cancer Epidemiology) supplemented by the new SAS algorithm using PROC MIXED is meant for confounding analysis of this trial on the outcome head neck cancer for the exposure no smoking by race being non-Hispanic, black, and Hispanic. The results indicate that since both the "acov" variable $P < 0.0001$ for type 2 and 3 tests effect and zxy has $P < 0.0001$ are significant (in figure 3), hence one may conclude that for fixed effects there is confounding by race. There is also confounding between outcome head neck cancer, and confounding variable race for type 3 effects for fixed effects with coefficients set to 0, which may let one conclude variability may exist between all parameters when different from 0 due to $P < 0.0001$ of the chi-square, and F-statistic's $P < 0.0001$ making the need of accurate x-rays or other laboratory reports more significant. If there was an error in this study, despite randomization, which was expected to be avoided normally then there may be confounding [Szklo, Nieto \(2007\)](#). One does not normally expect that races will show differences for head neck cancer rate as is found due to non-smoking. Confounding in the INHANCE indicates that for head neck cancer as outcome, the exposure of nonsmoking vs. non-drinking are significantly different showing risk variations due to the potential confounder by more than 10 percent with significant 95 CI. Race will confound the exposure of non-smoking for head neck cancer. An inference regarding the exposure related to one may infer a time correlation for this effect resulting in head neck cancer. Hazard analysis is shown that strata 1 of non-Hispanic has greater risk of cancer by 25 percent, vs. 4.6 percent over a period of time, in the question of whether the exposure of nonsmoking vs. non-drinking causes increase of head neck cancer for black race, and 1 percent increased risk for Hispanic race. There is confounding hence the study shows confounding by race. The "acov" variable P value criteria may be useful in the future for testing for confounding in mixed, random, or fixed effects with good fit because of its statistical value. In this study, the Null Model Likelihood Ratio Test shows that $P < 1$, therefore the null of homogeneous variance is not rejected for this new procedure for confounding with PROC MIXED and is important when there is risk. The ROC curve shows that the confidence in the test is good because the sensitivity is at 100 percent and C statistics is 1 which is excellent after data transformation method of the author. The power for the "acov" variable is 78 percent using PROC GENMOD (SAS)[®] using link log and the distribution is geometric.

```
proc genmod data=nonsmokernew descending ;
  weight count;
  class ca;
  title2 'Power of nonsmoker count data of INHANCE';
```

```

model cases= zxy acov / link=log dist=geometric type3 ;
ods output type3=tests;

run;
data power; set tests;
format chisq 6.5;
test = cinv(0.95,1);
power = 1-probchi(test,1, chisq);
proc print data=power;
run;

```

Criteria for Confounding:

- 1) The strength of association of risks across strata have to be different by 10 percent or more.
- 2)The risks have to be consistently different across strata different by 10 percent or more.
- 3)The confidence intervals have to be statistically significant.
- 4)"acov" has $P < \alpha$ for either chisquare or F-statistic significant

2.12 Comparison of Effect Modification and Confounding Analysis of INHANCE for Nondrinkers and Nonsmokers

Analysis of the effect modification and confounding of nondrinkers vs. nonsmokers as exposure the conclusion is that while there is effect modification $P < 0.0001$ and $P < 0.0091$ for "acov" [Agravat \(2011\)](#), there is confounding by no alcohol drinking vs non smoking $P < 0.0391$ chi-square $P < 0.1081$ for F-statistic for "acov" variable for type 1 and type 2 effects (see figure 7) that is not significant at $\alpha = .05$. The chi-square statistics are significant for both effect modification and confounding. For the exposure no smoking vs no drinking, the strength of the P value is more significant in terms of effect modification and confounding. The respective P values are: $P < 0.0001$ and $P < 0.01$ for chi-square and F-statistic for "aem". For confounding, the $P < 0.0001$ $P < 0.0001$ for chi-square and F -statistic for "acov". This presents a very important situation for the problem of exposures of drinking and smoking for head neck cancer because both may be risk factors since the question of the effect of actually drinking and actually smoking and combined may be worse for the outcome head neck cancer by exposure by race which i the multivariate statistic for nondrinking and is not significant for the exposure of nondrinkers and head neck cancer. Perhaps the exposure is not independent of the outcome that is head neck cancer a as supported by the lack of significance of the confounding variable since $P < 0.1081$ for nondrinkers and confounding.

Comparisons of Standard Methods vs. New Method Agravat's Method may show promise for small, moderate samples, and for larger samples, non-normal/normal distributions with advantages demonstrated by higher power, better C statistics, and moderate -2LL. Breslow Day and other methods are not meant at all for small sample sizes and especially for distributions like hypergeometric, which involve accounting for time, despite relying on count datasets that are similar to Agravat's method. The common error for Breslow Day for non-normal/normal, hyper-geometric distributions is "data are too sparse" or simply do not work except for fixed effects that have large sample size. Agravat's method

Table 5: INHANCE Comparison of Effect Modification and Confounding

Exposure	Chi-square(EM)	Chi-square(C)	F-Statistic(EM)	F-Statistic(C)
(No) Drinking / Smoking	$P < .0001$	$P < .0391$	$P < .0091$	$P < .1081$
(No) Smoking/ Drinking	$P < .0001$	$P < .01$	$P < .0001$	$P < .0001$

```

data nondrinkeracov;
input cases ca zxy zxy acov count;
datalines;
1 0 1 1 1 795
0 1 1 1 609.61 2586
1 1 332 569 1 763
0 0 1913 3279 0 4397
1 0 1 1 1 111
0 1 1 1 195.38 233
1 1 27 46 1 62
0 0 104 178 0 238
1 0 1 1 1 40
0 1 1 1 128.81 152
1 1 20 34 1 45
0 0 74 127 0 170
;
run;

proc mixed data=nondrinkeracov;
class ca zxy;
weight count;
model cases=acov zxy/solution ddfm=satterth covb chisq htype=1,2;
random int/type=un subject=cases;run;

```

Type 1 Tests of Fixed Effects						
Effect	Num DF	Den DF	Chi-Square	F Value	Pr > ChiSq	Pr > F
acov	1	4	0.98	0.98	0.3264	0.3820
zxy	6	4	9.16	1.53	0.1651	0.3555

Type 2 Tests of Fixed Effects						
Effect	Num DF	Den DF	Chi-Square	F Value	Pr > ChiSq	Pr > F
acov	1	4	4.28	4.28	0.0391	0.1081
zxy	6	4	9.15	1.53	0.1651	0.3555

Figure 7: Inhance Confounding for No Drinking vs. no Smoking

allows for inference of the study question when looking at the parameter that may involve the exposure and interaction term for variables when evaluating the risk from perspectives for the outcome, because the existing technique, Breslow Day test, does not work for these conditions leaving the statistician in the dark regarding causal inferences of heterogeneity, random effects and risk statistics, allowed in mixed effects or random effects analysis. Many risk estimates don't allow time inferences for case control studies. However causality can be inferred through generalized random effects inferences.

The question of risk can be addressed for non-normal data where cases with Agravat's method are analyzed better for regression, due to low -2LL. The author uses a survival function in a novel way for estimates of beta when beta can obviously be nonzero and censored events will be accounted for, hence regression other than linear and logistic models are possible more effective for unbiased risk statistics. "Acov" is supported by the ROC curves demonstrating that the area under the curve gives the evidence that the power, and C statistic are more in favor of the new method because they are well above Breslow Day's (C-statistics .5 for the count data), and sensitive to standard errors (Agravat (2009)). Confidence of the the author's new method may follow due to better sensitivity, due to better C-statistic) of the Agravat's method that works consistently for the non-normal distributions, but data could apply to linear data pending on Shapiro-Wilks statistics and other statistics, allowing inferences to be made regarding interaction and effect modification based on power and C-statistics. The Proc Mixed application, produces significant results that are accurate and reliable with more confidence from better results that may be potentially significant for future applications in studies and lower -2LL for this new method. The fact that Agravat's method works well and provides results for various distributions means that a broad use of this method is warranted if the need for random effects analysis is needed.

2.13 Comments on Matrices, Probability, and Concentric Circles

Agravat Series, Matrices, and Concentric Rings For a square matrix, which is also possible, for the matrices the commutative property of multiplication shows that if E1 or E2 are singular when 0 is a possible determinant when dealing with the O statistics determinants of certain elements, then if E1 and E2 are singular then concentric rings are possible which may be related to how the matrices shown demonstrates the involvement of complex and real numbers which shows properties of being both nonsingular and singular because of nonzero values and being square having 0 values Agravat (2011) (see figure 8 for similar results with $\pi(t)$) with the conditional probability algorithm. One "n" represents conditional probability algorithm of Agravat, and one for the n exponent. The matrices produce and work that include probability formula that are significant for inferences. The new P(t) allows calculations of floors and enclosure in a plane. The Probability or Pi using this P(t) theorem also produces a concentric circle (see figure 8, and 9). The outside ring represents P(t), (probability of time) and the inside P(z), (probability of event). The literature review of topics included "Ideals Containing Monics" Nashier et. al. (1987) and a "Note on Perfect Rings" Nashier et. al. (1991).

New Probability Algorithm and Distribution for Time and Event z The plot of time and probability of event for conditional probability algorithm of the author shows that times and slopes can be calculated for an event for instance at times given. The slope for a $n = 9$ and the i's will be shown in the plot for i from 5 -9. One can see for the probability algorithm given that conditional probability events can be seen. The changes can be seen as well. Tests can potentially be run for three body problems since the probability Pz is for three levels relating to *Poincarè* conjecture. The Agravat's algorithm can be for any levels Agravat (2011) and involve more than bivariate end point with infinite levels of conditional probability!

$$P(n|i) = \frac{(n-i) * (n-i)}{(n-i)^n} \quad (84)$$

$$P(n \cap i) = P(i) * P(n|i) \quad (85)$$

$$P(n \cap i) = P(i) * (n-i) * (n-i) \div (n-i)^n \quad (86)$$

the race is known to change the risks for head neck cancer independent of exposure with $P < 0.0001$.

New Corollaries to Heisenberg's Uncertainty Principle

The author suspects that while the Agravat's conditional probability algorithm is accurate and reliable, that with reference to Heisenberg's uncertainty principle comment in "Formulas Calculating Risk Estimates and Testing for Effect Modification and Confounding", the author further stipulates: 1) what exists in one state will not exist in others, when there is equilibrium or motion with regards to commutative properties of momentum and time; 2) that there may be commutative properties of the matrices but the system tend to disorder which may lead to decreases in energy, and order which may lead to increases in energy level without assuming independence in Agravat's algorithm and distribution; 3) certain phenomenon of nature such as light, e.g. rainbows are transmutable as in the prisms. The author believes that the Agravat series transformation of Dirac and special relativity of Einstein [Agravat \(2011\)](#) is important to gravitation in outer space where gravity is greater in the solar system between planets. Planets within a gravitational field and the time such as estimated time= 0 for events become more important for infinite events. This understanding allows for the conception of actually how much energy there is in space. Graphs and correction of special relativity in figures 12 and show that energy decreases with increases in probability. The greater energy is at lower probability levels hence the question of laws being appropriate to the solar systems in general may be questionable. Where electromagnetic and gravitational fields are greater there life may not be supportable. Where life is supportable, the laws of physics with respect to energy and gravitation may not be the rule because levels of forces in space are greater despite being in "zero gravity". The concept or belief in entropy increasing is relative to other events and defined as the disorder in a system. The first corollary has importance because Heisenberg Uncertainty Principle states that one may not know both the position and momentum of a particle. However, the author believes that separation of events by equilibrium and motion will clarify this situation followed by the second corollary. As the system tends to disorder there is a decrease of energy; and, if going towards order there is increase of energy at fixed points compared to overall system energy. As during expanding space, where there is no limitation or assumption of no harm, one may postulate that then new order (disorder) may increase energy [Agravat \(2011\)](#) for large changes in time or distances. Bose did not state photon behavior was independent [Agravat \(2011\)](#). Less perturbed systems may increase order or disorder and involve hypergeometric properties in terms of waves. The author believes that Einstein's special relativity is appropriate for his time travel hypothesis. However, subatomic hypergeometric forces as well as the interaction between dependent and independent may propel some particles faster than light which the author perceives is a medium and may potentially separate such as by refraction that happens in water. Gravitation or motion of particles if time is included and potential energy (mgh) is included, when destruction happens the potential energy may disperse to other particles which may sum to be faster than media such as light, and some energy is propagated as waves at that time.

$$f(z) = \sum_{n=1}^{\infty} \frac{-2}{z^2} - \frac{4y^n}{z^2} + \frac{4y^{n+1}}{z^2} \quad (87)$$

$$f(z) = \sum_{n=1}^{\infty} \frac{6}{z^2} - \frac{8y^n}{z^2} - \frac{8y^{n+1}}{z^2} \quad (88)$$

$$f(z) = \sum_{n=1}^{\infty} \frac{-12}{z^2} + \frac{16y^n}{z^2} + \frac{16y^{n+1}}{z^2} \quad (89)$$

Table 6: Agravat Series and Matrix

	3rd	4th	5th
Condition 1	-33.52	-6.67	-17.87
Condition 2	-42.15	-127.02	-241.51
Condition 3	-8.41	-36.58	-7.77

Agravat Series and Commutative Properties of Matrices Given by the formulas in equations of Agravat series [Agravat \(2011\)](#) for 1st, 2nd, and 3rd columns respectively when $n=3.16$ and: $z=.25$, $y=.25$ (first row); $n=3.16, z=-1.532$, $y=-1.422$ (second row); $n=3.16$, and $z=.468$, $y=.578$ (third row) (see figure . The top right 2x2 the matrix multiplied by the bottom left 2x2 matrix are the elements from the covariance matrix. Create the matrix first as shown in the table with Agravat Series. Next calculate the covariance. The implicit derivatives are hyper-geometric and may involve natural log so calculations are easily done from beta estimates. Agravat series shows that commutative properties do hold for matrices that conflict with Heisenberg's uncertainty principle regarding matrices. The ideal N statistic values are derived by solving the fifth implicit derivative or Agravat series for z and y set equal to each other and set to 0. The result is both z and y will equal .25. The base ten of log for n set to $2x.25$ gives $n = 3.16$. Thus that becomes the ideal exponent. The Agravat series as demonstrated before has ability to portray the *Poincairè* conjecture and may be appropriate to explain the commutative properties of the spheres different from hypothesized Heisenberg who did not calculate commutative porperties. Thus hyper-geometric properties of the series may hold within the spheres. This procedure may be set to replicate commutative properties of matrices showing that addition, subtraction, and multiplication of matrices are possible (see figures 10 and 11 for derivation and output)!

```
proc iml;

    COVM = 3|| 4||5;

    fzm={
    -33.52  -6.67  -17.87,
    -42.15  -127.02  -241.51,
    -8.41  -36.58  -7.77};

    fzmT=fzm';

print fzm;

    sscpsfmT=fzm*fzm';

    cov=sscpsfmT/8;

    print cov;

proc iml;
    * Read data into IML ;
    use passlungexp5d;
    read all ;
    EL1={
    821.98439  9529.7229,
    83.092713  859.67573};

    EL2={
    821.98439  83.092713,
    9529.7229  859.67573};

    E1=EL1*EL2;

print E1;

deter=det(E1);

print deter;
```

```

n = 0; z = y
-12/z^2 + 16y^n/z^2 + 16y^{n+1}/z^2 = 0

-12/z^2 + 16y^n/z^2 - 16y^{n+1}/z^2 = 0 >> if >> n = 0
-12/z^2 + 16y^0/z^2 - 16y^{0+1}/z^2 = 0
-12/z^2 + 16*1/z^2 - 16*y/z^2 = 0
-12/z^2 + 16/z^2 - 16y/z^2 =
4/z^2 - 16/z = 0
z = .25

-12/z^2 + 16y^n/z^2 + 16y^{n+1}/z^2 = 0
n = 0
-192 = 256 - 256y = 0
y = .25

Condition 1: z=y=.25 n ideal where log base 10 of n=.5 so n=3.16
Condition 2: actual z and y and calculated n
Condition 3: add 2 to beta estimates

```

Figure 10: Ideal Beta Estimate Proof for Z and Y

```

fzm
-33.52    -6.67    -17.87
-42.15   -127.02   -241.51
-8.41    -36.58    -7.77

cov
185.92703 821.98439 83.092713
821.98439 9529.7229 859.67573
E1
91491277 8260772.4
8260772.4 745946.76
deter
7.26083E9
E2r
682562.74 7904716.3
7904716.3 91554661
deter
7.26083E9
Es
1643.9688 9612.8156
9612.8156 1719.3515
deter
-89579664
Esr
1643.9688 9612.8156
9612.8156 1719.3515
deter
-89579664

```

Figure 11: Agravat Series and Commutative Properties of Matrices

```

E2r=EL2*EL1;

print E2r;

deter=det(E2r);

Print deter;

Es=EL1+EL2;
print Es;

deter=det(Es);
print deter;

Esr=EL2+EL1;
print Esr;

deter=det(Esr);
print deter;

Ess=EL1-EL2;
print Ess;

deter=det(Ess);
print deter;

Esrs=EL2-EL1;
print Esrs;

deter=det(Esrs);
print deter;

EsrsT=Esrs';
print EsrsT;

deter=det(EsrsT);
print deter;

```

3 Discussion

What will be appropriate when in the case of variables where the probability is not binomial? One may utilize a cumulative distribution or logit in logistic regression to observe the patterns of relationships of values. The probability equation of the author, for three level of confounder, is appropriate for odds ratios, relative risks, and hazard ratios and related calculations. In these risk statistics calculations, there are also no restrictions on prevalence being 10 percent or less or the need for time points with

Table 7: INHANCE Comparison of Effect Modification and Confounding

RACE	CASES (Never Drinkers)	Controls	CASES (Never Smokers)	Controls
Non-Central European	795	2586	763	4397
Black	111	233	62	238
Hispanic	40	152	45	170

these new methods for survival analysis. For hazard ratio, which deals with the baseline hazard and the chief covariate over time, there may be an algorithm, Agravat's conditional probability algorithm is more appropriate than binomial where there may not be confidence in the results due to the lack of an appropriate distribution to deal with heterogeneous covariates that are not normal for the outcome, but can be selected during the Novel R code, that allows events to occur to be taken into account and is a parametric method procedure. The most appropriate distribution can be chosen to obtain the best results. The hazard ratio from the equation (3) is .21 (95 CI: .32,.14) which means the risk for head neck cancer increases for one unit change in race level is 21 percent more for head neck cancer from no smoking due to race being non-Hispanic and statistically significant from not smoking. Subsequently, the strata 2(black) and 3(Hispanic) from equation (3) yield hazard ratios increases by .046 (95 CI:.069,.03) and .010 (95 CI: .015, .006) respectively also statistically significant for not smoking. The lowest cancer rate is in Hispanic race of the three for head neck cancer due to non-smoking as exposure. The chi-squares test shows $P < 0.0001$ for cases, zxy, and xzy, new variables and is statistically significant, hence the author states there may be independence as well as randomness among these variables. The probability algorithm may show nonlinear regression to be the case instead of linear due to probability value over 1 or 5.78, 27.91, and 130.2 for the strata's one, two, and three due to higher probabilities from the probability algorithm [Agravat 2011](#). Higher probabilities calculated by $P(z)_{new}$ yield more changes in the hazard logits that illustrate the problem for race and outcome head neck cancer for the exposure nondrinking vs. nonsmoking.

In the article, "Formulas Calculating Risk Estimates and Testing for Effect Modification and Confounding", the equation regarding $Bz=0$ and By not equal to 0 gives $odds(z) = (2 - y)/(1 - y)$ after inverse equations and incorporating the standard definition of probability for binomial covariates. Thus with Bz fixed to 0, no harm is assumed. Since there is confounding by race in this study of INHANCE, $P < 0.0001$ for "acov", the calculation of the hazard ratio may be a useful statistic when the risk of no harm is possible for this potential non-confounder trials at three levels and independence of exposure non-smoking is rejected and hypothesis is rejected. Using this equation and value and the $P(z)$ new formula, one observes that the $P(z)$ new is also 1 (eq. (4) in terms of odds (z)). Finally the new hazard ratio equals 2.31 for head neck cancer with races fixed to $\beta z = 0$ for nonsmoking vs. non-drinking indicating statistically higher risks and inferred for $Bz = 0$ and $By \neq 0$. The baseline hazard as calculated by the author's analysis shows a value of -2.30. Comparing the odds(z) above to the hazard ratios for the three strata (see table 9 and 10) using equation (3), there is a great difference comparing the hazard ratio when no harm is assumed. One might have thought that the risk is similar when assuming no harm due to non-smoking. Actually the risk is more for binomial probability for hazard ratios for head neck cancer for not smoking than the new probability formula with independence ($HR(z)$ is .21) when assuming no harm and the hypothesis is nonnormal than the multinomial distribution calculations. The author is based on independence assumption [Agravat 2011](#).

Pathology, Pathogenesis, and Treatment The risk factors of head neck cancer normally include smoking, alcohol, and human papilloma virus, mutation of TP53 gene, that is influenced by heavy drinking and heavy smoking including betel nuts which are commonly used in Asia [Maur Bunting-Blaustein \(2011\)](#). Other factors are vitamin deficiencies such as A and iron in Plummer-Vinson syndrome. Human Papilloma Virus is associated with squamous cell carcinoma while Epstein-Barr virus is linked with the non-keratinizing type, (type II and type III undifferentiated, nasopharyngeal carcinoma endemic to Africa and Asia). Squamous cell carcinoma is associated with 25 percent of HPV and 60 percent of oropharyngeal carcinoma of the lingual and palatine tonsils. Over amplification and cell expression is associated with tumorigenesis. CDKN2A is associated with regulating the cell cycle. High production of Epidermal Growth Factor Receptors are associated with head neck cancer as well and poor prognosis is associated for higher α tumorigenic transforming factor levels. Treatment of head neck cancer is done with cetuximab, ertotinib, or docetaxol combinations.

Tp53, a tumor suppressor gene, has been found to be involved in the etiology of head neck cancer as well as other cancers such as lung, breast and colon cancer. The lack of TP53 gene may be secondary to mutations or deletions in genes. Degradation of p53 and regulation of p53 is crucial to cell regulation and may affect gene function. Enhanced degradation of p53 is linked to complexes of viral proteins. The E6 protein of high risk HPV viruses may result in increased p53 degradation. HPV DNA that is oncogenic is present in over 80 percent of cervical SCC and association with p53 is found to transform viruses. P53 has a negative correlation with tonsillar carcinoma ($P = .03$). HPV is strongly associated with primary

tumors of oropharynx ($P = .005$). There is also a high correlation with HPV and ($P = .0001$) SCC of tonsil. 12.5 percent of oropharyngeal cancers that are not in tonsils were positive with HPV. There is an inverse relationship to heavy smoking history with patients with HPV ($P = .05$), though there is a strong association of heavy smoking history and p53 mutation. There is a correlation of HPV infection and race ($P = .015$), where white patients were more likely to be HPV positive. The author's results also conclude that there is more risk by hazard ratio of .216 (95CI: .32, .14) that is statistically significant for white race or non-central European versus other races, black and Hispanic. The new measure of the author where baseline hazard and probability or with survival time comparison shows greater heterogeneity (-.868 vs -1.07) (see table 9 and 10). Abnormal p53 has been found to be in 33-100 percent of head neck cancer specimens [Haraf,Nodzinski,Brachman, et.al.\(1996\)](#) as supported by literature this evidence in suggests that the exposure of alcohol and human papilloma virus are statistically significantly associated with head neck cancer where heavy drinking is the exposure. Hence the gene TP53 which is significant statistically for race and human papilloma virus may play a role that the exposure heavy drinking may possibly need to be analyzed in the future to determine the mechanism. The statistics of analysis for probability may be based on independence assumption or independence with respect to time and event per probability of time. In section 2.13 the equation for probability of event given time over probability of time shows that the mutually exclusive events may support the possibility of head neck cancer from nonsmoking/nondrinking by race for the white race according to time probabilities being closer for the comparison of Agravat's probability algorithm result vs. mutually exclusive events probability in equation (69) vs. equations (132 and 133) with values of (see equations 131-133) -1.42 vs. -.64 and -.52 being closer. The author also proves that the probability of mutually exclusive events equals 1, where all events may occur, not 0. The exposure according to [Haraf,Nodzinski,Brachman, et.al.\(1996\)](#) states the smoking is inversely related to human papilloma virus ($P < 0.05$).

The correlation of cases of head neck cancer with race is 0 as well as exposure nonsmokers from PROC CORR and PROC FREQ gives lack of independence based on Pearson correlation statistic and p value. R square is 0 for all variables. These standard statistical procedures reveal little in this case for this study on head neck cancer. The conclusion may show the difficulty of study of this disease from the point of view of heterogeneity and multi-collinearity as has been done in the past [MacComb, Fletcher \(1971\)](#). Predicting the outcome, head neck cancer, for risk factors and possible interaction between these covariates based on race may be difficult without taking into account the interaction based on the variable race by strata if necessary.

The new measure value is -5.01 for strata 1 in INHANCE (non-central). For the black race, the new measure is -12.19, and -15.91 for Hispanic. The corresponding survival time for strata 1 is .80; strata 2 has survival time of .95 for black race; Hispanic race has survival time for event of .98 and new survival time equation (11). In this case $\beta z = -1.532$ and $\beta y = -1.422$. The variability of By is not limited to the binomial distribution and the outcome statistic can be significant for handling more heterogeneity with three confounding levels of race where data is non-normal for testing hypothesis for extreme probabilities that are nonnormal.

A New Measure for Heterogeneity The survival statistic of $-HR'(z)/P(z)$ allows the first strata's hazard function for all survival calculations (table 9) the value is -.868 for non-Hispanic. This new statistic demonstrates that the measure of protection for negative derivative of Hazard Ratio over probability of the event with respect to confounder is lowest because it is -.868 for non-Hispanic subjects. For black race, the measure is -.179 or next lowest; then Hispanic group has (strata 3) a value of -.038 respective to first group. Using the standard definition of $P = z/1 + z$ in the place of $P(z)$ in [Agravat \(2011\)](#) and hazard ratio equation (3), the hazard ratio calculated compares 2.35 is calculated that compares to the odds ratio value 2.3. The baseline and equation for hazard ratio equations of the author are more sensitive. In fact the possibility of calculating risk based on no harm assumption may be a paradox in time because the baseline and stratified values that are based on actual values rather than assumption of standard errors being equal when regarding the subjects is better understood from the interaction of the exposure nonsmoking vs. nondrinking for head neck cancer and race and effect modification and confounding. Perhaps, there may be more factors over all to produce this risk from non-smoking for head neck cancer in black race other than race such as the exposure for inferences that need to be analyzed at stratified levels of non-normal data. The Hispanic race has slightly higher statistic versus strata 1 or 2. The survival time gives a measure of length of survival at time = 0 to event (at baseline) or a

potential fixed time point. Still the survival times are lowest in non-central Europe, .80; then, next is the black race with .95, and highest with Hispanic a survival time of .99 though the new measure shows that other new survival measures discussed imply that the events and survival are actually different at baseline (see tables 9 and 10). The new statistic, $-HR'(z)/P(z)/S(z)$ shows a nonlinear relationship a measure of heterogeneity due to race. This new statistical measure of risk versus probability compared to survival time gives a value of -1.07 for central European group, -.188 for black group, and -.038 for Hispanic group where greater risks among black, and non-Hispanic vs. Hispanic for baseline hazard vs. probability respectively exist. One may state that the difference in the new measures exist for the strata 1 where the statistics have values of -.868 to -1.07 for the the first strata and the stratified comparisons shows little difference hence there may be more heterogeneity for the first strata for non-central European race (table 9 and 10) that has higher hazard ratio of .216.

If the individual strata is compared independently, then the distribution analysis shows greater differences for non-central European race. The new measure for strata's: 1, -5.01; 2 or black group is -12.19; 3, and -15.91 for Hispanic group. The corresponding measure compared to Probability of event is -.868, -.437, and -.122 for strata's one, two and, three races being non-Hispanic, black, and Hispanic groups. This statistic versus probability compared to survival time is -1.07, -.46, and -.123 for the non-Hispanic, black, and Hispanic groups. Compared to the view of the first strata which may involve bias the results show less risk for head neck cancer from nonsmoking due to race, and is worse for black subjects and Hispanics compared to probability and actual strata's hazard functions which is alarming because the values are closer to 0 that hazard ratios are in fact reversed for seriousness of event from hazard ratios. Confounding exists by race $P < 0.0001$ in "acov" variable for chi-square showing an increase in heterogeneity. This negative relationship with confounding, "negative confounding," may decrease the prognosis of subjects because of the expected increase of extra variables that may have an impact in head neck cancer and exposure nonsmoking vs. nondrinking. Compared to Survival time, the difference are more negative values for black and Hispanic subjects and higher risk for non-central European group due to hazard ratios (see table 9 and 10).The $S(z)$ is more sharp for the latter groups. All races treated independently show a worse scenario of baseline hazard vs. probability and survival than based on the first strata a statistic needed to be followed more closely (see table 9 vs table 10). Since the confounding and issue of interaction of the new calculated P values for effect modification and confounding including both the chi-square and F-statistic and O_{cinf} statistic there has to be more close attention to the risks and for interaction of the exposure non-smoking vs non drinking than non-drinking vs. non-smoking for baseline hazard.

In this case-control study (INHANCE), there is a possibility of residual confounding where there are residual differences between cases and controls. In one step where nondrinkers or nonsmokers are compared in one datable versus each other, the relative risk is .23 in a fixed effects analysis using the logistic model for cases over controls Hashibe et. al.(2007). In the random effects analysis Hashibe et al.(2007), for never smokers to never drinkers directly the relative risk is .33 adjusted for age, sex, race, education, center, and years of smoking cigars or pipes. This analysis with hazard ratios for strata one for race compared to the relative risk .23 is similar question of never smokers compared to the question of head neck cancer for race being non-Hispanic or largely central European population. Strata two has hazard ratio equal to .066 for black race that were a small segment of the various populations studied. The Hispanic segment had hazard ratio equal to .01 compared to the same datable from INHANCE for fixed effects and are different across strata by much more than 10 percent. In that analysis of the case-control study, race is compared to the status of being ever drinkers vs. never smokers for head neck cancer who never used tobacco or were never drinkers.

The analysis of head neck cancer by exposure nonsmoking supports heterogeneity existing. The Shapiro-Wilks test shows non-normal data for outcome of cases of head neck cancer $P < 0.0003$. The

Table 8: Probability, Hazards and New Distribution Analysis for INHANCE and Non-smokers Based Compared to Race Non-Central European

Strata	P(z)	HR(z)	-HR'(z)	-HR'(z)/ P(z)	S(z)	-HR'(z)/P(z)/S(z)	F(z)
(1)White	5.78	.216	-5.01	-.868	.805	-1.07	.19
(2)Black	27.91	.046	-5.01	-.179	.95	-.188	.05
(3)Hispanic	130.2	.010	-5.01	-.038	.99	-.038	.01

Table 9: Probability, Hazards and New Distribution Analysis for INHANCE and Non-smokers based on Individual Strata of Non-normal Data

Strata	P(z)	HR(z)	-HR'(z)	-HR'(z)/ P(z)	S(z)	-HR'(z)/P(z)/S(z)	F(z)
(1)White	5.78	.216	-5.01	-.868	.805	-1.07	.19
(2)Black	27.91	.046	-12.19	-.437	.95	-.46	.05
(3)Hispanic	130.2	.010	-15.91	-.122	.99	-.123	.01

Table 10: Traditional Hazards and Survival Analysis for INHANCE and Non-smokers Based Compared to Race Non-Central European

Strata	HR(z)	S(z)	F(z)	$\Lambda(z)$	$\lambda(z)$	hz0
(1)White	.216	.805	.19	.094	5.74	26.57
(2)Black	.046	.95	.05	.022	22.54	490
(3)Hispanic	.010	.99	.01	.004	100.08	10008

type 2 tests are significant for fixed effects thus rejecting fixed effects model for "acov". The chi-squares are equal at 63.46 and F-statistics hence the denominator is approaching infinity explains the equivalence of the value. The type 3 effects model is also rejected for equal Beta estimates to 0 hence one suspects random effects because the $P < 0.0001$ for chi-square and F-statistics for "acov", therefore again one suspects a heterogeneous model to explain the effects of head neck cancer by exposure non-smoking for race and the O_c statistics is appropriate for calculating asymptotic chi-square for this heterogeneous data. Since the probability is also greater than one, one suspects that there is non-normal probability and a binomial formula for linear probability is not appropriate and the equation is appropriate based on independence proof of the author [Agravat\(2011\)](#).

The population is large for this study strata about 11592 still the Breslow-Day fixed effects test for testing homogenous odds null is not appropriate because this is a random effects model case that may be needed. Since there is effect modification and confounding as supported by P values for "aem" and "acov" one reports the stratified hazard ratios for hazard ratios and the subsequent statistics for hazard distribution available with the author's derivation as shown in tables 9 and 10. The baseline hazard function vs. probability shows a different trend. The measure shows that the black race group has a low value of baseline hazard vs. probability -.437 for black followed by -.868 for non-Hispanic and -.122 for Hispanic. Perhaps there is a difference in treatment or availability, or possibly prevention due to the risk of smoking and nonsmoking for head neck cancer by race in the International Head Neck Cancer Epidemiology study by race. Table 9 presents the survival statistics according to the effects for race being white for head neck cancer and nonsmoking that can be considered fixed by race being white and still random. Table 10 renders survival statistics that are appropriate for race and random effects since heterogeneity is shown to exist as well as effect modification by exposure non-smoking for race (for chi-square $P < 0.0001$, F-statistic $P < 0.01$) and confounding for chi-square $P < 0.0001$, F-statistic $P < 0.0001$ by race for the outcome head neck cancer. The baseline hazard, hz0 has power of 74 percent when comparing the outcome for strata and link =log for igaussian distribution with PROC GENMOD with $P < 0.0091$. Hence there is a statistical significance for strata and baseline hazard without $P(z)_{new}$ in the model as a predictor. The utilization of the first strata is comparable to binomial statistical analysis because the results are directly compared to one strata or not while the latter is multivariate and reflective of each individual strata.

3.1 Probability of Mutually Exclusive Statements and Agravat's Algorithm

$$P(z \cup t) = \frac{(1) \div (odds(z)) - (odds(y))}{(1 - odds(y))} - (2lnz(t+1)) \div (P(z) - 1) \quad (90)$$

$$P(z \cup t) = P(z) + P(t) \quad (91)$$

$$P(z|t)=P(z \cap t) \div P(t) \quad (92)$$

$$P(z|t)=(z - t) * (z - t) \div (z - t)^z \quad (93)$$

$$P(z|t)=z^2 - 2z * t + t^2 \div (z^{2z} - (2z * t)^z + t^{z^2}) \quad (94)$$

$$P(z|t)=2ln * z(1 - t) + 2lnt \div (4zln * t - 2z * lnzt) \quad (95)$$

$$P(z|t)=2lnz + 2zln t \quad (96)$$

$$\boxed{P(z|t) = -2lnz(t + 1)} \quad (97)$$

$$P(t)=[P(z \cap t)] \div (P(z|t)) \quad (98)$$

$$P(t)=[P(z) + P(t)] \div (-2lnz(t + 1)) \quad (99)$$

$$P(t)=[(P(z|t))] \div (P(z) - 1) \quad (100)$$

$$\boxed{P(t)=[(-2lnz(t + 1))] \div (P(z) - 1)} \quad (101)$$

$$P(z \cup t)=[P(z) + P(t)] \quad (102)$$

$$P(z \cup t)=\frac{(1) \div (odds(i * z)) - (odds(y))}{(1 - odds(y))} - (2lnz(t + 1)) \div (P(z) - 1) \quad (103)$$

The derivation of Probability of time and z the odds (z) or confounder obtained from Agravat's algorithm [Agravat \(2011\)](#) of probability that is conditional is derived as demonstrated where z and t are mutually exclusive equations (42,44). This statement is similar to random effects. The inferences are also related to the random effects model. From the probability statements, the new probability formulae for probability of z given time and time are derived that may be useful for many applications of survival statistics equation (44). The potential is there but the risk of varying the outcome y for a fixed non zero level of z shows that time approaches 0 as time increases and y increases with z hence the problem of risks that may burden any equilibrium or homeostasis stressing the body beyond its limits (see figure 10). The change in hazard ratios is: 1.59 vs. .21 for non-Hispanics; 2.53 vs. .046 for blacks; and 3.53 vs. .017 for Hispanics with respect to time 0. Of course, if the time becomes 0 then there is nothing that one can do to the subject to improve the human condition though one can measure the probability in the study for the confounder and time in term of random effects. For fixed effects the probability substituted likewise for P(z) by $P(z \cap t)$, the probabilities for strata 1,2,3 are 5.78, 27.91, and 130.2. The hazard ratios change to .187, .084, and .016 at time 0. Each group maintains improvement a decrease in hazard ratio after study versus at time 0 which show a significant change.

3.2 Tenets on the The Nature of Time Theorem Under Independence, and Agravat's Algorithm

$$P(z \cap t)=P(z) * P(t) \quad (104)$$

$$P(z \cap t)=\frac{(1) \div (odds(i * z)) - (odds(y))}{P(z) \div (P(z) - 1)} \quad (105)$$

$$P(z \cap t)=P(z)^2(P(z) - 1) \quad (106)$$

$$P(z|t) = \frac{1}{(P(t)^2 - 1)} + 1 = P(t) \quad (107)$$

$$1 = P(t) - \frac{1}{(P(t)^2 - 1)} \quad (108)$$

$$1 = \frac{P(t)^3 - P(t)}{P(t)^2 - 1} \quad (109)$$

$$P(t) + P(t)^2 - 1 + 1 = 1 \quad (110)$$

$$P(t) * (P(t) - 1) = 1 \quad (111)$$

$$\boxed{P(t)^2 + P(t) - 1 = 0} \quad (112)$$

$$\boxed{P(z|t) = \frac{(z-t) * (z-t)}{(z-t)^z}} \quad (113)$$

$$\ln P(z|t) = \ln(z) - \ln(zt) + 2\ln(t) - 2z\ln(z) + 2z\ln(zt) + 2z\ln t \quad (114)$$

$$= (\ln(2 - 2z) - 1) + 2\ln t + 2z\ln zt + 2z\ln t \quad (115)$$

$$= (\ln z(1 - 2z)) + 2\ln t + 2\ln z + 2z\ln z + 2z\ln z \quad (116)$$

$$= (\ln z - 2z\ln z) + 2\ln t + 2\ln z + 2z\ln z + 2z\ln t \quad (117)$$

$$= (3\ln z + 2\ln t + 2z\ln t) \quad (118)$$

$$= (3\ln z + 2\ln t(1 + z)) \quad (119)$$

$$\ln P(z|t) = 3\ln z + 2\ln t(1 + z) \quad (120)$$

$$\ln P(z|t) = 3\ln z + 2\ln t + 2z\ln t \quad (121)$$

$$P(z|t) = \frac{P(z) * P(t)}{P(t)} = P(z) \quad (122)$$

$$\ln P(z|t) = \ln P(z) \quad (123)$$

$$\ln P(z) = 3\ln z + 2\ln t + 2z\ln t \quad (124)$$

$$\exp^{\ln P(z)} = \exp^{3\ln z} + \exp^{2\ln t} + \exp^{2z\ln t} \quad (125)$$

$$P(z) = z^3 + t^2 + t^{2z} \quad (126)$$

$$P(z) - z^3 = t^2 + t^{2z} \quad (127)$$

$$P(z) - z^3 = t^2(1 + t^z) \quad (128)$$

$$t^2 = \frac{P(z) - z^3}{(1 + t^z)} \quad (129)$$

$$t = \sqrt{\frac{P(z) - z^3}{(1 + tz)}} \quad (130)$$

$$t^2(1 + tz) = P(z) - z^3 \quad (131)$$

$$\ln t^2(1 + tz) = \ln P(z) - \ln z^3 \quad (132)$$

$$t^2 = \frac{P(z) - z^3}{\exp^{1+tz}} \quad (133)$$

$$t = \sqrt{\frac{P(z) - z^3}{\exp^{1+tz}}} \quad (134)$$

If Probability of event given time is based on classic laws, then using the Agravat's $P(z)$ new as probability equation, and equation for $P(t)$, one can set the equation to 1 and mutually exclusive events laws gives that the probability of time is hypergeometric and involves complex numbers. The solution of $P(z|t)$ using the new conditional probability algorithm [Agravat \(2011\)](#) shows it is proportional to square root of $P(z)$ minus z cubed divided by $1 + t$ to the z power. Equation (123) shows that the probability of event given time equals probability of event and supports the independence assumption of Bayes. As $\exp(y)$ and $\exp(z)$ become larger, time becomes complex and for negative values of exponential of z and y , time is positive and probability of z is large (see table 11). For the time squared approaching 0 values of energy is decreasing along with probability of event (see figure 13). Time is largely involving complex numbers and roots that increase with decrease of probability.

$$\frac{P(z)}{P(t)} = P(z|t) - 1 \quad (135)$$

$$P(t) = \frac{P(z)}{(P(z|t) - 1)} \quad (136)$$

$$P(t)(P(z) * P(t) - 1) = P(z) \quad (137)$$

$$P(t)^2 * P(z) - P(t) = Pz \quad (138)$$

$$P(z) * P(t)^2 - P(t) = P(z) \quad (139)$$

$$P(z) = \frac{P(t)}{(P(t)^2 - 1)} \quad (140)$$

$$P(z|t) = \frac{P(z)}{P(t)} + 1 \quad (141)$$

$$P(z|t) = \frac{P(t)}{(P(t)^2 - 1)} P(t) + 1 \quad (142)$$

$$P(z|t) = \frac{1}{(P(t)^2 - 1)} + 1 \quad (143)$$

$$\frac{P(z)}{P(t)} + 1 = \frac{1}{(P(t)^2 - 1)} + 1 \quad (144)$$

$$P(z) = \frac{P(t)}{(P(t)^2 - 1)} \quad (145)$$

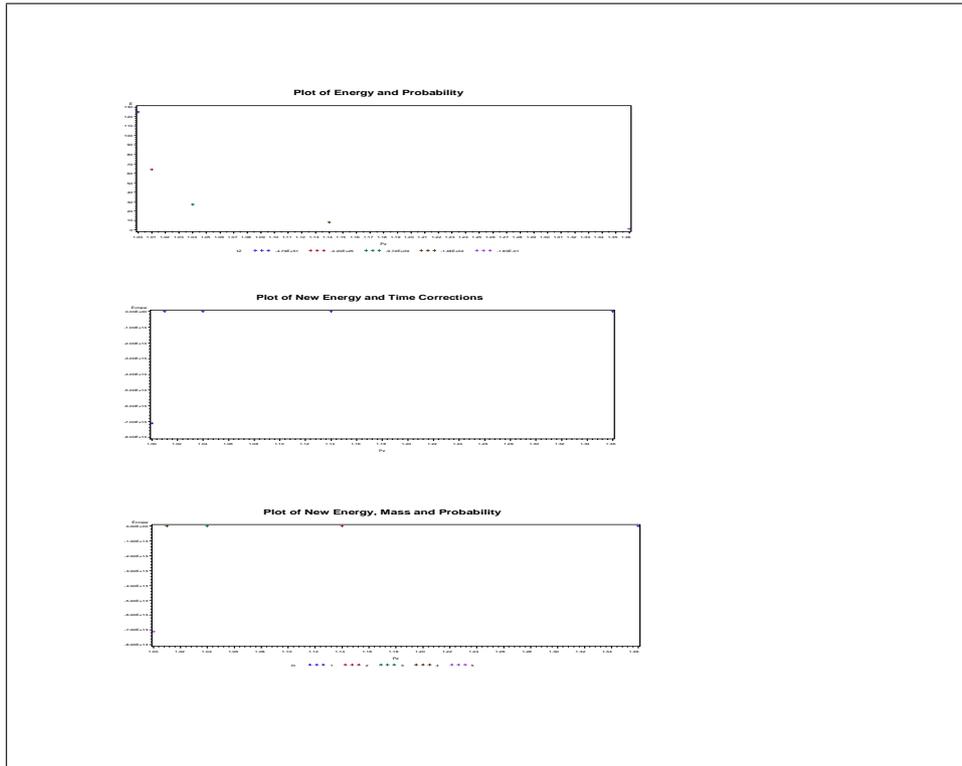


Figure 12: Plot of Energy, Energy Correction, Probability of Event, and Time Squared

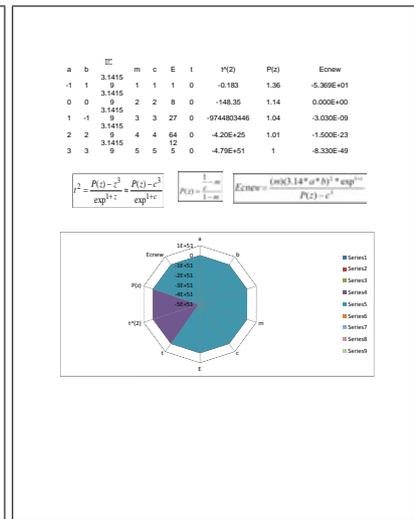
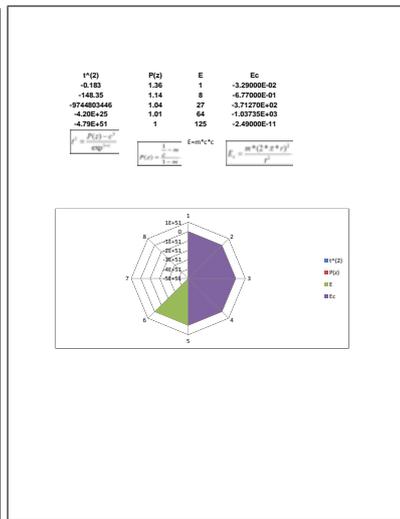
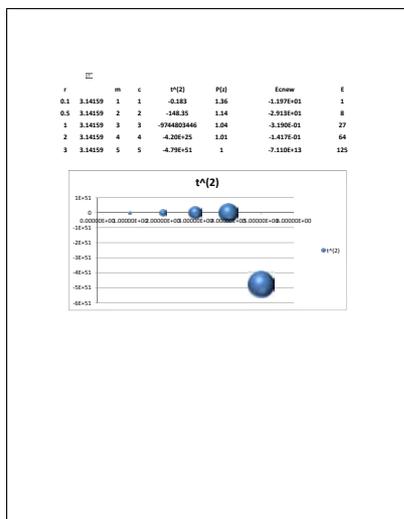


Figure 13: Agravat's Graph of Na-
ture of Time

Figure 14: Agravat's Plots of Spe-
cial Relativity vs. Correction

Figure 15: Plots for New Special
Relativity Correction

Table 11: Values of Time Squared and Probability for Agravat's Probability of Time Formula and Beta Estimates

Probability	Y	Z	Time
8.38	-2	-2	4.77
3.718	-1	-1	1.83
Omega	0	0	Omega
1.367	1	1	$\sqrt{(-.1828)}$
1.13	2	2	$\sqrt{-20.02}$
1.049	3	3	$\sqrt{-148.39}$
1.01	4	4	$\sqrt{-1096}$
1.006	5	5	$\sqrt{-8103.0}$

$$P(z|t) \div (P(t)) = \frac{-2 \ln z(t+1)}{\frac{-2 \ln z(t+1)}{\frac{P(t)}{(P(t)^2 - 1)}}} \quad (146)$$

$$P(z|t) \div (P(t)) = \frac{P(t)}{(P(t)^2 - 1)} \quad (147)$$

$$P(z|t) = (P(t)^2 - 1) \quad (148)$$

$$P(z|t) \div (P(t)) = P(z) - 1 \quad (149)$$

$$P(z|t) \div (P(t)) = \frac{P(t)}{(P(t)^2 - 1)} - 1 \quad (150)$$

3.3 Inferences on Time and Probability of an Event and Special Relativity

The bending of light as shown in "Formulas Calculating Risk Estimates and Testing for Effect Modification and Confounding" [Agravat \(2011\)](#) is supported by the tangential movement of time as the probability algorithm of $P(z)$ new loses appropriateness due to higher values. The calculations involves using a probability formula such as Agravat's $P(z)$ new. Figure 13 shows that special relativity calculations values are not completely calculated by $E = mc^2$. The new correction shows a different relationship or distribution that is nonnormal vs. normal (special relativity) and more of the value from the parameters (see figure 14). Another plot shows energy approaches 0 as time squared approaches 0 and probability. Probability and energy values are higher and larger and different by magnitude as time squared approaches 0 for the author's corrections. According to "Formulas Calculating Risks and Testing for Effect Modification and Confounding" [Agravat \(2011\)](#) z being proportional to y or c (speed of light) to mass that may yield allowable values that are not destructive in reference to the Manhattan project. Hence the question of is there more energy on planets more suitable for life or less energy on planets where life is suitable may be asked. Still one may have to wonder is the universe actually contracting or expanding may be unanswered on this view in our solar system. The decrease in Probability of event

'z' ($P(z)$) increase with time squared as shown (in table 12) for negative parameters (table 12)! Table 12 shows that as time is hypergeometric as parameters become positive and time is positive for negative parameters. At certain values for the variables, E_{cnew} may have lower values than the standard $E = mc^2$ or greater as the powers increase because time calculations according to the author's corrections show increases in energy that are higher than Einstein's special relativity. $E = mc^2$ shows a normal distribution by Shapiro-Wilks's test for calculations. Table 12 has $P(z)$ having $P < 0.0011$ is non-normal and time has $P < 1.0$ meaning time is normal. Energy decreases with probability according to Einstein's Special Relativity but is constant with regards to special relativity correction (see figure 12) as well as for time squared. First of all there is no time according to Einstein's special relativity formula due to derivatives and limits canceling to 0 though the new correction of energy does contain time squared. The new correction actually shows that time squared and probability may be somewhat constant compared to energy in figure 12. One may support the assertion that matter in neither created or destroyed within limits. Second, with reference to *Poincaré's* three body problem, as applied by the author where time may exist for special relativity correction. Third, energy levels vary differently from Einstein's special relativity formula by magnitude and sign. In some points in space and time, energy may be more than the time-squared values and probabilities in that area allowing for diversity in the cosmos! Or the areas habitable like earth for life allow more time squared and probabilities at lower energies. the author's correction for special relativity shows that Einstein's special relativity may be appropriate for three body mass problem but not the whole solar system whose orbits are in ellipse.

```
data plotnewGSF12III;
  input Pz m c t2 Ecnw E;
datalines;
1.36 1 1 -0.183 -1.197E+01 1
1.14 2 2 -148.35 -2.913E+01 8
1.04 3 3 -9744803446 -3.190E-01 27
1.01 4 4 -4.20E+25 -1.417E-01 64
1 5 5 -4.79E+51 -7.110E+13 125 ;
run;
axis1 value=none c=blue i=j w=2 l=1;
axis2 value=none c=red i=j w=2 l=2;
proc gplot data=plotnewGSF12III;
plot E*Pz =t2 / haxis=axis1 vaxis=axis2;
title'Plot of Energy and Probability'; run;
axis1 value=none c=blue i=j w=2 l=1;
axis2 value=none c=red i=j w=2 l=2;
proc gplot data=plotnewGSF12III;
plot Ecnw*Pz Ecnw*t2 / haxis=axis1 vaxis=axis2;
title'Plot of New Energy and Time Corrections'; run;
```

$$(t_c)^2 = \frac{m(2 * \pi * r)^2}{E} \quad (151)$$

$$(t)^2 = \frac{P(z) - z^3}{exp^{1+z}} \quad (152)$$

$$E_c = \frac{(m) * (2 * \pi * r)^2}{t_c^2} \quad (153)$$

$$P(z) = \frac{\left(\frac{1}{c} - m\right)}{1 - m} \quad (154)$$

$$t^2 = \frac{\left(\frac{1}{c} - m\right)}{\left(\frac{1}{1 - m}\right) - c^3} exp^{1+c} \quad (155)$$

$$E_{cnew} = \frac{(m * (\pi * a * b)^2 * exp^{1+c})}{P(z) - z^3} \quad (156)$$

$$E_{cnew} = \frac{(m * (2 * \pi r)^2 * exp^{1+c})}{P(z) - z^3} \quad (157)$$

$$E_c = \frac{(\pi * a * b)^2 * (exp^{1+c}) * (m - m^2)}{(\frac{1}{c} - m - c^3)} \quad (158)$$

3.4 Inferences on Atomic Particles from Probability of Time and Events

$$c^2 = \frac{d^2}{t^2} \quad (159)$$

$$c^2 = \frac{(2 * \pi i * r)^2}{t^2} \quad (160)$$

$$c^2 = \frac{(2 * \pi i * r)^2}{\frac{P(c) - c^3}{exp^{1+c}}} \quad (161)$$

$$c^2 = \frac{(2 * \pi i * r)^2}{(\frac{P(c) - c^3}{exp^{1+c}})} \quad (162)$$

$$c^2 * \frac{(P(c) - c^3)}{exp^{1+c}} = d^2 \quad (163)$$

$$2 \ln(c) + \ln P(c) - 3 \ln c - c = d^2 \quad (164)$$

$$-\ln(c) + \ln P(c) - c = d^2 \quad (165)$$

$$-\ln(c) + \ln\left(\frac{\frac{1}{c} - m}{(1 - m)}\right) - c = d^2 \quad (166)$$

$$-\ln(c) + \ln\left(\frac{\frac{1}{c} - m}{(1 - m)}\right) - c = (2 * \pi * r)^2 \quad (167)$$

$$-\ln(c) + \ln c - c + \ln m - \ln m = (2 * \pi * r)^2 \quad (168)$$

$$c = - (2 * \pi * r)^2 \quad (169)$$

$$c = - (2 * \pi * 5.29^{-11})^2 \quad (170)$$

$$c = - 1.1x10^{-19} m^2 \quad (171)$$

Charge of Electron from New Time and Probability Calculations The importance of the time equations (eq.129 or 154), Probability of event P(z), and, and rate x time equals distance a basic law of physics is that the author is able to calculate a value similar to charge of an electron. In the case of the inferences referenced about less harm in [Agravat \(2011\)](#), one transforms P(z) into P(c) and recalculates c that may stand for charge of electron in this case is $-1.1x10^{-19}$ very similar to Millikan's estimate of $1.5x10^{-19}$. The author is able to use the value of accepted radii of an electron of $5.29x10^{-11}m^2$ (Bohr radius) from wikipedia of Bohr. The author believes that the centripetal force may hold the electron in orbit thus the Bohr radius becomes the pertinent radius to calculate the charge of the electron. The author may not be sure of the units but the charge is negative and the

magnitude is very similar. With the radius of the proton being $.8768x10^{-15}m$, equation (169) yields a value of $-3.63x10^{-29}m^2$ for its charge that is different from the same for 1 electron charge. This may be true because the proton is in the nucleus with neutrons. The electron divided by proton charge is 3030303030. The electron radius ($2.81x10^{-15}$) gives a value of $3.13x10^{-28}m$ for antiparticle of lepton. The quantum of charge of the electron may be $n * charge * mass * velocity$ in orbit. The mass of electron is $9.1x10^{-31}kg$. The calculation of electron speed is: 1) Bohr radius/time; 2) energy correction formula with P(c) as 0; 3). $E = n * m * v$; 4 the energy solved for may allow for calculation of velocity that yields $-1.43x10 - 59m^2 * kg^2/time^2$ (the units for time was not kept) for the first orbit. The time of the electron may be calculated and with P(c) as 0 gives $\sqrt{-.135}$ or $.3698i$. Velocity then becomes $\Delta D/time$ or $5.29x10^{-11}m/.3698(time) = -1.43x10 - 10m/time$ (perhaps a complex number i kept). The kinetic energy of electron in orbit 1 may be thus $-9.3x10^{-51}$. The proton radius of $2x10^{-16}$ yields a value of $1.579x10^{-30}$. Equation (172) yields a value of (with $-1.1x10^{-19}$ for charge) $-2.73x10^{-49}$ for energy. The numerator is same but Energy with velocity differs by a factor of $1/c^3$. The energy of electron according to equation (174) is $-9.34x10^{-20}kg^2 * m^2/time^2$ (or joules).

$$E = \frac{m * (2 * \pi * r)^2 * exp^{1+c}}{P(c) - c^3} \quad (172)$$

$$E = \frac{-2.73x10 - 49}{-c^3} \quad (173)$$

$$E = -9.34x10^{-20}kg^2 * m^2/time^2 \quad (174)$$

$$E = n * m * v \quad (175)$$

$$v_q = \frac{E}{n * m} \quad (176)$$

$$v = \frac{\Delta D}{time} \quad (177)$$

3.5 Agravat's Proof of Right Triangle Dimensions

$$E(Y) = P * Q = R \quad (178)$$

$$E(Y)^2 = (P * Q)^2 \quad (179)$$

$$E(Y)^2 = \hat{P}^2 * \hat{Q}^2 \quad (180)$$

$$(P * Q)^2 = \hat{P}^2 * \hat{Q}^2 \quad (181)$$

$$\hat{R}^2 = \hat{P}^2 * \hat{Q}^2 \quad (182)$$

$$\ln(P * Q)^2 = \ln \hat{P}^2 + \ln \hat{Q}^2 \quad (183)$$

$$\ln R^2 = \ln \hat{P}^2 + \ln \hat{Q}^2 \quad (184)$$

$$exp^{\ln R^2} = exp^{\ln \hat{P}^2} + exp^{\ln \hat{Q}^2} \quad (185)$$

$$\hat{R}^2 = \hat{P}^2 + \hat{Q}^2 \quad (186)$$

Based on the definition of the estimate of mean in the binomial distribution, the proof of $P^2 + Q^2 = R^2$ is shown that works for right triangles. Vector mathematics is utilized to give definition of sums. The estimate of means is utilized with vector sum to show this feature of right triangles.

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