International STD Research & Reviews
4(1): 1-14, 2016; Article no.ISRR.25609
ISSN: 2347-5196, NLM ID: 101666147

SCIENCEDOMAIN *international*
www.sciencedomain.org

Modeling the Global Public Health Determinants of HIV/AIDS-Related Deaths

Jandryle U. Trondillo[1*]

[1]College of Governance and Business, University of Southeastern Philippines, Obrero, Davao City, 8000, Philippines.

Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

Article Information

DOI: 10.9734/ISRR/2016/25609
Editor(s):
(1) Kailash Gupta, Division of AIDS, NIAID, NIH, USA.
Reviewers:
(1) Nélida Virginia Gómez, Buenos Aires University, Argentina.
(2) Anonymous, State University of Maringa, Brazil.
Complete Peer review History: http://sciencedomain.org/review-history/14109

Original Research Article

Received 11th March 2016
Accepted 24th March 2016
Published 9th April 2016

ABSTRACT

The alarming global incidence of HIV/AIDS epidemic prompted this research undertaking to explore several health indicators that lead to and mediate reported HIV/AIDS mortality. A number of studies attempted to explore and give light to the epidemic incidence, however, the understanding of the causality issue at the policy level is still immature. To address this issue, this paper took into account a number of health indicators linked to HIV/AIDS prognosis and explore its direct and mediating effect to the global mortality prevalence. In particular, utilizing the panel data gathered from World Health Organization (WHO) and World Bank data repository, this paper modeled the determinants of global HIV/AIDS mortality. Goodness-of-fit statistics revealed a satisfactory model fitting indices ($\chi 2$=12.711,>0.05, NFI=.966, CFI=.989, RMSEA=.064, SRMR =.044, and HOELTER=225 and 326 at 0.05 & 0.01, respectively). Furthermore, this paper developed a structural equations model to investigate quantitatively the interrelationships among identified exogenous variables and its effect on worldwide HIV/AIDS mortality. Three health indicators were identified to have indirect effect while two indicators have a direct effect on HIV/AIDS mortality. Further complicated dismantling and constructive designs are described based on the empirical findings of this paper. Mediation analysis for the final outcome is also outlined.

Keywords: HIV; AIDS; modeling; structural equations modeling.

**Corresponding author: Email: jandryle@gmail.com;*

1. INTRODUCTION

The global prevalence of Acquired Immune Deficiency Syndrome (AIDS) caused by Human Immunodeficiency Virus (HIV) has become the stumbling stone of developmental progress and one of the most talked about topic in global health governance [1,2]. Since the inception of HIV studies in 1980's, there is an abundance of literature talking about the etiology, mode of transmission, and organ-specific pathology specifically its mechanism to alter normal immune system functioning that made human life vulnerable to opportunistic infection. While technological breakthroughs in the development of antiretroviral treatments (ART) have confirmed its efficacy in suppressing HIV and AIDS-related intervention [3-6] the epidemic control remains disproportionately attained among countries [7].

Currently, based on the estimates of UNAIDS [6] there were 36.9 million people estimated worldwide who are living with HIV in the year 2014, which is 5 million higher compared to 29.8 million people that were appraised in 2001. The worldwide prevalence slayed 1.2 million of people in 2014, which is 42% lower compared to 2004. However, it is still the leading cause of mortality globally and the primary cause of death in the African continent. Alarmingly, there were reported 2 million cases of HIV infections in 2015, which corresponds to 5,600 cases of new infection daily. The circumstances of new infection were attributed to heterosexual transmission, although several risk factors vary. On the other hand, in some countries, it was reported that the epidemic is attributed to men who have sex with men (MSM), shared parenteral drugs, and commercial sex workers.

Even though HIV detection capability has improved over time, increasing the number of people to learn about their HIV status is still vital since nearly half of the total HIV-infected individuals are still naïve that they are infected [8]. This gives importance to the presence of testing and counseling facilities that are competent enough to handle HIV-related cases. HIV infection has led to the renaissance of tuberculosis infection (TB), especially in the African continent, and TB became the leading cause of mortality among people with HIV/AIDS worldwide [8]. Moreover, more than half (51%) of all adults living with HIV globally are women. In fact, HIV-related deaths are the leading cause of deaths among women of reproductive age [6], which are attributed to differential access to public health services, gender inequalities, and sexual violence. As a result, women are twice more likely to acquire HIV infection compared to males internationally. Additionally, there were 2.6 million children all over the globe living with HIV in 2014 with more than 200,000 new cases of HIV infection and 150,000 HIV/AIDS-related mortality [6].

Countries that are fighting to the concentrated HIV/AIDS epidemic are facing new challenges in the economic dimension [9,10]. The simplest explanation of the causality between health and the economy is through the productivity of the workers. Those who are healthier can be able to work more efficiently, both physically and psychologically [11]. Several pieces of evidence link this to numerous countries, but the largest part of those existing with HIV (97%) exist in low to middle revenue nations, predominantly in sub-Saharan Africa [12]. The upward relationship between lower socioeconomic status and HIV progress was well documented in a number of literature [13-15]. The majority who were identified to have HIV or at risk of HIV infection does not have access to facilities that offers preventive and curative services [12]. This makes HIV/AIDS a burning public health concern around the globe. Although there is a scarcity of literature talking about the link between the numbers of facilities that caters to HIV/AIDS cases, we can clearly say that there is an obvious link between the two.

The prevalence of HIV/AIDS disease levels varies accordingly between countries and population groups within a certain country as well. The variation was specifically observed in socio-demographic and health factors than in projections about the pandemic effects based on crude epidemiological data. It was also noted in several studies that the difference between cultures in terms of the utilization and availability of contraceptives, educational status, and access to sexually transmitted diseases (STD) and sexually transmitted infections (STI) facilities, and out-of-pocket health care spending can explain why there is disparity in the HIV/AIDS prevalence levels and mortality levels. The said differences can be reflected in the styles of living and social standards of people in a certain demographic group. This diversity is linked to a wide range of biological, socioeconomic, behavioral and demographic factors [16].

As discussed above, several studies have been shepherded in the worldwide scale and

particularly in sub-Saharan Africa talking about the risk factors associated with HIV infection. In a number of studies, the determinants or HIV/AIDS risk factors were grouped into two categories namely: sexual behaviors [17-20] and influential risk factors [21-24]. Accordingly, many kinds of literature determined the mechanism of HIV/AIDS spread as well as the prevention to contribute to a global decrease in incidence and prevalence. Most of the studies focus more on the clinical side of the topic and only a few kinds of literature dig deeper in the significance of policies and programs offered by individual public health services to contribute to the reduction of HIV/AIDS mortality ratio. One important parameter of policies is the availability of physical resources to cater to the growing needs of the individuals living with HIV.

Only a few researchers contribute to the body of knowledge about HIV/AIDS in relation to health policy, particularly on modeling the public health determinants of HIV/AIDS mortality. Thus, this study was conceptualized to emphasize the importance of existing public health services such as facilities and testing center, antiretroviral coverage provided by governments to people living with HIV, and even out-of-pocket health spending to enhance the lives of people living with HIV in the present time. The researcher explored these indicators and associated health outcomes and verified if such has a direct or indirect effect towards global incidence of HIV/AIDS-related deaths. Understanding this concept in an empirical manner will provide grounded evidence for policy makers to revisit their existing policies or device new policies to strengthen their public health spending particularly to public health services that cater to people living with HIV. Finally, this study explored the influence of the identified health indicators in relation to the accounted HIV/AIDS deaths in the global scale and an empirical model was developed as final output of this paper to illustrate relationships of exogenous variables to HIV/AIDS-related mortality and portray the direct and indirect effect of several health indicators as contributing factor to HIV/AIDS mortality.

2. METHODS

2.1 Data

Data for this study were gathered from the global health observatory resources of World Health Organization (WHO) [70] and World Bank [69] (WB) data repository of 193 UN member countries except Nauru and Vatican with lacking data from the six identified determinants of HIV deaths. A logarithmic functional form was used to achieve the best fit of both the exogenous and endogenous variables used in this study. The appropriate lag structure was selected according to the direction of the skewness and kurtosis of the original metadata acquired from World Health Organization data repository particularly HIV/AIDS-related statistical database and out-of-pocket healthcare spending from World Bank. Although it is not necessary to perform data transformation since the researcher utilized the population in the analysis of this study, still, the researcher believed that normal distribution would be better for ease of analysis and interpretation even though the distribution is assumed normally distributed. A brief description of the data used, which was adopted from the data source is discussed below.

- $Faci_{Log}$ is the lagged logarithm of estimated number of testing and counseling facilities per 100 000 adult population in UN countries averaged from 2010 to 2011 acquired from the global health observatory of World Health Organization.
- OOP_{Log} is the lagged logarithm of out-of-pocket expenditure as a percentage of total expenditure on health from 1995 to 2013 acquired from World Bank data repository.
- $Test_{Log}$ is the lagged logarithm of estimated number of people aged 15 years and over who received HIV testing and counseling per 1000 adult population from 2012 to 2013 gathered from World Health Organization global health observatory.
- $Preg_{Log}$ is the lagged logarithm of estimated percentage of pregnant women living with HIV who received antiretrovirals for preventing mother-to-child transmission in 2013 from WHO global health observatory.
- $Child_{Log}$ is the lagged logarithm of reported number of children receiving antiretroviral therapy in the year 2012 from World Health Organization global health observatory.
- Cov_{Log} is the lagged logarithm of estimated antiretroviral therapy coverage among people living with HIV in percentage for the year 2013 gathered from World Health Organization global health observatory of data.
- $Death_{Log}$ is the reported number of deaths due to HIV/AIDS averaged from 2001 to

2013 and acquired from the global health observatory of World Health Organization.

In the following chapters, the researcher will be discussing the model fit, particularly the use of the chi-square test based on the possibility of numerous assumptions indicated in several kinds of literature, mainly those which stipulates that the "sample size is sufficiently large" [25]. However, there is a deficiency of established criterion in the literature about the connotation "sufficiently large." Although there is no established range that prescribes the essential sampling size, it was recommended that the sample size should be at least larger than the number of covariances in the input data matrix [26]. The best ratio is 10 observations per factor, with an increment in the sampling size as the complexity of the model increases. In relation to this research, the path model has 7 determinants which correspond to 70 observations. Thus, since the researcher employed all UN countries in this study, it is safe to say that the number of observations is more than enough with recommended number [26].

2.2 Structural Equations Modeling

Structural equations modeling is an effective and comprehensive multivariate analysis procedure from the family of multivariate analysis. To be specific on the applicability of the tool, it functions more as an extension of a generalized linear model which permits researchers to test a set of regression equations altogether [27], and to concurrently explore the relationships between diverse variables [28]. Hence, grounded on the said assumption, this research utilized the above-mentioned statistical technique with the aid of Amos software to model the determinants of HIV/AIDS-related mortality in 193 UN countries. In the preliminary procedure done to carry out this study, the researcher utilized an OLS regression to determine the causal relationship of variables to a number of deaths due to HIV/ADIS. However, this study found out that even tough variables are independent of each other and there is no presence of correlation upon inspection of data; still, if there is an assumption that there might be existing mediating effects of variables, it can only be only be tested and detected utilizing a different statistical approach. As such, it was then realized that structural equations modeling is the best tool to explore these set of variables. To provide a clear picture of the above description of SEM, the hypothetical structural model for HIV/AIDS-related deaths globally was shaped in the proposed model as shown in Fig. 1.

3. RESULTS

3.1 Model Fitting

According to several published literature, it is not necessary to report all fit indices generated in the text output file of the AMOS software [29,26,30,31]. Several recommendations are being presented by several authors in reporting the goodness of fit of the model. Hooper, Coughlan, and Mullen [30] recommended the absolute fit indices which include Chi-Squared test, RMSEA, GFI, AGFI, the RMR and the SRMR. For this paper. the researcher opted to utilize the Chi-Squared test, Goodness-of-Fit Index (GFI), Adjusted Goodness-of-Fit Index (AGFI), Normed Fit Index (NFI), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), Standardized Root Mean Square Residual (SRMR), and Hoelter's [32] Critical N indices which utilizes both 0.01 and 0.05 parameters (see Table 1 in appendices section). Each will be discussed in the succeeding paragraphs.

The minimum discrepancy (CMIN) represented as $\chi 2$ is 15.999 with a probability value (*p*) of .067 which is insignificant at 0.05 alpha. $\chi 2$/DF was generated by dividing the value of $\chi 2$ by its df (9). The goal here is to yield a chi-square test that is more than the 0.05 alpha to accept the likelihood ratio that the data fits the hypothesized model. The $\chi 2$ value of 15.999 signifies the incongruity amongst the unrestricted sample covariance and the restricted covariance $\Sigma(\theta)$. In principle, it represents the likelihood ratio test statistic, which is usually conveyed as the model's $\chi 2$ statistic [29]. Generally, the null hypothesis is expressed as $\Sigma = \Sigma(\theta)$ which is equivalent to the hypothesis that $\Sigma - \Sigma(\theta) = 0$, and according to Bollen [33], testing the $\chi 2$ represents simultaneous testing of all residuals in $\Sigma - \Sigma(\theta)$ which is zero. In other words, the H0 assumes that the specification of the factor variances, factor loadings, and covariances (including error variances) for the hypothesized model presented in Fig. 1 is assumed valid in this case.

The Goodness-of-Fit Index (GFI) is a statistical measure of the relative amount covariance and variance in the S that is mutually explicated by

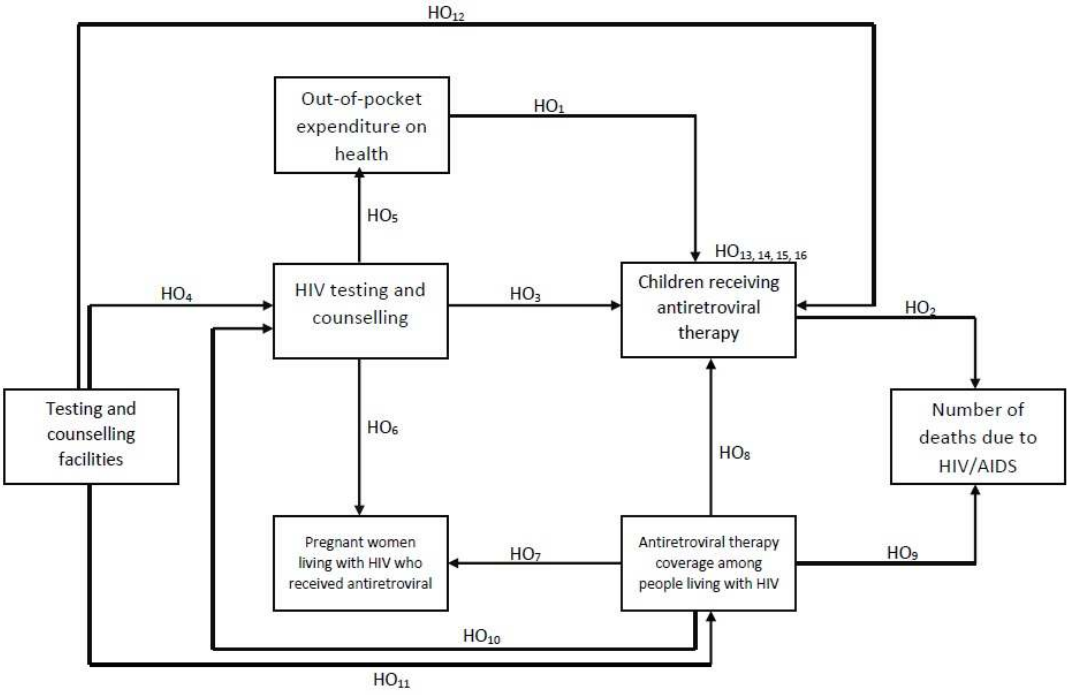

Fig. 1. Hypothesized structural equations model of HIV/AIDS-related mortality

Note: Non-shaded boxes and the hypothesized direction of effects are based on the literature examined in published studies

the Σ. Otherwise, the Adjusted Goodness-of-Fit Index (AGFI) just differ in the manipulation of degrees of freedom in the proposed model. Both addresses the issue on parsimony by integrating a punishment for the incorporation of more parameters and both can be categorized as absolute indices of fit since they fundamentally compare the proposed model with the null model [34]. Based on the findings of this paper, the GFI and AGFI values described in Table 1 (.977and .930, respectively), suggest that the proposed structural model fits the data utilized fairly well.

Moving on to the next column, the Normed Fit Index (NFI) criterion was presented as recommended by Bentler and Bonett. In some literature, it was discussed that the NFI has the capability to underestimate the fit in small samples [29], and since this study utilizes only 193 observations, Comparative Fit Index (CFI) in the following column was also presented since the latter takes sample size into account [35]. The values of both NFI and CFI range from 0 to 1 and are consequential from the comparison of the hypothetical model with the null model.

Hence, both provide a measure of wide-ranging covariation in the data. Bentler [36] originally proposed a value of greater than .90 in both NFI and CFI for the model to be considered a well-fitting model; however, Hu and Bentler [34] suggested that both indices should have a cutoff value of close to .95. In addition, CFI should be the index of choice in reporting as discussed above in relation to sampling adequacy [35]. Going back to Table 1, the CFI value is .991 which indicates that the hypothetical model fitted the data utilized. Considering the NFI (.980) which is above the .95 cutoff, the model can also be described as adequately fitted.

The following column provides the result for the root mean square error of approximation (RMSEA). This index was described as one of the most informative criteria in understanding the covariance of a structural model undertaking [29]. Steiger and Lind [37] originally proposed the index and it takes into account the approximation error in the population [38] which can be translated into a basic form of inquiry involving how well the model, with unknown but optimally chosen parameter values, would fit the population covariance matrix. The RMSEA measures this specific type of discrepancy paying particular attention to the sensitivity to the estimated parameters in the model like in the

case of this paper's hypothesized model which is very complex. Browne and Cudeck [38] indicated that RMSEA values which are less than .05 can indicate a good fitting model while values which are less than .08 are still tolerable (with reasonable errors of population approximation). On the other hand, values .08 up to .10 indicate a mediocre fitting model while values that go beyond .10 suggest a poorly fitting model [39]. Going back to the findings of this study, the RMSEA is .064 which can be interpreted that the proposed model is a well-fitting model. In the appendices section where the RMSEA table was presented completely, we can note that the confidence interval indicates that this study is 90% confident that the true RMSEA value in the population falls within the bounds of .00 to .114 indicating a tolerable degree of precision.

The last goodness of fit statistical criterion that this paper considered was Hoelter's [32] Critical N (CN) indices. This principle differs greatly from the discussed criteria since it focuses unswervingly on sampling adequacy than on the model fitting. Based on the development of this index, it rose from an effort to look for a fit index that can be independent of the size of the sample. Specifically, the index is for estimation of a sample that is sufficient enough to produce a suitable model fit with chi-square test [34]. A value of more than 200 is an indication of a model that has satisfactorily represented the sample data [32]. In this study, both the .01 and .05 Critical N values (column eight and nine) are greater than 200 (202 and 259, respectively), which imply that the hypothesized model's sampling adequacy was satisfactory and acceptable. Finally, on the basis of the nine goodness-of-fit measures presented above, this paper can very well conclude that the hypothesized model as shown in Fig. 1 fits the sample data gathered from World Health Organization (WHO) and World Bank satisfactorily.

3.2 Regression Weights

Table 2 presents the regression weights which contains the unstandardized coefficient estimates generated utilizing a maximum likelihood technique. Accompanying with each of the calculated unstandardized coefficients are its corresponding standard error (S.E.) and critical ratio (C.R.) value. The standard error represents the predictable variation of each estimated coefficients and is the index of "efficacy" of the predictor variables in estimating the endogenous variable, which in the case of this paper is the outcomes of death related to HIV/AIDS. This can be simplified into the idea that the smaller the S.E. means the more effective the exogenous variables is. Every C.R. value was calculated by dividing the parameter estimate to its respective standard error, and it is approximately distributed as the z [26]. Furthermore, a C.R. that falls outside ±1.96 designates a statistically significant path ($p<.05$). Utilizing this criterion, findings of this study reveal that all the exogenous variables are highly and statistically significant (see table 2 in appendices section) with all variables having less than 0.01 probability value. This implies that all the identified predictor variables contribute to the HIV/AIDS-related mortality at the international level.

Table 2 also presents the standardized regression weights (β) which represent the standardized coefficient estimates. The values presented in the table are independent of the units in which all variables are being measured [26]. The purpose of presenting this table is to demonstrate the direct relative relationship happening between the exogenous variables and the endogenous variables. The empirical result suggests that an increase in the number of testing and counselling facilities will significantly contribute to antiretroviral therapy coverage among people living with HIV and in the number of people aged 15 years and over who received HIV testing and counselling; while a reduction will consequence to an increased reported number of children receiving antiretroviral therapy ($\beta = 0.39$; $\beta = 0.52$; $\beta = -0.28$, respectively). For the estimated number of people aged 15 years and over who received HIV testing and counselling, an increase will also strengthen the reported number of children receiving antiretroviral therapy and estimated percentage of pregnant women living with HIV who received antiretrovirals for preventing mother-to-child transmission; while its decline will raise the out-of-pocket expenditure as a percentage of total health expenditure ($\beta = 0.35$; $\beta = 0.32$; $\beta = -0.28$, respectively).

In addition, significant growth of the antiretroviral therapy coverage among people living with HIV will rise the reported number of children receiving antiretroviral therapy, estimated percentage of pregnant women living with HIV who received antiretrovirals for preventing mother-to-child transmission, and estimated number of people aged 15 years and over who received HIV testing and counselling; while a significant reduction will

consequence on increased reported number of deaths due to HIV/AIDS (β = 0.32; β = 0.64; β = 0.22; β = -0.26, respectively). Lastly, improved out-of-pocket expenditure as a percentage of total health expenditure will result in a lessening of reported number of children receiving antiretroviral therapy, while increasing the latter will impact on increased percentage of reported number of deaths due to HIV/AIDS globally (β = -0.27; β = 0.98, respectively).

3.3 Empirical Model

3.3.1 Direct and indirect effects

Two exogenous variables have been found to have direct effect to the number of deaths due to HIV/AIDS namely estimated antiretroviral therapy coverage among people living with HIV (Cov_{Log}) and reported number of children receiving antiretroviral therapy ($Child_{Log}$). The direct effect of Cov_{Log} on $Death_{Log}$ is -.823 (*p=.012*). Thus, a percentage increase in Cov_{Log} will result to DeathLog's percentage to go down by 0.823. Moreover, the direct effect of $Child_{Log}$ to $Death_{Log}$ is .729 (*p=.025*). This implies that when $Child_{Log}$ goes up by 1 percent, the percentage of $Death_{Log}$ will also increase by 0.729. The direct (unmediated) effect of both Cov_{Log} and $Child_{Log}$ on $Death_{Log}$ are both statistically significant at the 0.05 level (two-tailed). This is a bootstrap estimation developed after creating the confidence intervals (bias-corrected). See Table 4 in the reference section.

In contrast, four variables have been identified to have indirect effect on HIV/AIDS global mortality estimate. First on the list is antiretroviral therapy coverage among people living with HIV (Cov_{Log}). The mediated effect of CovLog on $Death_{Log}$ is 1.283 (*p*=.007). This can be interpreted that when Cov_{Log} goes up by 1 percent, the percentage $Death_{Log}$ also goes up by 1.283. Next is the estimated number of people aged 15 years and over who received HIV testing and counseling ($Test_{Log}$). The indirect effect of $Test_{Log}$ on $Death_{Log}$ is .636 (*p*=.006). The relationship implies that a percentage increase in $Test_{Log}$, $Death_{Log}$'s percentage will upsurge by 0.636. Additionally, the effect of OOP_{Log} on $Death_{Log}$ is inverse (-1.402, *p=.026*). This suggests that when the out-of-pocket expenditure on health increases in percentage, the number of HIV/AIDS-related mortality in percentage goes down by 1.402. Finally, the last identified variable is a number of testing and counseling facilities ($Faci_{Log}$). However, the researcher opts not to report its mediated effect since the coefficient is not statistically significant (*p*=.997) at 0.01 or .05 level (two-tailed). The determination of statistical significance is made possible by the two-sided bias- corrected confidence intervals constructed.

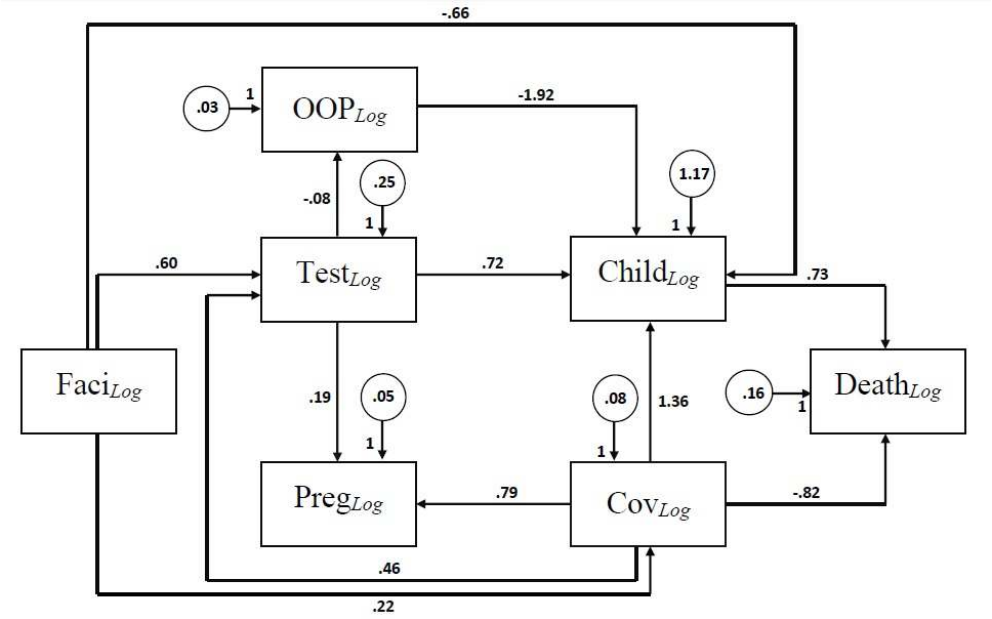

Fig. 2. Path model determining the HIV/AIDS mortality, unstandardized regression weights
Note: All paths are significant at 99% (p <0.01). See table 2 in appendices for specific value

3.4 Methodological and Theoretical Limitations

Data gathered from World Health Organization global health observatory contains a number of missing cases per country. To address the missing cases and to proceed with the data analysis, the researcher opted to perform maximum likelihood estimation using the Amos software feature by estimating means and intercepts in the estimation tab embedded in the AMOS software that utilizes the cases available to compute maximum likelihood estimates [40,41]. Nevertheless, the results generated provide unbiased parameter estimates and standard errors such as not having the necessity perform careful variable selection applied to impute values that are being carried out in a multiple imputation procedures. In addition, to minimize error in the data representation, especially in panel data, the researcher opted to acquire the mean value of the respective starting year until the last reported year of the global health observatory.

Like any other statistical methodologies, structural equations modeling has corresponding assumptions as elaborated in the book of Ho [26]. In this study, all the exogenous variables are independent to each other and the data represents a unique measurement. Next, sampling adequacy is not an issue in this study since the researcher opted to utilize the 193 UN member countries except Nauru and Vatican and the data used were observed. There is no latent variable in the model since it is more appropriate to make use of observed data for observations less than 200 as recommended to employ the structural equations modeling [42].

There is a scarcity of literature to further support the different null hypothesis of this research paper. However, a number of studies as specified in the literature review and author's personal knowledge of public health services support the different hypothesis established in the construction of the hypothetical model of this paper - in relation to the interrelationships of the different exogenous variables.

4. DISCUSSION

Both estimated antiretroviral therapy coverage among people living with HIV and reported a number of children receiving antiretroviral therapy have direct effect on the HIV/AIDS-related deaths. It was noted that antiretroviral therapy coverage among people living with HIV positively impacts HIV/AIDS mortality because as coverage increase, the number of deaths decreases. True enough, antiretroviral therapy has been reported to have been contributing to the improving the life expectancy of individuals living with HIV [43-45]. However, early initiation of the antiretroviral therapy can reduce rates of sexual transmission of HIV-1 and clinical events, demonstrating both public and personal health welfares from such the said therapy [46].

Although antiretroviral therapy showed to have a significant contribution to the quality of life of children living with HIV/AIDS [47,44] it doesn't guarantee that it will not result to death especially that the antiretrovirals are only for slowing the activity of the virus. It just provide a better quality of life and enhanced life expectancy while living with HIV [48]. Also, increasing the reported number of children receiving antiretroviral therapy significantly contribute to the accuracy of accounting the number of deaths due to HIV/AIDS globally as estimates are important in developing priorities for public health interventions. It is evident that there are countries that do not have a complete data of their reported number of children over the years and even lacking data at all.

Moreover, antiretroviral therapy coverage among people living with HIV on HIV/AIDS deaths has been found out to have a negative direct effect (-.823) and positive indirect effect (1.283) as mediated by reported number of children receiving antiretroviral therapy with a total effect of .461 ($p=.044$). Based on literature, antiretroviral therapy coverage plays an important role in the prognosis of individuals living with HIV and decreasing the transmission especially to low and middle income countries [49-55]. Thus, empirical findings suggest that antiretroviral therapy coverage should be strengthened across countries and that several effects should be developed to extract all cases of HIV infection incidence to ensure that all people living with HIV/AIDS can benefit to antiretroviral coverages. In less developed countries, it was observed that there is still a stigma about HIV, creating barriers to seeking antiretroviral coverage, thus increasing the incidence of death among HIV-infected individuals. Careful monitoring and accounting of the cases partnered with greater antiretroviral therapy coverage would improve the number of deaths due to HIV/AIDS. Also, since antiretroviral therapy coverage have direct ($Test_{Log}$, OOP_{Log},

$Child_{Log}$ and $Preg_{Log}$) and indirect (OOP_{Log}, $Child_{Log}$, and $Preg_{Log}$) effect to other factors, improving it will also improve the incidence of HIV among specific population groups and HIV/AIDS-related expenditure.

Additionally, the estimated number of people aged 15 years and over who received HIV testing and counseling exhibit an indirect effect to HIV/AIDS-related mortality and mediated the effect of antiretroviral therapy coverage to deaths having a total effect of .636 (*p=.006*). Several studies suggest that HIV testing and counselling correlates to decreased incidence of HIV/AIDs related deaths and transmission [56-66]. Indeed, the availability of the facilities catering HIV/AIDs testing and counseling would greatly affect the accuracy of accounting the actual number of HIV/AIDS-related incidence than of those who died with HIV unaccounted. Also, improving the number of people aged 15 years and over who received HIV testing and counseling will encourage safe sexual practices, thus, can positively contribute to the reduction of HIV incidence.

Lastly, the out-of-pocket expenditure as a percentage of total expenditure on health showed to have an indirect effect on global HIV/AIDS-related mortality with total a total effect of -1.402. This evidence signifies that there is a poor health care spending worldwide since there is an increased incidence of out-of-pocket spending. Ideally, governments should be the primary responders to cater to patients living with HIV. Governments should offer wellness to its constituents by ensuring that the out-of-pocket health spending is decreased [67,68]. Yes, it can be a good sign that when out-of-pocket health spending rises, the incidence of mortality related to HIV/AIDs decreases. However, this is a manifestation of poor public health policy making. Governments should invest in HIV/AIDS health programs and coverage to provide appropriate health care services to its constituents. This, in turn, would contribute to the economic stability as evidenced by a number of studies examined in a different publication.

5. CONCLUSION

To conclude, several health indicators were found to have an indirect and direct relationship towards the number of HIVAIDS related deaths on the global scale. The data significantly predicted the model ($\chi 2=12.711,>0.05$, NFI=.966, CFI=.989, RMSEA=.064, SRMR=.044, and HOELTER=225 and 326 at 0.05 & 0.01 respectively) with all corresponding paths statistically significant at 99% (*p*=.01). Three health indicators established in the hypothetical model were identified to have indirect effect to $Death_{Log}$ (Cov_{Log}, $Test_{Log}$, and OOP_{Log}) with all three variables significant at 0.05 alpha (0.007, 0.006, and 0.026, respectively) while two indicators have direct effect on HIV/AIDS mortality (Cov_{Log} and $Child_{Log}$); both are statistically significant at 95% (p=<.05). It is surprising that reported number of children receiving antiretroviral therapy has a positive correlation to the global mortality of HIV/AIDS-related deaths. However, analysis of this paper suggested that the relationship can be attributed to the problem of inaccuracy in reporting the number of cases causing a significant decrease in the observed cases of HIV/AIDS mortality. Thus, if there will be a more accurate recording of the number of children receiving antiretroviral therapy, the accuracy of reported cases of HIV/AIDS-related mortality will significantly increase as well. Accuracy in the reporting of the number of cases will lead to better prioritization and make health care policies.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. McInnes C, Lee K, (Eds). Framing Global Health Governance. Routledge; 2016.
2. Mondal MNI, Shitan M. Factors affecting the HIV/AIDS epidemic: An ecological analysis of global data. African Health Sciences. 2013;13(2):301-310.
3. Boyd MA, Cooper DA. Novel antiretroviral agents and universal access to HIV care. The Lancet HIV; 2016.
4. Granich R, Williams B. HIV treatment: Time to lean forward. The Lancet. 2016;387:10013.
5. Sheth AN, Ofotokun I, Buchacz K, Armon C, Chmiel JS, Hart RL, Palella Jr FJ.

Antiretroviral regimen durability and success in treatment-naive and treatment-experienced patients by year of treatment initiation, United States, 1996–2011. JAIDS Journal of Acquired Immune Deficiency Syndromes. 2016;71(1):47-56.
6. UNAIDS. Epidemiological slides – How AIDS Changed Everything report; 2015.
7. Maynard G, Ong C. Economic dependency and HIV/AIDS prevalence in the developing world: A comparative, longitudinal analysis. Sociological Inquiry; 2016. DOI: 10.1111/soin.12105
8. World Health Organization. Global tuberculosis report; 2015.
9. Haacker M. The macroeconomics of HIV/AIDS. International Monetary Fund; 2004.
10. Veenstra N, Whiteside A. Economic impact of HIV. Best Practice & Research Clinical Obstetrics & Gynaecology. 2005;19(2): 197-210.
11. Trondillo JU. Estimating the effect of maternal and child health outcomes to GDP per Capita. British Journal of Economics, Management and Trade. 2016;12(1):1-12
12. World Health Organization. Global HIV/AIDS response: Epidemic update and health sector progress towards universal access: progress report; 2011.
13. Ackermann L, Klerk GWD. Social factors that make South African women vulnerable to HIV infection. Health Care for Women International. 2002;*23*(2):163-172.
14. Muula AS. HIV infection and AIDS among young women in South Africa. Croat Med J. 2008;49(3):423-435.
15. Tladi LS. Poverty and HIV/AIDS in South Africa: An empirical contribution. SAHARA-J: Journal of Social Aspects of HIV/AIDS. 2006;3(1):369-381.
16. No CM. HIV risk factors: A review of the demographic, socio-economic, biomedical and behavioural determinants of HIV prevalence in South Africa; 2002.
17. Bassett MT, McFarland WC, Ray S, Mbizvo MT, Machekano R, van de Wijgert JH, Katzenstein DA. Risk factors for HIV infection at enrollment in an urban male factory cohort in Harare, Zimbabwe. JAIDS Journal of Acquired Immune Deficiency Syndromes. 1996;13(3):287-293.
18. Chao A, Bulterys M, Musanganire F, Abimana P, Nawrocki P, Taylor E, Saah A. Risk factors associated with prevalent HIV-1 infection among pregnant women in Rwanda. International Journal of Epidemiology. 1994;23(2):371-380.
19. Nunn AJ, Kengeya-Kayondo JF, Malamba SS, Seeley JA, Mulder DW. Risk factors for HIV-1 infection in adults in a rural Ugandan community: A population study. Aids. 1994;8(1):81-86.
20. Quigley M, Munguti K, Grosskurth H, Todd J, Mosha F, Senkoro K, Gavyole A. Sexual behaviour patterns and other risk factors for HIV infection in rural Tanzania: A case–control study. Aids. 1997;11(2):237-248.
21. Andoh SY, Umezaki M, Nakamura K, Kizuki M, Takano T. Correlation between national income, HIV/AIDS and political status and mortalities in African countries. Public Health. 2006;120(7):624-633.
22. Buve A, Carael M, Hayes R, Robinson NJ. Variations in HIV prevalence between urban areas in sub-Saharan Africa: Do we understand them? AIDS (London, England). 1994;9:S103-9.
23. Craven BM, Stewart GT. Economic implications of socio-cultural correlates of HIV/AIDS: An analysis of global data. Applied Economics. 2013;45(14):1789-1800.
24. Drain PK, Smith JS, Hughes JP, Halperin DT, Holmes KK. Correlates of national HIV seroprevalence: An ecologic analysis of 122 developing countries. JAIDS Journal of Acquired Immune Deficiency Syndromes. 2004;35(4):407-420.
25. Jöreskog KG, Sörbom D. LISREL 8: Structural equation modeling with the SIMPLIS command language. Hillsdale, NJ: Lawrence Erlbaum Associates; 1993.
26. Ho R. Handbook of univariate and multivariate data analysis with IBM SPSS. CRC Press. 2014;421-505.
27. Hooman HA. Structural equation modeling with LISREL application. Tehran: SAMT Publication. 2009;235-247.
28. Hoyle RH, (Ed). Handbook of structural equation modeling. Guilford Press; 2012.
29. Byrne BM. Structural equation modeling with AMOS: Basic concepts, applications, and programming. Routledge; 2010.
30. Hooper D, Coughlan J, Mullen M. Structural equation modelling: Guidelines for determining model fit. 2008;2.
31. Schreiber JB, Nora A, Stage FK, Barlow EA, King J. Reporting structural equation modeling and confirmatory factor analysis results: A review. The Journal of Educational Research. 2006;99(6):323-338.

32. Hoelter JW. The analysis of covariance structures: Goodness-of-fit indices. Sociological Methods & Research. 1983; 11:325–344.
33. Bollen KA. Structural equations with latent variables. New York: Wiley; 1989a.
34. Hu L-T, Bentler PM. Evaluating model fit. In Hoyle RH, (Ed.), Structural equation modeling: Concepts, issues, and applications. Thousand Oaks, CA: Sage. 1995;76–99.
35. Bentler PM. Comparative fit indexes in structural models. Psychological Bulletin. 1990;107:238–246.
36. Bentler PM. On the fit of models to covariances and methodology to the bulletin. Psychological Bulletin. 1992;112: 400–404.
37. Steiger JH, Lind JC. Statistically based tests for the number of common factors. Paper presented at the Psychometric Society annual meeting, Iowa City, IA; 1980.
38. Browne MW, Cudeck R. Alternative ways of assessing model fit. In Bollen KA, Long JS, (Eds). Testing structural equation models. 1993;136–162.
39. MacCallum RC, Browne MW, Sugawara HM. Power analysis and determination of sample size for covariance structure modeling. Psychological Methods. 1996;1: 130-149.
40. Allison P. Multiple imputation for missing data: A cautionary tale, sociological methods and research. 2000;28:301-309.
41. Hox JJ. A review of current software for handling missing data. Kwantitatieve Methoden. 1999;62:123-138.
42. Kenny DA. Measuring model fit; 2012.
43. Calcagno A, Montrucchio C, Capetti A, Guaraldi G, Cenderello G, Calza L, Avolio A. Raltegravir plus nevirapine as maintenance antiretroviral therapy in HIV-positive patients: Safety, efficacy and pharmacokinetics. Current HIV Research. 2016;14(1):54-60.
44. World Health Organization. Antiretroviral therapy of HIV infection in infants and children: towards universal access: recommendations for a public health approach-2010 revision. World Health Organization; 2010.
45. Zolopa AR, Andersen J, Komarow L, Sanne I, Sanchez A, Hogg E, ACTG A5164 study team. Early antiretroviral therapy reduces AIDS progression/death in individuals with acute opportunistic infections: A multicenter randomized strategy trial. PloS one. 2009;4(5):e5575.
46. Cohen MS, Chen YQ, McCauley M, Gamble T, Hosseinipour MC, Kumarasamy N, Godbole SV. Prevention of HIV-1 infection with early antiretroviral therapy. New England Journal of Medicine. 2011;365(6):493-505.
47. de Martino M, Tovo PA, Balducci M, Galli L, Gabiano C, Rezza G, Italian Register for HIV Infection in Children. Reduction in mortality with availability of antiretroviral therapy for children with perinatal HIV-1 infection. Jama. 2000;284(2):190-197.
48. Antiretroviral Therapy Cohort Collaboration. Life expectancy of individuals on combination antiretroviral therapy in high-income countries: A collaborative analysis of 14 cohort studies. The Lancet. 2008; 372(9635):293-299.
49. Boerma J, Stanecki KA, Newell ML, Luo C, Beusenberg M, Garnett GP, Zaniewski E. Monitoring the scale-up of antiretroviral therapy programmes: Methods to estimate coverage. Bulletin of the World Health Organization. 2006;84(2):145-150.
50. Bozzette SA, Joyce G, McCaffrey DF, Leibowitz AA, Morton SC, Berry SH, Goldman DP. Expenditures for the care of HIV-infected patients in the era of highly active antiretroviral therapy. New England Journal of Medicine. 2001;344(11):817-823.
51. Brinkhof MW, Dabis F, Myer L, Bangsberg DR, Boulle A, Nash D, Sprinz E. Early loss of HIV-infected patients on potent antiretroviral therapy programmes in lower-income countries. Bulletin of the World Health Organization. 2008;86(7):559-567.
52. Lima VD, Johnston K, Hogg RS, Levy AR, Harrigan PR, Anema A, McIntyre D, Thiede M, Dahlgren G, Whitehead M. What are the economic consequences for households of illness and of paying for health care in low-and middle-income country contexts? Social Science & Medicine. 2006;62(4):858-865.
53. Montaner JS, Hogg R, Wood E, Kerr T, Tyndall M, Levy AR, Harrigan PR. The case for expanding access to highly active antiretroviral therapy to curb the growth of the HIV epidemic. The Lancet. 2006;368(9534):531-536.
54. Montaner JS, Lima VD, Barrios R, Yip B, Wood E, Kerr T, Kendall P. Association of highly active antiretroviral therapy coverage, population viral load, and yearly

new HIV diagnoses in British Columbia, Canada: A population-based study. The Lancet. 2010;376(9740):532-539.

55. Montaner JS, Wood E, Kerr T, Lima V, Barrios R, Shannon K, Hogg R. Expanded highly active antiretroviral therapy coverage among HIV-positive drug users to improve individual and public health outcomes. JAIDS Journal of Acquired Immune Deficiency Syndromes. 2010;55: S5-S9.
56. Bwambale FM, Ssali SN, Byaruhanga S, Kalyango JN, Karamagi CA. Voluntary HIV counselling and testing among men in rural western Uganda: Implications for HIV prevention. BMC Public Health. 2008;8(1): 1.
57. Day JH, Miyamura K, Grant AD, Leeuw A, Munsamy J, Baggaley R, Churchyard GJ. Attitudes to HIV voluntary counselling and testing among mineworkers in South Africa: Will availability of antiretroviral therapy encourage testing? Aids Care. 2003;15(5):665-672.
58. De Paoli MM, Manongi R, Klepp KI. Factors influencing acceptability of voluntary counselling and HIV-testing among pregnant women in Northern Tanzania. AIDS Care. 2004;16(4):411-425.
59. Evans C, Ndirangu E. The nursing implications of routine provider-initiated HIV testing and counselling in sub-Saharan Africa: a critical review of new policy guidance from WHO/UNAIDS. International Journal of Nursing Studies. 2009;46(5):723-731.
60. Maman S, Mbwambo J, Hogan NM, Kilonzo GP, Sweat M. Women's barriers to HIV-1 testing and disclosure: Challenges for HIV-1 voluntary counselling and testing. AIDS Care. 2001;13(5):595-603.
61. Meiberg AE, Bos AE, Onya HE, Schaalma HP. Fear of stigmatization as barrier to voluntary HIV counselling and testing in South Africa; 2008.
62. Müller O, Barugahare L, Schwartländer B, Byaruhanga E, Kataaha P, Kyeyune D, Ankrah M. HIV prevalence, attitudes and behaviour in clients of a confidential HIV testing and counselling centre in Uganda. Aids. 1992;6(8):869-874.
63. Obare F, Fleming P, Anglewicz P, Thornton R, Martinson F, Kapatuka A, Kohler HP. Acceptance of repeat population-based voluntary counselling and testing for HIV in rural Malawi. Sexually Transmitted Infections. 2009; 85(2):139-144.
64. Shetty AK, Mhazo M, Moyo S, Von Lieven A, Mateta P, Katzenstein DA, Bassett MT. The feasibility of voluntary counselling and HIV testing for pregnant women using community volunteers in Zimbabwe. International Journal of STD & AIDS. 2005; 16(11):755-759.
65. Sweat M, Gregorich S, Sangiwa G, Furlonge C, Balmer D, Kamenga C, Coates T. Cost-effectiveness of voluntary HIV-1 counselling and testing in reducing sexual transmission of HIV-1 in Kenya and Tanzania. The Lancet. 2000;356(9224): 113-121.
66. Van Dyk AC, Van Dyk PJ. What is the point of knowing?: Psychosocial barriers to HIV/AIDS voluntary counselling and testing programmes in South Africa. South African Journal of Psychology. 2003;33(2):118-125.
67. Fairfield KM, Eisenberg DM, Davis RB, Libman H, Phillips RS. Patterns of use, expenditures, and perceived efficacy of complementary and alternative therapies in HIV-infected patients. Archives of Internal Medicine. 1998;158(20):2257-2264.
68. Yu D, Souteyrand Y, Banda MA, Kaufman J, Perriëns JH. Investment in HIV/AIDS programs: does it help strengthen health systems in developing countries? Globalization and Health. 2008;4(1);8.
69. The World Bank, World Development Indicators. Out-of-pocket health expenditure. Atlas method [Data file]; 2016. Available:http://api.worldbank.org/v2/en/indicator/sh.xpd.oopc.zs?downloadformat=excel
70. World Health Organization. Global Health Observatory resources. Atlas method [Data file]; 2015.
Available:http://apps.who.int/gho/data/node.resources

APPENDICES

Table 1. Goodness of fit statistics

Model	χ2	*p*	χ2/DF	GFI	AGFI	NFI	CFI	RMSEA	SRMR	HOELTER .05	HOELTER .01
Default model	15.99	.067	1.778	.977	.930	.980	.991	.064	.044	202	259
Saturated model	.000			1.00		1.00	1.00				
Independence model	802.42	.000	38.21	.475	.300	.00	.00	.441		8	10

a. Note: N=193, b. α ≤ 0.05

Table 2. Unstandardized regression weights of the direct model

			b (β) *Unstandardized*	*b* (β) *Standardized*	*Standard error*	*Critical ratio*
Cov_{Log}	<---	$Faci_{Log}$	.216***	.393	.037	5.900
$Test_{Log}$	<---	$Faci_{Log}$	..600***	.520	.070	8.588
$Child_{Log}$	<---	$Faci_{Log}$	-.660***	-.280	.179	-3.683
OOP_{Log}	<---	$Test_{Log}$	-.080***	-.281	.020	-4.040
$Child_{Log}$	<---	$Test_{Log}$	.719***	.351	.162	4.448
$Preg_{Log}$	<---	$Test_{Log}$	.192***	.324	.026	7.282
$Child_{Log}$	<---	Cov_{Log}	1.362***	.318	.286	4.768
$Preg_{Log}$	<---	Cov_{Log}	.794***	.640	.055	14.403
$Death_{Log}$	<---	Cov_{Log}	-.823***	-.258	.102	-8.028
$Test_{Log}$	<---	Cov_{Log}	.456***	.218	.127	3.591
$Child_{Log}$	<---	OOP_{Log}	-1.922***	-.267	.445	-4.318
$Death_{Log}$	<---	$Child_{Log}$	.729***	.981	.024	30.496

Source: *Author's calculation*

Table 3. Total, direct and indirect unstandardized effects of the direct model

	Effect	$Faci_{Log}$	Cov_{Log}	$Test_{Log}$	OOP_{Log}	$Child_{Log}$
Cov_{Log}	*Total*	.216				
	Direct	.216				
	Indirect					
$Test_{Log}$	*Total*	.699	.456			
	Direct	.600	.456			
	Indirect	099				
OOP_{Log}	*Total*	-.056	-.036	-.080		
	Direct			-.080		
	Indirect	056	-.036			
$Child_{Log}$	*Total*	.244	1.760	.872	-1.922	
	Direct	660	1.362	.719	-1.922	
	Indirect	904	.397	.153		
$Death_{Log}$	*Total*		.461	.636	-1.402	.729
	Direct		-.823			.729
	Indirect		1.283	.636	-1.402	
$Preg_{Log}$	*Total*	.305	.881	.192		
	Direct		.794	.192		
	Indirect	305	.087			

Source: *Author's calculation*

Table 4. Unstandardized two tailed significance (Bias corrected)

	Effects	$Faci_{Log}$	Cov_{Log}	$Test_{Log}$	OOP_{Log}	$Child_{Log}$
Cov_{Log}	*Total*	.009				
	Direct	.009				
	Indirect					
$Test_{Log}$	*Total*	.013	.018			
	Direct	.006	.018			
	Indirect	.020				
OOP_{Log}	*Total*	.015	.013	.012		
	Direct			.012		
	Indirect	.015	.013			
$Child_{Log}$	*Total*	.133	.009	.007	.028	
	Direct	.010	.006	.008	.028	
	Indirect	.010	.007	.017		
$Death_{Log}$	*Total*	.997	.044	.006	.026	.025
	Direct		.012			.025
	Indirect	.997	.007	.006	.026	
$Preg_{Log}$	*Total*	.010	.016	.006		
	Direct		.012	.006		
	Indirect	.010	.012			

Source: *Author's calculation*

Peer-review history:
The peer review history for this paper can be accessed here:
http://sciencedomain.org/review-history/14109