ZNPHI
ZAMBIA NATIONAL
PUBLIC HEALTH INSTITUTE

The Health Press

ISSN 2520-4378

VOL. 02
ISSUE 03
31 MARCH 2018

ZAMBIA'S JOURNAL ON PUBLIC HEALTH, DISEASE SURVEILLANCE, PREVENTION AND CONTROL

The Health Press - Zambia is published by Zambia National Public Health Institute, Ministry of Health Zambia since Jan 31, 2017.

Address: Plot 13, Reedbuck Road, Kabulonga, Lusaka.

Editor-in-Chief:
Ms Mazyanga L. Mazaba
Managing Editor:
Dr Raymond Hamoonga
Editorial Team:
Prof Paul Kelly
Prof Seter Siziya
Prof Mudenda Hangómbe
Dr Jeremiah Banda
Mr Stanley Banda
Dr Alwyn Mwinga
Dr Victor Mukonka
Dr John S Moran

Email: editor.heathpress@znphi.co.zm
Website: http://znphi.co.zm/thehealthpress/
Suggested Citation:
[Author Surname, Initial].[Article title].Health Press Zambia Bull 2018;02(3):[inclusive page numbers].

TABLE OF CONTENTS

CHRONIC KIDNEY DISEASE – SHOULD WE BE CONCERNED?

Editorial

By ML Mazaba [1]

1. Zambia National Public Health Institute, Lusaka, Zambia.

Correspondence: Mazyanga Lucy Mazaba (mazyanga.mazaba@znphi.co.zm)

Citation style for this article: Mazyanga ML. Chronic Kidney Disease – Should we be concerned?. Health Press Zambia Bull. 2018;2(3); pp 1-3.

For over 11 years, up to 90 countries have participated in celebrating World Kidney Day, a joint initiative of the International Society of Nephrology (ISN) and the International Federation of Kidney Foundations (IFKF). This year on the 8th March, health organisations, medical professionals, government officials and the general public joined in the awareness drive that was themed Kidney's & Women's Health – Include, Value and Empower [1].

Why the concern and need to create awareness?

Some Facts

Globally, an estimated 10% of the population is affected by chronic kidney disease (CKD) [2]. Millions of people die annually because they do not have access to affordable treatment [3]. While over 80% of all kidney failure patients on treatment are in developed countries [2], treatment with dialysis or kidney transplantation creates a huge financial burden for most of the patients in medium income countries, and sadly in up to 112 low income countries, the treatment is not affordable resulting in the death of over 1 million people annually from untreated kidney failure [3,4]. The number of kidney failure cases will increase more in developing than developed countries, such as China and India that are experiencing increased life expectancy [3].

Prevalence

Chronic Kidney Disease (CKD) is an increasing public health issue with an estimated prevalence between 6 and 8%; 14% among women and 12% among men. Every year about 195 million women worldwide are affected with CKD with an average of 600,000 deaths per annum currently placing it as the 8th leading cause of death among women [5,6]. Lozano et al. [8] indicated that CKD was the 27th then 18th most common cause of death globally in 1990 and 2010, respectively [7] and by 2015 had risen to be the 12th most common cause of death, recording a 31.7% increase in a decade from 2005.

In Africa - there is paucity in epidemiological information on CKD; more so in sub-Saharan Africa. A systematic review of 90 studies done in sub-Saharan Africa indicated an overall prevalence of 9% (95% CI 12.2 – 15.7). In Zambia, the CKD prevalence measured as proteinuria was estimated in the range of 4 – 24%. The review also revealed a substantial prevalence of CKD amongst HIV patients [9]. The prevalence of CKD is bound to increase with the increase in diabetes and hypertension in Zambia.

Risk factors & health problems resulting

According to the CDC in Atlanta, Diabetes and high blood pressure have been identified as risk factors for CKD, considering the high prevalence of these two non-communicable diseases, all should be concerned [10].

Kidney failure, heart disease and stroke are the common consequences of CKD. Anaemia, recurring infections, low calcium level, high phosphorous level, high potassium levels, loss of appetite, excess body fluids causing oedema and depression have also been listed as consequences of CKD. CKD is also considered a risk factor for adverse pregnancy outcome and reduced fertility. Following such conditions is more often premature death compared to persons without CKD [10].

Not all hope is lost

There are still opportunities to prevent CKD and its complications; control risk factors including high blood pressure and high blood sugar levels, monitoring for kidney disease among the high risk groups, continuous care and management of CKD through lifestyle changes and adherence to treatment are measures encouraged to prevent the diseases and its consequences. It is evident that following the causes, risk and prevention measures surrounding CKD that higher awareness, timely diagnosis and proper follow up of CKD is critical and must be encouraged. Response to these indications is critical for improved patient care and management as well as policy decision. Public health practitioners, health

care workers and policy makers have an important role in the prevention of Chronic Kidney Disease.

The Health Press Zambia (THP-Z) pledges to play a role in the awareness drive

In this issue, we feature two original articles: Determinants of foetal mortalities, and Phytoestrogens and early onset androgenic alopecia. The authors of the manuscript on determinants of foetal mortalities aimed at evaluating factors associated with foetal mortalities. They determined maternal age and parity being factors associated with foetal outcomes. The objective in the article on Phytoestrogens and early onset androgenic alopecia was to determine the association between phytoestrogen containing foods and early onset androgenic alopecia. The authors conclude that individuals who develop early onset androgenic alopecia have a lower consumption of soya foods. Please access their articles and enjoy the research findings of the authors.

On an exciting note, THP-Z has made good strides in its journey to get indexed on MEDLINE and other scholarly indexes following acceptance as a member of the Africa Journal Partnership Program. Refer to the letter from the Co-Director Annette Flanagin posted in this issue. An immediate benefit is increased visibility THP-Z as it is published on Africa Journal Online platform.

LIST OF REFERENCES

1. World Kidney Day. URL: http://www.worldkidneyday.org.

2. World Kidney Day: Chronic Kidney Disease. 2015. URL: http://www.worldkidneyday.org/faqs/chronic-kidney-disease/

3. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet 2013;382(9888):260-272.

4. Couser WG, Remuzzi G, Mendis S, Tonelli M. The contribution of chronic kidney disease to the global burden of major non-communicable diseases. Kidney Int 2011;80(12):1258-1270.

5. Institute for Health Metrics and Evaluation (IHME). GBD Compare Data Visualization. Seattle, WA: IHME, University of Washington, 2016. URL: http:// vizhub.healthdata.org/gbd-compare. URL: https://vizhub.healthdata.org/gbd-compare/.

6. Hill NR, Fatoba ST, Oke JL, Hirst JA, O'Callaghan CA, Lasserson DS, et al. Global prevalence of chronic kidney disease – a systematic review and meta-analysis. PLoS One 2016;11(7):e0158765.

7. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380(9859):2095-128.

8. Neuen BL, Chadban SJ1, Demaio AR, Johnson DW, Perkovic V. Chronic kidney disease and the global NCDs agenda. BMJ Glob Health 2017;2(2):e000380

9. Stanifer JW, Jing B, Tolan S, Helmke N, Mukerjee R, Naicker S, et al. The epidemiology of chronic kidney disease in sub-Saharan Africa: a systematic review and meta-analysis. Lancet Glob Health 2014;2(3):e174-81.

10. Centers for Disease Control and Prevention. National Chronic Kidney Disease Fact Sheet, 2017. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2017. URL: https://www.cdc.gov/kidneydisease/pdf/kidney_factsheet.pdf

AFRICAN JOURNAL PARTNERSHIP PROGRAM WELCOMES THE HEALTH PRESS

To the Editor - On behalf of the African Journal Partnership Program (AJPP), I write to welcome The Health Press into AJPP. The mission of AJPP is to promote wider dissemination of African health and medical research published in African health and medical journals. AJPP meets its mission by facilitating collaboration and mutual learning between African journal editors and international journal and publishing partners, improving the visibility of African research by working to get African journals accepted into MEDLINE and other scholarly indexes, improving editorial practices and the technical production capability of African journals, and supporting the training of medical and health editors, researchers, authors, reviewers, and journalists in Africa. AJPP is sponsored by the US National Library of Medicine and the Fogarty International Center with support from the Council of Science Editors, the Elsevier Foundation, African Journals Online (AJOL), Clarivate Analytics, Kaufman Wills Fusting & Company (KWF), SPi Global, 5 leading international journals (noted below), and many others.

AJPP, a small but mighty organization, operates with formal partnerships among the journal members. The Health Press will be partnered with JAMA and the Malawi Medical Journal (one of the founding African journal members of AJPP). Other partnerships include

- ***African Health Sciences, the Rwandan Journal of Medicine and Health Sciences, and The BMJ***
- ***Ghana Medical Journal, Sierra Leone Journal of Biomedical Research, and The Lancet***
- ***Ethiopian Journal of Health Sciences, Annals of African Surgery, and Annals of Internal Medicine***
- ***Le Mali Medical, Annales Africaines de Medicine, Environmental Health Perspectives, and the New England Journal of Medicine***

We hope AJPP will support The Health Press' vision "to address the challenges in making informed decisions of public health within Zambia and the global world in general."

Annette Flanagin, RN, MA, FAAN
Co-Director, African Journal Partnership Program
Executive Managing Editor
Vice President, Editorial Operations
JAMA and The JAMA Network
330 N Wabash Ave
Chicago, IL 60611
United States
annette.flanagin@jamanetwork.org

DETERMINANTS OF FETAL MORTALITY IN ZAMBIA

PERSPECTIVE

By H T Nyirenda[1], D Mulenga[1], H B C Nyirenda[2]

1. Copperbelt University, School of Medicine, Department of Clinical Sciences, Public Health Unit, Ndola, Zambia
2. The University of Zambia, School of Education, Department of Adult Education and Extension studies

Citation style for this article: Nyirenda H T, Mulenga D, Nyirenda H B C. Determinants of fetal mortality in Zambia. Health Press Zambia Bull. 2018 2(3); pp 5-16.

A pregnancy that does not terminate into a live birth is a public health concern. The aim of the paper was to determine factors associated with fetal deaths in Zambia.

This paper uses data from the 2013/2014 Zambia Demographic Health Survey (ZDHS) and used a cross sectional study design. The study was purely quantitative and was conducted through structured interviews. A representative sample of 18,052 households was drawn and interviewed women in the reproductive age group 15-49.

The results showed that only 5.3% women in Zambia had a fetal death. The multivariate logistic regression findings indicate that the odds of having a fetal death was 1.46 (CI: 1.20-1.79) higher for women whose health care was decided by their partner; increasing maternal age increased the odds of having a fetal death by 1.02 (CI: 1.01-1.03) and the odds of having a fetal death was lower for women who had children or a child alive.

Evaluating factors associated with fetal death makes it possible to recognize that interventions in some social, economic, demographic and maternal factors is key in the reduction and prevention of adverse birth outcomes such as fetal deaths.

Keywords: Fetal death, Determinants, Maternal health, factors,

Introduction

Fetal death is a public health challenge in the care of pregnant women worldwide, particularly in developing countries. Unlike in most developed countries, pregnancies in most developing countries are unplanned and usually present with complications which end up in adverse outcomes for both an infant and mother (1).

Fetal deaths are grossly underreported in most developing countries, and this makes comparisons difficult. This situation hinders attempts to adapt interventions and set health care priorities to meet local needs. For instance, researchers in Jamaica found that compared with 94 percent of live births, only 13 percent of late fetal deaths and 25 percent of infant deaths had been registered (2). While in Thailand, only 55 percent of infant deaths and none of the late fetal deaths were recorded in official registers (3).

Worldwide, the rate of fetal mortality varies considerably depending on the definitions applied for classifying fetal deaths (4). The most devastating adverse pregnancy outcome is when the pregnancy does not terminate into a live birth but ends up as an abortion or stillbirth. This is devastating for the mother but also of concern for clinical practice.

Fetal mortality is said to be an important indicator of the quality of antenatal and obstetric care (5,6). However, the contribution of other factors such as socio-demographic factors cannot be overlooked. Research in developing countries has been and is still being carried out to establish the factors associated with fetal death. In Zimbabwe results show that perinatal mortality is unacceptably high and associated factors vary across demographic subgroups (7,8). Other studies also indicate that socio-economic factors largely operate through proximate factors such as maternal biological, antenatal, and intrapartum factors (9).

The gap in fetal mortality between developing and developed countries (10) can only be reduced if preventable factors are identified and well addressed in developing countries. Most fetal deaths can be averted by implementing programmes and policies that support women's access to affordable and high-quality family planning, antenatal delivery and postnatal care (11). Therefore, In order to address the problem of fetal mortality in Zambia, there is need to identify factors associated with pregnancies that do not end up in live births. This study was carried out to determine factors that are associated with fetal death among women in Zambia.

Methods

Zambia covers a land area of 752,612 square kilometres. This study was conducted in Zambia's 10 provinces. The provinces include Central, Copperbelt, Eastern, Lusaka, Southern, Luapula, Muchinga, Northern, North-Western and Western Provinces.

This paper used data from the 2013/2014 Zambia Demographic Health Survey (ZDHS) which is a national sample survey designed to provide up-to-date information on health status and behaviour.

The study adopted a cross sectional study design targeting all women aged 15-49 who were either permanent residents

of the households or visitors present in the households on the night before the survey. It was purely quantitative and was conducted through structured interviews.

A representative sample of 18,052 households was drawn for the 2013-14 ZDHS to provide estimates at the national, provincial and regional (Rural/Urban) levels. An updated list of enumeration areas (EAs) for the 2010 Population and Housing Census provided the sampling frame for the survey comprising 25,631 EAs and 2,815,897 households. The survey used a two-stage stratified cluster sample design, with EAs (or clusters) selected during the first stage and households selected during the second stage. In the first stage, 722 EAs (305 in urban areas and 417 in rural areas) were selected with probability proportional to size. The sample was representative of women in the reproductive age group. The total number of women sampled and interviewed were 16,411. However, for this study, all women who were both nulliparous and had never had a fetal death were excluded from the study. This study also focused only on fetal deaths that occurred 5 years prior to the survey. Thus fetal deaths that occurred more than 5 years from the study period were also excluded from the study. Therefore, after the exclusion of the afore mentioned, the total number of women included in the sample for this study was 11, 486 and a weighted estimate of 11, 546 women in the reproductive age group was derived. Hence, all statistics presented under results reflect weighted numbers.

Fetal death which was a dependent variable is defined as a pregnancy that was terminated in a miscarriage, abortion, or still birth, or any pregnancy that did not result in a live birth.

The independent variables included respondents'; Age, Region, Years lived in place of residence, Highest educational level, Religion, Wealth index, Total children ever born, Number of living children, Currently/formerly/never in union, marital status, fertility preference (desired number of children) and Person who usually decides on respondent's health care.

Data analysis was done using Stata version 13 and the sample data was weighted in order to come up with population estimates. Bivariate analysis or Chi-square analysis was conducted in an attempt to describe and establish the relationship between fetal deaths and socio-economic and demographic factors. A multivariate logistic regression analysis was conducted to ascertain association between fetal deaths and socio-economic and demographic factors that were significant at bivariate analysis level.

Ethical Consideration

The paper used secondary data hence posed no risk or harm to the respondents. The data did not contain any of the respondent's names nor traces of the respondents. This paper, therefore holds respondents information with the highest confidentiality. Permission to use the data was sought from the Zambian Central Statistics Office (CSO).

Results

Socio-economic and Demographic Characteristics

Twenty one percent of the women were in the age group 25 to 29; 57.2% of the women were from rural areas; 51.7% had a primary education; 81.2% were protestant; 41.9% were rich based on the wealth index; 34.1% bore 5 and more children; 28.4% had 5 and more living children; three quarters (75.4%) were in a union; over half (56.1%) preferred having another child and 42.8% of the women's health care was decided by both the partner and themselves as a couple (Refer to table1).

Prevalence of fetal deaths

The results show that only 5.3 (612) percent of women in Zambia had a fetal death or a pregnancy that did not result in a live birth within 5 years prior to the survey.

Association between fetal deaths and socio-economic and demographic variables

The chi-square results in table 1 with a p-value less than 0.05 at 95% confidence interval (CI) indicate that there was a statistically significant relationship between each of the following independent variables and the dependent variable (fetal deaths); age of mother, years lived in a place of residence, children ever born, number of living children, marital status, fertility preference, person who makes decisions on the mothers health care. The percentage of women with fetal deaths increased with increasing age; more women in rural areas (5.5%) had fetal deaths compared to urban women (5.1%); 8.3% of women who lived in a place of residence less than a year had a fetal death; women with a higher education had a fetal death (6%); the percentage of fetal deaths reduced with increase in the number of children ever born and the number of children alive; 5.8% of fetal deaths were among women in a union; 6.5% of fetal deaths were among women who were undecided about fertility preference (undecided about having another child); and 11.4% of fetal deaths occurred to women's whose health care was determined by someone else. However, women's socio-economic characteristics such as; region, education status, religion and wealth index were not significantly associated with fetal deaths.

Table 1: Associations between socio-economic and demographic factors on one hand and fetal death on the other.

	Fetal death					
	No	No	Yes	Yes	Total	Total
	%	95% CI	%	95% CI	%	95% CI
Age in 5-year groups						
15-19	7.5	[6.9-8.1]	9.4	[6.9-12.6]	7.6	[7.0-8.1]
20-24	18.9	[18.1-19.8]	23.8	[20.0-28.2]	19.2	[18.3-20.1]
25-29	21.4	[20.5-22.4]	20.1	[16.4-24.4]	21.4	[20.5-22.3]
30-34	18.9	[18.1-19.8]	19.7	[16.2-23.6]	19	[18.1-19.9]
35-39	15.2	[14.4-16.1]	13.7	[10.8-17.3]	15.1	[14.3-16.0]
40-44	10.6	[10.0-11.3]	9	[6.6-12.1]	10.5	[9.9-11.2]
45-49	7.4	[6.8-8.0]	4.3	[3.0-6.3]	7.2	[6.7-7.8]
Total	100		100		100	

Pearson: Uncorrected chi2(6) = 20.6353
Design-based F(5.84, 4095.30) = 2.5330 Pr = 0.020

Region						
urban	42.8	[41.1-44.6]	41.2	[36.7-45.8]	42.8	[41.1-44.4]
rural	57.2	[55.4-58.9]	58.8	[54.2-63.3]	57.2	[55.6-58.9]
Total	100		100		100	

Pearson: Uncorrected chi2(1) = 0.6634
Design-based F(1.00, 701.00) = 0.4661 Pr = 0.495

Years lived in place of residence						
Less than a year	8.3	[7.6-9.2]	13.4	[10.5-17.1]	8.6	[7.8-9.5]
One to Three years	19.3	[18.3-20.5]	23.1	[19.5-27.2]	19.5	[18.5-20.7]
Four to ten years	23.3	[22.3-24.4]	23.9	[20.0-28.4]	23.3	[22.3-24.4]
Eleven to fourty one years	14.8	[13.9-15.7]	10.7	[8.1-13.9]	14.6	[13.7-15.4]
Always	31.7	[29.8-33.6]	27.2	[23.2-31.6]	31.4	[29.6-33.3]
Visitor	2.6	[2.2-2.9]	1.6	[0.8-3.2]	2.5	[2.2-2.9]
Total	100		100		100	

Pearson: Uncorrected chi2(5) = 34.0194
Design-based F(4.94, 3459.52) = 5.2270 Pr = 0.000

Highest educational level						
no education	10.1	[9.3-11.1]	10.5	[7.6-14.3]	10.2	[9.3-11.1]
primary	51.7	[50.1-53.4]	50.1	[45.2-54.9]	51.7	[50.1-53.2]
secondary	33.4	[32.0-34.9]	34.2	[29.7-38.9]	33.5	[32.0-35.0]
higher	4.7	[3.9-5.6]	5.3	[3.4-8.2]	4.7	[3.9-5.7]
Total	100		100		100	

Pearson: Uncorrected chi2(3) = 0.9529
Design-based F(2.98, 2089.73) = 0.2113 Pr = 0.888

Religion						
catholic	17.7	[16.5-19.0]	15.1	[12.0-18.8]	17.5	[16.3-18.8]
protestant	81	[79.7-82.3]	84	[80.3-87.2]	81.2	[79.9-82.4]
muslim	0.6	[0.3-1.2]	0		0.6	[0.3-1.2]
other	0.7	[0.5-1.0]	0.9	[0.3-2.5]	0.7	[0.5-1.0]
Total	100		100		100	

Pearson: Uncorrected chi2(3) = 7.2397
Design-based F(1.74, 1218.98) = 0.7084 Pr = 0.474

Wealth index						
Poor	38.6	[37.1-40.2]	38.1	[34.1-42.2]	38.6	[37.1-40.1]
Middle	19.4	[18.1-20.8]	20.9	[17.8-24.4]	19.5	[18.2-20.9]
Rich	41.9	[40.0-43.9]	41	[36.4-45.8]	41.9	[40.0-43.8]
Total	100		100		100	

Pearson: Uncorrected chi2(2) = 0.8360
Design-based F(1.90, 1333.43) = 0.3497 Pr = 0.694

Total children ever born						
Zero	0		15.3	[12.3-19.0]	0.8	[0.6-1.0]
One	19.5	[18.6-20.5]	19.4	[16.1-23.2]	19.5	[18.6-20.5]
Two	17.7	[16.8-18.5]	16.6	[13.3-20.6]	17.6	[16.8-18.4]
Three	15.5	[14.7-16.3]	12.1	[9.3-15.7]	15.3	[14.5-16.1]
Four	12.8	[12.1-13.6]	10.9	[8.4-14.1]	12.7	[12.0-13.5]
Five & above	34.5	[33.4-35.7]	25.6	[21.8-29.7]	34.1	[32.9-35.2]
Total	100		100		100	

Pearson: Uncorrected chi2(2) = 0.8360
Design-based F(1.90, 1333.43) = 0.3497 Pr = 0.694

Number of living children						
Zero	1.3	[1.0-1.6]	17.5	[14.2-21.4]	2.1	[1.8-2.5]
One	21.1	[20.1-22.2]	21.1	[17.4-25.3]	21.1	[20.1-22.2]
Two	18.9	[18.0-19.7]	17.4	[13.9-21.4]	18.8	[17.9-19.6]
Three	16	[15.2-16.9]	14.2	[11.2-17.7]	15.9	[15.1-16.7]
Four	13.9	[13.1-14.7]	10.2	[7.8-13.1]	13.7	[12.9-14.5]
Five & above	28.9	[27.8-30.0]	19.7	[16.4-23.5]	28.4	[27.3-29.5]
Total	100		100		100	

Pearson: Uncorrected chi2(5) = 735.7369
Design-based F(4.94, 3460.51) = 104.4339 Pr = 0.000

Currently/formerly/ never in union						
never in union	9.5	[8.7-10.2]	8.2	[6.0-11.0]	9.4	[8.7-10.1]
currently in union/living with a man	75	[73.8-76.2]	82.5	[78.8-85.7]	75.4	[74.2-76.6]
formerly in union/living with a man	15.5	[14.7-16.5]	9.3	[7.0-12.2]	15.2	[14.4-16.1]
Total	100		100		100	

Pearson: Uncorrected chi2(2) = 20.2786
Design-based F(2.00, 1399.16) = 7.5913 Pr = 0.001

Fertility preference						
have another	55.5	[54.1-56.8]	67.6	[62.8-72.0]	56.1	[54.8-57.4]
undecided	5.3	[4.7-5.9]	6.5	[4.4-9.5]	5.3	[4.8-6.0]
no more	36.2	[35.0-37.4]	24.3	[20.7-28.1]	35.5	[34.3-36.7]
sterilized (respondent or partner)	1.7	[1.4-2.1]	0.8	[0.3-2.0]	1.7	[1.4-2.0]
declared infecund	1.4	[1.1-1.7]	0.9	[0.3-2.5]	1.3	[1.1-1.6]
Total	100		100		100	

Pearson: Uncorrected chi2(4) = 43.8033
Design-based F(3.81, 2672.33) = 7.9938 Pr = 0.000

Fertility preference						
have another	55.5	[54.1-56.8]	67.6	[62.8-72.0]	56.1	[54.8-57.4]
undecided	5.3	[4.7-5.9]	6.5	[4.4-9.5]	5.3	[4.8-6.0]
no more	36.2	[35.0-37.4]	24.3	[20.7-28.1]	35.5	[34.3-36.7]
sterilized (respondent or partner)	1.7	[1.4-2.1]	0.8	[0.3-2.0]	1.7	[1.4-2.0]
declared infecund	1.4	[1.1-1.7]	0.9	[0.3-2.5]	1.3	[1.1-1.6]
Total	100		100		100	

Pearson: Uncorrected chi2(5) = 735.7369
Design-based F(4.94, 3460.51) = 104.4339 Pr = 0.000

Person who usually decides on respondent's health care						
respondent alone	31.7	[30.0-33.5]	29.4	[24.6-34.7]	31.6	[29.9-33.3]
respondent and husband/partner	43.1	[41.3-45.0]	38.4	[33.5-43.6]	42.8	[41.0-44.7]
husband/partner alone	24.8	[23.3-26.3]	31.3	[26.8-36.2]	25.2	[23.7-26.6]
someone else	0.4	[0.3-0.6]	0.9	[0.3-2.3]	0.5	[0.3-0.7]
Total	100		100		100	

Pearson: Uncorrected chi2(3) = 13.1704
Design-based F(2.94, 2060.06) = 3.4106 Pr = 0.018

Multivariate logistic regression: Determinants of fetal deaths

After taking care of multicollinearity by taking care of variables with a variance inflation factor of above 10 and factors that were not significant at bivariate (chi-square) analysis, a multivariate logistic regression was fitted as shown in table 2. Using stepwise regression and backwards elimination method based on p-values to explain determinants of fetal deaths, table 2 shows that fetal deaths in Zambia can be explained by factors that were significantly associated with fetal deaths as shown in table 2 model 4. The final model (model 4) reveals that fetal deaths could be explained by three factors which include; a women's age, a person who makes decisions on a woman's health care and the number of living children that a woman has. The model thus shows that the odds of having a fetal death was 1.46 (1.20 - 1.79) higher for women whose health care was decided by their partner compared to those who made the decisions on their health care by themselves. The model also shows that increasing a woman age by 1 unit increases the odds of having a fetal death by 1.02 (CI: 1.01-1.03). Finally, the model shows that the more living children that a woman has, the less the odds of having a fetal death, this is in comparison with women who never had any living children. Therefore, women who had one, two, three, four, five and more living children were 91% (OR: 0.09, CI: 0.06-0.14), 94% (OR: 0.06, CI: 0.04-0.08), 95% (OR: 0.05, CI: 0.03-0.08), 96% (OR: 0.04, CI: 0.03-0.07) and 96% (OR: 0.04,CI: 0.02-0.06) less likely to have a fetal death respectively compared to women who never had any living children.

Table 2: Factors associated with fetal deaths-Multivariate logistic regression (odds ratios)

Factors	Model1	Model2	Model3	Model4
	OR	OR	OR	OR
Fertility preference				
Have another	1			
Undecided	1.33			
	(0.8 - 2.3)			
No more	0.73**			
	(0.6 - 1.0)			
Declared infecund	0.54			
	(0.2 - 1.8)			
Years lived in a place of residence				
Less than a year	1	1		
One to Three years	0.78	0.79		
	(0.5 - 1.1)	(0.5 - 1.1)		
Four to ten years	0.73	0.74		
	(0.5 - 1.1)	(0.5 - 1.1)		
Eleven to forty one years	0.54**	0.55**		
	(0.3 - 0.9)	(0.3 - 0.9)		
Always	0.74	0.75		
	(0.5 - 1.1)	(0.5 - 1.1)		
Visitor	0.53	0.53		
	(0.2 - 1.2)	(0.2 - 1.2)		

Decisions on respondents health care				
Respondent alone	1	1	1	1
Respondent and husband/ partner	0.93	0.95	0.97	
	(0.7 - 1.2)	(0.7 - 1.3)	(0.7 - 1.3)	
husband/partner alone	1.42**	1.42**	1.44**	1.46***
	(1.1 - 1.9)	(1.1 - 1.9)	(1.1 - 1.9)	(1.2 - 1.8)
Someone else	1.67	1.53	1.53	
	(0.4 - 6.4)	(0.4 - 5.7)	(0.4 - 5.8)	
Age	1.03***	1.02***	1.02**	1.02**
	(1.0 - 1.1)	(1.0 - 1.0)	(1.0 - 1.0)	(1.0 - 1.0)
Number of living children				
Zero	1	1	1	1
One	0.12***	0.12***	0.12***	0.09***
	(0.1 - 0.2)	(0.1 - 0.2)	(0.1 - 0.2)	(0.1 - 0.1)
Two	0.08***	0.08***	0.08***	0.06***
	(0.1 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)
Three	0.07***	0.07***	0.07***	0.05***
	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)
Four	0.05***	0.05***	0.05***	0.04***
	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)
Five and above	0.05***	0.05***	0.04***	0.04***
	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)	(0.0 - 0.1)

Confidence interval in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Discussion

Fetal death refers to the intrauterine death of a fetus prior to delivery (WHO). In Zambia's new constitution Article 28, "life begins at conception". By implication, every pregnancy counts. However, the study indicates that close to 6 percent of women had a fetal death within 5 years prior to the study. There are so many factors that have been attributed to fetal deaths in many different studies, however, this study found that social and demographic factors such as maternal age, parity and person's responsible in decision making about a woman's health care play a key role on the pregnancy outcome as they were found to be associated with a risk of intrauterine fetal deaths. Several studies conducted both in developing and developed countries have observed that increasing maternal age has an impact on the risk of fetal mortality (12,13). These findings are similar with findings of (Kliman, 2004) that indicated that the odds of having a fetal death for women aged above 34 were 3.5 times higher compared to the controls (14). According to (Johnson, 2012) maternal age is an important factor in fertility because obstetric and perinatal risks increase with maternal age and that women are not knowledgeable of the increased medical risks of delayed child-bearing such as multiple births, preterm delivery, stillbirth, and Caesarean section (15). The influence of maternal age on fetal deaths can also be attributed to the fact that fertility is inversely related to maternal age. This means that as a woman grows older, her fertility declines. It is therefore imperative that women must be educated on the dangers of having children at older ages as they are at a higher risk of experiencing fetal deaths.

In the past, low fecundity among women or challenges/difficulties in having children due to physiological incapability's was associated with old age and higher parity but nowadays many women delay childbearing for social reasons (17) which posses a negative impact that can be explained by both biological mechanisms and forces of selection leading to an increase in fetal deaths. In a study on determinants of fetal death in Greece, (18) found a significantly higher risk of fetal death for higher maternal age and (7,20) other researchers observed that mothers aged 40 years or more were at higher risk of having a fetal death than younger mothers. Andersen and colleagues (17) also observed that fetal loss is high in women in their late 30s or older, irrespective of their reproductive history. However, in another study (21), even though agrees with the rest of the findings, provides an additional contrary finding that is not mentioned by other researchers in which it states that age below 20 years puts women at high risk of fetal death.

Women's decision's regarding health care are cardinal as they are an integral part of maternal and child health outcomes (19). Dual commitment in reproductive health decision making is cardinal for health concerns such as control of STDs including AIDS, family planning and infertility investigation (22). This implies that women need men as partners in reproductive health who understand the risks they might be exposed to and strategies for their prevention. For instance, preventive reproductive health initiatives and information should not be left for female alone but should involve both sexes. However, the current study found that women also require autonomy in their health care decisions if they are to avoid fetal deaths. Our current study found that the odds of having a fetal death was 1.458 higher for women whose healthcare was decided by their partner compared to those who made the decisions on their health care by themselves. In a study to explore women's level of satisfaction with their involvement in health care decisions during a high-risk pregnancy, it was observed that although most women want to be actively involved in health decision-making during a high-risk pregnancy, some prefer a passive role (23). A Nepal Demographic Health Survey (NDHS) shows that 37% of currently married women participated in important household decisions including their own health care (24). The Nepal DHS findings are similar to the study findings that found that about 4 in 10 women participate in decision making on their health care.

Having living children was inversely related to having a fetal death in this study. Therefore, women without children had higher odds of having a fetal death. The study findings were consistent with findings by Kozuki et al that found that nulliparous women had significant associations with adverse outcomes (25). However, a study by Lima et al in Cuiabá Showed that having live children was not associated with fetal death in the univariate analysis that was conducted (26).

Limitations

This study had some limitations. The study was a cross sectional study that collected data about past cases. The study also used secondary data hence not all factors that could potentially influence fetal deaths were captured. The analysis, therefore, was limited to the available indicators (variables in the dataset) that had potential to influence the health outcome.

Conclusion

There are various factors influencing fetal deaths. Maternal age being associated with fetal deaths mirrors the number of births affected by a weakened reproductive health system. Parity being negatively related to fetal deaths means that women's maternal experiences have a positive impact on health outcomes. Decision making inequalities (inability of women to make decisions on their health) have negatively

affected fetal deaths and women's access to reproductive health services. This study has implications on sensitization programs on the timing and appropriate age for conception. Sensitization programs should also be extended to the community on the importance of male involvement in maternal and child health as this has a positive effect on women's access to health care. However, male involvement should be a pillar of support for a woman's decisions regarding health and health care.

Declarations

Acknowledgements

We wish to thank the Zambia Central Statistics Office (CSO) for granting us permission to use the data. More specifically, we thank the Dissemination Office for the quick response to the request.

Availability of data and materials

The data is available in soft copy in different formats from the Central Statistics Office and the questionnaires are available in soft copy as well.

Competing interests

The authors declare that they have no competing interests.

LIST OF REFERENCES

1. Glasier A, Gülmezoglu AM, Schmid GP, Moreno CG, Van Look PF. Sexual and reproductive health: a matter of life and death. The Lancet. 2006 Nov 10;368 (9547):1595-607.

2. McCaw-Binns AM, Fox K, Foster-Williams KE, Ashley DE, Irons B. 1996. Registration of births, stillbirths and infant deaths in Jamaica. International Journal of Epidemiology 25(4):807–813.

3. Lumbiganon P, Panamonta M, Laopaiboon M, Pothinam S, Patithat N. 1990. Why are Thai official perinatal and infant mortality rates so low? International Journal of Epidemiology 19(4):997–1000

4. ICD-10 Mortality Manual Part 2K, 2014. https://www.cdc.gov/nchs/data/dvs/2k_2014

5. Shingairai A Feresu,Siobán D Harlow,Kathy Welch, Brenda W Gillespie: Incidence of stillbirth and perinatal mortality and their associated factors among women delivering at Harare Maternity Hospital, Zimbabwe: a cross-sectional retrospective analysis. BMC Pregnancy and Childbirth 2005, 5:9

6. Kambarami RA: Levels and risk factors for mortality in infants with birth weights between 500 and 1800 grams in a developing country: a hospital based study. Cent Afr J Med. 2002, 48 (11/127): 133-136.

7. Feresu SA, Welch K, Gillespie B, Harlow SD: Incidence of and Sociodemographic Risk Factors for Stillbirth, Pre-term birth and Low Birthweight in Zimbabwean Women. Paediatr Perinat Epidemiol. 2004, 18: 154-163.

8. Tachiweyika E, Gombe N, Shambira G, Chadambuka A, Tshimamga M, Zizhou S. Determinants of perinatal mortality in Marondera district, Mashonaland East Province of Zimbabwe, 2009: a case control study. Pan African Medical Journal. 2011;8(1).

9. Mavalankar DV, C. R. Trivedi, and R. H. Gray: Levels and risk factors for perinatal mortality in Ahmedabad, India. Bull World Health Organ. 1991; 69(4): 435–442.

10. Goffinet F, Combier E, Bucourt M, de Caunes F, Papiernik E. Epidemiology of fetal deaths in the Seine-Saint-Denis perinatal survey. J Gynecol Obstet Biol Reprod 1996;25:153-9.

11. WHO 2017. Women, newborns, children and adolescents: life-saving momentum after a slow start, www.who.int/publications/10-year-review/en/

12. Fretts RC, Schmittdiel J, McLean FH, Usher RH, Goldman MB. Increased maternal age and the risk of foetal death. N Engl J Med 1995; 333: 953–957.

13. Petitti DB. The epidemiology of foetal death. Clin Obstet Gynecol 1987; 30: 253–258.

14. Gregory, M. F. M. a. E. C., 2015. Fetal and Perinatal Mortality: United States, 2013. National Vital Statistics Reports, Volume Volume 64, Number 8 .

15. Johnson, J.-A., 2012. Delayed Child-Bearing:SOGC GENETICS COMMITTEE. J Obstet Gynaecol Can 2012;34(1):80–93, January.Volume No. 271, .

16. Kliman, H. J., 2004. Intrauterine fetal death. Department of OB/GYN. Yale University School of Medicine.14 January.

17. Andersen AM, Wohlfahrt J, Christens P, Olsen J, Melbye M. Maternal age and fetal loss: population based register linkage study. Bmj. 2000 Jun 24;320(7251):1708-12.

18. Petridou E, Kotsifakis G, Revinthi K, Polychronopoulou A, Trichopoulos D. Determinants of stillbirth mortality in Greece. Soz Praventivmed 1996;41:70-8.

19. Huang DY, Usher RH, Kramer MS, Yang H, Morin L, Fretts RC. Determinants of unexplained antepartum fetal deaths. http://dx.doi.org/10.1016/S0029-7844(99)00536-0

20. Raymond EG, Cnattingius S, Kiely JL. Effects of maternal age, parity and smoking on the risk of stillbirth. Br J Obstet Gynaecol 1994;101:301-6.

21. Mbizvo MT, Bassett MT. Reproductive health and AIDS prevention in sub-Saharan Africa: the case for increased male participation. Health policy and planning. 1996;11(1):84-92

22. Harrison MJ, Kushner KE, Benzies K, Rempel G, Kimak C. Women's satisfaction with their involvement in health care decisions during a high□risk pregnancy. Birth. 2003 Jun 1;30(2):109-15.

23. Acharya DR, Bell JS, Simkhada P, Van Teijlingen ER, Regmi PR. Women's autonomy in household decision-making: a demographic study in Nepal. Reproductive health. 2010 Jul 15;7(1):15. http://www.reproductive-health-journal.com/content/7/1/15

24. Kozuki N, Lee AC, Silveira MF, Sania A, Vogel JP, Adair L, Barros F, Caulfield LE, Christian P, Fawzi W, Humphrey J. The associations of parity and maternal age with small-for-gestational-age, preterm, and neonatal and infant mortality: a meta-analysis. BMC Public Health. 2013 Sep 17;13(3):S2. https://doi.org/10.1186/1471-2458-13-S3-S2

25. Lima JC, Oliveira GJ, Takano OA (2006-2010). Factors associated to fetal death in Cuiabá, Mato. Rev. Bras. Saude Mater. Infant. vol.16 no.3 Recife July/Sept. 2016. http://dx.doi.org/10.1590/1806-93042016000300008

PHYTOESTROGENS AND EARLY ONSET ANDROGINIC ALOPECIA: A STUDY IN NDOLA, ZAMBIA.

PERSPECTIVE

K Mukumbwa[1]

1. Public Health Unit, School of Medicine, The Copperbelt University, Ndola, Zambia

Correspondence: Kalenge Mukumbwa (kalengealpha@gmail.com)

Citation style for this article: Mukumbwa K. Phytoestrogens and early onset androginic alopecia: a study in ndola, zambia. 2018;2(3), pp19-24.

Androgenic alopecia is the most common type of hair loss experienced in males, presenting in genetically susceptible males with high free testosterone levels. Early onset androgenic alopecia refers to androgenic alopecia with an onset before thirty years. Phytoestrogens are plant derived substances that exhibit estrogen like properties in the body. The aim of the study was to determine the association between phytoestrogen containing foods and early onset androgenic alopecia.

A retrograde case control study was conducted in Ndola, Zambia. The study was conducted from the 10th of September 2016 to the 22nd of January 2017.Control group comprised individuals with a positive familial susceptibility to androgenic alopecia, with no androgenic alopecia. Case group comprised individuals who had visible androgenic alopecia.

A total of 194 individual participated in this study of which 97 were Cases and 97 Controls. The high intake of Soy foods was found to be significantly associated with decreased early onset androgenic alopecia (OR=3.5, 95% CI [1.862-6.546], other food however showed no significance.

The study shows that individuals who develop early onset androgenic alopecia have a lower consumption of soya foods. The low consumption of soy foods is significantly associated with the presence of early onset androgenic alopecia. Other commonly consumed phytoestrogen containing foods in the study were not significantly associated with early onset androgenic alopecia.

Switching to a high Soya diet may prevent early onset androgenic alopecia in genetically susceptible individuals. Further research is required into other environmental factors associated with early onset androgenic alopecia.

Key Words:

Phytoestrogens, early onset Androgenic alopecia.

Introduction

Androgenic alopecia is the gradual patterned decrease in scalp hair density and loss of due to the transformation of terminal scalp hair to vellus hair in individuals with familial increased susceptible of hair follicles to androgen effects following puberty[1], it is the most common form of hair loss[2,3]. World-wide prevalence thought to be as high as 50%[3]. Early onset androgenic alopecia is that beginning before the age of thirty [4,]. Androgenic alopecia is assumed to be caused by a combination of genetic predisposition and sufficient circulating androgen levels [1]. The mode of inheritance of androgenic alopecia is complex and some have proposed that the genetic component is autosomal dominant while others argue its mode of inheritance is polygenic [1].

Regardless of the mode of inheritance it has been established that high circulating androgen levels are needed for androgenic alopecia expression [5,6]. Increased hair growth has been noted in estrogen treated hair obtained from the temporal region of balding men [7,8].

Phytoestrogens are plant derived estrogen like substances. They are thought to be hormonally active in the body. Numerous health benefits have been attributed to their consumption such as a lowered risk of osteoporosis, heart disease, breast cancer, prostate cancer and menopausal symptoms [9,10,11,12]. Phytoestrogens are also considered to be endocrine disruptors. Phytoestrogen containing foods include Soya and Soya products, Beans (black beans, white beans and mung beans), Nuts including peanut butter, lentils, peas, carrots and rice. [13]

Consuming phytoestrogen rich foods such as soya among others may act as an endocrine disruptor preventing the expression of androgenic alopecia in genetically susceptible individuals. Relatively little is known of the environmental factors relating to androgenic alopecia, though they may play act to slow, speed up or possibly prevent androgenic alopecia. Phytoestrogens are an important possible factor in androgenic alopecia. [14]

Methodology

Study Site: The study was done at Northern Technical College which is located in Ndola on the Copperbelt province of Zambia. The area was chosen as it has a collective large population of young adults who are known to consume a lot of phytoestrogen rich foods.

Study Design:

Study Site: The study was done at Northern Technical College which is located in Ndola on the Copperbelt province of Zambia. The area was chosen as it has a collective large population of young adults who are known to consume a lot of phytoestrogen rich foods.

Study Design: The study was a retrograde case-control study looking for association between a phytoestrogen rich diet and early onset androgenic alopecia. . With the aid of a Chart showing the Hamilton-Norwood scale students were asked to identify if any immediate family members had hair loss ,the scale was also used

Study population: Males aged 15years to 30years. The study included individuals who had consented to the study, individuals who did not give their consent were not included in the study. The controls were individuals with a familial susceptibility to androgenic alopecia but with no visible androgenic alopecia, while the cases were individuals with visible androgenic alopecia.

Study population: Total number of participants was 194 of which 97 were Cases and 97 were Controls. The sample size was computed from a pilot study .The pilot study had 30 cases and 30 controls. Parameter used to determine the sample size was the intake of Soy and Soya products association with androgenic alopecia were P1 case prevalence= 44% and P2 case prevalence= 25% f(αβ) was taken as 7.8% at power 80% and significant level at 5% and this gave a minimum sample size of 94.

Variables

The dependent variable for this study was perception androgenic alopecia, participants were classified as either visible hair loss (case) or no hair loss (control). Other domains considered in this study were the level of alopecia and the time taken to reach the level of alopecia .The independent variable was the intake of phytoestrogen rich foods.

Ethical considerations

Permission to conduct the study was obtained from the Tropical diseases research centre, the Copperbelt University School of Medicine and the Students at Northern Technical College who participated in the study.

Data Entry and analysis

The data obtained using the questionnaire was entered into SPSS version 20 for analysis. Descriptive statistics and logistic regression were used for the analysis. The chi square test was used to determine associations between phytoestrogen intake and early onset androgenic alopecia. A p value of less than 0.05 was considered to be statistically significant, 95% Confidence interval was used and the adjusted odds ratio was computed.

Results

A total of 194 respondents (97 cases and 97 controls) all aged between 15years and 30years.

Table 1 shows the association between commonly eaten phytoestrogen containing foods and the androgenic alopecia. There was a significant association between intake of Soy foods and androgenic alopecia however; other foods had no significance to androgenic alopecia.

Table1: Phytoestrogen food intake association with perception of androgenic alopecia

Phytoestrogen Food Intake		Perception Of Androgenic Alopecia		P - Value
		Hair Loss (Case)	No Hair Loss (control)	
Soy & Soya	Low	69(73.4)	42(44.7)	<0.001
	High	25(26.6)	52(55.3)	
Beans	Low	70(72.2)	66(68.8)	0.603
	High	27(27.8)	30(31.2)	
Nuts	Low	40(41.2)	48(52.2)	0.132
	High	57(58.8)	44(47.8)	
Peas	Low	84(86.6)	85(89.5)	0.540
	High	13(13.4)	10(10.5)	
Carrots	Low	78(80.4)	85(89.5)	0.080
	High	19(19.6)	10(10.5)	
Oats	Low	85(87.6)	82(85.4)	0.653
	High	12(12.4)	14(14.6)	
Lentils	Low	91(92.9)	84(87.5)	0.209
	High	7(7.1)	12(12.5)	
Rice	Low	21(21.6)	18(18.9)	0.642
	High	76(78.4)	77(81.1)	

Table 2 this shows the intake of soya to be independently significant to androgenic alopecia. Individuals with a high intake of soy were 3.5 (95% CI [1.862-6.546]) times less likely to have androgenic alopecia compared to those who had a low intake.

Table 2: Independently significant to androgenic alopecia

Factor	P-value	Model 1 AOR(95%CI)	Model 2 AOR(95%CI)
Soya intake	<0.001	3.511(1.870-6.591)	3.491(1.862-6.546)
Beans	0.525	1.259(0.618-2.563)	
Nuts	0.373	0.745(0.391-1.422)	
Peas	0.759	0.841(0.278-2.546)	
Carrots	0.223	0.530(0.191-1.472)	
Oats	0.475	1.445(0.527-3.963)	
Lentils	0.224	2.022(0.60-3.047)	
Rice	0.462	1.355(0.603-3.047)	

Discussion

The aim of this study was to investigate the association between commonly consumed phytoestrogen containing foods and early onset androgenic alopecia. The study showed a significant association between the consumption of soy foods and androgenic alopecia. Other foods (Rice, oats, carrots, beans, peas and nuts) were not found to have a significant association with androgenic alopecia.

A high consumption of soy foods was found to be significantly associated with the absence of androgenic alopecia and a low consumption with the presence of androgenic alopecia. This finding has been postulated and documented in other studies, none of which have been conducted in Zambia. [15, 16]

Despite been said to be phytoestrogen containing foods and been locally commonly consumed, food such as beans, nuts, lentils ,peas, oats ,carrots and rice showed no significant association with the presence of androgenic alopecia [13]. This finding can be explained by looking at the relative amounts of phytoestrogens that the foods contain. [13] It is easily evident that the levels of phytoestrogens that these foods contain relative to soya are very little; as such it is no surprise that were found to be insignificant in this study.

Study Limitation

The diet of individuals in tertiary education systems such as the one of this study does not necessarily reflect the diet patterns of the rest of the population. The students also have a break from the dieting pattern that was investigated for a period of about three months when they are on school break, however very few mentioned this. The food history taken was based on a weekly consumption which was taken as a reflection of chronic diet.

Conclusion

In this study two observations were made. It was found that individuals who had a diet rich in soya were less likely to have early onset androgenic alopecia despite been genetically susceptible and those with low soya intake more likely to have early onset androgenic alopecia.it was also found that other phytoestrogen containing foods did not have a significance on early onset androgenic alopecia.

Acknowledgement

Sincerest thanks to all the students who participated in the study and also to individuals who aided in the data collection. Special thanks to Dr Alfred Sichilima for his invaluable contribution in his capacity as project supervisor

LIST OF REFERENCES

1. Sinclair, R. (1998). Male pattern androgenetic alopecia. British Medical Journal v.317 (7162)

2. Axel H, Sandra H, Sibylle R, Tim B, Jan F, Felix F, et all (2005). Genetic Variation in the Human Androgen Receptor Gene Is the Major Determinant of Common Early-Onset Androgenetic Alopecia. American Journal of Human Genetics, Volume 77, Issue 1, pages 140-148

3. Ellis J, Sinclair R and Harrap S (2004). Androgenetic alopecia: pathogenesis and potential for therapy. Expert Reviews in Molecular Medicine v.2002,pages 1-11

4. Pengsalae N, Tanglertsampan C and Lee S (2013).Association of early onset androgenic alopecia and metabolic syndrome in Thai men. Journal Medical Association Thailand. 96(8):pages 947-51

5. Randall V. (2008). Androgens and hair growth. Dermatologic Therapy Volume 21, Issue 5, pages 314–328.

6. Hanss Schweikert & Jean Wilson. (2013). Regulation of human hair growth by steroid hormones - testosterone metabolism in Isolated Hairs. The Journal of Clinical Endocrinology & Metabolism Volume 38, Issue 5

7. Conrad, F, Ohnemus U, Bodo E, Bettermann A & Paul R. (2004). Estrogens and human scalp hair growth—still more questions than answers. Journal of Investigative Dermatology volume 122, pages 840–842.

8. Randall V, Thornton M & Messenger A.(1992) Cultured dermal papilla cells from androgen-dependent human hair follicles contain more androgen receptors than those from non-balding areas of scalp. Journal of Endocrinology volume 133, pages 141–147

9. Patisaul H. & Jefferson11 W. (2010). The pros and cons of phytoestrogens. Front Neuroendocrinol. 31(4): pages 400–419.

10. Shidhaye S, Malke S, Mandal S, Sakhare N & Kadam V (2008). Soy - A Hidden Treasure For Therapeutic, Cosmetic And Pharmaceutical Use. The Internet Journal of Alternative Medicine Volume 7,Number 2

11. Kuhnle GG, Ward HA, Vogiatzoglou A, et al; Association between dietary phyto-oestrogens and bone density in men and postmenopausal women. British Journal of Nutrition. 2011 Oct; 106(7):1063-9. doi: 10.1017/S0007114511001309. Epub 2011 May 17.

12. Holzbeierlein JM, McIntosh J, Thrasher JB; The role of soy phytoestrogens in prostate cancer. Curr Opin Urol. 2005 Jan;15(1):17-22.

13. Thompson LU, Boucher BA, Liu .Z, Cotterchio M & Kreiger N.(2006).Phytoestrogen Content of foods consumed in Canada, including isoflavons, lignans and coumestan. Nutrition and Cancer.v54,number2 pages 184-201

14. Severi G, Sinclair R, Hoper JL, English DR, McCredie MR, Boyle P, et al (2003). Androgenetic alopecia in men aged 40–69 years: prevalence and risk factors. British Journal of Dermatology Volume 149, Issue 6, pages 1207–1213.

15. Blanchello (2007). Soybean and baldness prevention: apparently, there is a link. The Science Creative Quarterly. 04.03.2007

16. Lai H, Chu F, Chang W, Wang L, Yang C et al.(2013).Androgenic alopecia is Associated with less dietary Soy, Higher Blood Vanadium and rs1160312 1 Polymorphism in Taiwanese Communities. PloS ONE 8(12):e79789.doi:10.137i/journal.pone.0079789