

**Non-Communicable Disease (NCD) Rates in Urbanized versus Rural
Populations in Developing Countries**

A thesis submitted to the University of Arizona College of Medicine – Phoenix
in partial fulfillment of the requirements for the degree of Doctor of Medicine

Now Bahar Alam
Class of 2018

Mentor: Cody Conklin, MD

ACKNOWLEDGEMENTS

Thank you to Dr. Cody Conklin for her advice and expertise in completing this systematic review.

Thank you to Paul Kang for his help and contribution to the project.

Thank you to Dr. McEchron for his support in the Scholarly Project course at University of Arizona College of Medicine-Phoenix.

ABSTRACT

Background: Studies estimating the current prevalence rates and future demographics of being overweight or obese and non-communicable diseases initially demonstrated Western countries had the highest rates of obesity. Now, obesity is more prevalent in urban populations of Africa, Central and South America, Asia, and Caribbean and Pacific Islands.

Objective: Determine if any differences exist with the NCD rates in urbanized versus rural populations in developing countries. Specifically, any differences pertaining to being overweight or obese or BMI levels; hypertension rates or blood pressure levels; hypercholesterolemia rates or cholesterol levels; and diabetes rates or blood glucose levels were studied.

Methods: Using PubMed, a thorough review of the literature was conducted using various search terms related to the research topic. The database was systemically searched from 1995 up to December 2016.

Results: The effect size using Cohen's d was utilized to measure the size of associations or differences between the urban and rural populations. Nine studies compared BMI values based on gender and reported the means with standard deviations. In the total male population ($N=17402$), there was a Cohen's $d=0.64$, $p < 0.001$, CI.95 (0.45, 0.83). In the total female population ($N=18126$), there was a Cohen's $d=0.56$, $p < 0.001$, CI.95 (0.31, 0.81). Overall, there was Cohen's $d=0.60$, $p < 0.001$, CI.95 (0.46, 0.74). Five studies compared mean systolic and diastolic blood pressures. In the total male population ($N=16155$), there was a Cohen's $d=0.17$, $p = 0.001$, CI.95 (0.02, 0.32) for systolic pressures and a Cohen's $d=0.32$, $p < 0.001$, CI.95 (-0.08, 0.71) for diastolic pressures. In the total female population ($N=16852$), there was a Cohen's $d=0.22$, $p = 0.005$, CI.95 (0.09, 0.35) for systolic pressures and a Cohen's $d=0.38$, $p < 0.001$, CI.95 (-0.10, 0.86) for diastolic pressures. Overall, there was Cohen's $d=0.20$, $p < 0.001$, CI.95 (0.13, 0.28) for systolic pressures and Cohen's $d=0.36$, $p < 0.001$, CI.95 (0.17, 0.55) for diastolic pressures. Eight studies compared total cholesterol levels. In the total male population ($N=4961$), there was a Cohen's $d=0.64$, $p < 0.001$, CI.95 (0.38, 0.90). In the total female population ($N=5581$), there was a Cohen's $d=0.55$, $p < 0.001$, CI.95 (0.29, 0.81). Overall, there was Cohen's $d=0.59$, $p < 0.001$, CI.95 (0.42, 0.77). Four studies compared blood glucose levels. In the total male population ($N=1687$), there was a Cohen's $d=-0.03$, $p < 0.001$, CI.95 (-0.55,

0.49). In the total female population (N=2100), there was a Cohen's $d=-0.09$, $p < 0.001$, CI.95 (-0.56, 0.38). Overall, there was Cohen's $d=-0.07$, $p < 0.001$, CI.95 (-0.36, 0.22).

Conclusion: Cross-sectional and observational studies comparing BMI values, blood pressure levels, cholesterol levels, and blood glucose levels have addressed if living in urban versus rural areas increases the prevalence of NCDs related to these variables. Urban populations and living a sedentary lifestyle does increase the likelihood of being overweight or obese, hypertension, hypercholesterolemia, and diabetes.

TABLE OF CONTENTS

	Page
CHAPTER 1: COMPREHENSIVE REVIEW	
HISTORY	1
EFFECTS OF NUTRITION ON HEALTH	4
NUTRITION AND EXERCISE PROGRAMS AND POLICY CHANGES	8
REFERENCES	11
CHAPTER 2: SYSTEMATIC REVIEW	
INTRODUCTION	20
MATERIALS AND METHODS	22
RESULTS	27
DISCUSSION	39
FUTURE DIRECTIONS	42
CONCLUSIONS	43
REFERENCES	44

LIST OF FIGURES

Figure 1: Quality assessment tool for observational cohort and cross-sectional studies

Figure 2: PRISMA Flow Diagram

Figure 3: Effect sizes using Cohen's d of studies reporting mean BMI values with standard deviations in males and females in urban vs rural populations

Figure 4: Effect sizes using Cohen's d of studies reporting mean systolic and diastolic blood pressures with standard deviations in males and females in urban vs rural populations

Figure 5: Effect sizes using Cohen's d of studies reporting mean total cholesterol levels with standard deviations in males and females in urban vs rural populations

Figure 6: Effect sizes using Cohen's d of studies reporting mean blood glucose levels with standard deviations in males and females in urban vs rural populations

LIST OF TABLES

Table 1: Assessment of risk within individual studies

HISTORY

Throughout the world, the prevalence of non-communicable diseases is increasing at an alarming rate. It is estimated that 1.1 billion adults globally are overweight, and 312 million of them are obese.¹ Being overweight and obese has contributed to an increase in cases of diabetes and hypertension, which have predisposed individuals to other chronic diseases such as cardiovascular disease. Obesity rates have tripled in the past couple of decades in developing countries due to lack of physical activity and overconsumption of cheap, energy-dense foods.¹ Low-energy dense foods provide fewer calories; therefore, more can be eaten without consuming excess calories. High-energy dense foods have a high concentration of calories per gram of food.² This review examines the research on the global trends, which have resulted in an increase in non-communicable disease rates and the efforts that are already being taken to halt the increase in non-communicable disease rates.

The shifts in diet, activity patterns and body composition are occurring more rapidly than they did in Western countries. Modernization of the economy has been occurring in much of Asia, the Middle East, Latin America, northern Africa and many areas in sub-Saharan African in a short time frame of 10-20 years. In many Western societies, these changes occurred over many decades to a century or more.³ For instance, in the 1970s: many developing countries had food supply concerns; most households had no television; walking and biking was the main form of transportation; food trade was at a minimum; consumption of processed food was low; and most rural and urban occupations consisted of heavy labor.³ In the present day: laborers use machines like small gas-powered tractors; industrial technology has grown; sugary drinks and processed foods are more readily accessible; televisions, computers and cell phones are commonplace; and mass transit has replaced walking and biking.³ Even the poorest nations had access to a high fat diet (diet obtaining 20% of energy from fat) by 1990. An increase in the consumption of vegetable fats in low and high-income nations and an increase in consumption of animal fats in poor countries caused an increase in total fat consumption for both low and high-income countries.³ However, there was a decrease in fat intake for moderate-income countries.³

The main studies have estimated current prevalence rates and future demographics of being overweight, obesity and non-communicable diseases.⁴⁻⁷ Initially, Western countries were associated with the highest rates of obesity. Now, obesity is more prevalent in urban populations of Africa, Central and South America, Asia, and Caribbean and Pacific Islands.⁸⁻¹¹ In a 2011 study, the prevalence of obesity exceeded 40% in a population of middle-aged urban Indian women.¹² Another study looked at trends in obesity and compared any rate of change in BMI of adolescents and adults from 1989 to 1997 in China.³ Not only did BMI increase, but the shape of the BMI distribution curve changed as well. When compared to the results from 1989, there were less underweight men and women and more overweight and obese adults in 1997. The proportion of overweight/obese men increased from 6.4% to 14.5% and in women from 11.5% to 16.2%. In the past three decades, the age-standardized mean BMI increased by 0.4–0.5 kg/m² per year.³

However, a bimodal distribution of under- and over-nutrition exists in low and middle-income nations due to the rapid urbanization and modernization.^{9,13,14} Bangladesh is one of many countries facing the double burden of underweight and overweight malnutrition. While there has been a significant decline in chronic nutritional deficiencies among Bangladeshi women in a 15 year period from 1996 to 1997, malnutrition continues to be an issue. Approximately 28% of rural women and 13.5% of urban women are considered underweight.¹ On the other hand, there has been an increase in the prevalence of being overweight from 11.4% to 28.9% in urban women and 1.7% to 12.1% in rural women. Among urban women in seven African countries (Burkina Faso, Ghana, Kenya, Malawi, Niger, Senegal and Tanzania), the prevalence of being overweight and obese increased by approximately 35% between 1992 and 2006.^{15,16}

The prevalence of overweight/obese mothers has increased steadily since 1980 to reach 40% and has now surpassed the prevalence of under-nutrition in Africa.¹⁷ This change has occurred as a result of the rapid economic development and urbanization of sub-Saharan Africa along with unhealthy diets and decreased physical activity. Sub-Saharan Africa also has the highest prevalence of under-nutrition. As much as 50% of young children in East Africa have stunted

growth and maternal under-nutrition is over 10% in some regions.¹⁸ Even with the high prevalence of under-nutrition, the pediatric population has experienced a greater increase in being overweight compared to the adult population; the younger generations have been documented to have higher BMIs at earlier ages than ever before.¹⁹⁻²¹ In China, the prevalence of underweight individuals has been rapidly declining, but there has been an increase in the proportion of overweight individuals over the past 30-40 years due to the introduction of westernized diets and sedentary lifestyles.^{19,22-27} Nevertheless, there still exists the double burden of under- and over-nutrition, which are affected by age, gender, urban versus rural environment and socioeconomic status.^{24,28,29}

A growing number of lower-income countries are facing a double burden of malnutrition. Under-nutrition coexists with over-nutrition and the non-communicable diseases that are associated with it. While there are well documented and evidence based benefits of maintaining a healthy weight, high physical activity and balanced diet, being overweight/obese is the fifth leading cause of global mortality and linked to multiple non-communicable diseases.³⁰ Children who are overweight/obese have more issues due to an increased risk of obesity in adulthood, physical and psychosocial morbidity, and premature mortality in adulthood. Being overweight/obese in childhood is also associated with impaired social and economic productivity in adulthood.³¹ It is no wonder that the increasing prevalence of non-communicable diseases is concerning in the developing world.¹⁵ The exact effects of nutrition and diet on the body will be the next topic to be discussed.

EFFECTS OF NUTRITION ON HEALTH

A 2013 report by U.S. Burden of Disease collaborators discovered that out of 17 risk factors, the largest risk factor for related deaths and disability-adjusted life-years (DALYs) is composition of diet. (Years of Life Lost and Years Lived with Disability are combined into DALYs). It contributed to 26% of deaths and 14% of DALYs, which is more than the deaths and DALYs tobacco is associated with. Diets low in fruits, low in nuts and seeds, high in sodium, high in processed meats, low in vegetables, and high in trans fats were noted to be detrimental to health. Other risk factors included tobacco, high blood pressure, high BMI and physical inactivity.³²

Another study demonstrated that consumption of excess sugar directly promotes the development of cardiovascular disease and type 2 diabetes. Mechanisms through which these chronic diseases occur involves unregulated hepatic uptake and metabolism of fructose-containing sugars, sucrose and high fructose corn syrup. This in turn causes decreased insulin sensitivity and increased uric acid levels, fatty liver disease and dyslipidemia. Consumption of excess sugar may also promote the development of cardiovascular disease and type 2 diabetes indirectly by causing increased body weight and fat gain. It is also possible that fructose consumption causes increased energy intake and reduced energy expenditure due to its failure to stimulate leptin production.³³

Also contributing to a high BMI and greater prevalence of both excess weight and obesity are the consumption of ultra-processed foods. One researcher discovered that Brazilian adolescents who consumed 56% of their total energy intake from ultra-processed foods had a mean BMI 0.94 kg/m² higher than those who consumed 6.0%. The adjusted odds ratio of being obese was 1.98 in those consuming the most ultra-processed foods. Even after adjusting for the amount of fruits, vegetables and beans that the adolescents consume did not affect the risk estimate.³⁴ While ultra-processed foods provide the convenience of being consumed anywhere and are typically ready-to-consume dishes that can replace home-cooked meals, the processing techniques and additives make these foods extremely hyper-palatable. As a result, individuals

tend to “mindlessly eat”. However, these foods could cause damage to the body response systems in place that control satiety and appetite. ^{35,36}

One particular type of ultra-processed food is sugar-sweetened beverages that lead to weight gain. ³⁷ The consumption of soft drinks and energy-dense fast foods has been positively associated with overweight and obese adolescents. ³⁸ In a group of Mexican adolescents, subject’s BMI increased an average of 0.33 for each additional sweetened beverage consumed daily. Those who drank more than three daily servings of the beverages were 2.1 times more likely to have excess body fat than the subjects who drank less than one serving. ³⁹ Furthermore, it has been shown that for each additional daily sweetened beverage serving consumed, individuals experienced an average increase of 0.49 mmol/l in triglyceride and a decrease of 0.31 mmol/l in HDL cholesterol. Those who drank more than two servings of the beverages each day were at twice the risk of developing metabolic syndrome than those who did not consume the sweetened drinks. ⁴⁰ In fact, higher sweetened beverage consumption increased the risk of developing all of the components of metabolic syndrome. ⁴⁰ In another study, overweight/obese children were significantly more likely to have diastolic prehypertension, systolic prehypertension and the lowest quintile of HDL cholesterol. Being obese as a child is strongly associated with cardiovascular disease risk factors. ⁴¹

Another possible relationship between the consumption of ultra-processed foods with obesity is the portion size. Portion sizes of many ultra-processed foods have increased dramatically in the past several decades. Some studies have even correlated the increases in obesity to increased total energy intake. ⁴²⁻⁴⁴ Ultra- processed foods have high glycemic loads due to the lack of water and the type of carbohydrates. ^{35,45} Similarly, high glycemic loads can cause increased insulin secretion, which might promote weight gain by directing nutrients away from oxidation in muscle and towards storage as fat. ^{46,47} In one study, subjects in an overfeeding trial with high glycemic foods had increased body weight, plasma proinsulin and insulin. ⁴⁸ It is evident that ultra-processed foods are nutritionally unbalanced. ^{49,50} They contain poor quality fat and low contents of fiber, micronutrients, and phytochemicals.

Diet and nutrients play a role in the risk of cancers as well. Persons who consume diets high in vegetables and fruits were shown to have a decreased risk of oral and pharyngeal (OCP) cancers. While those consuming diets high in milk and dairy products, eggs, red meat, potatoes and desserts had an increase in OCP cancers. However, the risk was only significant for potatoes and desserts. In regards to micronutrients, vegetable protein, vegetable fat, polyunsaturated fatty acids, a-carotene, b-carotene, b-cryptoxanthin, lutein and zeaxanthin, vitamin E, vitamin C and folate were inversely related with the incidence of OCP cancer. Animal protein, animal fat, saturated fatty acids, cholesterol, and retinol were directly related to the incidence of OCP cancer. These results were adjusted for BMI, tobacco smoking, alcohol drinking, etc. Combinations of low consumption of fruits and vegetables with a high consumption of meat and high tobacco and alcohol use resulted in a 10 to 20 fold excess risk of developing OCP cancer.⁵¹ Consuming these nutrients in excess could lead to increased weight, which has also been shown to increase one's susceptibility to thyroid cancer. Excess calorie intake was more frequent in patients with thyroid cancer than in controls. Increased protein and carbohydrate consumption was also associated with increased risk of thyroid cancer.⁵² It is evident that a diet rich in fruits and vegetables and low in meat and animal products does contribute to a decreased incidence of certain types of cancer.

Finally, mothers should be very mindful of their diet and the type of nutrition they are getting. For instance, children of overweight/obese mothers and children of normal weight mothers both had similar gestational age, birth weight, and Tanner stage. However, the offspring of overweight/obese mothers had a significantly higher BMI, percentage body fat, visceral fat with no difference in lean body mass compared to offspring of normal weight mothers. Moreover, children of overweight/obese mothers had lower insulin sensitivity and an adverse cardiovascular disease risk profile. This includes higher blood pressure, triglycerides to high-density lipoprotein ratio, and hs-C-reactive protein. High BMI during pregnancy contributed to excess adiposity, insulin resistance, and cardiometabolic disease risk in the offspring.⁵³ Both an excess or a deficit in maternal nutrition will have lasting impacts on offspring development and susceptibility to metabolic disorders. Various epigenetic mechanisms might rationalize how diet

during early critical development stages affects one's susceptibility to metabolic diseases later in life. As evidence, microRNAs regulate gene expression and contribute to tissue homeostasis and disease. Diet can actually alter microRNA expression. This means a mother's nutritional status during pregnancy affects how susceptible offspring are to develop cardiometabolic risk factors due to the contributions of microRNAs.⁵⁴ Likewise, children who had a high birth weight were more likely to have a higher waist circumference, triceps skinfold thickness, fat mass and lean body mass compared to children born with a normal weight. The children born with the highest birth weight were more at risk for obesity and possibly chronic diseases in adulthood.⁵⁵

After reviewing these articles, it is apparent that the effects nutrition and diet have on an individual's health are profound. With the increasing prevalence of being overweight and obesity related non-communicable diseases, it is more important than ever for future health education programs to prevent excess weight gain while targeting unhealthy eating habits. The next part of the review will focus on reviewing programs and system changes geared towards halting this unsettling trend.

NUTRITION AND EXERCISE PROGRAMS AND POLICY CHANGES

Some countries have started initiatives and implemented programs to combat the rise in non-communicable diseases. And evidence is promising, demonstrating a decrease in non-communicable disease risk factors. Examples of local programs have been studied in Iran and India. In Iran, a community-based program promoted healthy lifestyle behaviors.⁵⁶ Its purpose was to prevent cardiovascular disease through interventions that targeted healthy nutrition, increased physical activity, tobacco control and coping with stress. Both urban and rural areas were included in the intervention. Intervention activities encompassed public education through mass media, collaboration with community health and non-health related partners, professional education, marketing, organizational development, legislation and policy development, and research and evaluation. Results showed a significant decrease in the prevalence of abdominal obesity, hypertension, hypercholesterolemia, and hypertriglyceridemia compared to the control group in both males and females. The decline in being overweight and obese was only significant in females.⁵⁶

In India, another example targeting children utilized school-based health and nutrition education programs that measured the impact of education on the behavior and knowledge of urban children, parents and teachers in government run and private schools. Before the intervention, participants were given a knowledge and behavior assessment questionnaire that included the following themes: health, nutrition, diseases, physical activity and healthy cooking practices. Low knowledge and behavior scores were reported in 75-94% government and 48-78% of private school children of all ages. After program completion, scores improved in all of the children regardless of the type of school they attended.⁵⁷ A lack of health education and knowledge about common non-communicable diseases is evident in populations with double burden diseases. In more than two thirds of students surveyed, familial risk factors of diabetes and hypertension and lifestyle-related risk factors were not known.⁵⁸ Additionally, many of students had poor knowledge about malaria prevention despite living in an endemic area.⁵⁸ Many communities would benefit from a comprehensive health education for prevention of double burden diseases.

On a national level, other countries have developed a national physical activity plan that could be utilized as a reference in countries lacking a plan. For instance, Kuwait has ten-step template for a national plan that is adaptable to other countries.⁵⁹ Programs that were proven to be effective in preventing and controlling obesity in western countries could also be implemented in developing countries. For instance, in an observational study of 20 childcare centers in the U.S., it was reported that equipment like hula-hoops, balls and jump ropes, areas to run in and structures to play on promoted moderate to vigorous physical activity over sedentary behavior.⁶⁰ Another U.S. study enrolled overweight children in a 12-week intervention program to decrease sedentary behaviors and increase physical activity. Both children and caregivers were to meet weekly for two hour group sessions to set goals for exercise, diet and behavior change. Children with parents who were the least involved were eight times less likely to lose weight than children whose parents were involved.⁶¹

Education interventions could be utilized to prevent obesity and other non-communicable diseases. Education creates awareness and highlights the importance and benefits from investing in obesity prevention; prevention of obesity does lead to higher work productivity later in life.⁶² Individuals have also indicated that they would like to make better nutritional choices. They expressed an interest in reading food labels correctly.⁶³ Education through nutrition and exercise programs are proven methods to decrease the prevalence of non-communicable diseases.

Regulating the food industry is another means to combat the rise observed in the number of individuals with non-communicable diseases. One good example of this is the regulation of salt in processed foods. A link between increased salt intake and cardiovascular disease has been demonstrated.⁶⁴ Research conducted in five different countries demonstrated that salt regulation is cost-effective or even cost saving, meaning that costs of treatment avoided outweigh costs of intervention.⁶⁵⁻⁶⁹ However, it is not an easy task to improve policies in the food industry. Global nutrition is increasingly in the hands of a few large multinational food companies.⁷⁰ The companies are able to spend on lobbying the public and political leaders to

argue against effective interventions, such as taxes and regulation. They also distract attention toward interventions that center on the individual and his/her responsibility to change behavior and achieve a balanced lifestyle. Even with the evidence that regulation of salt in processed foods is cost-effective, there is resistance to its implementation on a global scale.⁷¹ Some countries have been successful in resisting industry pressures. Finland and Sweden have a low consumption of soft drinks due to national policies that limit the exposure to unhealthy foods and drinks.⁷² It is possible for policies to be effective in reversing the trends of the obesity epidemic as well as the increase in non-communicable disease rates.

More countries need to incorporate a plan and involve different government sectors to combat non-communicable diseases and the obesity epidemic. While it will not be an easy task, analyzing research from past studies will be beneficial in determining what will be the best plan of action. With over 1 billion overweight/obese people in the world and the non-communicable diseases associated with being overweight/obese, it is more important than ever to combat this health crisis.

REFERENCES

1. Hossain P, Kavar B, El Nahas M. Obesity and diabetes in the developing world--a growing challenge. *N Engl J Med*. 2007;356(3):213-215.
2. British Nutrition Foundation. What is energy density?
<http://www.nutrition.org.uk/healthyliving/fuller/what-is-energy-density.html>. Updated 2015.
Accessed October 18, 2015.
3. Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr*. 2002;5(1A):205-214.
4. Kastorini CM, Milionis HJ, Ioannidi A, et al. Adherence to the mediterranean diet in relation to acute coronary syndrome or stroke nonfatal events: A comparative analysis of a case/case-control study. *Am Heart J*. 2011;162(4):717-724.
5. Nguyen TT, Adair LS, Suchindran CM, He K, Popkin BM. The association between body mass index and hypertension is different between east and southeast asians. *Am J Clin Nutr*. 2009;89(6):1905-1912.
6. WHO Expert Consultation. Appropriate body-mass index for asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363(9403):157-163.
7. Subramanian SV, Perkins JM, Ozaltin E, Davey Smith G. Weight of nations: A socioeconomic analysis of women in low- to middle-income countries. *Am J Clin Nutr*. 2011;93(2):413-421.
8. Martorell R, Khan LK, Hughes ML, Grummer-Strawn LM. Obesity in women from developing countries. *Eur J Clin Nutr*. 2000;54(3):247-252.

9. Popkin BM. The nutrition transition and obesity in the developing world. *J Nutr.* 2001;131(3):871S-873S.
10. Popkin BM. The world is fat. *Sci Am.* 2007;297(3):88-95.
11. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. *J Clin Endocrinol Metab.* 2008;93(11 Suppl 1):S9-30.
12. Pandey RM, Gupta R, Misra A, et al. Determinants of urban-rural differences in cardiovascular risk factors in middle-aged women in india: A cross-sectional study. *Int J Cardiol.* 2013;163(2):157-162.
13. Piernas C, Wang D, Du S, et al. The double burden of under- and overnutrition and nutrient adequacy among chinese preschool and school-aged children in 2009–2011 *European Journal of Clinical Nutrition.* 2015:1.
14. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev.* 2012;70(1):3-21.
15. Kimani-Murage EW, Muthuri SK, Oti SO, Mutua MK, van de Vijver S, Kyobutungi C. Evidence of a double burden of malnutrition in urban poor settings in nairobi, kenya. *PLoS One.* 2015;10(6):e0129943.
16. Ziraba AK, Fotso JC, Ochako R. Overweight and obesity in urban africa: A problem of the rich or the poor? *BMC Public Health.* 2009;9:465-2458-9-465.
17. Black RE, Victora CG, Walker SP, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. *Lancet.* 2013;382(9890):427-451.

18. United Nations Standing Committee on Nutrition (SCN). Sixth report on the world nutrition situation: Progress in nutrition. geneva: United nations. geneva: United nations. www.unscn.org/files/Publications/RWNS6/report/SCN_report.pdf. Updated 2010.
19. Wang Z, Zhai F, Zhang B, Popkin BM. Trends in chinese snacking behaviors and patterns and the social-demographic role between 1991 and 2009. *Asia Pac J Clin Nutr*. 2012;21(2):253-262.
20. Popkin BM, Conde W, Hou N, Monteiro C. Is there a lag globally in overweight trends for children compared with adults? *Obesity (Silver Spring)*. 2006;14(10):1846-1853.
21. Popkin BM. Recent dynamics suggest selected countries catching up to US obesity. *Am J Clin Nutr*. 2010;91(1):284S-288S.
22. Adair LS, Gordon-Larsen P, Du SF, Zhang B, Popkin BM. The emergence of cardiometabolic disease risk in chinese children and adults: Consequences of changes in diet, physical activity and obesity. *Obes Rev*. 2014;15 Suppl 1:49-59.
23. Dearth-Wesley T, Wang H, Popkin BM. Under- and overnutrition dynamics in chinese children and adults (1991-2004). *Eur J Clin Nutr*. 2008;62(11):1302-1307.
24. Gordon-Larsen P, Wang H, Popkin BM. Overweight dynamics in chinese children and adults. *Obes Rev*. 2014;15 Suppl 1:37-48.
25. Drewnowski A, Popkin BM. The nutrition transition: New trends in the global diet. *Nutr Rev*. 1997;55(2):31-43.
26. Zhai FY, Du SF, Wang ZH, Zhang JG, Du WW, Popkin BM. Dynamics of the chinese diet and the role of urbanicity, 1991-2011. *Obes Rev*. 2014;15 Suppl 1:16-26.

27. Ng SW, Howard AG, Wang HJ, Su C, Zhang B. The physical activity transition among adults in china: 1991-2011. *Obes Rev.* 2014;15 Suppl 1:27-36.
28. Jones-Smith JC, Popkin BM. Understanding community context and adult health changes in china: Development of an urbanicity scale. *Soc Sci Med.* 2010;71(8):1436-1446.
29. Zhang B, Zhai FY, Du SF, Popkin BM. The china health and nutrition survey, 1989-2011. *Obes Rev.* 2014;15 Suppl 1:2-7.
30. World Health Organization. Global health risks: Mortality and burden of disease attributable to selected major risks. geneva, switzerland: WHO.
http://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_full.pdf.
Updated 2009.
31. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: Systematic review. *Int J Obes (Lond).* 2011;35(7):891-898.
32. Murray CJ, Atkinson C, Bhalla K, et al. The state of US health, 1990-2010: Burden of diseases, injuries, and risk factors. *JAMA.* 2013;310(6):591-608.
33. Stanhope KL. Sugar consumption, metabolic disease and obesity: The state of the controversy. *Crit Rev Clin Lab Sci.* 2015:1-16.
34. Louzada ML, Baraldi LG, Steele EM, et al. Consumption of ultra-processed foods and obesity in brazilian adolescents and adults. *Prev Med.* 2015;81:9-15.
35. Ludwig DS. Technology, diet, and the burden of chronic disease. *JAMA.* 2011;305(13):1352-1353.

36. Ogden J, Coop N, Cousins C, et al. Distraction, the desire to eat and food intake. towards an expanded model of mindless eating. *Appetite*. 2013;62:119-126.
37. DiMiglio DP, Mattes RD. Liquid versus solid carbohydrate: Effects on food intake and body weight. *Int J Obes Relat Metab Disord*. 2000;24(6):794-800.
38. Li M, Dibley MJ, Sibbritt DW, Yan H. Dietary habits and overweight/obesity in adolescents in xi'an city, china. *Asia Pac J Clin Nutr*. 2010;19(1):76-82.
39. Denova-Gutierrez E, Jimenez-Aguilar A, Halley-Castillo E, et al. Association between sweetened beverage consumption and body mass index, proportion of body fat and body fat distribution in mexican adolescents. *Ann Nutr Metab*. 2008;53(3-4):245-251.
40. Denova-Gutierrez E, Talavera JO, Huitron-Bravo G, Mendez-Hernandez P, Salmeron J. Sweetened beverage consumption and increased risk of metabolic syndrome in mexican adults. *Public Health Nutr*. 2010;13(6):835-842.
41. Messiah SE, Vidot DC, Gurnurkar S, Alhezayen R, Natale RA, Arheart KL. Obesity is significantly associated with cardiovascular disease risk factors in 2- to 9-year-olds. *J Clin Hypertens (Greenwich)*. 2014;16(12):889-894.
42. Albar SA, Alwan NA, Evans CE, Cade JE. Is there an association between food portion size and BMI among british adolescents? *Br J Nutr*. 2014;112(5):841-851.
43. Vermeer WM, Steenhuis IH, Leeuwis FH, Bos AE, de Boer M, Seidell JC. Portion size labeling and intended soft drink consumption: The impact of labeling format and size portfolio. *J Nutr Educ Behav*. 2010;42(6):422-426.

44. Diliberti N, Bordi PL, Conklin MT, Roe LS, Rolls BJ. Increased portion size leads to increased energy intake in a restaurant meal. *Obes Res.* 2004;12(3):562-568.
45. Monteiro CA, Cannon G. The impact of transnational "big food" companies on the south: A view from brazil. *PLoS Med.* 2012;9(7):e1001252.
46. Ludwig DS. The glycemic index: Physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA.* 2002;287(18):2414-2423.
47. Brand-Miller J, McMillan-Price J, Steinbeck K, Caterson I. Dietary glycemic index: Health implications. *J Am Coll Nutr.* 2009;28 Suppl:446S-449S.
48. Iggman D, Rosqvist F, Larsson A, et al. Role of dietary fats in modulating cardiometabolic risk during moderate weight gain: A randomized double-blind overfeeding trial (LIPOGAIN study). *J Am Heart Assoc.* 2014;3(5):e001095.
49. Monteiro CA, Levy RB, Claro RM, de Castro IR, Cannon G. Increasing consumption of ultra-processed foods and likely impact on human health: Evidence from brazil. *Public Health Nutr.* 2011;14(1):5-13.
50. Moubarac JC, Martins AP, Claro RM, Levy RB, Cannon G, Monteiro CA. Consumption of ultra-processed foods and likely impact on human health. evidence from canada. *Public Health Nutr.* 2013;16(12):2240-2248.
51. Bravi F, Bosetti C, Filomeno M, et al. Foods, nutrients and the risk of oral and pharyngeal cancer. *Br J Cancer.* 2013;109(11):2904-2910.

52. Marcello MA, Sampaio AC, Geloneze B, Vasques AC, Assumpcao LV, Ward LS. Obesity and excess protein and carbohydrate consumption are risk factors for thyroid cancer. *Nutr Cancer*. 2012;64(8):1190-1195.
53. Tan HC, Roberts J, Catov J, Krishnamurthy R, Shypailo R, Bacha F. Mother's pre-pregnancy BMI is an important determinant of adverse cardiometabolic risk in childhood. *Pediatr Diabetes*. 2015;16(6):419-426.
54. Casas-Agustench P, Iglesias-Gutierrez E, Davalos A. Mother's nutritional miRNA legacy: Nutrition during pregnancy and its possible implications to develop cardiometabolic disease in later life. *Pharmacol Res*. 2015;100:322-334.
55. Pereira-Freire JA, Lemos JO, de Sousa AF, Meneses CC, Rondo PH. Association between weight at birth and body composition in childhood: A brazilian cohort study. *Early Hum Dev*. 2015;91(8):445-449.
56. Sarrafzadegan N, Kelishadi R, Sadri G, et al. Outcomes of a comprehensive healthy lifestyle program on cardiometabolic risk factors in a developing country: The isfahan healthy heart program. *Arch Iran Med*. 2013;16(1):4-11.
57. Shah P, Misra A, Gupta N, et al. Improvement in nutrition-related knowledge and behaviour of urban asian indian school children: Findings from the 'medical education for children/adolescents for realistic prevention of obesity and diabetes and for healthy ageing' (MARG) intervention study. *Br J Nutr*. 2010;104(3):427-436.
58. Lorga T, Aung MN, Naunboonruang P, Junlapeeya P, Payaprom A. Knowledge of communicable and noncommunicable diseases among karen ethnic high school students in rural thasongyang, the far northwest of thailand. *Int J Gen Med*. 2013;6:519-526.

59. Ramadan J, Vuori I, Lankenau B, Schmid T, Pratt M. Developing a national physical activity plan: The kuwait example. *Glob Health Promot*. 2010;17(2):52-57.
60. Bower JK, Hales DP, Tate DF, Rubin DA, Benjamin SE, Ward DS. The childcare environment and children's physical activity. *Am J Prev Med*. 2008;34(1):23-29.
61. Heinberg LJ, Kutchman EM, Berger NA, et al. Parent involvement is associated with early success in obesity treatment. *Clin Pediatr (Phila)*. 2010;49(5):457-465.
62. Hendriks AM, Gubbels JS, De Vries NK, Seidell JC, Kremers SP, Jansen MW. Interventions to promote an integrated approach to public health problems: An application to childhood obesity. *J Environ Public Health*. 2012;2012:913236.
63. Chopera P, Chagwena DT, Mushonga NG. Food label reading and understanding in parts of rural and urban zimbabwe. *Afr Health Sci*. 2014;14(3):576-584.
64. Godlee F. The food industry fights for salt. *BMJ*. 1996;312(7041):1239-1240.
65. Bibbins-Domingo K, Chertow GM, Coxson PG, et al. Projected effect of dietary salt reductions on future cardiovascular disease. *N Engl J Med*. 2010;362(7):590-599.
66. Cobiac LJ, Vos T, Veerman JL. Cost-effectiveness of interventions to reduce dietary salt intake. *Heart*. 2010;96(23):1920-1925.
67. Murray CJ, Lauer JA, Hutubessy RC, et al. Effectiveness and costs of interventions to lower systolic blood pressure and cholesterol: A global and regional analysis on reduction of cardiovascular-disease risk. *Lancet*. 2003;361(9359):717-725.

68. Rubinstein A, Colantonio L, Bardach A, et al. Estimation of the burden of cardiovascular disease attributable to modifiable risk factors and cost-effectiveness analysis of preventative interventions to reduce this burden in argentina. *BMC Public Health*. 2010;10:627-2458-10-627.
69. Smith-Spangler CM, Juusola JL, Enns EA, Owens DK, Garber AM. Population strategies to decrease sodium intake and the burden of cardiovascular disease: A cost-effectiveness analysis. *Ann Intern Med*. 2010;152(8):481-7, W170-3.
70. Stuckler D, Nestle M. Big food, food systems, and global health. *PLoS Med*. 2012;9(6):e1001242.
71. Cobiac LJ, Veerman L, Vos T. The role of cost-effectiveness analysis in developing nutrition policy. *Annu Rev Nutr*. 2013;33:373-393.
72. Stuckler D, McKee M, Ebrahim S, Basu S. Manufacturing epidemics: The role of global producers in increased consumption of unhealthy commodities including processed foods, alcohol, and tobacco. *PLoS Med*. 2012;9(6):e1001235.

INTRODUCTION

Throughout the world, the prevalence of non-communicable diseases (NCD) are increasing at an alarming rate. It is estimated that 1.1 billion adults globally are overweight, and 312 million of them are obese.¹ Being overweight and obese has contributed to an increase in cases of diabetes and hypertension, which have predisposed individuals to other chronic diseases such as cardiovascular disease. Obesity rates have tripled in the past couple of decades in developing countries due to lack of physical activity and overconsumption of cheap, energy-dense foods.¹ Low-energy dense foods provide fewer calories; therefore, more can be eaten without consuming excess calories. High-energy dense foods have a high concentration of calories per gram of food.²

The shifts in diet, activity patterns and body composition are occurring more rapidly than they did in Western countries. Modernization of the economy has been occurring in much of Asia, the Middle East, Latin America, northern Africa and many areas in sub-Saharan African in a short time frame of 10-20 years. In many Western societies, these changes occurred over many decades to a century or more.³ For instance, in the 1970s: many developing countries had food supply concerns; most households had no television; walking and biking was the main form of transportation; food trade was at a minimum; consumption of processed food was low; and most rural and urban occupations consisted of heavy labor.³ In the present day: laborers use machines like small gas-powered tractors; industrial technology has grown; sugary drinks and processed foods are more readily accessible; televisions, computers and cell phones are commonplace; and mass transit has replaced walking and biking.³ Even the poorest nations had access to a high fat diet (diet obtaining 20% of energy from fat) by 1990. An increase in the consumption of vegetable fats in low and high-income nations and an increase in consumption of animal fats in poor countries caused an increase in total fat consumption for both low and high-income countries.³ However, there was a decrease in fat intake for moderate-income countries.³

The main studies have estimated current prevalence rates and future demographics of being overweight or obese and non-communicable diseases.⁴⁻⁷ Initially, Western countries were associated with the highest rates of obesity. Now, obesity is more prevalent in urban populations of Africa, Central and South America, Asia, and Caribbean and Pacific Islands.⁸⁻¹¹ In the next two decades, developing countries will impact worldwide cardiovascular mortality at unprecedented proportions as predicted by the World Health Organization (WHO). NCDs account for 35% of all deaths in low- and middle-income countries. In one study, the prevalence of obesity exceeded 40% in a population of middle-aged urban Indian women.¹² Another study looked at trends in obesity and compared any rate of change in BMI of adolescents and adults from 1989 to 1997 in China.³ Not only did BMI increase, but the shape of the BMI distribution curve changed as well. When compared to the results from 1989, there were less underweight men and women and more overweight and obese adults in 1997. The proportion of overweight/obese men increased from 6.4% to 14.5% and in women from 11.5% to 16.2%. In the past three decades, the age-standardized mean BMI increased by 0.4–0.5 kg/m² per year.³ Being overweight/obese is the fifth leading cause of global mortality and linked to multiple non-communicable diseases.

In light of these findings, this review was started to determine if any differences exist with the NCD rates in urbanized versus rural populations in developing countries. Articles comparing these two populations were systematically reviewed. Specifically, any differences pertaining to being overweight or obese or BMI levels; hypertension rates or blood pressure levels; hypercholesterolemia rates or cholesterol levels; and diabetes rates or blood glucose levels were studied.

MATERIALS AND METHODS

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were utilized to complete the systematic review. The flow diagram was used. At each step of the diagram, the articles included or excluded from the study as well as the reasons why were recorded.

Methodological Approach

Using PubMed, a thorough review of the literature was conducted using various search terms related to the research topic. The database was systemically searched from 1995 up to December 2016. From these search terms, articles that pertained to differences in NCD rates and what factors may have influenced them were used in this systematic review. A variety of combinations were used including using the Mesh function for some of the search terms. The following are the search terms that were used:

1. (("Developing Countries"[Mesh]) AND "Diet"[Mesh]) AND "Hypertension"[Mesh] – 22 articles
2. (("Developing Countries"[Mesh]) AND "Hypertension"[Mesh]) AND diet – 35 articles
3. (("Developing Countries"[Mesh]) AND cardiovascular) AND diet – 89 articles
4. ((cardiovascular) AND urbanization) AND developing countries – 67 articles
5. (("Diet"[Mesh]) AND "Urbanization"[Mesh]) AND "Hypertension"[Mesh] – 8 articles
6. ((diet) AND urbanization) AND hypertension – 45 articles
7. ((cardiovascular) AND "Urbanization"[Mesh]) AND "Diet"[Mesh] – 14 articles
8. ((diet) AND cardiovascular) AND developing countries – 252 articles
9. (("Diet"[Mesh]) AND "Developing Countries"[Mesh]) AND cardiovascular – 46 articles
10. ((intervention) AND "Hypertension"[Mesh]) AND "Developing Countries"[Mesh] – 21 articles
11. (((("Diet"[Mesh]) AND urbanization) AND "Obesity"[Mesh]) AND "Developing Countries"[Mesh]) – 11 articles
12. (("Developing Countries"[Mesh]) AND "Obesity"[Mesh]) AND noncommunicable diseases – 19 articles
13. (("Diet"[Mesh]) AND "Developing Countries"[Mesh]) AND noncommunicable diseases – 13 articles

14. (("Smoking"[Mesh]) AND cardiovascular) AND "Developing Countries"[Mesh] – 64 articles
15. (("Diet"[Mesh]) AND "Smoking"[Mesh]) AND "Developing Countries"[Mesh] – 30 articles
16. (("Hypertension"[Mesh]) AND "Smoking"[Mesh]) AND "Developing Countries"[Mesh] – 32 articles
17. (("Rural Population"[Mesh]) AND "Urban Population"[Mesh]) AND cardiovascular – 441 articles
18. (("Rural Population"[Mesh]) AND "Urban Population"[Mesh]) AND cardiovascular developing countries – 26 articles
19. (("Rural Population"[Mesh]) AND "Urban Population"[Mesh]) AND non communicable diseases – 67 articles
20. rural population urban population developing countries diabetes – 87 articles
21. (((("Rural Population"[Mesh]) AND "Urban Population"[Mesh]) AND "Obesity"[Mesh]) AND developing countries – 22 articles
22. (((("Obesity"[Mesh]) AND "Hypertension"[Mesh]) AND "Developing Countries"[Mesh]) AND rural population – 12 articles
23. (("Dyslipidemias"[Mesh]) AND "Urban Population"[Mesh]) AND "Rural Population"[Mesh] – 78 articles
24. (((("Rural Population"[Mesh]) AND "Urban Population"[Mesh]) AND "Developing Countries"[Mesh]) AND obesity – 17 articles

The searches yielded 1518 articles, which 40 of those were determined to be relevant to the research question and were assessed for full eligibility.

The reasons why the majority of articles were excluded were based on the inclusion and exclusion criteria that were placed.

Inclusion criteria:

- Articles published after 1990 (only two articles are from before 2000; the rest are from after 2000)
- Urban versus rural groups were compared

- Groups were compared using either obesity rates, lipid levels, BMI, fasting glucose, blood pressure, and prevalence of hypertension, diabetes or other cardiovascular diseases.
- The studies had to be conducted in a developing country with a developing economy according to the International Monetary Fund or a newly industrialized country.
- Articles had to be published in the English language

By adhering to the inclusion criteria guidelines, the 40 articles were analyzed in depth to determine if they could be used in the review. Of those 9 of the articles were excluded. The reasons these were not used in the review were only one gender was studied, they focused more on nutritional epidemiology, measured mortality or did not report results in the form of prevalence or the mean of a population.

Data Collection and Analysis

Data extraction

Data from the 31 articles were extracted and collected onto a collection form. The data was organized in a way, which results pertaining to hypertension, BMI, dyslipidemia, and hyperglycemia could easily be identified. These results from each individual study were analyzed. Then, the results were analyzed as a whole to determine if any trends or significant differences occurred among the groups compared.

Assessment of risk of bias in individual studies

The author assessed the risk of bias in the studies that met the inclusion criteria using the Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies. This tool includes the specific criteria in Figure 1. If a “yes” was answered for all the criteria, it indicated a low risk of bias. If a “no” was answered for the criteria, it indicated a high risk of bias.

Summary measures

The effect size (Cohen’s d) between the rural and urban populations were utilized to demonstrate any differences in hypertension, obesity, hyperlipidemia, and hyperglycemia rates.

A Cohen's d of 0.2 is a 'small' effect size, 0.5 represents a 'medium' effect size and 0.8 a 'large' effect size. This means that if two groups' means don't differ by 0.2 standard deviations or more, the difference is trivial, even if it is statistically significant.

FIGURE 1

Criteria	Yes	No
1. Was the research question or objective in this paper clearly stated?		
2. Was the study population clearly specified and defined?		
3. Was the participation rate of eligible persons at least 50%?		
4. Were all the subjects selected or recruited from the same or similar populations (including the same time period)? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants?		
5. Was a sample size justification, power description, or variance and effect estimates provided?		
6. For the analyses in this paper, were the exposures(s) of interest measured prior to the outcome(s) being measured?		
7. Was the timeframe sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed?		
8. For the exposures that can vary in amount or level, did the study examine different levels of the exposure as related to the outcome?		
9. Were the exposure measures (independent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?		
10. Was the exposure(s) assessed more than once over time?		
11. Were the outcome measures (dependent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?		
12. Were the outcome assessors blinded to the exposure status of participants?		
13. Was loss to follow-up after baseline 20% or less?		
14. Were key potential confounding variables measured and adjusted statistically for their impact on the relationship between exposure(s) and outcome(s)?		

Figure 1: Quality assessment tool for observational cohort and cross-sectional studies.

RESULTS

Included studies

After a review of the literature, 31 articles met the inclusion and eligibility criteria of this systematic review (Figure 2). The results from the majority of the 31 articles did demonstrate differences in hypertension, obesity, hyperlipidemia, and hyperglycemia rates between urban versus rural populations. Either mean values or prevalence rates of the four mentioned variables were reported in the articles that were included for this study.

Twenty-six articles studied any differences in BMI values, obesity and/or being overweight rates, 21 articles studied any differences in blood pressure or hypertension rates, 24 articles studied any differences in cholesterol levels or hypercholesterolemia prevalence, and 12 articles studied any differences in glucose levels and/or rate of diabetes.¹³⁻⁴³ These studies were conducted in countries primarily in Africa, Southeast Asia, or Latin America. All participants, except in one study, were age ≥ 18 years old. All the included studies are observational or cross-sectional studies.

Risk of bias within included studies

The results of the assessment of risk in the individual studies are demonstrated in Table 1.

FIGURE 2

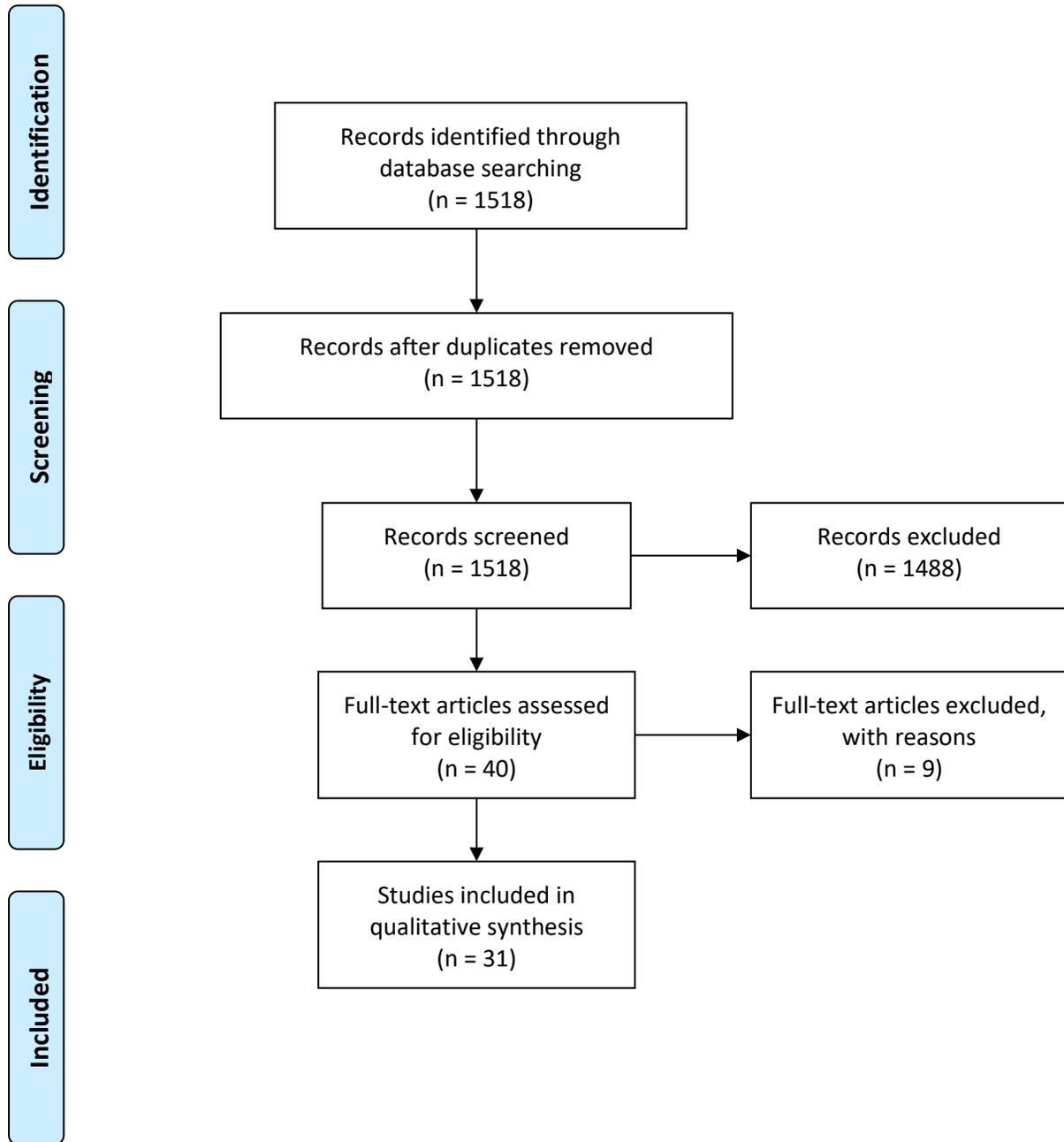


Figure 2: PRISMA Flow Diagram

Table 1: Assessment of risk within individual studies

Name of authors	Total number of criteria = 14		
	Number of yes (n)	Number of no (n)	Number of unknown (n)
Ulası et al. 2010	10	1	3
Adediran et al. 2013	9	1	4
Kinra et al. 2011	9	1	4
Vardavas et al. 2010	11	2	1
Saha et al. 2013	9	2	3
The InterASIA Collaborative Group et al. 2003	11	1	2
Gupta et al. 1997	11	1	2
Yajnik et al. 2013	11	1	2
Schutte et al. 2004	10	1	3
Sobngwi et al. 2004	12	1	1
Ramachandran et al. 2008	12	1	1
Adediran et al. 2012	9	1	4
Mbalilaki et al. 2007	10	1	3
Midha et al. 2009	11	1	2
Yamamoto-Kimura et al. 2006	11	2	1
Mendis et al. 1994	11	1	2
Joshi et al. 2014	12	1	1
Njelekela et al. 2002	9	1	4
Nongkynrih et al. 2008	10	1	3
Glew et al. 2004	10	1	3
Lissock et al. 2011	9	1	4
Torun et al. 2002	11	1	2
Zhang et al. 2013	8	1	5
Pongchaiyakul et al. 2006	10	1	3
Sabir et al. 2013	9	1	4
Pisa et al. 2012	10	1	3
Kende 2001	9	1	4
Abdul-Rahim et al. 2001	11	1	2
Mohan et al. 2008	10	1	3
Kumar et al. 2006	10	1	3
Mathenge et al. 2010	9	2	3

Comparing urban versus rural populations

BMI, obesity, and being overweight

Out of the 6 articles that studied mean BMI values, 4 articles did demonstrate significant differences ($p\text{-value} \leq 0.05$) between the urban and rural populations, with higher BMIs in the urban groups.¹⁶⁻²¹ One of these 4 articles studied the same population when the community was rural and then again 45 years later when it became urbanized.¹⁶ There was a statistically significant difference in the mean BMI between the two points in time. Two of the articles demonstrated no significant differences between the urban versus rural groups.^{17,20}

Fifteen articles separated BMI values based on gender as well. Out of the 15 studies, 12 of them showed statistically significant differences with higher BMIs in both genders residing in urban areas versus both genders in rural areas.^{14,15,23-25,27-29,34-36,38} In two of the studies considering gender differences, women in urban versus rural communities had no statistically significant differences in BMI whereas men in the same studies did.^{30,31} In one study, men had no statistically significant differences whereas women did.³³

Three studies demonstrated statistically significant differences in the rates of obesity and being overweight between the urban and rural populations.^{13,22,26} Two studies also considered gender differences and found a statistically significant increase in obesity rates in males residing in urban compared to rural communities.^{32,37} One of these studies demonstrated a significant increase in obesity rates in females living in urban communities versus rural, but the other study did not show a statistically significant increase.

To further assess for potential differences between the urban and rural populations, the effect size using Cohen's d was utilized to measure the size of associations or differences. Of the 15 articles that compared BMI values based on gender, only 9 studies reported the means with standard deviations. From these studies, the effect sizes of the urban versus rural male and female population and the two genders combined were measured. In the total male population ($N=17402$), there was a Cohen's $d=0.64$, $p < 0.001$, $CI.95 (0.45, 0.83)$. In the total female

population (N=18126), there was a Cohen's $d=0.56$, $p < 0.001$, CI.95 (0.31, 0.81). Overall, there was Cohen's $d=0.60$, $p < 0.001$, CI.95 (0.46, 0.74).^{14,24,25,27,29-31,34,36} (Figure 3)

Hypertension

Seven articles studied mean systolic and diastolic blood pressure values in the overall urban versus rural population. Four of these articles demonstrated statistically significant differences in systolic blood pressure.^{14,19-21} Five articles demonstrated significant differences in diastolic blood pressure in the urban versus rural groups.^{14,19-21,35} In all these cases, the urban population had higher blood pressures. There were no significant differences in the other studies.

Additionally, 8 articles sought to determine if differences in mean systolic and diastolic blood pressure existed between males and females in urban versus rural communities. While only 5 articles found statistically significant differences in systolic blood pressures between the urban versus rural genders, 6 of these articles showed statistically significant differences in diastolic blood pressures.^{23,25,28,33,36,38} The urban populations had the higher blood pressures.

Additionally, 6 articles studied any potential differences in the prevalence of hypertension between urban and rural populations. The cut off for hypertension in all these studies were 140/90 mmHg. One of these articles considered any differences in the prevalence of high blood pressure between males and females in the urban and rural communities.³⁷ It found a statistically significant increase of hypertension prevalence in both genders in the urban populations of $p\text{-value} \leq 0.05$. Four of the 6 articles showed a significant increase in the prevalence of hypertension in the overall urban versus the rural populations.^{13,18,22,39} One article showed a slight increase in the prevalence of hypertension in the urban population, but it was not statistically significant.²⁶

To further assess for potential differences between the urban and rural populations, the effect size using Cohen's d was utilized to measure the size of associations or differences. Of the 8 articles that compared mean systolic and diastolic blood pressures between males and females in urban versus rural communities, only 5 studies reported the means with standard deviations.

From these studies, the effect sizes of the urban versus rural male and female population and the two genders combined were measured. In the total male population (N=16155), there was a Cohen's $d=0.17$, $p = 0.001$, CI.95 (0.02, 0.32) for systolic pressures and a Cohen's $d=0.32$, $p < 0.001$, CI.95 (-0.08, 0.71) for diastolic pressures. In the total female population (N=16852), there was a Cohen's $d=0.22$, $p = 0.005$, CI.95 (0.09, 0.35) for systolic pressures and a Cohen's $d=0.38$, $p < 0.001$, CI.95 (-0.10, 0.86) for diastolic pressures. Overall, there was Cohen's $d=0.20$, $p < 0.001$, CI.95 (0.13, 0.28) for systolic pressures and Cohen's $d=0.36$, $p < 0.001$, CI.95 (0.17, 0.55) for diastolic pressures.^{14,25,29,30,36} (Figure 4)

Hypercholesterolemia

Ten articles studied the mean total cholesterol levels in the urban versus rural population. Eight of these articles showed a statistically significant increase in the total cholesterol value in the urban populations.^{17-19,22,37,40-42} There were no significant differences in the other two studies.^{20,35} Twelve articles looked into the mean total cholesterol levels between males and females in the urban versus rural groups. In females, 8 articles showed a significant increase in mean total cholesterol levels; four studies showed no differences between the females living in urban versus rural communities.^{23-25,27-31,33,34,38,43} In males, 10 articles showed a significant increase in mean total cholesterol levels; two studies showed no differences between the males living in urban versus rural communities.

One study looked specifically in the mean total triglyceride levels between the same population, but at two different points in time.¹⁶ At the first point in time, the population was rural, but became urbanized 45 years later. There was a significant increase in the mean total triglyceride level at the latest time point with a p -value < 0.001 . Another study determined if there was a difference in the prevalence of hypercholesterolemia in urban versus rural communities.²⁶ It showed there a significant increase in prevalence in the urban community with a p -value < 0.001 .

To further assess for potential differences between the urban and rural populations, the effect size using Cohen's *d* was utilized to measure the size of associations or differences. Of the 12 articles that compared total cholesterol levels based on gender, only 8 studies reported the means with standard deviations. From these studies, the effect sizes of the urban versus rural male and female population and the two genders combined were measured. In the total male population (N=4961), there was a Cohen's *d*=0.64, $p < 0.001$, CI.95 (0.38, 0.90). In the total female population (N=5581), there was a Cohen's *d*=0.55, $p < 0.001$, CI.95 (0.29, 0.81). Overall, there was Cohen's *d*=0.59, $p < 0.001$, CI.95 (0.42, 0.77).^{23-25,27,29,31,34,38} (Figure 5)

Hyperglycemia

Three articles studied mean total blood glucose levels in the urban versus rural population. All these articles showed a statistically significant increase in blood glucose values in the urban populations with a p -value < 0.001 .^{21,35,37} Seven articles looked into the mean total blood glucose levels between males and females in the urban versus rural groups. In females, five articles showed a significant increase in mean total glucose levels; two studies showed no differences between the females living in urban versus rural communities.^{25,33,34,38,43} In males, only four articles showed a significant increase in mean total blood glucose levels; three studies showed no differences between the males living in urban versus rural communities.^{25,34,38,43} In two other studies, the prevalence of diabetes was significantly increased in the urban compared to the rural population with a p -value = 0.02 in one study and p -value < 0.001 in the second study.^{18,22}

To further assess for potential differences between the urban and rural populations, the effect size using Cohen's *d* was utilized to measure the size of associations or differences. Of the 7 articles that compared blood glucose levels based on gender, only 4 studies reported the means with standard deviations. From these studies, the effect sizes of the urban versus rural male and female population and the two genders combined were measured. In the total male population (N=1687), there was a Cohen's *d*=-0.03, $p < 0.001$, CI.95 (-0.55, 0.49). In the total

female population (N=2100), there was a Cohen's $d=-0.09$, $p < 0.001$, CI.95 (-0.56, 0.38).

Overall, there was Cohen's $d=-0.07$, $p < 0.001$, CI.95 (-0.36, 0.22).^{25,29,30,34} (Figure 6)

FIGURE 3

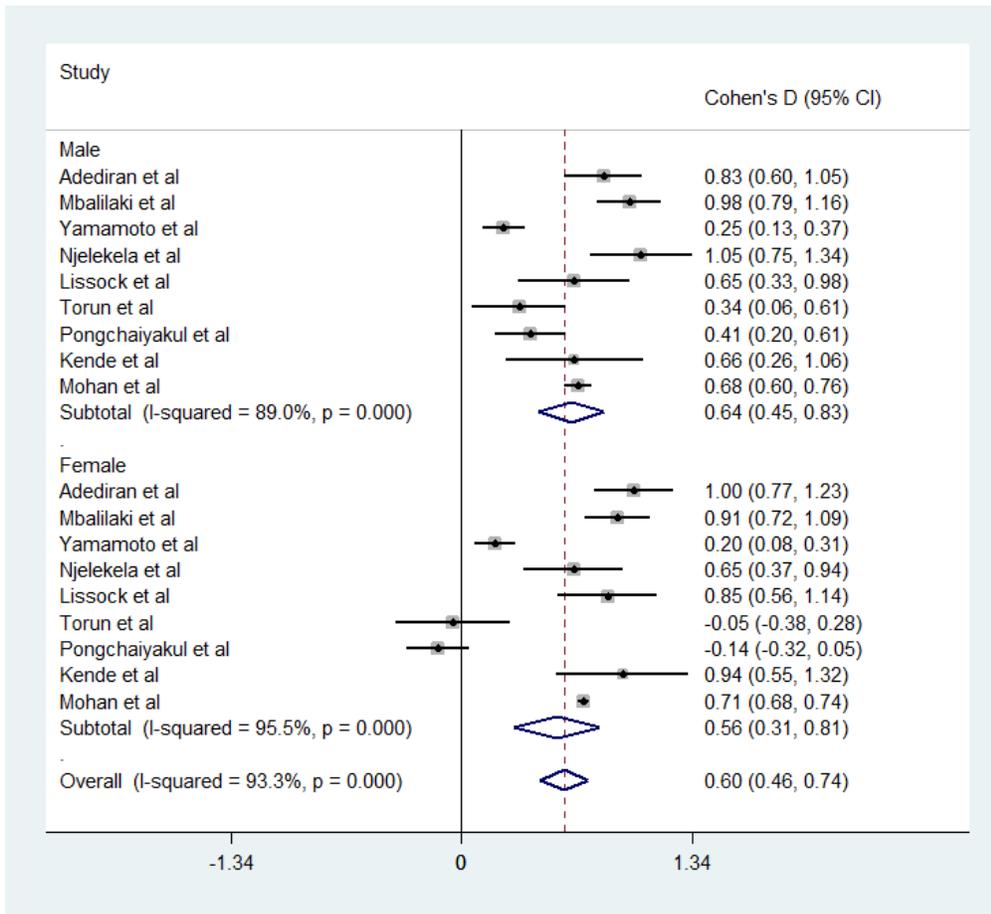


Figure 3: Effect sizes using Cohen's d of studies reporting mean BMI values with standard deviations in males and females in urban vs rural populations

FIGURE 4

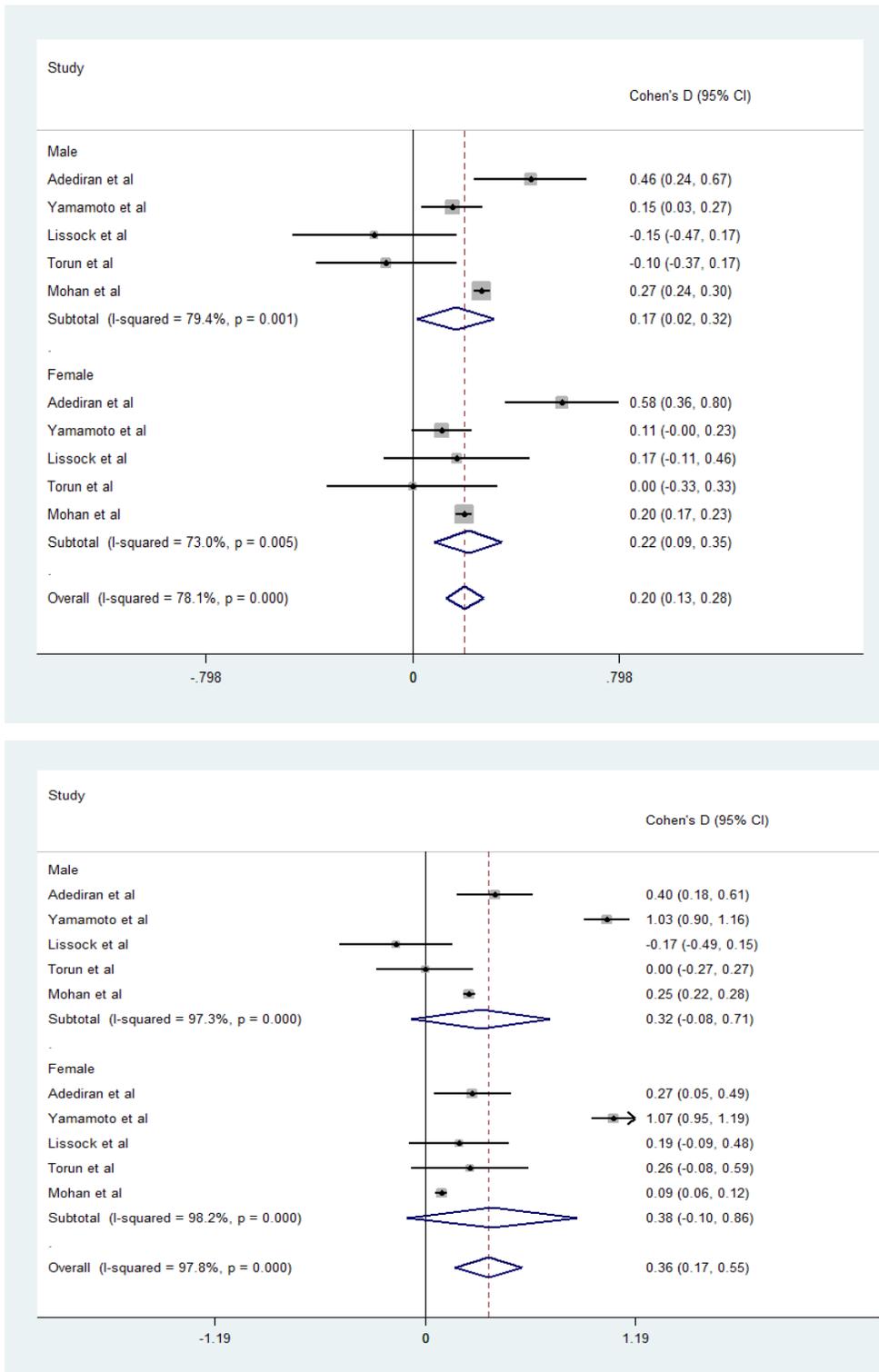


Figure 4: Effect sizes using Cohen's d of studies reporting mean systolic and diastolic blood pressures with standard deviations in males and females in urban vs rural populations

FIGURE 5

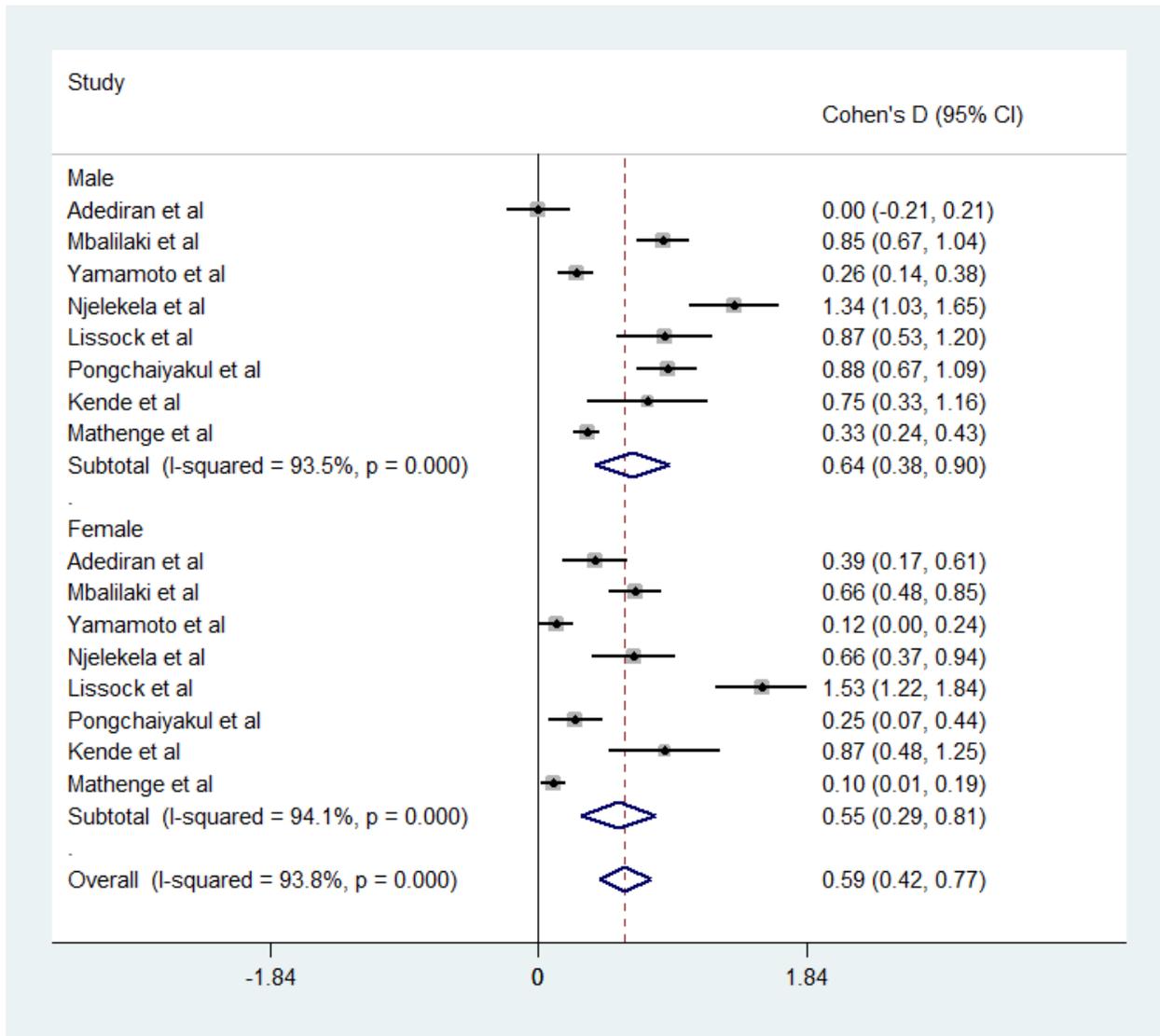


Figure 5: Effect sizes using Cohen's d of studies reporting mean total cholesterol levels with standard deviations in males and females in urban vs rural populations

FIGURE 6

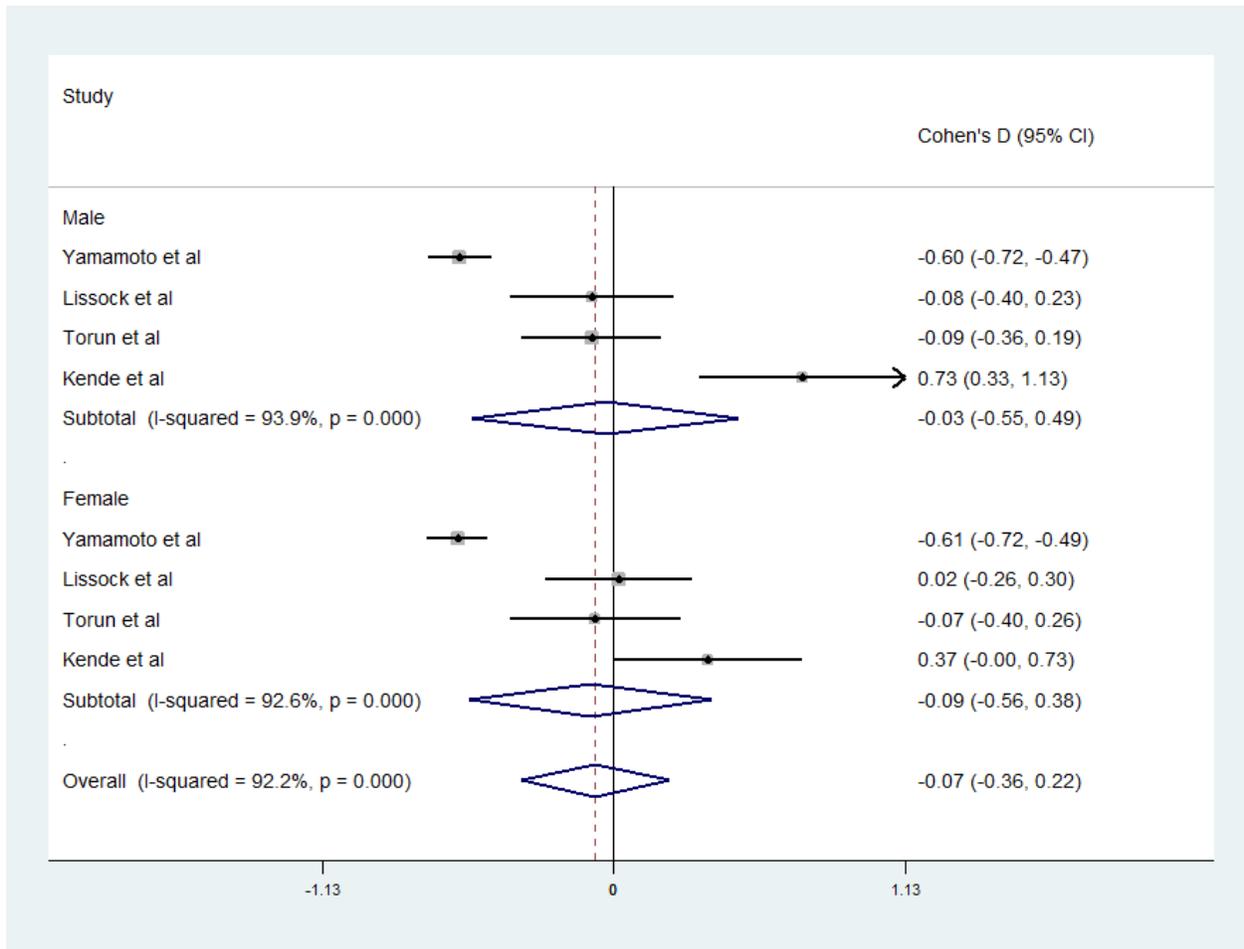


Figure 6: Effect sizes using Cohen's d of studies reporting mean blood glucose levels with standard deviations in males and females in urban vs rural populations

DISCUSSION

Summary of evidence

The objective of this review was to determine if any differences exist with the NCD rates in urbanized versus rural populations in developing countries. Articles pertaining to being overweight or obese or BMI levels; hypertension rates or blood pressure levels; hypercholesterolemia rates or cholesterol levels; and diabetes rates or blood glucose levels were reviewed. Only studies done in developing countries as defined by the IMF were included in the review. The vast majority of studies included in this review demonstrated statistically significant increases in BMI and rates of being overweight or obese in urban versus rural populations using effect sizes. A total of 9 studies reported the means with standard deviations. From these studies, the effect sizes of the urban versus rural male and female population and the two genders combined were measured. In the total male population (N=17402), there was a Cohen's $d=0.64$, $p < 0.001$, CI.95 (0.45, 0.83). In the total female population (N=18126), there was a Cohen's $d=0.56$, $p < 0.001$, CI.95 (0.31, 0.81). Overall, there was Cohen's $d=0.60$, $p < 0.001$, CI.95 (0.46, 0.74) with a medium effect size that indicates a significant difference between the urban and rural populations.

Most of the studies also demonstrated statistically significant increases in blood pressure values or rates of hypertension. Five studies reported mean systolic and diastolic blood pressures. In the total male population (N=16155), there was a Cohen's $d=0.17$, $p = 0.001$, CI.95 (0.02, 0.32) for systolic pressures and a Cohen's $d=0.32$, $p < 0.001$, CI.95 (-0.08, 0.71) for diastolic pressures. In the total female population (N=16852), there was a Cohen's $d=0.22$, $p = 0.005$, CI.95 (0.09, 0.35) for systolic pressures and a Cohen's $d=0.38$, $p < 0.001$, CI.95 (-0.10, 0.86) for diastolic pressures. Overall, there was Cohen's $d=0.20$, $p < 0.001$, CI.95 (0.13, 0.28) for systolic pressures and Cohen's $d=0.36$, $p < 0.001$, CI.95 (0.17, 0.55) for diastolic pressures with a small effect size that indicates a significant difference between the urban and rural populations. Eight studies reported mean cholesterol levels. In the total male population (N=4961), there was a Cohen's $d=0.64$, $p < 0.001$, CI.95 (0.38, 0.90). In the total female population (N=5581), there was a Cohen's $d=0.55$, $p < 0.001$, CI.95 (0.29, 0.81). Overall, there was Cohen's $d=0.59$, $p < 0.001$, CI.95 (0.42, 0.77) with a medium effect size that indicates a significant difference between the

urban and rural populations. Four studies reported mean blood glucose levels. In the total male population (N=1687), there was a Cohen's $d=-0.03$, $p < 0.001$, CI.95 (-0.55, 0.49). In the total female population (N=2100), there was a Cohen's $d=-0.09$, $p < 0.001$, CI.95 (-0.56, 0.38). Overall, there was Cohen's $d=-0.07$, $p < 0.001$, CI.95 (-0.36, 0.22) with an effect size that indicates no significant differences between the urban and rural populations.

The review specifically only looked at differences among being overweight or obese, hypertension, hypercholesterolemia or diabetes in urban versus rural communities in developing or recently developed countries to determine if a difference in NCD rates exists. Other NCDs exist in these countries such as chronic respiratory illnesses, coronary artery disease, chronic kidney disease, cancer, mental illnesses, etc. that were not looked at in this review. However, this review demonstrates that most of the evidence reported statistically significant increases in the four categories of NCDs listed above in urban versus rural populations. These results cannot be generalized to the other NCDs. A systematic review studying all these diseases will provide more definitive answers. If differences exist with most of the NCDs in urban versus rural areas, methods to address the issue could be implemented.

Limitations

One limitation of this systematic review was that no meta-analysis or measures of consistency were executed. This does limit the validity of the evidence as no statistical computations were done to combine the results of the studies. All the studies included in the systematic review were observational or cross-sectional studies. There is more strength in randomized control trials. As a result, randomization was not used to control for unknown and unmeasured differences between urban and rural populations. There was a potential for confounding variables.

Interpretation of results in the context of similar studies

One review by Okrainec et al. looked at risk factors for coronary artery disease, such as diabetes, genetic factors, hypercholesterolemia, hypertension, and smoking in the developing

world.⁴⁴ This differs from the current study as this review did not exclusively include only articles comparing the urban versus the rural populations. While there were a few articles with populations in India and Sub-Saharan Africa that compared the urban community with the rural one, the primary focus was to report risk factors for coronary artery disease in developing countries. Additionally, the Okrainec et al review studied the impact of socioeconomic status on coronary artery disease and prevention and control.⁴⁴ This current review focused solely on differences in NCD rates in urban versus rural populations. However, with the articles that compared the two populations, the review demonstrated there was an increase in prevalence of hypercholesterolemia, hypertension, unfavorable BMIs, and high blood glucose levels in the urban communities. This finding is consistent with the results of the present review. The conclusion of the review was the prevalence of coronary artery disease in developing countries are at epidemic levels.

FUTURE DIRECTIONS

A systematic review studying more types of NCDs will provide more definitive answers about whether living in an urbanized community influences the prevalence of these illnesses. If differences exist with most of the NCDs in urban versus rural areas, methods to address the issue could be evaluated and implemented. Programs that promote healthy lifestyle behaviors could be created to lower the prevalence of NCDs.

CONCLUSION

Cross-sectional and observational studies comparing BMI values, blood pressure levels, cholesterol levels, and blood glucose levels have addressed if living in urban versus rural areas increases the prevalence of NCDs related to these variables. Urban populations and living a sedentary lifestyle does increase the likelihood of being overweight or obese and having hypertension, hypercholesterolemia, but not diabetes.

REFERENCES

1. Hossain P, Kavar B, El Nahas M. Obesity and diabetes in the developing world — A growing challenge. *New England Journal of Medicine*. 2007;356(3):213-215.
<http://dx.doi.org.ezproxy4.library.arizona.edu/10.1056/NEJMp068177>. Accessed Dec 6, 2017.
doi: 10.1056/NEJMp068177.
2. British Nutrition Foundation. What is energy density? . Accessed 10/24/, 2016.
3. Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr*. 2002;5(1A):205-214. Accessed Dec 6, 2017. doi: 10.1079/PHN2001295.
4. Kastorini C, Milionis HJ, Ioannidi A, et al. Adherence to the mediterranean diet in relation to acute coronary syndrome or stroke nonfatal events: A comparative analysis of a case/case-control study. *Am Heart J*. 2011;162(4):717-724. Accessed Nov 30, 2017. doi: 10.1016/j.ahj.2011.07.012.
5. Nguyen TT, Adair LS, Suchindran CM, He K, Popkin BM. The association between body mass index and hypertension is different between east and southeast asians. *Am J Clin Nutr*. 2009;89(6):1905-1912. Accessed Nov 30, 2017. doi: 10.3945/ajcn.2008.26809.
6. Appropriate body-mass index for asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363(9403):157-163. Accessed Nov 30, 2017. doi: 10.1016/S0140-6736(03)15268-3.
7. Subramanian SV, Perkins JM, Özaltin E, Davey Smith G. Weight of nations: A socioeconomic analysis of women in low- to middle-income countries. *Am J Clin Nutr*. 2011;93(2):413-421. Accessed Nov 30, 2017. doi: 10.3945/ajcn.110.004820.
8. Martorell R, Khan LK, Hughes ML, Grummer-Strawn LM. Obesity in women from developing countries. *Eur J Clin Nutr*. 2000;54(3):247-252. Accessed Nov 30, 2017.

9. Popkin BM. The nutrition transition and obesity in the developing world. *J Nutr.* 2001;131(3):873S. Accessed Nov 30, 2017.
10. Popkin BM. The world is fat. *Sci Am.* 2007;297(3):88-95. Accessed Nov 30, 2017.
11. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. *J Clin Endocrinol Metab.* 2008;93(11 Suppl 1):9. Accessed Nov 30, 2017. doi: 10.1210/jc.2008-1595.
12. Pandey RM, Gupta R, Misra A, et al. Determinants of urban-rural differences in cardiovascular risk factors in middle-aged women in india: A cross-sectional study. *Int J Cardiol.* 2013;163(2):157-162. Accessed Nov 30, 2017. doi: 10.1016/j.ijcard.2011.06.008.
13. Ulasi II, Ijoma CK, Onodugo OD. A community-based study of hypertension and cardio-metabolic syndrome in semi-urban and rural communities in nigeria. *BMC Health Serv Res.* 2010;10:71. Accessed Nov 30, 2017. doi: 10.1186/1472-6963-10-71.
14. Adediran OS, Adebayo PB, Akintunde AA. Anthropometric differences among natives of abuja living in urban and rural communities: Correlations with other cardiovascular risk factors. *BMC Res Notes.* 2013;6:123. Accessed Nov 30, 2017. doi: 10.1186/1756-0500-6-123.
15. Kinra S, Andersen E, Ben-Shlomo Y, et al. Association between urban life-years and cardiometabolic risk: The indian migration study. *Am J Epidemiol.* 2011;174(2):154-164. Accessed Nov 30, 2017. doi: 10.1093/aje/kwr053.
16. Vardavas CI, Linardakis MK, Hatzis CM, Saris WHM, Kafatos AG. Cardiovascular disease risk factors and dietary habits of farmers from crete 45 years after the first description of the mediterranean diet. *Eur J Cardiovasc Prev Rehabil.* 2010;17(4):440-446. Accessed Nov 30, 2017. doi: 10.1097/HJR.0b013e32833692ea.
17. Saha S, Gupta K, Kumar S. Cardiovascular health among healthy population of northeast region of india: A cross-sectional study comparing urban-tribal difference. *J Indian Med Assoc.* 2013;111(12):814, 816. Accessed Nov 30, 2017.

18. Cardiovascular risk factor levels in urban and rural thailand--the international collaborative study of cardiovascular disease in asia (InterASIA). *Eur J Cardiovasc Prev Rehabil.* 2003;10(4):249-257. Accessed Nov 30, 2017. doi: 10.1097/01.hjr.0000085254.65733.21.
19. Yajnik CS, Joglekar CV, Chinchwadkar MC, et al. Conventional and novel cardiovascular risk factors and markers of vascular damage in rural and urban indian men. *Int J Cardiol.* 2013;165(2):255-259. Accessed Nov 30, 2017. doi: 10.1016/j.ijcard.2011.08.053.
20. Schutte R, Huisman HW, Malan L, et al. Differences in cardiovascular function of rural and urban african males: The THUSA study. *Cardiovasc J S Afr.* 2004;15(4):161-165. Accessed Nov 30, 2017.
21. Sobngwi E, Mbanya J, Unwin NC, et al. Exposure over the life course to an urban environment and its relation with obesity, diabetes, and hypertension in rural and urban cameroon. *Int J Epidemiol.* 2004;33(4):769-776. Accessed Nov 30, 2017. doi: 10.1093/ije/dyh044.
22. Ramachandran A, Mary S, Yamuna A, Murugesan N, Snehalatha C. High prevalence of diabetes and cardiovascular risk factors associated with urbanization in india. *Diabetes Care.* 2008;31(5):893-898. Accessed Nov 30, 2017. doi: 10.2337/dc07-1207.
23. Adediran O, Akintunde AA, Edo AE, Opadijo OG, Araoye AM. Impact of urbanization and gender on frequency of metabolic syndrome among native abuja settlers in nigeria. *J Cardiovasc Dis Res.* 2012;3(3):191-196. Accessed Nov 30, 2017. doi: 10.4103/0975-3583.98890.
24. Mbalilaki JA, Hellènius M-, Masesa Z, Høstmark AT, Sundquist J, Strømme SB. Physical activity and blood lipids in rural and urban tanzanians. *Nutr Metab Cardiovasc Dis.* 2007;17(5):344-348. Accessed Nov 30, 2017. doi: 10.1016/j.numecd.2006.03.003.
25. Yamamoto-Kimura L, Posadas-Romero C, Posadas-Sánchez R, Zamora-González J, Cardoso-Saldaña G, Méndez Ramírez I. Prevalence and interrelations of cardiovascular risk factors in

- urban and rural mexican adolescents. *J Adolesc Health*. 2006;38(5):591-598. Accessed Nov 30, 2017. doi: 10.1016/j.jadohealth.2005.04.004.
26. Mendis S, Ekanayake EM. Prevalence of coronary heart disease and cardiovascular risk factors in middle aged males in a defined population in central sri lanka. *Int J Cardiol*. 1994;46(2):135-142. Accessed Nov 30, 2017.
27. Njelekela M, Kuga S, Nara Y, et al. Prevalence of obesity and dyslipidemia in middle-aged men and women in tanzania, africa: Relationship with resting energy expenditure and dietary factors. *J Nutr Sci Vitaminol*. 2002;48(5):352-358. Accessed Nov 30, 2017.
28. Glew RH, Conn CA, Vanderjagt TA, et al. Risk factors for cardiovascular disease and diet of urban and rural dwellers in northern nigeria. *J Health Popul Nutr*. 2004;22(4):357-369. Accessed Nov 30, 2017.
29. Lissock, Clarisse Noël Ayina Ayina, Sobngwi E, Ngassam E, Ngoa Etoundi LS. Rural and urban differences in metabolic profiles in a cameroonian population. *Pan Afr Med J*. 2011;10:1. Accessed Nov 30, 2017.
30. Torun B, Stein AD, Schroeder D, et al. Rural-to-urban migration and cardiovascular disease risk factors in young guatemalan adults. *Int J Epidemiol*. 2002;31(1):218-226. Accessed Nov 30, 2017.
31. Pongchaiyakul C, Hongsprabhas P, Pisprasert V, Pongchaiyakul C. Rural-urban difference in lipid levels and prevalence of dyslipidemia: A population-based study in khon kaen province, thailand. *J Med Assoc Thai*. 2006;89(11):1835-1844. Accessed Nov 30, 2017.
32. Zhang Y, Wang S. Rural-urban comparison in prevalence of overweight and obesity among adolescents in shandong, china. *Ann Hum Biol*. 2013;40(3):294-297. Accessed Nov 30, 2017. doi: 10.3109/03014460.2013.772654.

33. Pisa PT, Behanan R, Vorster HH, Kruger A. Social drift of cardiovascular disease risk factors in africans from the north west province of south africa: The PURE study. *Cardiovasc J Afr*. 2012;23(7):388. Accessed Nov 30, 2017. doi: 10.5830/CVJA-2012-018.
34. Kende M. Superiority of traditional village diet and lifestyle in minimizing cardiovascular disease risk in papua new guineans. *P N G Med J*. 2001;44(3-4):135-150. Accessed Nov 30, 2017.
35. Abdul-Rahim HF, Hussein A, Bjertness E, Giacaman R, Gordon NH, Jervell J. The metabolic syndrome in the west bank population: An urban-rural comparison. *Diabetes Care*. 2001;24(2):275-279. Accessed Nov 30, 2017.
36. Mohan V, Mathur P, Deepa R, et al. Urban rural differences in prevalence of self-reported diabetes in india--the WHO-ICMR indian NCD risk factor surveillance. *Diabetes Res Clin Pract*. 2008;80(1):159-168. Accessed Nov 30, 2017. doi: 10.1016/j.diabres.2007.11.018.
37. Kumar R, Singh MC, Singh MC, et al. Urbanization and coronary heart disease: A study of urban-rural differences in northern india. *Indian Heart J*. 2006;58(2):126-130. Accessed Nov 30, 2017.
38. Mathenge W, Foster A, Kuper H. Urbanization, ethnicity and cardiovascular risk in a population in transition in nakuru, kenya: A population-based survey. *BMC Public Health*. 2010;10:569. Accessed Nov 30, 2017. doi: 10.1186/1471-2458-10-569.
39. Midha T, Idris MZ, Saran RK, Srivastav AK, Singh SK. Prevalence and determinants of hypertension in the urban and rural population of a north indian district. *East Afr J Public Health*. 2009;6(3):268-273. Accessed Nov 30, 2017.
40. Sabir AA, Isezuo SA, Ohwovoriole AE, et al. Rural-urban difference in plasma lipid levels and prevalence of dyslipidemia in hausa-fulani of north-western nigeria. *Ethn Dis*. 2013;23(3):374-378. Accessed Nov 30, 2017.

41. Gupta R, Prakash H, Kaul V. Cholesterol lipoproteins, triglycerides, rural-urban differences and prevalence of dyslipidaemia among males in rajasthan. *J Assoc Physicians India*. 1997;45(4):275-279. Accessed Nov 30, 2017.
42. Joshi SR, Anjana RM, Deepa M, et al. Prevalence of dyslipidemia in urban and rural india: The ICMR-INDIAB study. *PLoS ONE*. 2014;9(5):e96808. Accessed Nov 30, 2017. doi: 10.1371/journal.pone.0096808.
43. Nongkynrih B, Acharya A, Ramakrishnan L, Ritvik n, Anand K, Shah B. Profile of biochemical risk factors for non communicable diseases in urban, rural and periurban haryana, india. *J Assoc Physicians India*. 2008;56:165-170. Accessed Nov 30, 2017.
44. Okrainec K, Banerjee DK, Eisenberg MJ. Coronary artery disease in the developing world. *Am Heart J*. 2004;148(1):7-15. doi: 10.1016/j.ahj.2003.11.027 [doi].