

**INTERNATIONAL JOURNAL OF FOOD AND
NUTRITIONAL SCIENCES**

IMPACT FACTOR ~ 1.021



Official Journal of IIFANS

METABOLIC SYNDROME-CONCEPTS AND CRITERIAS: A REVIEW

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Received on: 14th March, 2017

Accepted on: 13th April, 2017

Metabolic syndrome, defined as a “constellation of an interconnected physiological, biochemical, clinical and metabolic factors that directly increases the risk of atherosclerotic cardiovascular disease, Type 2 Diabetes Mellitus and all cause mortality” is one of the most escalating public health problems effecting populations worldwide. At present it is gaining recognition because of increasing understanding of its association with cardiovascular mortality and morbidity. Present review is an attempt to glance into the historical prospects of the syndrome and describe the characteristics of the four most extensively accepted criteria’s documented by WHO (1998), EGIR (1999), NCEP-ATPIII (2005, Revised), IDF (2005). The common components of the metabolic syndrome included in these defining criteria’s include insulin resistance, abdominal obesity, lipid abnormalities and elevated blood pressure however, their defining components and diagnostic thresholds are emphasized differently highlighting the need to evolve criteria which is uniform for all the populations worldwide which will help in comparing different populations.

Keywords: Metabolic syndrome, Insulin resistance syndrome, Prevalence

INTRODUCTION

Nutrition, demographic, epidemiological and socioeconomic transitions are occurring in many developing countries .Continuing under nutrition and escalating over nutrition has created double jeopardy of communicable and non communicable diseases (Misra, 2002; and Reddy, 2002).It is a major escalating public health challenge worldwide (Zimmet and Alberti, 2008) .

Metabolic syndrome is defined by a constellation of an interconnected physiological, biochemical, clinical and metabolic factors that directly increases the risk of atherosclerotic cardiovascular disease (ASCVD), Type 2Diabetes Mellitus and all cause mortality (Grundy *et al.*, 2005; and Wilson *et al.*, 2005). It is a clinical challenge worldwide in the wake of urbanization, surplus energy intake, increasing obesity and sedentary life habits (Kaur, 2014). The underlying causes of the syndrome are genetic

and environmental, overweight, obesity, and physical inactivity, which lead to insulin resistance, hyperinsulinemia, endothelial dysfunction and inflammation (Ridker *et al.*, 2003). Affected individuals have a two to five of developing Type 2 diabetes and to suffer cardiovascular related mortality leading to a great impact on public health costs and planning (Eckel *et al.*, 2005; and Alberti *et al.*, 2009).

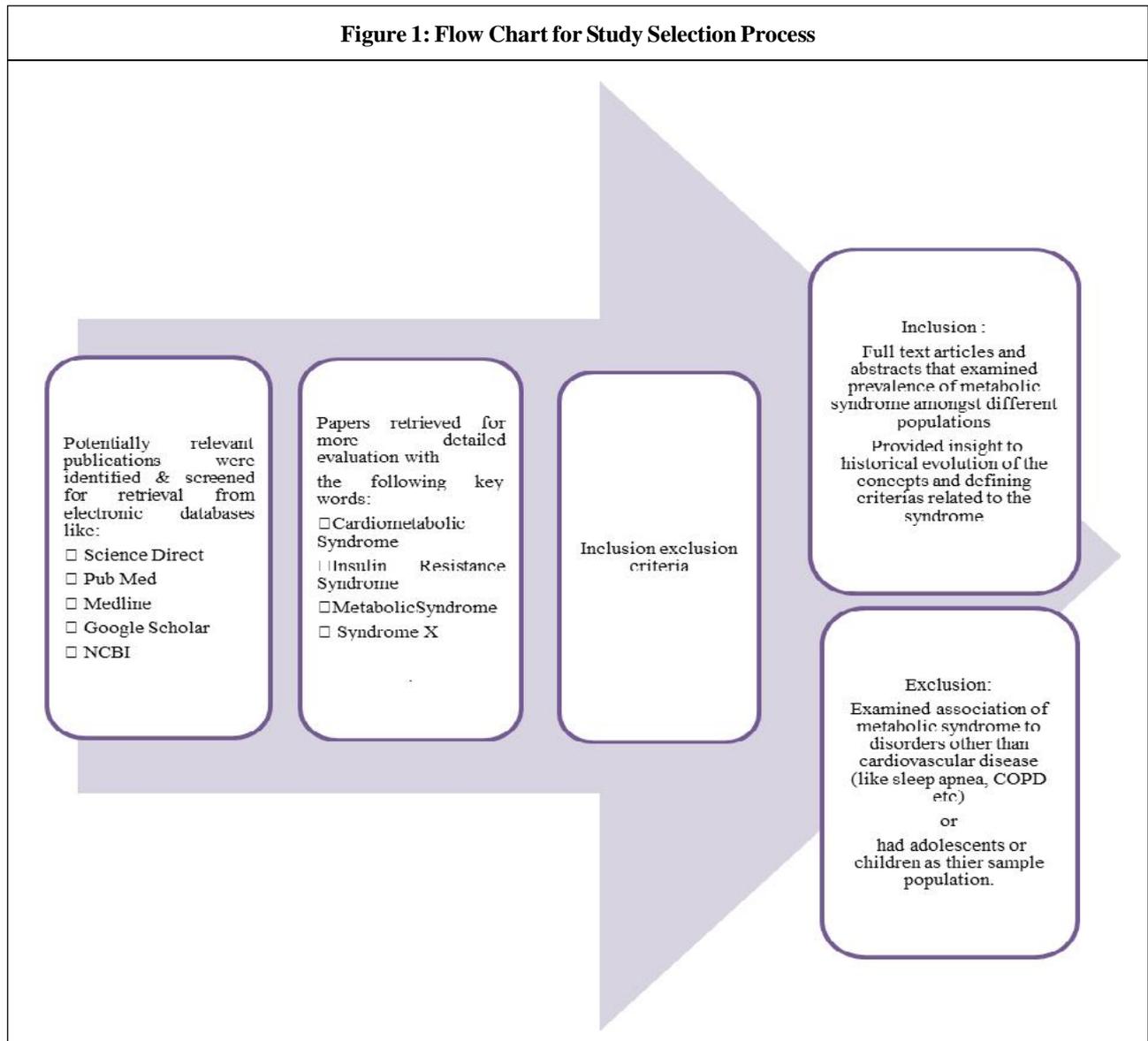
METHODOLOGY

Relevant articles were identified by searching the PubMed, Science Direct, Google Scholar, NCBI and Medline databases. Research articles including cross-sectional, behavioural and longitudinal studies, reviews were selected. Key words such as “Metabolic Syndrome”, “Insulin Resistance Syndrome”, “Prevalence” were used in the search strategy (Figure 1).

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Figure 1: Flow Chart for Study Selection Process



Studies considered for inclusion in this review were full text articles and abstracts that examined prevalence of metabolic syndrome and had adults as their sample population. Articles which were not in English were translated and then used for the present review. We excluded studies that examined association of metabolic syndrome to disorders other than cardiovascular disease (like sleep apnea, COPD, etc.), or were conducted on children or adolescents as sample population.

Since the review is an attempt to highlight the historical insight associated with the development of the concepts and criterias of the syndrome, attempts were made to retrieve earliest possible documentations.

RESULTS AND DISCUSSIONS

Historical Insight

Although the present era of 'Metabolic Syndrome' or Insulin Resistance Syndrome' seems to have started few decades ago with the description of Syndrome X by Reaven in the late 1980's the history of this syndrome is much longer (Sarafidis and Nisson, 2006), where it started off as a concept rather than diagnosis (Shaw and Chilsom, 2003). It was as early as 250 years ago, long before Metabolic Syndrome description when the Italian physician and anatomist Morgagni identified the association between visceral obesity, hypertension (arterial hypertension), atherosclerosis, the high level of uric acid in the blood and

the frequent respiratory disorders during sleep (the obstructive sleep apnea) (Crepaldi and Stefania, 2006).

Nicole Paulesci 1920, has said about obesity and diabetes that most frequently the obese people become glycosuric, as if the two affections (obesity and fat diabetes) represent two consequent phases of the same pathological process (Milici, 2010). Few years later its existence was demonstrated by a Swedish physician Kylin in 1923 where he described syndrome characterized by hypertension, hyperglycemia, obesity and hyperurecemia (Oladejo, 2011). He identified a triad of hypertension, hyperglycemia and gout in his patients (Dang and Deswal, 2014). In 1927, Maranon, the founder of modern endocrinology in Spain precisely described that arterial hypertension and obesity is a pre-diabetic condition. He also believed that food is essential for preventing and treating these metabolic disturbances (Sharma *et al.*, 2015). French Physician Jean Vague drew attention to upper body adiposity (android or male type obesity) as obesity phenotype that was commonly associated with abnormalities found in Type II diabetes and cardiovascular disease (Vague, 1947). Thereafter, almost two decades later, Avogadro, Crepaldi and co-workers presented an abstract at the annual meeting of the European Association for the study of diabetes (1965) where they reported the positive affect of hypocaloric, low carbohydrate diet on obese patients with diabetes, hypercholesterolemia and hyperglyceredemia and confirmed a strict relationship among these three metabolic disorders (Okafor, 2012). This frequent simultaneous presence of obesity, high blood fat, diabetes and hypertension was termed as ‘Plurimetabolic Syndrome’ (Crepaldi and Maggi, 2006).

This was followed by a Dresden Study where Herman Haller pioneered the term ‘Metabolic Syndrome’ in his scientific literature as association of obesity, diabetes mellitus, hyperlipoproteinemia, hyperurecemia, stenosis hepatitis) with increase of viscosity of the blood and plasma as well as disturbances of coagulation together with other factors of risk further the development of arteriosclerosis or has directing influence on it (Haller, 1977). Singer, in the very same year suggested that hyperlipoproteinemia are frequently associated with other metabolic diseases (obesity, gout, diabetes mellitus, and hypertension) and so called Metabolic Syndrome (Singer, 1977). A year later Gerald & Philips conceptualized that “Constellation of Abnormalities”, i.e., glucose intolerance and hyperinsulinemia, hyperglycemia and hypertension is associated with myocardial infarction, obesity and ageing

(Philips, 1978). In 1985, Michael Modan proposed that hyperinsulinemia was the link between hypertension, obesity and glucose intolerance. They signified that insulin resistance and/or hyperinsulinemia are present in the majority of hypertensives, constitute a common pathophysiologic features of obesity, glucose intolerance and hypertension, possibly explaining their ubiquitous association (Modan *et al.*, 1985).

It was in 1988, when Reaven, an endocrinologist from Stanford University at the banting lecture of American Diabetes Association linked the constellation of metabolic abnormalities to insulin resistance as a key component .He described “a cluster of risk factors for diabetes and cardiovascular disease” and named it “Syndrome X”. However, he missed obesity/visceral obesity from the definition which was later added as a crucial abnormality (Reaven, 1988; and Kaur, 2014). Syndrome X has also been called ‘Reaven’s Syndrome’ (Leslie, 2005). The syndrome was renamed as the “Deadly Quartet” in by Kaplan (1989) where he referred the term to the four clinical findings, i.e., obesity, Insulin resistance/intolerance, hypertriglycerdemia and hypertension which are common and coexist oftenly and are associated with cardiovascular mortality and morbidity (Kaplan, 1989). Since then number of studies depicted the broad spectrum of metabolic disorders. In 1991, Defronzo and Ferrannini renamed Syndrome X as IRS (Insulin Resistance syndrome) proposing that Insulin Resistance appears to be a syndrome associated with clustering metabolic disorders including NIDDM, Obesity, Hypertension, lipid abnormalities and atherosclerotic cardiovascular disease (Defronzo and Ferrannini, 1991). Later, Namakura *et al.* studied associations between intra-abdominal visceral fat accumulation and coronary risk factors and proposed the term “Visceral fat syndrome” that encompasses fat accumulation, glucose intolerance,

Table 1: Synonyms for Metabolic Syndrome

<p>Insulin Resistance Syndrome Cardiometabolic Syndrome Plurimetabolic Syndrome Syndrome X The Deadly Quartet Visceral Fat Syndrome Atherogenic Metabolic Triad</p>

Table 2: Defining Criteria for Metabolic Syndrome

Criteria	WHO (1998)	EGIR (1999)	NCEP-ATPIII (2005, Revised)	IDF (2005)
Mandatory component	Insulin Resistance /Diabetes/IGT/IFG/T2 DM or other evidence of IR*	Hyperinsulinemia ^s (Plasma Insulin >75 th percentile)	None	Central obesity (defined as waist circumference with ethnicity-specific values [†]) But; if BMI is > 30 kg/m ² central obesity can be assumed and WC doesn't need to be measured
Other additional components	Plus Any 2 of the following:	Plus Any 2 of the following:	Any 3 of the following:	Plus Any 2 of the following:
	WHR	WC	WC	BP/HTN
	>0.90 (men), 0.85 (women) or BMI >30 Kg/m ²	>94 cm (men) >80 cm (women)	>102 cm (men) >88 cm (women)	≥130/85 mmHg or BP ≥130/85 mmHg or treatment of previously diagnosed HTN
	HTN	TG	TG	HDL
	≥140/90 mmHg	≥177 mg/dl or	>150 mg/dl or R _x	<40 mg/dl or <1.03 mmol/L (men) <50 mg/dl or <1.29 mmol/L (women)
Dyslipidemia				
	TG	HDL-C	BP	Raised TGL
	150 mg/dl (or greater) or ≥1.7 mmol/L	<39 mg/dl in men or women	≥130/85 mmHg or R _x	≥150 mg/dl or ≥1.7 mmol/L or specific treatment for this lipid abnormality
	HDL-C	BP	HDL	Raised fasting plasma glucose
	<35 mg/dl or <0.9 mmol/L (men) <39 mg/dl or <1.0 mmol/L (women)	≥140/90 mmHg or on anti-hypertensive drugs	<40 mg/dl (men) <50 mg/dl (women) or R _x	≥100 mg/dl (5.6 mmol/L) or previously diagnosed type 2 diabetes (If above 5.6 mmol/L/100 mg/dL, OGTT is strongly recommended but is not necessary to define the presence of the syndrome)
	Microalbuminuria	Fasting glucose	Fasting Glucose	
	(Albumin: creatinine ratio >30 mg/g or greater) or urinary albumin excretion rate ≥20 g/min	110 mg/dl or greater.	100 mg/dl or greater or R _x	

Note: *IR (Other evince of IR (Family history of type 2 diabetes, age, ethnicity, sedentary lifestyle); R_x pharmacological treatment; ^shyperinsulinemia: reliable only in patient without diabetes; [†]Waist circumference specific for each population (IDF, 2005; South Asian/Chinese/Japanese: Men90 cm and Women-80 cm; Europids: Men94 cm and Women-80 cm; In USA, the ATP III values (102 cm male; 88 cm female) are likely to continue to be used for clinical purposes.

hyperlipidemia and hypertension (Nakamura *et al.*, 1994). In Lamarche *et al.* (1998) reported a combination of hyperinsulinemia, elevated adipolipoprotein B, and small dense Low Density Lipoprotein (LDL) cholesterol as the “Atherogenic Metabolic Triad” (Lamarche *et al.*, 1998; and Oda, 2008).

Present Day Defining Criteria for Metabolic Syndrome

In an effort to define clinical criteria for the metabolic syndrome many International expert groups have recommended different clinical criteria for the metabolic syndrome (Table 2). The most extensively accepted criteria’s are documented by:

- World Health Organization, WHO (1998)
- European Group for the study of Insulin Resistance, EGIR (1999)
- National Cholesterol Education Program–Adult Treatment Panel III, NCEP-ATPIII (2005, Revised)
- International Diabetes Federation, IDF (2005)

The major components of the metabolic syndrome included in these definitions are similar, but components and their diagnostic threshold values are emphasized differently in different definitions (Qiao, 2006).

World health organization first developed its definition in 1998 (Alberti and Zimmet, 1998) tying together the key components of Insulin Resistance, obesity, dislipidemia and hypertension. The definition mandates that IR to be present ,without it even if all the other criteria were met, the patient would not have metabolic syndrome (Huang, 2009). Potential disadvantage of WHO criteria is that special testing of glucose status beyond routine clinical assessment may be necessary to diagnose metabolic syndrome (Grundy *et al.*, 2004).

One year later, European Group for the study of Insulin resistance proposed a modification to the WHO definition (Balkau and Charles, 1999). EGIR focused more on abdominal obesity than did WHO but in contrast to WHO, it excluded patients with Type II diabetes mellitus from their syndrome because Insulin Resistance was viewed primarily as a risk factor for Diabetes (Parikh and Mohan, 2012). It excluded microalbuminuria as an integral component of the syndrome ,in addition waist circumference and not BMI was regarded as the main indicator to assess obesity (94 cm in men and 80 cm in women) (Balkau and Charles, 1999).

NCEP ATPIII presented a new approach that was less ‘glucentric’ and treated all components with equal importance, i.e., central obesity, hypertriglycerdemia, HDL, hypertension, and fasting plasma glucose. Waist circumference was used to assess obesity but higher cut offs were used to assess the same (i.e., 102 cm for men and 88 cm for women). International Diabetes federation mandates the presence of ‘central obesity’ to be an essential component for the presence of metabolic syndrome defined as waist circumference waist ethnicity specific values (Table 3).

Table 3: Ethnic Specific Values for Waist Circumference (IDF, 2005) (Gender and Ethnic Group Specific)

Country/Ethnic Group	Waist Circumference (as Measure of Central Obesity)	
		Male
Europids*	Male	≥94 cm
	Female	≥80 cm
South Asians**	Male	≥90 cm
	Female	≥80 cm
Chinese	Male	≥90 cm
	Female	≥80 cm
Japanese***	Male	≥90 cm
	Female	≥80 cm
Ethnic South and Central Americans	Using South Asian recommendations until more specific data are available	
Sub-Saharan Africans	Use European data until more specific data are available	
Eastern Mediterranean and Middle East (Arab) population	Use European data until more specific data are available	

Note: *In the USA, the ATP III values (102 cm male; 88 cm female) are likely to continue to be used for clinical purposes; **Based on a Chinese, Malay and Asian Indian population; ***Subsequent data analyses suggest that Asian values (male, 90 cm; female 80 cm) should be used for Japanese populations until more data are available.

Worldwide

The prevalence of MS is increasing in epidemic proportions in both developed countries and developing countries (Cornier *et al.*, 2008). Table 4 represents the prevalence of

Table 4: Prevalence of Metabolic Syndrome Worldwide

Author	Year [€]	State/Population	Sample Size (n)	Prevalence (%)	Criteria Used*
Tan <i>et al</i>	2004	Singapore	4723	17.9	*M-NCEP-ATPIII
		(used data from Singapore National Health Survey)		Female 15.35	
		Male 20.9			
OH & Associates	2004	South Korea	774	22.9	*M-NCEP-ATPIII
		(Urban population)		Female 16.8	
		Male 29			
Sidorenkov	2010	Russia	3705	15.6	NCEP-ATPIII
		(adults 18-19 years) used survey data conducted in 2000, Arkhagelak)		Female 19.8	
		Male 11.5			
Metelskaya <i>et al</i>	2012	Russia	1788	34.2	NCEP-ATPIII
		(Elderly population 55 years and above)		Female 41.7	
		Male 26.8			
Ali <i>et al</i>	2012	Pakistan	1329	63.7	*IDF
		(≥18 years, attending routine annual physical checkups)		Female 57.8	
		Male 66.2			
Binno <i>et al</i>	2015	Italy	9647	29	AHA-NHLBI
		(Ogliastro)		Female 31.8	
		Male 26.4			
Bonora <i>et al</i>	2003	Italy	888	34.1	WHO
		(40-79 years)		Female 35.9	
		Male 32.9			
Kaykhaei <i>et al</i>	2012	Iran (Zahadan)	1802	21	NCEP-ATPIII
		(≥19 years)		Female 24.9	
		Male 15.4			
Riedger <i>et al</i>	2011	Canada	1800	17.7	ATPIII
		(≥18 years)		Female 19.5	
		Male 15.9			
Ervin	2003	United States	3423	34	NCEP-ATPIII
		NHANES (2003-2006)		Female 32.6	
		Male 35.1			

Table 4 (Cont.)

Loizou <i>et al</i>	2006	Cyprus	1200	22.2	NCEP-ATPIII
		(20-80 Years)		Female 18.3	
				Male 26.5	
Ogbera	2010	South Africa	963	86	IDF
		(35-85 years diabetic population)		Female 86	
				Male 83	
Athyros <i>et al</i>	2005	Greece	4153	23.6	NCEP-ATPIII
		(≥18 years)		Female 22.8	
				Male 24.4	
Pannier <i>et al</i>	2006	France	101697	8.15	NCEP-ATPIII 2001
		(aged 18-80 years)		Female 6.1	
				Male 10.2	
AlDaghri <i>et al</i>	2010	Saudi Arab	2850	35.3	NCEP-ATPIII
		(18-55 years)		Female 34.1	
				Male 36.6	
Bhowmik <i>et al</i>	2015	Bangladesh	2293	30.7	*NCEP-ATPIII
		(≥20 years rural population)		Female 30.5	
				Male 30.5	
Vojarova de courten <i>et al</i>	2003	Slovakia	570	Gypsies 20 Non - gypsies 4	WHO
		(Gypsies and non gypsies)			
Al-lawati & associates	2003	Oman	1419	21	NCEP-ATPIII
		(>20 years)		Female 23	
				Male 19.5	
Soewondo <i>et al</i>	2010	Indonesia	1591	28.4	*NCEP-ATPIII
		(25-64 years)		Female 30.4	
				Male 25.4	
Katulanda <i>et al</i>	2012	Sri Lanka	4,485	27.1	*IDF
		(Adults over 18 years of age)		Female 28.3	
				Male 18.4	
Wang <i>et al</i>	2013	China	22,457	33.9	*IDF
		(adults aged ≥32 years)		Female 32.5	
				Male 35.1	

Note: *Asian specific cut off used to diagnose metabolic syndrome in Asian population/s; †date of publishing.

metabolic syndrome in different parts of the world; however, these are reliant on the different defining criteria taken into consideration.

Other important factors that appear to affect the prevalence are ethnicity, socioeconomic status, gender and lifestyle habits.

India

Metabolic syndrome is a crucial factor in causation of diabetes and coronary artery disease in Asian Indians.

The prevalence of metabolic syndrome varies by the criteria used and the population studied with greater prevalence in urban areas and in people with diabetes and obesity (Reddy *et al.*, 2006). Significant difference exist in the prevalence of various component of the metabolic syndrome within an urban environment and this appears to be influenced by socio-economic status (Mohan *et al.*, 2001). Table 5 depicts the prevalence of metabolic syndrome in different regions of India across different populations.

Table 5: Prevalence of Metabolic Syndrome in India					
Author	Year[€]	State/Population	Sample Size (n)	Prevalence (%)	Criteria Used*
Prasad <i>et al</i>	2012	Orissa	509	43.2	IDF
		Eastern India		Females 52.5	
				Males 34.2	
Sathyanarayan and Subbalakshmi	2012	Old age homes	189	57.67	IDF
				Females 61.01	
				Males 52.11	
Thiruvagounder <i>et al</i>	2010	Salem (patients attending routine medical checkups)	1558	27.62	NCEP-ATPIII
				Females 27.04	
				Males 33.17	
Kanjilal	2008	Mumbai	2316	40.3	ATPIII
		Bangalore			
		(IARS Families)			
Pathania <i>et al</i>	2013	Ambala, Haryana	1200	9.2	IDF
				Females 11.64	
				Males 6.45	
Deepa <i>et al</i>	2007	CURES-Chennai	23001	25.8	IDF
Bansal and Joshi	2015	Delhi (Muslim Population)	406	75.12	NCEP-ATPIII
				Females 77.58	
				Males 67.6	
Kaur	2014	Punjab	351	17.38	NCEP-ATPIII
				Females 57.38	
				Males 42.63	
Kamble <i>et al</i>	2010	Aji (Rural Wardha, village of PHC)	300	9.3	NCEP-ATPIII
				Females 10.7	
				Males 8.2	
Gupta <i>et al</i>	2004	Urban Indian population	1091	31.6	NCEP-ATPIII
				Females 39.9	
				Males 22.9	
Surana <i>et al</i>	2008	Mumbai	5088	77.2	NCEP-ATPIII
		(Diabetic population)		Females 87.71	
				Males 69.33	

Table 5 (Cont.)

Sawant <i>et al</i>	2011	Mumbai	548	19.52	NCEP-ATPIII
		(adults attending cardiac evaluation camps)		Females 12.6	
				Males 25.16	
Mangat <i>et al</i>	2010	Chandigarh	605	47.4	IDF
		(urban and rural slum population)		Females 59.6	
				Males 40.4	
Chow <i>et al</i>	2007	Andhra Pradesh	4535	28.2	NCEP-ATPIII
				Females 23.9	
				Males 32.5	
Yadav <i>et al</i>	2013	Gwalior Chambal (Diabetic participants)	700	57.7	IDF
				Females 70.4	
				Males 52.7	
Raman <i>et al</i>	2010	Chennai	1414	73.3	IDF
		(Diabetic Type II)		Females 83.3	
				Males 65.3	
Reddy <i>et al</i>	2006	10 Industrial Companies, India	19973	26.6	NCEP-ATPIII
				Females 36.3	
				Males 20.9	
Ismail <i>et al</i>	2016	Kerala	120	28.3	NCEP-ATPIII
		(Tribal area, Kannur district)		Females 32.5	
				Males 21	
Walvekar <i>et al</i>	2015	Karnataka Bank employees (only males)	73	Males 45.2	NCEP-ATPIII
Padmavathi <i>et al</i>	2013	Adults attending OPD, routine medical checkup	206	46.3	NCEP-ATPIII
				Females 42.2	
				Males 45	
Peixoto <i>et al</i>	2014	Goa	325	36.9	*NCEP-ATPIII
		(rural population)		Females 39.8	
				Males 33.6	
Jain <i>et al</i>	2015	Gujarat	1500	16	NCEP-ATPIII
		(apparently healthy adults 20-40 years)		Females 10.8	
				Males 21.5	
Madan and Narsaria	2016	Mumbai (Apparently healthy men 18-65 years)	313	Males 39.9	*NCEP-ATPIII

Note: *Asian specific cut off used to diagnose metabolic syndrome; €date of publishing.

Can and Bersot (2007) asserted the agreement among WHO, EGIR definition was very good. Metabolic syndrome that do not require measurement of insulin levels (NCEP, ACEE and IDF) identify twice more patients with insulin

resistance and increased Framingham risk score and are more useful than the definition that require measurement of insulin levels (WHO, EGIR). Both WHO and ATPIII definitions yielded similar estimates for the entire population

but masked difference for various population sub groups (i.e., in African-American men) (Ford and Giles, 2003). The results of the study conducted by Vinluan et al, 2012, show that all the definitions of metabolic syndrome with the exception of EGIR are associated with increased risks of cardiovascular events in elderly subjects. Only the modified WHO definition significantly (didn't include microalbuminuria) predicts all three endpoints, total cardiovascular events, coronary and cerebrovascular events. In an attempt to assess the performance of five different criterias of metabolic syndrome the level of agreement appears to be poor though IDF criteria shows a fair level of agreement with the WHO criteria (Onesi and Ignatius, 2014). Misra *et al.* (2005) showed the candidate definition MS 4 which included modified cut-offs of waist circumference (90 cms in men and >80 cm), BMI (>23 Kg/m²) and SST (>18 mm) in addition to the criteria a given by ATP definition demonstrated the maximum percentage of prevalence and gain in prevalence. Bhowmik *et al.* (2015), noted a higher prevalence of metabolic syndrome in Bangladeshi population using NCEP definition and was strongly associated with diabetes, hypertension and CVD. In a cross sectional study conducted by Moy and Bulgiba (2010), it was observed that modified NCEP-ATP III criteria may be more suitable in the diagnosis of metabolic syndrome for the Malay Cohort in Kuala Lumpur. In the rural Chinese population, the JIS, IDF and CDs criteria may not be more suitable than WHO and updated NCEPATPIII definition for screening high risk individuals and estimating the risk of CHD and stroke from metabolic syndrome especially in Men.

CONCLUSION

The review summarizes the different evolutionary and evolving concepts related to the syndrome and provides insight to the most widely used defining criteria's at present. Each one of the stated criteria has its own defining components and those which are common to all have different threshold values to identify and define metabolic syndrome that may or may not be specific to different ethnic populations. These differences make it difficult to analyze and compare the prevalence across different studies/ population groups.

Data across different populations shows an increasing trend of the prevalence of the deadly syndrome in both the developed and developing nations urging a need to develop a uniform criterion that is applicable to all populations which will help in comparing different studies/population and to aptly recognize people at risk.

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