Journal of Diabetes Science and Technology
Volume 5, Issue 2, March 2011

Prevalence and Clinical Profile of Metabolic Obesity and Phenotypic Obesity in Asian Indians

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Abstract

Background:

We estimated the prevalence of metabolically obese nonobese (MONO), metabolically obese obese (MOO), and metabolically healthy obese (MHO) individuals and correlated this with the prevalence of coronary artery disease (CAD) compared to metabolically healthy nonobese (MHNO) in urban South Indians.

Method:

Study subjects (n = 2350) were recruited from the Chennai Urban Rural Epidemiology Study. Generalized obesity was defined as a body mass index (BMI) ≥25 kg/m^2, based on the World Health Organization Asia Pacific guidelines. Metabolic syndrome (MS) was diagnosed based on the South Asian Modified-National Cholesterol Education Programme criteria. Coronary artery disease was defined by known myocardial infarction or Q waves on resting electrocardiogram.

Results:

Metabolically obese nonobese was defined as nonobese subjects (BMI < 25 kg/m^2) with MS, MOO as obesity (BMI ≥ 25 kg/m^2) with MS, MHO as obese subjects (BMI ≥ 25 kg/m^2) with no MS, and MHNO as no obesity or MS. Metabolically obese nonobese was identified in 355 (15.1%), MOO in 348 (14.8%), MHO in 312 (13.3%), and MHNO in 1335 (56.8%) subjects. The prevalence of CAD among the MONO, MOO, MHO, and MHNO was 5.5%, 4.2%, 1.4%, and 2.6%. However, when age standardization was done, there was no statistically significant increase in the risk of CAD among MONO [odds ratio (OR) = 1.300, 95% confidence interval (CI) 0.706–2.394, p = .339], MOO (OR = 1.651, 95% CI 0.852–3.199, p = .137), and MHO (OR = 0.524, 95% CI 0.250–2.130, p = .564) groups compared to MHNO, perhaps due to small numbers.

Conclusion:

Metabolic obesity may have different clinical implications than phenotypic obesity.

J Diabetes Sci Technol 2011;5(2):439-446

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Abbreviations: (BMI) body mass index, (BP) blood pressure, (CAD) coronary artery disease, (CI) confidence interval, (CURES) Chennai Urban Rural Epidemiology Study, (ECG) electrocardiogram, (HbA1c) glycated hemoglobin, (HDL) high-density lipoprotein, (MHNO) metabolically healthy nonobese, (MHO) metabolically healthy obese, (MONO) metabolically obese nonobese, (MONW) metabolically obese normal weight, (MOO) metabolically obese obese, (MS) metabolic syndrome, (OR) odds ratio

Keywords: Asian Indians, coronary artery disease, diabetes, metabolic syndrome, metabolically obese, nonobese, obesity, South Asians

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Introduction

Obesity is a growing public health problem leading to significant morbidity and mortality worldwide. However, all obese individuals do not present with metabolic risk factors, and conversely, all nonobese individuals need not be metabolically healthy. This has led to the identification of different subtypes of obesity such as metabolically obese normal weight (MONW) and metabolically healthy obese (MHO) subtypes of obesity.[1]

The concept of MONW individuals was identified in the 1980s.[2–5] These are individuals who, despite having a normal weight and body mass index (BMI), present with metabolic abnormalities and features of metabolic syndrome (MS).[6] These abnormalities include increased visceral adiposity, increased blood pressure (BP), low levels of high-density lipoprotein (HDL) cholesterol, elevated levels of triglycerides, and impaired fasting blood glucose.

Other investigators[7–10] have described the MHO entity. Metabolically healthy obese is defined as those individuals who, despite having obesity (based on BMI), lack any metabolic abnormalities or MS.

There are studies to suggest that an abnormal metabolic profile, rather than a high BMI, is associated with a higher risk for cardiovascular disease.[11] However, to our knowledge, there are no such studies in South Asians and, specifically, Asian Indians. Hence the current study was undertaken to estimate the prevalence of metabolically obese nonobese (MONO; MONW is renamed as MONO in this article), MHO, metabolically obese obese (MOO), and metabolically healthy nonobese (MHNO) subjects in an urban South Indian population and to compare the prevalence of coronary artery disease (CAD) in these subtypes of obesity.

Study Design

The Chennai Urban Rural Epidemiology Study (CURES) is a large cross-sectional study done on a representative population of the metropolitan city of Chennai (formerly Madras) in Southern India. The detailed study design of the CURES is described elsewhere,[12] and the sampling frame is shown on our Web site (*http://www.drmohansdiabetes.com/bio/CURES.pdf*). Briefly, of the 155 corporation wards in Chennai, 46 wards were randomly selected to provide a total sample size of 26,001 individuals who were ≥20 years of age.

Phase 1 of the CURES was conducted in the field and involved a door-to-door survey of 26,001 individuals. A detailed questionnaire was administered to all study subjects to collect information regarding demographic, socioeconomic, behavioral, and health status. Fasting capillary blood sugar, BP, and basic anthropometric measures were taken in all eligible individuals.

Phase 2 of the CURES deals with studies on the prevalence of microvascular and macrovascular complications among those with diabetes. Phases 1 and 2 are not discussed further in this article.

In Phase 3 of the CURES, every 10th subject recruited in phase 1 (n = 2600) was invited to our center for detailed anthropometric measurements and biochemical tests. Of these, 2350 participated in the present study (response rate, 90.4%). Institutional ethical committee approval was obtained, and written informed consent was obtained from all study subjects.

All the study subjects underwent an oral glucose tolerance test using a 75 g glucose load, except self-reported diabetes subjects for whom fasting venous plasma glucose was measured. The fasting blood sample was taken after ensuring 8 h of overnight fasting for estimation of plasma glucose and serum lipids using a Hitachi 912 Autoanalyser (Roche Diagnostics GmbH, Mannheim, Germany) and utilizing kits supplied by Boehringer Mannheim (Mannheim, Germany). The intra-assay and interassay coefficient of variation for the biochemical assays ranged between 3.1% and 7.6%. Glycated hemoglobin (HbA1c) was measured by the high-pressure liquid chromatography method using the Variant machine (BIORAD, Hercules, CA). The intra-assay and interassay coefficient of variation of HbA1c was <10%.

Anthropometric measurements and BP were obtained using standardized techniques by two trained interviewers. Interobserver and intraobserver coefficients of variation were less than 5%.

Height was measured with a tape to the nearest centimeter. Subjects were asked to stand upright without shoes with their back against the wall, heels together, and eyes directed forward.

Weight was measured with a spring balance that was kept on a firm horizontal surface. Subjects wore light clothing and stood upright without shoes, and weight was recorded to the nearest 0.5 kg. The scale was calibrated every day with "standard" weights.

Body mass index was calculated using the following formula: weight (kg)/height $(m)^2$.

Waist circumference was measured using a nonstretchable fiber measuring tape. The subjects were asked to stand erect in a relaxed position with both feet together on a flat surface; one layer of clothing was accepted. Waist girth was measured as the smallest horizontal girth between the costal margins and the iliac crests at minimal respiration.

Hip circumference was taken as the greatest circumference at the level of greater trochanters (the widest portion of the hip) on both sides. Measurements were made to the nearest 0.1 cm.

For both waist and hip circumference, two measurements were made, and the mean of the two readings was taken as the final value.

Blood pressure was recorded in the sitting position in the right arm, to the nearest 2 mmHg, using the mercury sphygmomanometer. Two readings were taken 5 minutes apart, and the mean of the two was taken as the BP. If the difference between the first and the second reading was greater than 10 mmHg for systolic pressure and/or greater than 6 mmHg for diastolic pressure, then a third measurement was made; and the mean of all three measurements was taken as the BP.

Definitions

Normal Glucose Tolerance

Subjects were confirmed to have normal glucose tolerance if the fasting plasma glucose was less than 100 mg/dl (<5.6 mmol/liter) and 2 h postload plasma glucose was less than 140 mg/dl (<7.8 mmol/liter).[13]

Impaired Fasting Glucose

Impaired fasting glucose was diagnosed if the fasting plasma glucose was 100 mg/dl or greater (≥5.6 mmol/liter) and less than 126 mg/dl (≤7.0 mmol/liter) based on American Diabetes Association definition.[14]

Impaired Glucose Tolerance

Impaired glucose tolerance was diagnosed if the 2-hour postload plasma glucose was at least 140 mg/dl (≥7.8 mmol/liter) but less than 200 mg/dl (<11.1 mmol/liter).[13]

Diabetes

Diagnosis of diabetes was based on the World Health Organization Consulting Group criteria,[13] that is, fasting plasma glucose of ≥126 mg/dl (≥7.0 mmol/liter) and/or 2 h postload plasma glucose of ≥200 mg/dl (≥11.1 mmol/liter) or self-reported diabetes on treatment with a physician.

Hypertension

Hypertension was diagnosed if the BP was at least 130/85 mmHg (for adolescents) and/or based on drug treatment of hypertension.[15]

Hypertriglyceridemia

Hypertriglyceridemia was diagnosed if serum triglyceride levels were 150 mg/dl or greater (≥1.7 mmol/liter) or if subjects were taking drugs for hypertriglyceridemia.[15]

Low High-Density Lipoprotein Cholesterol

Low HDL cholesterol was diagnosed if HDL cholesterol levels were less than 40 mg/dl (<1.04 mmol/liter) for men and less than 50 mg/dl (<1.3 mmol/liter) for women.[15]

Coronary Artery Disease

Coronary artery disease was diagnosed on the basis of known myocardial infarction or Q waves on resting electrocardiogram (ECG).

Metabolic Syndrome

Metabolic syndrome was defined based on the criteria of the South Asian Modified National Cholesterol Education Programme.[16] A subject was classified as having MS by the presence of any three of the following clinical features: abdominal obesity (waist circumference ≥90 cm for males; ≥80 cm for females), BP ≥130 and/or ≥85 mmHg, pre-existing diabetes or fasting glucose of ≥100 mg/dl (≥5.6 mmol/liter), serum triglycerides ≥150 mg/dl (≥1.7 mmol/liter), or HDL cholesterol <40 mg/dl (<1.04 mmol/liter) for males and <50 mg/dl (<1.3 mmol/liter) for females.

Metabolically Obese Nonobese

Metabolically obese nonobese was diagnosed in nonobese (BMI <25 kg/m²) subjects who fulfilled the criteria for MS.

Metabolically Obese Obese

Metabolically obese obese was defined as those who were obese (BMI ≥25 kg/m²) and had MS.

Metabolically Healthy Obese

Metabolically healthy obese was defined as those who were obese (BMI ≥25 kg/m²) but did not have MS.

Metabolically Healthy Nonobese

Metabolically healthy nonobese was defined as those who were nonobese (BMI <25 kg/m²) and did not have MS.

Statistics

Statistical analyses were performed using SPSS for Windows, version 15.0, software. Preliminary descriptive analyses were conducted to check for the distribution of the variables of interest. Values were expressed as mean ± standard deviation. Analysis of variance was used to compare the difference in means between groups. Chi square test statistic was used for comparing proportions among groups. Logistic regression analysis was done using CAD as the dependant variable and MONO, MOO, and MHO as independent variables using MHNO as the reference. A p value of <0.05 was considered statistically significant.

Results

The prevalence of the various obesity subtypes in the study population is presented in **Figure 1**. The prevalence of MONO was 15.1% (n = 355), while that of MOO was 14.8% (n = 348), MHO was 13.3% (n = 312), and MHNO was 56.8% (n = 1335).

Table 1 shows the clinical and biochemical characteristics of the study subjects in the MONO, MOO, MHO, and MHNO groups. All the obesity subtypes were found

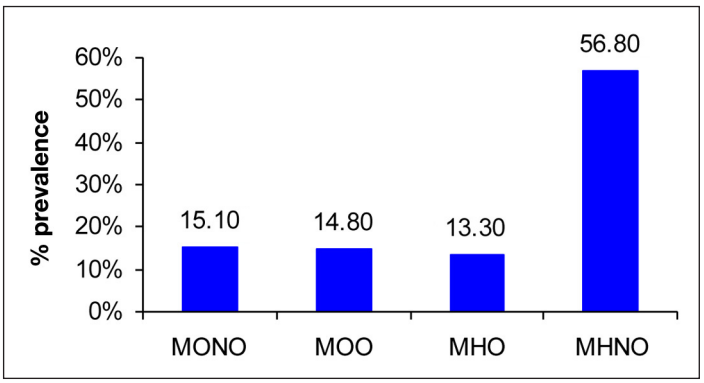

Figure 1. Prevalence of the various obesity subtypes.

Table 1.
Clinical and Biochemical Characteristics of MONO, MOO, MHO, and MHNO

Variables	MONO n = 355	MOO n = 348	MHO n = 312	MHNO n = 1335	Analysis of variance p value
Female n (%)	186 (52.4)	193 (55.5)	198 (63.5)	677 (50.8)	<0.05
Age in years	48 ± 12	42 ± 10	36 ± 10	38 ± 13	<0.001
BMI (kg/m²)	22.8 ± 1.7	27.7 ± 2.5	27.5 ± 2.3	20.4 ± 2.6	<0.001
Waist circumference (cm) Females Males	 85.3 ± 6.9 89.9 ± 5.8	 92.5 ± 7.9 98.6 ± 6.3	 89.5 ± 9.3 95.5 ± 7.5	 75.2 ± 8.8 79.2 ± 9.0	 <0.001 <0.001
Systolic BP (mmHg)	129 ± 18	129 ± 19	116 ± 14	114 ± 17	<0.001
Diastolic BP (mmHg)	79 ± 11	81 ± 11	73 ± 9	71 ± 11	<0.001
Fasting plasma glucose (mg/dl)	121 ± 53	111 ± 48	87 ± 17	88 ± 26	<0.001
Serum triglyceride (mg/dl)[a]	158 ± 2	158 ± 2	100 ± 1	79 ± 1	<0.001
HDL cholesterol (mg/dl) Females Males	 42 ± 8 37 ± 8	 40 ± 7 36 ± 7	 45 ± 10 39 ± 7	 47 ± 10 42 ± 10	 <0.001 <0.001
Low-density lipoprotein cholesterol (mg/dl)	121 ± 36	116 ± 33	115 ± 30	107 ± 32	<0.001
HbA1c (%)	7.1 ± 1.9	6.6 ± 1.6	5.6 ± 0.8	5.6 ± 1.0	<0.001

[a] Log-transformed values.

to be more common in females. The MHO group was younger and had significantly lower systolic and diastolic BPs, fasting plasma glucose, serum triglycerides, and HbA1c levels as compared to the MONO and MOO groups.

Figure 2 shows the age-wise distribution of the MONO, MOO, MHO, and MHNO subtypes. The prevalence of MONO increased with age. In contrast, the prevalence of MHO decreased with age. There was no clear trend with respect to age distribution of MOO and MHNO groups.

Figure 3 shows the prevalence CAD in the different subgroups. Overall, CAD was present in 68 subjects (2.9%). The prevalence of CAD in MONO, MOO, MHO, and MHNO were 5.5%, 4.2%, 1.4%, and 2.6%, respectively.

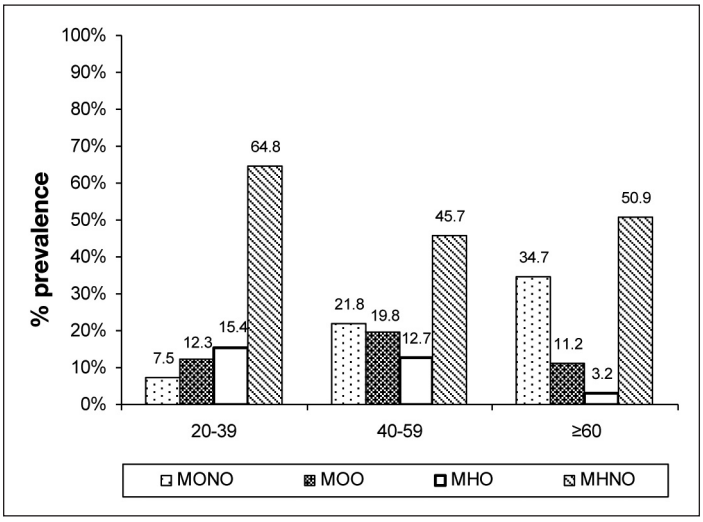

Figure 2. Prevalence of MONO, MOO, MHO, and MHNO by age group.

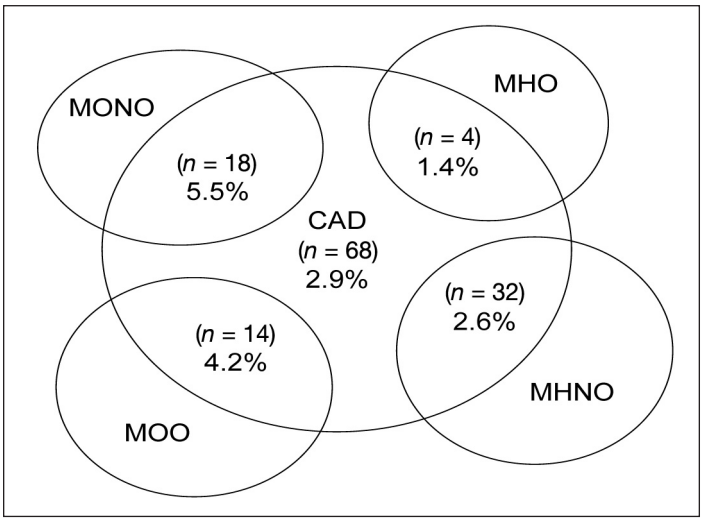

Figure 3. Prevalence of CAD among MONO, MOO, MHO, and MHNO.

Table 2 shows the risk of CAD among the various groups. Taking MHNO as the reference group [odds ratio (OR) = 1], there was a significantly increased risk of CAD among the MONO subjects [OR = 2.183, 95% confidence interval (CI) 1.209–3.941, p = .010], while the MHO subjects did not have a statistically significant increase in the risk for CAD (OR = 0.524, 95% CI 0.184–1.493, p = 0.226). The MOO subjects showed an increase in the CAD risk (OR = 1.66, 95% CI 0.876–3.150), although it was not statistically significant (p = 0.120), probably due to small numbers.

However, after age standardization (**Table 2**), there was no statistically significant increase in the risk of CAD among MONO, MOO, and MHO groups, perhaps due to small numbers.

The risk of CAD among the MONO, MOO, and MHO groups was studied by gender (**Table 2**). Compared to the MHNO males, the MONO males had an increased risk of CAD (OR = 2.552, 95% CI 1.212–5.377, p = .014). However, compared to the MHNO females, MOO

Table 2.
Risk of CAD among the MONO, MOO, and MHO Groups

Subtypes		OR	CI		p value
			Lower	Upper	
MONO	Overall (n = 355)	2.183	1.209	3.941	p = .01
		1.300[a]	0.706[a]	2.394[a]	p = .339[a]
	Females (n = 186)	1.727	0.646	4.611	p = .276
	Males (n = 169)	2.552	1.212	5.377	p = .014
MOO	Overall (348)	1.66	0.876	3.150	p = .120
		1.651[a]	0.852[a]	3.199[a]	p = .137[a]
	Females (n = 193)	2.123	0.866	5.202	p = .100
	Males (n = 155)	1.340	0.526	3.419	p = .540
MHO	Overall (312)	0.524	0.184	1.493	p = .226
		0.524[a]	0.250[a]	2.130[a]	p = .564[a]
	Females (n = 198)	0.796	0.224	2.825	p = .724
	Males (n = 114)	0.285	0.038	2.150	p = .223
MHNO (n = 1335) Reference group (OR=1)					

[a]Age adjusted.

females had a higher risk for CAD (OR = 2.123, 95% CI 0.866–5.202), though not statistically significant (p = .10).

Discussion

The relationship between body weight and metabolic abnormalities is not straight forward. Indeed, the MONO individuals often go undetected, because body weight or BMI is the usual method of assessing obesity. However, these nonobese individuals who are metabolically abnormal are exposed to an increased risk of cardiovascular disease. Studies also show that Asian Indians have very high rates of premature CAD.[17] Most of the earlier studies on subtypes of obesity have been on Western populations.[11] There is one study that has been done in a Korean population.[18] To our knowledge, this is the first study that reports on the presented subtypes of obesity in Asian Indians. In addition, we have looked at the prevalence of CAD among these subtypes of obesity.

In our study, the prevalence of the MONO subtype was 15.1%, which is high when compared to a community-based prospective observational study of Caucasian adults in the Framingham Offspring Study (2.6%)[11] and a community-based cross-sectional survey done on Korean adults (8.7%).[18]

Our earlier studies have shown that even those with a BMI of 23 have metabolic abnormalities in our population.[19] In this study also, 21% of nonobese subjects were metabolically obese, which is much higher compared to the Framingham Offspring Study (7.1%)[11] and the Korean population (12.7%).[18] The prevalence of MS among a normal-weight Iranian population was 9.9% in males and 11.0% in females.[20] A cross-sectional study done among a nonobese Taiwanese population showed that 18.7% of nonobese subjects had MS.[21] Park and colleagues[22] showed that, in the United States, 4.6% of normal-weight males and 6.2% of normal-weight females had MS. This suggests that physicians should screen for metabolic abnormalities even in persons with a normal BMI since their early detection and treatment among MONO individuals may be beneficial in the prevention of cardiovascular disease.[23]

In our study, the prevalence of the MHO subtype was 13.3%, which is higher compared to a Caucasian study (8.1%)[11] but slightly lower than a Korean study (15.2%).[18] When stratified by BMI category, 47.3% of our obese subjects had a healthy metabolic status, which is similar to the Korean population (47.9%)[18] but slightly higher than the Framingham Offspring Study (37%).[11] A cross-sectional study done in an obese Italian population showed that 27.5% of obese subjects were metabolically healthy.[24] Among a cohort of obese postmenopausal Canadian women, 12.3% of obese women were metabolically healthy.[25] A population-based study done in Portugal among males and females using different MS criteria showed that 3.3–32.1% of obese males and 11.4–43.3% of obese females were metabolically healthy.[26]

We found the prevalence of the MOO subtype was 14.8%, which is similar to that reported in the Caucasian study (13.9%). A study conducted among patients attending an obesity clinic[27] showed that the overall prevalence of MS among obese patients was 40.2%. A study conducted among obese patients referred for weight reduction surgery in Taiwan[28] showed 50.7% of obese patients had MS and were significantly older. A cross-sectional analysis of data from a large Italian database[29] of treatment-seeking obese subjects showed that 53% of cases met the criteria of MS.

It was found that 7.5% and 12.3% of our MONO and MOO subjects were in the age group of 20–30 years. The higher prevalence of metabolic abnormalities in a younger age group indicates that these individuals will have prolonged exposure to several atherosclerotic risk factors. This could contribute to the excess mortality in Asian Indians, as observed in a study that compared Indians, Malays, and Chinese.[30]

Since MS and obesity are both strongly associated with CAD, the risk of CAD was compared among these three subtypes of obesity. Overall, the prevalence and risk of CAD was significantly higher in the MONO subjects. The possible explanation could be that, in the absence of metabolic abnormalities, phenotypic obesity alone does not increase the risk for CAD, though it is premature to assert the risk of CAD based on phenotypic obesity alone. However, it is also reported that MHO subjects may have subclinical vascular disease.[31,32] Given that the risk of CAD is higher among the MONO subjects, it appears that identification and treatment of metabolic abnormalities could reduce the risk of cardiovascular disease among the MONO individuals, although prospective studies would be needed to prove this.

The strengths of the present study are that the subjects were representative of the urban population of Chennai, the sample size is fairly large (n = 2350), and the response rate is very good (90.4%). One of the limitations of the study is that the cross-sectional nature of the design does not allow any cause–effect conclusions to be made. Second, the number of CAD subjects was small, which

reduces the strength of the study. Finally, the diagnosis of CAD was made on the basis of known myocardial infarction or Q waves on resting ECG. It is possible there may be some false positives and false negatives that could affect the results. However, as these are the first results from India, the results are of interest.

In summary, we report on the prevalence of the MONO, MOO, MHO, and MHNO subtypes of obesity in an Asian Indian population and show that the prevalence and the risk of CAD is higher among the MONO individuals. This suggests that even nonobese subjects who have MS should be considered high-risk subjects for CAD. These MONO subjects could benefit from lifestyle intervention along with appropriate pharmacotherapy, which could help prevent or delay the development of CAD.

Funding:

We are grateful to the Chennai Willingdon Corporate Foundation, Chennai, for the financial support provided for the study.

Acknowledgments:

We thank the epidemiology team members for conducting the CURES field studies. This is the 93rd publication from the CURES (CURES-93).

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