Pregnancy outcomes decline with increasing body mass index: analysis of 239,127 fresh autologous in vitro fertilization cycles from the 2008–2010 Society for Assisted Reproductive Technology registry

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Objective: To examine the effect of body mass index (BMI) on IVF outcomes in fresh autologous cycles.
Design: Retrospective cohort study.
Setting: Not applicable.
Patient(s): A total of 239,127 fresh IVF cycles from the 2008–2010 Society for Assisted Reproductive Technology registry were stratified into cohorts based on World Health Organization BMI guidelines. Cycles reporting normal BMI (18.5–24.9 kg/m^2) were used as the reference group (REF). Subanalyses were performed on cycles reporting purely polycystic ovary syndrome (PCOS)–related infertility and those with purely male-factor infertility (34,137 and 89,354 cycles, respectively).
Intervention(s): None.
Main Outcome Measure(s): Implantation rate, clinical pregnancy rate, pregnancy loss rate, and live birth rate.
Result(s): Success rates and adjusted odds ratios (ORs) with 95% confidence intervals (CIs) for all pregnancy outcomes were most favorable in cohorts with low and normal BMIs and progressively worsened as BMI increased. Obesity also had a negative impact on IVF outcomes in cycles performed for PCOS and male-factor infertility, although it did not always reach statistical significance.
Conclusion(s): Success rates in fresh autologous cycles, including those done for specifically PCOS or male-factor infertility, are highest in those with low and normal BMIs. Furthermore, there is a progressive and statistically significant worsening of outcomes in groups with higher BMIs. More research is needed to determine the causes and extent of the influence of BMI on IVF success rates in other patient populations. (Fertil Steril® 2016;105:663–9. ©2016 by American Society for Reproductive Medicine.)
Key Words: Obesity, BMI, IVF, ART

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Obesity is a global issue that affects millions worldwide and is increasing in severity. The proportion of women worldwide with a body mass index (BMI) above the normal cutoff of 25 kg/m^2 has increased from 29.8% in 1980 to 38% in 2013 (1). Reproductive-age women are not invulnerable to the obesity epidemic. The National Health and Nutrition Examination Survey found that as of 2010, more than one-half of pregnant women in the United States were overweight or obese, and 8% of reproductive-aged women were extremely obese (2). Unfortunately, national incentive programs to

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decrease the prevalence of obesity have yet to demonstrate tangible success (1).

The effects of obesity are particularly evident during pregnancy, prompting the American College of Obstetrics and Gynecology to revise their Committee Opinion on Obesity in Pregnancy in January 2013 (3). As they discuss, prepregnancy BMI significantly affects pregnancy outcomes such as gestational diabetes mellitus, gestational hypertension, preeclampsia, macrosomia, and cesarean delivery (3–7). Additionally, offspring of obese mothers are more likely to experience prematurity, stillbirth, and congenital abnormalities, such as neural tube defects, and in the long term have higher risks for childhood and adolescent obesity (3).

Given the population trends, it is not surprising that an increasing percentage of women who are seeking fertility treatment are also obese. In the setting of IVF, clinical outcomes such as implantation rate, clinical pregnancy rate, pregnancy loss rate, and live birth rate could all potentially be affected by BMI. Early studies suggested that BMI did not affect IVF outcomes (8–10), but more recent studies seem to suggest the opposite (11–14), including a relatively recent analysis of Society for Assisted Reproductive Technology (SART) data from 2007 (14).

To further complicate the nature of the relationship between BMI and IVF cycle outcome, a recently published article by Schliep et al. analyzed 721 couples and, like the early studies in the field, found no relationship between increasing BMI and poor IVF outcomes (15). Other studies have focused exclusively on pregnancy loss as a primary outcome in obese patients undergoing IVF and found pregnancy loss rates to be higher with increasing BMI (12–16).

Our objective was to examine the impact of BMI on IVF outcomes. To isolate the effects of obesity from those of other underlying diseases, we also examined two subgroups of patients: those with only ovulatory disorders/polycystic ovary syndrome (PCOS) as a diagnosis, and those with only male-factor infertility.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board at Duke University. A retrospective cohort study of 239,127 fresh autologous IVF cycles was performed with the use of data from the SART Clinic Outcome Reporting System (CORS) database from 2008 to 2010. SART CORS is a self-reported database in the United States that represents ~97% of the clinical activity of United States IVF clinics (17). Of note, BMI data (height and weight) have been included as a category in the database since 2007.

All fresh cycles from this time period for which physiologically reasonable data had been entered for height and weight were included. Patients with height <48 inches and weight <70 pounds were excluded. The cycles were then stratified into cohorts based on female BMI according to the following World Health Organization (WHO) BMI guidelines: underweight (16.0–18.4 kg/m^2), normal (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), obese (class I, 30.0–34.9 kg/m^2; class II, 35.0–39.5 kg/m^2; class III, 40.0–45.9 kg/m^2 and 46.0–49.9 kg/m^2, and superobese (>50.0 kg/m^2) (18). Cycles in patients with a normal BMI (18.5–24.9 kg/m^2) were used as the reference group (REF).

Outcomes for this study included implantation rate, which was observed as a continuous variable, and clinical pregnancy, pregnancy loss, and live birth, which where included as binary variables. Implantation rate was calculated as the ratio of fetal heart beats to the number of embryos transferred. Clinical pregnancy was defined as an intrauterine gestational sac visible by means of transvaginal ultrasound coincident with a positive serum β-hCG concentration. Pregnancy loss was defined as a clinical pregnancy ending before 24 completed weeks of gestation, including both spontaneous and therapeutic loss. Pregnancy loss rate was calculated only for those cases in which a pregnancy outcome was recorded in SART CORS. Live birth was defined as delivery of a live-born infant at $\geq$24 weeks' gestational age. All outcomes were calculated per cycle start except for pregnancy loss, which was calculated per clinical pregnancy. Adjusted odds ratios (ORs) were obtained by fitting regression models with age, smoking status, number of oocytes retrieved, number of embryos transferred, and percentage of blastocyst transfers. ORs were considered to be statistically significant when 95% confidence intervals (CIs) did not cross the null value (OR 1). Logistic regression was used for binary outcomes, and linear regression was used for continuous outcomes. An analysis of variance (ANOVA) was carried out to assess significant variance across the BMI categories, as well as between the individual BMI categories and the reference values for all outcome results. These were considered to be significant if the P value was $<.05$. All statistical analysis was done in the R statistical environment (R Core Team).

One potential limitation of looking at the impact of BMI on IVF outcomes is that a significant percentage of obese patients in the infertility population also have polycystic ovary syndrome (PCOS). Where the literature suggests a correlation between increasing BMI and poorer IVF outcomes, it has not been investigated whether or not these outcomes are actually attributable to the underlying pathologies of PCOS and not to obesity alone. We therefore performed two subgroup analyses to isolate the effect of obesity from PCOS on IVF outcomes. First, we performed the same analyses on those cycles for which "ovulation disorders/polycystic ovaries" was the patient's only listed infertility diagnosis in the SART database. Although we will henceforth refer to this category simply as "PCOS" we want to be clear that the category is defined by SART as containing patients with one or more of the following characteristics: multiple ovarian cysts affecting fertility; oligo-ovulation (<6 cycles per year); and anovulation (of hypothalamic or nonhypothalamic causes). This distinction means that this category would likely include the majority of PCOS patients (and PCOS patients contribute to the majority of the subgroup), but also includes other ovulatory disorders. Although both PCOS and hypothalamic dysfunction leading to amenorrhea can be attributable to BMI, we think that it is a subgroup worth examining. Second, we performed an analysis of those cycles with purely male-factor infertility in an attempt to exclude patients with PCOS or any other female pathology. The outcomes measured

were the same as for the primary group and included implantation rate, clinical pregnancy rate, pregnancy loss rate, and live birth rate.

RESULTS

Among the sample size of 239,127 cycles, more than one-half of the patients (134,588) fell into the normal BMI reference range (Table 1). The mean patient age was 35 years across all groups except for the underweight patients, who on average were slightly younger. The mean baseline FSH decreased with increasing BMI from 8.6 mIU/mL in underweight patients to 6.8 mIU/mL in the highest BMI categories. The mean number of oocytes retrieved was inversely proportional to BMI, ranging from 12.4 oocytes retrieved in normal and underweight patients to 10.5 in the highest BMI category. The percentage of cycle cancellations increased with increasing BMI until the largest BMI cohort, but it was not statistically significant. Across all BMI categories, a mean of 2.4 embryos were transferred, but the percentage of blastocyst transfers decreased slightly with increasing BMI from 27.8% to 21.5%. The proportion of cycles whose only infertility diagnosis was "PCOS" increased with increasing BMI category. The proportion of cycles with purely male-factor infertility was largest in the lower BMI categories.

Implantation Rate

Implantation rate (Table 2) decreased with increasing BMI, from 29.5% (REF) in normal-BMI patients to a low of 20.3% (OR 0.91–95% CI 0.88–0.95; $P<.001$) in patients in the highest BMI category. Statistical significance of outcomes was confirmed with the use of ANOVA.

Clinical Pregnancy Rate

Clinical pregnancy rates (Table 2) were highest in the normal and underweight BMI categories. There was a statistically significant progressive decrease in pregnancy rate with increasing BMI category. The adjusted ORs demonstrated decreasing odds of clinical pregnancy with higher BMIs, reaching a low of 0.75 (95% CI 0.65–0.85; $P<.001$) in cycles with BMI >50 kg/m^2.

Pregnancy Loss Rate

Pregnancy loss rate (Table 2) significantly increased with increasing BMI categories, from a low of 11.3% (REF) in normal-BMI cohorts to a high of 20.3% (OR 1.87–95% CI 1.18–2.95; $P<.007$) in the highest BMI cohort, with statistical significance across all of the cohorts.

Live Birth Rate

Live birth rate (Table 2) also decreased with increasing BMI, from a high of 31% in low- and normal-BMI cycles to a low of 21% (OR 0.52–95% CI 0.41–0.66; $P<.001$) in cycles with the highest BMI (>50 kg/m^2). Once again, there was a statistically significant difference between each cohort and the reference cohort.

Subgroup Analyses

PCOS. In cycles for which the only infertility diagnosis was listed as "ovulation disorders/polycystic ovaries" (Table 3), implantation rate, clinical pregnancy rate, pregnancy loss rate, and live birth rate all trended toward poorer outcomes with increasing BMI, the majority of which reached statistical significance, especially in patients with BMI >30 kg/m^2. Implantation rates reached statistical significance in patients with BMI >30 kg/m^2 and ranged from 42.6% in patients with normal BMI to a low of 26% in patients with BMI 45–49.9 kg/m^2 (OR 0.89–95% CI 0.83–0.94; $P<.001$). Clinical pregnancy rate was also statistically worse in patients with BMI >30 kg/m^2. Rates of pregnancy loss increased with BMI from 8.8% (REF) in normal-BMI cycles, to as high as 30% (OR 4.39–95% CI 1.87–10.3; $P<.001$) in patients with BMI >50 kg/m^2. Finally, live birth rates dropped from 44% in the reference

TABLE 1

Stimulation characteristics by BMI category.

	BMI, kg/m^2								
Parameter	**< 18.5**	**18.5–24.9**	**25–29.9**	**30–34.9**	**35–39.9**	**40–44.9**	**45–49.9**	**> 50**	***P* value**[a]
n	7,149	134,588	54,822	24,922	11,747	4,084	1,292	463	NA
Age, y (SEM)*	34.7 (0.057)	35.4 (0.013)	35.6 (0.020)	35.5 (0.031)	35.4 (0.045)	35.5 (0.075)	35.5 (0.134)	35.4 (0.221)	<.001
FSH, mIU/mL (SEM)	8.6 (0.079)	8.5 (0.065)	8.2 (0.161)	8.6 (0.675)	7.2 (0.035)	7.0 (0.075)	6.8 (0.120)	6.8 (0.151)	.028
Oocytes retrieved (SEM)	12.4 (0.099)	12.4 (0.022)	12.3 (0.035)	12.3 (0.051)	12.1 (0.074)	11.6 (0.122)	11.2 (0.218)	10.5 (0.349)	<.001
Cancellation rate, %	9.8	10.3	11.3	11.3	12.2	13.3	14.2	11.7	.92
Embryos transferred (SEM)	2.3 (0.012)	2.4 (0.003)	2.4 (0.005)	2.4 (0.007)	2.4 (0.010)	2.4 (0.018)	2.5 (0.035)	2.3 (0.052)	<.001
Blastocysts transferred, %	27.8	26.6	25.9	26.1	25.1	22.0	20.3	21.5	<.001
PCOS, %	6.9	5.6	6.4	9.4	13.3	13.6	16.5	19.4	<.001
Male factor, %	19.7	19.5	19.2	18.1	16.6	16.6	16.3	14.9	<.001

Note: BMI = body mass index; PCOS = polycystic ovary syndrome; SEM = standard error of the mean.
[a] *P* value calculated by means of analysis of variance comparing variance across the entire group. $P<.05$ denotes statistical significance.

Provost. Pregnancy declines with increasing BMI. Fertil Steril 2016.

TABLE 2

Results for all autologous cycles by BMI category.[a]

	BMI, kg/m^2							
Parameter	**< 18.5**	**18.5–24.9**	**25–29.9**	**30–34.9**	**35–39.9**	**40–44.9**	**45–49.9**	**> 50**
n	7,149	134,588	54,822	24,922	11,747	4,084	1,292	463
Implantation rate, %	30.4	29.5	28.3	26.9	25.8	23.6	22.9	20.3
aOR (95% CI)[b]	0.99 (0.98–1.00)	REF	0.99 (0.99–0.996)	0.98 (0.97–0.99)	0.96 (0.95–0.97)	0.95 (0.93–0.97)	0.91 (0.88–0.95)	0.91 (0.88,0.95)
P value[c]	.26		< .001	< .001	< .001	< .001	< .001	< .001
Clinical pregnancy rate, %	37.7	37.9	36.8	35.7	33.7	32.0	30.6	30.0
aOR (95% CI)[b]	0.93 (0.88–0.99)	REF	0.97 (0.95–0.99)	0.90 (0.87–0.93)	0.81 (0.76–0.85)	0.80 (0.74–0.87)	0.75 (0.65–0.85)	0.66 (0.53–0.82)
P value[c]	.06		.013	< .001	< .001	< .001	< .001	.002
Pregnancy loss rate, %[d]	11.4	11.3	12.7	14.6	15.3	14.8	17.6	20.3
aOR (95% CI)[b]	1.11 (0.97–1.28)	REF	1.14 (1.08–1.21)	1.33 (1.23–1.43)	1.40 (1.26–1.56)	1.26 (1.06–1.51)	1.59 (1.19–2.14)	1.87 (1.18–2.95)
P value[c]	.21		< .001	< .001	< .001	.009	.002	.007
Live birth rate, %	31.2	31.4	29.8	28.0	26.3	24.3	22.8	21.2
aOR (95% CI)[b]	0.92 (0.86–0.97)	REF	0.94 (0.91–0.96)	0.84 (0.81–0.87)	0.76 (0.72–0.79)	0.73 (0.67–0.77)	0.67 (0.58–0.77)	0.52 (0.41–0.66)
P value[c]	.022		< .001	< .001	< .001	< .001	< .001	< .001

Note: aOR = adjusted odds ratio; BMI = body mass index; CI = confidence interval.
[a] All outcomes are per cycle start except for pregnancy loss (per clinical pregnancy).
[b] Odds ratios adjusted for age, smoking status, number of oocytes retrieved, number of embryos transferred, and percentage of blastocysts transferred.
[c] *P* values calculated by means of analysis of variance for each category compared to the reference group. Statistical significance defined as $P < .05$.
[d] Calculated for cases in which a pregnancy outcome was recorded in SART CORS.

Provost. Pregnancy declines with increasing BMI. Fertil Steril 2016.

TABLE 3

Results per cycle start[a] for diagnosis ovulatory disorders/PCOS only by BMI category.[a]

	BMI, kg/m^2							
Parameter	**< 18.5**	**18.5–24.9**	**25–29.9**	**30–34.9**	**35–39.9**	**40–44.9**	**45–49.9**	**> 50**
n	490	7,472	3,502	2,337	1,561	557	213	90
Implantation rate, %	42.4	42.6	40.1	35.5	32.5	29	26	29.2
aOR (95% CI)[b]	0.99 (0.94–1.03)	REF	0.98 (0.97–1.00)	0.96 (0.93–0.98)	0.92 (0.89–0.94)	0.91 (0.88–0.95)	0.88 (0.83–0.94)	0.91 (0.82–1.00)
P value[c]	.67		.08	< .001	< .001	< .001	< .001	.07
Clinical pregnancy rate, %	48.9	51.1	49.1	46.7	42.1	37.2	36.6	36.7
aOR (95% CI)[b]	0.92 (0.74–1.14)	REF	0.92 (0.84–1.01)	0.83 (0.75–0.92)	0.67 (0.59–0.77)	0.62 (0.51–0.76)	0.60 (0.44–0.82)	0.67 (0.40–1.10)
P value[c]	.60		.09	.002	< .001	< .001	0.002	.14
Pregnancy loss rate, %[d]	7.0	8.8	11.1	14.5	13.0	12.8	17.0	30.0
aOR (95% CI)[b]	0.75 (0.41–1.37)	REF	1.25 (1.01–1.54)	1.67 (1.33–2.09)	1.51 (1.13–2.01)	1.19 (0.73–1.93)	1.93 (1.01–3.68)	4.39 (1.87–10.30)
P value[c]	.44		.04	< .001	0.006	.41	.047	< .001
Live birth rate, %	43.3	44.1	40.6	37.3	33.4	29.4	27.7	23.3
aOR (95% CI)[b]	0.96 (0.77–1.20)	REF	0.87 (0.79–0.95)	0.75 (0.67–0.83)	0.62 (0.54–0.70)	0.59 (0.48–0.23)	0.52 (0.37–0.72)	0.43 (0.25–0.74)
P value[c]	.81		.003	< .001	< .001	< .001	< .001	.003

Note: Abbreviations as in Table 2.
[a] All outcomes are per cycle start except for pregnancy loss (per clinical pregnancy).
[b] Odds ratios adjusted for age, smoking status, number of oocytes retrieved, number of embryos transferred, and percentage of blastocysts transferred.
[c] *P* values calculated by means of analysis of variance for each category compared to the reference group. Statistical significance defined as $P < .05$.
[d] Calculated for cases in which a pregnancy outcome was recorded in SART CORS.

Provost. Pregnancy declines with increasing BMI. Fertil Steril 2016.

category to a low of 23.3% in patients with BMI >50 kg/m^2 (OR 0.43–95% CI 0.25–0.74; P=.003).

Male-factor infertility. Purely male-factor cycles were then analyzed, again comparing the underweight, overweight, and obese BMI cohorts to the reference cohort of normal BMI (Table 4). The data trend suggested that clinical pregnancies and live births decreased with increasing BMI category, although the ORs failed to reach statistical significance in some of the cohorts. Of note, all outcomes were statistically worse in patients with BMI 30–39.9 kg/m^2. Implantation rates ranged from 34% in the normal-BMI category to 20.1% (OR 0.86–95% CI 0.80–0.98, P=.02) in the BMI >50 kg/m^2 category, though it failed to demonstrate statistical significance for BMI >40 kg/m^2. Clinical pregnancy rate was 45% in patients with normal BMI, whereas cycles with BMI >50 kg/m^2 reported a clinical pregnancy rate of only 30.4% (OR 0.54–95% CI 0.30–0.98; P=.04). There was also an increase in the rates of pregnancy loss with increasing BMI, from 8.8% in the reference group to 20% in patients with BMI 45–49.9 (OR 2.35–95% CI 1.20–4.60; P=.01). Live birth rate in male-factor patients fell from 38.7% in the reference category to 24.6% in patients with BMI >50 kg/m^2 (OR 0.45–95% CI 0.24–0.83; P=.07) but failed to reach statistical significance in any of the BMI categories.

DISCUSSION

As our patient population becomes increasingly obese, it is important to continue to look at the impact that obesity has on IVF outcomes. The present study, representing the largest cohort study of BMI and IVF outcomes to date, with 239,127 fresh autologous cycles analyzed, strengthens claims that IVF success rates are most favorable in patients with low and normal BMIs. With increasing BMI, there was a steady and significant decrease in implantation rate, clinical pregnancy rate, and live birth rate and an increase in pregnancy loss. Whereas increasing BMI seemed to have a detrimental effect on IVF outcomes, the same was not true of cycles in underweight patients, suggesting that high BMI is more detrimental to IVF outcomes than low BMI.

We went a step further with the present study by looking at two additional subgroup populations with the goal of isolating obesity from other underlying pathologies in IVF, such as PCOS. The large cohort size allowed for subgroup analysis of patients with PCOS and male-factor infertility to isolate female obesity from other underlying pathologies. In patients with purely PCOS infertility, pregnancy loss was the only outcome that showed statistically significant changes with increasing BMI, although trends for all other outcomes also worsened with increasing BMI. When looking at patients with purely male-factor infertility, similar results were obtained. These results suggest that it is BMI itself rather than underlying pathologies that contribute to the worsening outcomes with increasing BMI, conflicting with the hypothesis that PCOS is the underlying pathology affecting IVF success rates in obese patients (19) and confirming the findings of other studies with smaller datasets (14–22).

TABLE 4

Results for diagnosis male factor only by BMI category.[a]

Parameter	BMI, kg/m^2						
	< 18.5	18.5–24.9	25–29.9	30–34.9	35–39.9	40–44.9	45–49.9	> 50
n	1411	26282	10549	4512	1947	678	210	69
Implantation rate, %	35.0	34.0	33.0	30.2	28.5	28.0	23.5	20.1
aOR (95% CI)[b]	0.99 (0.97–1.02)	REF	0.99 (0.99–1.00)	0.98 (0.96–0.99)	0.96 (0.94–0.98)	0.98 (0.94–1.01)	0.94 (0.89–1.00)	0.89 (0.80–0.98)
P value[c]	.74		.41	<.001	<.001	.17	.06	.02
Clinical pregnancy rate, %	44.2	45.0	44.5	41.5	38.4	37.6	30.5	30.4
aOR (95% CI)[b]	0.96 (0.84–1.09)	REF	0.99 (0.95–1.05)	0.87 (0.81–0.94)	0.79 (0.71–0.88)	0.89 (0.74–1.07)	0.65 (0.47–0.91)	0.54 (0.30–0.98)
P value[c]	.51		.92	<.001	<.001	.23	.01	.04
Pregnancy loss rate, %[d]	9.2	8.8	10.3	12.5	12.0	12.0	20.0	15.0
aOR (95% CI)[b]	1.10 (0.80–1.51)	REF	1.15 (1.01–1.30)	1.36 (1.15–1.62)	1.40 (1.08–1.81)	1.06 (0.68–1.67)	2.35 (1.20–4.60)	1.05 (0.24–4.67)
P value[c]	.56		.04	<.001	.01	.79	.01	.05
Live birth rate, %	38.0	38.7	37.7	33.9	31.6	30.2	22.8	24.6
aOR (95% CI)[b]	0.97 (0.85–1.10)	REF	0.97 (0.92–1.02)	0.82 (0.76–0.89)	0.76 (0.68–0.85)	0.85 (0.71–1.03)	0.58 (0.40–0.82)	0.55 (0.29–1.03)
P value[c]	.62		.28	<.001	<.001	.10	.002	.07

Note: Abbreviations as in Table 2.

[a] All outcomes are per cycle start except for pregnancy loss (per clinical pregnancy).

[b] Odds ratios adjusted for age, smoking status, number of oocytes retrieved, number of embryos transferred, and percentage of blastocysts transferred.

[c] *P* values calculated by means of analysis of variance for each category compared to the reference group. Statistical significance defined as $P<.05$.

[d] Calculated for cases in which a pregnancy outcome was recorded in SART CORS.

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The mechanism of the negative effect of BMI on IVF outcome is unclear. Recent studies have suggested decreased oocyte quality in obese patients (23) and looked to find molecular causes of decreased oocyte quality (24). The theories are diverse and include differing follicular fluid contents (25–27) and altered microenvironment oocyte/embryo metabolism (28). Alternatively, altered endometrial receptivity may play a role, a topic of research we are currently investigating in donor-recipient cycles.

SART CORS is the most recent and comprehensive assisted reproductive technology registry in the United States. The strength of the present study is the large sample size, which also allowed for subset analyses of cycles with PCOS and male-factor infertility. Additionally, we were able to use logistic regression modeling to control for a number of potential confounding variables, such as patient age, smoking status, number of oocytes retrieved, number of embryos transferred, and percentage of blastocyst transfers.

There are a number of limitations of this study. First, only female BMI could be analyzed, because male BMI is not recorded in the SART dataset. Although previous studies have linked female obesity and infertility, there are fewer data on the impact of male obesity on infertility. Several studies have seen no link between male BMI and semen parameters, and the association between male obesity and IVF outcome is unclear (15–30) but seem to suggest mild or no impact on IVF compared with female BMI. Second, our subset analyses, like any analysis of the SART dataset, are limited by user database input error of patient diagnosis. There is a verification process of a sampling of SART data performed by a third-party verification team, but this process does not include any assessment of practice or overall record keeping (31). In general, it is our assumption that this would likely mean that we would be missing patients in each category that did not have a diagnosis entered, and that it is possible that incorrect diagnoses were entered. Third, the heterogeneity of the "ovulation disorders/polycystic ovaries" category, which includes both patients with PCOS as well as hypothalamic or pituitary dysfunction, makes drawing conclusions about BMI in only patients with PCOS challenging. Although we would surmise that PCOS patients comprised the majority of this group, it is impossible to determine the percentage of PCOS patients in the category. Furthermore, the definition of PCOS can be interpreted liberally by individual clinics, because a number of conflicting but overlapping diagnostic criteria exist. It is impossible to determine the criteria of individual clinics for diagnosing PCOS. Fourth, this study did not control for multiple cycles in the same patient. Additionally, we were unable to adjust for patient race among BMI categories, because although race is included in the SART database, we unfortunately did not have that information in our dataset. Earlier studies have seen an interaction between race and BMI, suggesting that this may be a true limitation (32). Finally, although this represents the most recent analysis of BMI in the SART database, the most recent data are from 2010, attributed in part to the 2-year lag in release of data from the database (17).

CONCLUSION

The size of the dataset allowed for a broad analysis of the impact of BMI on IVF outcomes that has not been possible before, with results suggesting that BMI does in fact affect IVF outcomes and rates of pregnancy loss. Clinically, some of these differences were small, especially when comparing successive BMI categories, but the clinical significance at the highest BMI categories, which represent an increasingly higher percentage of our patients, are dramatic. Given these results, we think that a discussion of the impact of BMI on IVF outcomes is warranted in obese patients. Although we can recommend weight loss, this study does not specifically examine data that would recommend a certain percentage of weight loss to improve outcomes. This study demonstrates the impact of BMI on IVF to be gradual and progressive with increasing BMI. Despite live birth rates as much as 10% lower in the highest BMI groups, however, IVF success rates in the highest BMI group are still higher than for some of our poor-prognosis patients with diagnoses such as diminished ovarian reserve. For this reason, we think that limiting access for poorer prognosis alone seems unwarranted. Separately from IVF outcomes statistics, however, are the health concerns known to be common in obese pregnant patients, such as gestatational diabetes, preeclampsia, and preterm birth. Therefore, we propose that patients with obesity be dealt with on an individual basis, taking into account both IVF success rates as well as health, and that of the fetus, during pregnancy. Future research is needed to further define the mechanism of the impact of obesity on IVF outcome.

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