In a placebo-controlled pilot study, naltrexone (ReVia) produced weight loss in women with schizophrenia who were gaining weight while taking antipsychotic medication.

**Methods:** Participants were women, aged 18–70 years, who met DSM-IV criteria for schizophrenia or schizoaffective disorder, and were overweight (body mass index [BMI], ≥27) and actively gaining weight (>2% weight gain in the past year). Patients were randomly assigned to double-blind treatment with either 25 mg/day naltrexone or placebo and followed for 8 weeks. Eating behaviors and desires were measured with the Three-Factor Eating Questionnaire (a.k.a., the Eating Inventory) and the Questionnaire on Craving for Sweet or Rich Foods.

**Results:** Of 24 women enrolled in the trial, 3 left the study before receiving any treatment; the remaining 21 completed the study. Because more women with diabetes were assigned to the naltrexone group (6 vs. 1 to placebo), the analysis was adjusted for presence of diabetes. A total of 15 women were taking atypical antipsychotics.

After 8 weeks, women in the naltrexone group lost a mean of 7.5 lbs. (p=0.001 compared with the placebo group). Women who received placebo gained an average of 3 lbs. Only women without diabetes in the naltrexone group lost weight. Changes in BMI and waist circumference showed a similar pattern to changes in weight. There were no changes from baseline in metabolic laboratory measurements in either group. There were no differences between the groups in Positive and Negative Syndrome Scale scores or in quality of life of measurements. Women who received naltrexone reported a significant reduction in craving for sweet and rich foods. The groups did not differ in reinforcement, cognitive restraint, disinhibition, or susceptibility to hunger.

**Discussion:** The mechanism of weight change in these patients—decreased craving for sweet and rich foods—is consistent with the hypothesis that antipsychotic medications, via partial dopamine D2 receptor blockade, lead to a reward dilution so that patients eat more food to achieve the same reward. It is interesting to note that opioid receptor blockers such as...
naltrexone do not induce weight loss in people who are not taking antipsychotic medications. Endorphin release is particularly pronounced for high-fat, high-sugar foods, and preference for these foods is decreased by opioid receptor blockade with naltrexone. Effects of weight loss on metabolic parameters may take longer than 8 weeks to observe. Because it was a proof-of-concept trial, the study enrolled only women to maximize the likelihood of observing an effect. A larger study is currently underway that addresses some of the weaknesses of this study.

Study Rating*—17 (100%): This study met all criteria for a randomized controlled trial.

Tek C, Ratliff J, Reutenauer E, Ganguli R, et al: A randomized, double-blind, placebo-controlled pilot study of naltrexone to counteract antipsychotic-associated weight gain: proof of concept. *Journal of Clinical Psychopharmacology* 2014; doi 10.1097/JCP.0000000000000192. From Yale University School of Medicine, New Haven, CT; and the University of Toronto, Ont., Canada. Funded by Yale University; and other sources. Three study authors disclosed relationships with commercial sources; the remaining 2 declared no conflicts of interest.

*See Reference Guide.

SSRIs, NSAIDs, and Upper GI Bleeding

According to results of a meta-analysis, SSRIs are associated with increased risk of upper gastrointestinal bleeding, although the risk is more modest than a previous estimate.¹

Methods: Using an exhaustive search strategy that included 4 literature databases, gastroenterology conference proceedings, and bibliographies, the investigators identified studies of SSRIs and GI bleeding in patients aged >16 years. Randomized controlled trials, cohort, case-control, and cross-sectional studies were all included in the analysis. Studies of select groups of patients with organic diseases were excluded, but there were no restrictions on drug indication, dosage, or treatment duration.

Results: The search identified 15 case-control studies and 4 cohort studies. There were no randomized trials, because these are generally too small and brief to identify uncommon side effects. The investigators found no evidence of publication bias. However, a high risk of bias in terms of study design (i.e., study group selection, comparability of the groups, and ascertainment of the exposure or outcome of interest) was found in 9 case-control studies and in all 4 cohort studies. Six studies included only hospitalized patients, and the rest used population databases.

The 15 case-control studies included nearly 400,000 subjects, typically with many controls per case. The 4 cohort studies were inconsistent in their reporting of sample size but typically included thousands to tens of thousands of SSRI-treated patients. The combined odds ratio* for upper GI bleeding with SSRI therapy was 1.66 in the case-control studies and 1.68 in the cohort studies. Various statistical adjustments did not have a major effect on the SSRI-associated risk of bleeding. Based on the case-control studies, the number needed to harm* per 1 case of upper GI bleeding with SSRI treatment was 3177 for low-risk patients and nearly 881 for higher-risk patients.

Risk of upper GI bleeding was more pronounced when SSRIs were used concomitantly with NSAIDs. In the case-control studies, the odds ratio was 4.25 for users of both kinds of drug versus neither. Excess risk in the cohort studies was 6.08 (p<0.0001). Risk estimates for upper GI bleeding related to SSRIs and SSRIs plus NSAIDs were similar in hospital-based and population-based studies.

Discussion: SSRIs may cause upper GI bleeding by impairing platelet aggregation or by increasing gastric acidity. A previous meta-analysis identified substantial risk of upper GI bleeding with SSRI use (odds ratio, 2.4).² The present study, based on a much larger collection of data, suggests the risks are less than previously believed. Treatment risks and benefits should
be carefully weighed in older patients and in those with risk factors or a history of GI bleeding. The combination of SSRIs and NSAIDs should be avoided wherever possible. Proton pump inhibitor therapy should be considered in patients who cannot avoid the combination.

**Study Rating**—18 (100%): This study met all criteria for a systematic review/meta-analysis.

1. Anglin R, Yuan Y, Moayyedi P, Tse F, et al: Risk of upper gastrointestinal bleeding with selective serotonin reuptake inhibitors with or without concurrent nonsteroidal anti-inflammatory use: a systematic review and meta-analysis. *American Journal of Gastroenterology* 2014;109 (June):811–819. From McMaster University, Hamilton, Ont., Canada. This study was conducted without funding. The study authors declared no conflicts of interest.


*See Reference Guide.*

### Clonidine and Sensory Gating in Schizophrenia

In a small, pseudorandomized crossover study, clonidine (*Catapres*) normalized an EEG measure of sensory gating in patients with schizophrenia. These results suggest that clonidine, via its noradrenergic effects, may help restore normal prefrontal cortex filtering of sensory information in the disorder.

**Methods:** Study participants were 20 adult males, aged 25–50 years, receiving stable medication for schizophrenia. The majority (n=15) were receiving atypical antipsychotics, while 2 received conventional agents and 3 received both. Patients were allowed to continue concomitant benzodiazepines (n=5) or antidepressants lacking significant noradrenergic affinity (i.e., SSRIs; n=5). Those with substance dependence were excluded. Patients were age-matched to healthy male controls with no history of psychiatric illness. Patients were tested on 5 occasions in randomized order, with placebo and 4 different doses of clonidine: 0.025, 0.050, 0.075, and 0.15 mg. Controls were tested only once, unmedicated. Four hours after drug administration, study subjects were administered a test battery that included an assessment of P50 gating. Patients were administered a series of 2 consecutive, closely spaced bursts of white noise, the C stimulus and the T stimulus. The ratio of the amplitudes of P50 waves following the C and T stimuli (the T/C ratio), a measure of P50 suppression, was the primary outcome of this analysis.

**Results:** During the placebo session, patients with schizophrenia had a significantly increased T/C ratio compared with controls (p=0.008), indicating P50 gating deficits in spite of medical treatment. Compared with placebo, patients had a significant decrease in the T/C ratio during the clonidine sessions (p=0.012). There were no significant order effects of the 5 tests in patients and no difference in the results with different clonidine doses. The T/C ratio with all 4 doses of clonidine did not differ statistically from the ratio in controls. The effect in patients was largely due to a reduction in the amplitude of P50 response to the T stimuli, not the C stimuli.

**Discussion:** In schizophrenia, deficits in sensory gating—the ability to filter out irrelevant sensory stimuli—have been proposed to lead to cognitive fragmentation and ultimately hallucinations and delusions. Sensory gating is influenced by noradrenergic alpha1 and alpha2 receptors in the prefrontal cortex. The role of P50 in cognition has not been well studied, but there is some evidence that deficits are associated with impairments in sustained attention and vigilance. Clonidine, a specific noradrenergic alpha2 agonist, showed early promise in improving cognition decades ago, but these studies were never followed up. The present study suggests clonidine improves P50, a measure of sensory gating, while other results from the same experiment, published elsewhere, suggest it may also improve prepulse inhibition, a measure of sensorimotor gating.

Oranje B, Glenthoj B: Clonidine normalizes levels of P50 gating in patients with schizophrenia on stable medication. *Schizophrenia Bulletin* 2014;40 (September):1022–1029. From Copenhagen University Hospital, Glostrup; and the University of Copenhagen, Denmark. Funded by the Danish Council for Independent Research-Medical Sciences; and the Lundbeck Foundation. The authors declared no conflicts of interest.
Patient-Reported Outcomes in Depression

Second-line depression treatments had robust effects on patient-reported outcomes of functioning, quality of life, and depressive symptoms, according to an analysis of data from the STAR*D trial. Despite this success, large proportions of patients failed to achieve normal outcomes after second-line therapy.1

**Background:** Clinician-rated remission was the primary efficacy endpoint of the STAR*D trial, the largest major depression study conducted to date. Patient-reported outcome measures provide additional information, some of which cannot be captured with clinician ratings.

**Methods:** The investigators analyzed data from the randomized STAR*D trial of multiple levels of pharmacotherapy and psychotherapy in patients with major depressive disorder. The present analysis included patients who failed the initial trial of citalopram monotherapy. Level 2 consisted of either augmentation of citalopram with bupropion, buspirone, or cognitive therapy; or a switch to bupropion, sertraline, venlafaxine, or cognitive therapy. Treatment effects on clinician-reported outcomes have been reported previously;2,3 the focus of the present analysis was patient-reported outcomes. These were measured with the Quality of Life Enjoyment and Satisfaction Questionnaire (Q-LES-Q) short form, the Work and Social Adjustment Scale (WSAS), the Quick Inventory of Depressive Symptomatology–Self Report (QIDS-SR), and the Individual Burden of Illness for Depression (IBI-D).

**Results:** A total of 749 patients were included in the dataset. All 7 treatments led to statistically significant improvement in quality of life, with effect sizes* (Cohen’s d) ranging from 0.73 for switching to cognitive therapy to 0.42 for switching to bupropion or augmentation with cognitive therapy (p<0.01 for all). For functioning, the effect size was highest for switching to cognitive therapy (0.78) and lowest for bupropion alone (0.47). For depressive symptoms, effect sizes were largest for venlafaxine (0.88), followed by cognitive therapy (0.83), and smallest for citalopram plus buspirone (0.48).

Despite these generally positive effects, many patients remained significantly impaired after second-tier treatments. The proportion with normal quality of life and functioning increased significantly after treatment; however, only 19% of patients had Q-LES-Q scores in the normal range and 32% had WSAS scores in the normal range. Severe impairment was defined as scores 2 standard deviations below community norms. After Level 2 treatment, nearly 2 of 3 patients still struggled with severe impairment in quality of life and somewhat less than half had severely impaired functioning.

On average, patients in all treatment groups had a significant reduction in the overall burden of illness (p<0.0001), and the effects on patient-reported outcomes did not differ statistically among the 7 treatments. Switching to cognitive therapy stood out numerically, with the largest effect sizes, the largest proportion of patients finishing with normal function and quality of life (31%), the lowest proportion severely impaired, and the lowest ratings for burden of disease.

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1Ishak WW, Mirocha J, Pi S, Tobia G, et al: Patient-reported outcomes before and after treatment of major depressive disorder. Dialogues in Clinical Neuroscience 2014;16:171–183. From Cedars-Sinai Department of Psychiatry and Behavioral Neurosciences, Los Angeles, CA; and other institutions. The STAR*D study was funded by the NIMH. The funding source of the present analysis was not stated. The authors did not include disclosure of potential conflicts of interest.


**Drug Trade Names:** bupropion—Wellbutrin; buspirone—BuSpar; citalopram—Celexa; sertraline—Zoloft; venlafaxine—Effexor

*See Reference Guide.
According to results of an analysis of electronic health records, differences among antidepressants in their propensity to cause long-term weight gain are modest but potentially of interest in patients who are concerned about their weight or with other weight gain risk factors.

**Methods:** Study subjects were >19,000 adult patients, aged 18–65 years, who received treatment with any 1 of 11 different antidepressants for ≥3 months between 1990 and 2011 in a Boston health care system. Patients who received >1 of the study drugs were excluded. For comparison, the analysis also included >3000 patients who received a prescription for a nonpsychiatric intervention (i.e., weight loss drugs or an asthma medication). Weight measurements were obtained at baseline (within 1 month of the index prescription) and at 3, 6, 9, and 12 months. The primary analysis compared rates of weight change (weight trajectories) over the 12 months following prescription, using citalopram as the reference drug. Weight change in patients who completed the 12 months of follow-up and those who dropped out of treatment were calculated separately, and a weight average was computed.

**Results:** In addition to drug treatment, significant predictors of weight gain included male gender, younger age, lower baseline body mass index, anxiety or depression, and receiving a concomitant atypical antipsychotic. After adjustment for all of these factors, 3 antidepressants were associated with significantly less weight gain than citalopram. Bupropion was weight neutral (p=0.02 vs. citalopram). Amitriptyline and nortriptyline were associated with some weight gain, but not as much as citalopram (p<0.001 for both). Duloxetine was associated with a similar magnitude of difference from citalopram as the 2 tricyclics, but the difference was not statistically significant. There was no difference in weight gain among the SSRIs. (See table.)

Excluding patients taking antipsychotic drugs did not affect the results of the analysis. Adjusting the analysis to compensate for treatment dropouts attenuated the effects of bupropion somewhat but did not otherwise affect the findings. As expected, weight loss medications were associated with weight loss and the asthma drug was weight neutral.

**Discussion:** Previous studies of antidepressant-related weight gain suggest greater liability with tricyclics. These studies have mostly been confined to the acute treatment period and to clinical trial patient populations with specific disorders. The observational nature of the present study makes the results generalizable but may also have permitted certain biases. For example, patients' baseline weight may have

<table>
<thead>
<tr>
<th>Observed Mean Weight Change Over 12 Months of Antidepressant Use</th>
<th>3 Months</th>
<th>6 Months</th>
<th>9 Months</th>
<th>12 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram</td>
<td>-0.2 lb</td>
<td>0.9 lb</td>
<td>1.8 lbs</td>
<td>2.6 lbs</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>-0.2 lb</td>
<td>1.5 lbs</td>
<td>2.4 lbs</td>
<td>2.0 lbs</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>-0.7 lb</td>
<td>0.4 lb</td>
<td>1.5 lbs</td>
<td>1.5 lbs</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>0.2 lb</td>
<td>0.9 lb</td>
<td>2.2 lbs</td>
<td>2.2 lbs</td>
</tr>
<tr>
<td>Sertraline</td>
<td>0.0 lb</td>
<td>1.8 lbs</td>
<td>2.2 lbs</td>
<td>2.0 lbs</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>0.9 lb</td>
<td>1.1 lbs</td>
<td>1.3 lbs</td>
<td>2.0 lbs</td>
</tr>
<tr>
<td>Bupropion</td>
<td>-0.9 lb</td>
<td>0.0 lb</td>
<td>0.0 lb</td>
<td>-0.4 lb</td>
</tr>
<tr>
<td>Duloxetine</td>
<td>-0.2 lb</td>
<td>1.3 lbs</td>
<td>1.1 lbs</td>
<td>0.2 lb</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>0.9 lb</td>
<td>1.1 lbs</td>
<td>3.1 lbs</td>
<td>4.9 lbs</td>
</tr>
<tr>
<td>Nortriptyline</td>
<td>0.7 lb</td>
<td>1.1 lbs</td>
<td>1.1 lbs</td>
<td>0.7 lb</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>0.2 lb</td>
<td>0.4 lb</td>
<td>0.7 lb</td>
<td>2.6 lbs</td>
</tr>
</tbody>
</table>
influenced the drug they were prescribed. Overweight patients may have been over-represented because their physicians weigh them more frequently. Bias from nonrandom treatment discontinuation by patients who gained weight, which would lead to an underestimation of weight gain, cannot be excluded. In addition, antidepressant dose effects were not directly examined, because a substantially larger sample would be required for the analysis to have adequate statistical power.

Blumenthal S, Castro V, Clements C, Rosenfield H, et al: An electronic health records study of long-term weight gain following antidepressant use. JAMA Psychiatry 2014;71 (August):889–896. From Massachusetts General Hospital, Boston, and other institutions. Funded by the NIMH. Three study authors disclosed financial relationships with commercial sources; the remaining 9 authors declared no conflicts of interest.

Drug Trade Names: amitriptyline—Elavil, Endep, Enovil; bupropion—Wellbutrin; citalopram—Celexa; duloxetine—Cymbalta; escitalopram—Lexapro; fluoxetine—Prozac; mirtazapine—Remeron; nortriptyline—Aventyl, Pamelor; paroxetine—Paxil; sertraline—Zoloft; venlafaxine—Effexor

Depot Aripiprazole for Acute Treatment of Schizophrenia

In a phase III multicenter trial, extended-release injectable aripiprazole (Abilify Maintena) was effective and well tolerated in the treatment of acute exacerbations of schizophrenia.

Methods: Study subjects were adults with schizophrenia (DSM-IV-TR) who were currently experiencing an acute psychotic episode, defined as a quantifiable exacerbation of psychotic symptoms with a deterioration in clinical and/or functional status. All patients were living in a stable situation, had experienced response to an antipsychotic in the last 12 months, and were not treatment resistant. Patients without previous aripiprazole exposure (about 94% of the sample) received open-label oral aripiprazole for 3 days to establish tolerability. All patients then underwent a 7-day antipsychotic-free washout. Patients were then randomly assigned to aripiprazole once-monthly (AOM) injection or placebo. For the first 14 days, patients also received an oral formulation of their assigned medication (aripiprazole at 10–20 mg/day based on clinical judgment). All study participants were treated as inpatients for the first 2 weeks of the 12-week study, after which they could be discharged, returning to the clinic biweekly to complete follow-up. The primary efficacy outcome was change from baseline in the Positive and Negative Syndrome Scale (PANSS) total score.

Results: A total of 340 patients received randomized treatment. None failed the screen for aripiprazole tolerance. Sixty-four percent of AOM patients completed ≥10 weeks of treatment, as did 49% of the placebo group. The most common reasons for discontinuation were withdrawal of consent for aripiprazole (19%) and lack of efficacy for placebo (29%). Final analysis included all patients who received ≥1 injection and completed ≥1 postbaseline assessment.

Baseline PANSS total scores were 103 and 104 in the AOM and placebo groups, respectively. Improvements were significantly greater with AOM than placebo; at the 12-week evaluation, scores had decreased by 27 and 13 points, respectively (p<0.0001). The between-group difference reached statistical significance by the 1-week evaluation. By week 12, average Clinical Global Impression–Severity* scores decreased from a mean of 5.2 in both groups by 0.6 points with placebo and 1.4 points with AOM (p<0.0001). Response (≥30% reduction in PANSS total score) occurred in 35% of the aripiprazole group and 16% of the placebo group at week 12 (p<0.0001). Secondary endpoints of PANSS positive and negative symptoms and scores on the Personal and Social Performance Scale also favored aripiprazole.

The adverse event profile of AOM was similar to that previously reported. An exception was the unusual weight gain in study participants who received aripiprazole: nearly 8 lbs. on average, with 22% of patients gaining ≥7% of their initial weight. Analysis by race indicated that the 59 African-American participants who received aripiprazole gained a mean of 11 lbs., while white participants did not gain more weight than the placebo group. The authors point
out that African Americans have a relatively high prevalence of a risk allele for antipsychotic-induced weight gain, and this ethnic group was overrepresented in the study relative to previous AOM studies.

**Discussion:** Extended-release injectable aripiprazole has demonstrated efficacy as maintenance treatment for schizophrenia. The present study results suggest it may also be a viable option for management of acute episodes.

**Study Rating**—17 (100%): This study met all criteria for a randomized controlled trial.

Kane J, Peters-Strickland T, Baker R, Hertel P, et al: Aripiprazole once-monthly in the acute treatment of schizophrenia: findings from a 12-week, randomized, double-blind, placebo-controlled study. *Journal of Clinical Psychiatry* 2014; doi 10.4088/JCP.14m09168. From the Zucker Hillside Hospital, Glen Oaks, NY; and other institutions. Funded by Otsuka and Lundbeck. All study authors disclosed financial relationships with commercial sources, including 10 who are employed by either Otsuka or Lundbeck.

*See Reference Guide.

### Erythropoietin and Cognition in Bipolar Disorder

In a randomized phase-II study, weekly infusions of erythropoietin resulted in improvement in some areas of cognition in patients with bipolar disorder. Erythropoietin did not affect verbal memory, the primary study outcome, but had positive effects on a number of secondary and tertiary measures.

**Methods:** Study participants were adults with cognitive difficulties associated with bipolar disorder, which was in full or partial remission with mood-stabilizing medication, who were recruited from either a specialty clinic for affective disorders or via internet advertisement. They were randomly assigned to receive 8 weekly infusions of 40,000 IU erythropoietin or placebo. Cognitive function was measured with a comprehensive neuropsychological test battery at baseline, week 9 (following the last treatment), and at week 14. The primary outcome was change in verbal memory, measured as words recalled in the Rey Auditory Verbal Learning Test (RAVLT). This test also measured word recall following interference, word recall after a 30-minute delay, and word recognition. Secondary outcomes were sustained attention and facial-expression recognition. A variety of tertiary outcomes were also analyzed.

**Results:** Of 44 patients randomly assigned to treatment, 3 discontinued erythropoietin early after developing increased thrombocytes; however, all completed the cognitive assessments. One patient in the placebo group discontinued treatment and was lost to follow-up.

At week 9, erythropoietin was associated with small, nonsignificant improvement in word recall, which disappeared by week 14. The active treatment was also associated with improvement in word recall after interference, but not in the other word recall measures. Erythropoietin had mixed effects on the other outcomes, including significant improvement in speed for correct target detection; recognition of happy facial expressions; attention; executive function; and verbal fluency.

An exploratory analysis was conducted by assembling a composite score of the outcomes that were most strongly affected by erythropoietin. This score, which reflects overall speed of complex cognitive processing, including attention, memory, and executive function, showed significant improvement with erythropoietin from baseline to week 9 (p=0.01) and week 14 (p=0.01). Erythropoietin was associated with transient increases in blood pressure, hemoglobin, erythrocytes, and thrombocytes.

**Discussion:** Erythropoietin is produced in the brain, where it mediates neuroprotection and development. In previous studies, single-dose erythropoietin improved memory and
executive function in both healthy subjects and patients with depression. The present results suggest further trials of the non-hematopoietic analogs of erythropoietin may be warranted, considering the hematopoietic effects of erythropoietin may limit its clinical use.

Study Rating*—17 (100%): This study met all criteria for a randomized controlled trial.

Miskowiak K, Ehrenreich H, Christensen E, Kessing L, et al: Recombinant human erythropoietin to target cognitive dysfunction in bipolar disorder: a double-blind, randomized, placebo-controlled phase 2 trial. Journal of Clinical Psychiatry 2014; doi 10.4088/JCP.13m08839. From Copenhagen University Hospital, Denmark; and the Max Planck Institute of Experimental Medicine, Gottingen, Germany. Funded by the Danish Ministry of Science, Innovation, and Higher Education; Novo Foundation; and other sources. All 5 study authors disclosed relationships with commercial sources.

Reference Guide

Clinical Global Impression–Severity (CGI-S) Scale: A 7-point rating of the severity of illness. A score of 1 corresponds to a rating of normal; 2=borderline mentally ill; 3=mildly ill; 4=moderately ill; 5=markedly ill; 6=severely ill; 7=extremely ill.

Effect Size: The effect size represents the amount of change in outcome that can be attributed to treatment, where 0.2 indicates a small effect, 0.5 a medium effect, and 0.8 a large effect. It is relatively independent of clinical significance and large effect sizes do not ensure treatment efficacy.

Number Needed to Harm: A measure of how many patients need to be exposed to a risk-factor to cause harm in 1 patient that would not otherwise have been harmed. Lower NNH, indicates more attributable risk.

Odds Ratio: A comparison of the probability of an event in 2 groups. An odds ratio of 1 implies that the event is equally likely in both groups. An odds ratio greater than 1 indicates that the event is more likely to occur in that group than in the comparison group.

Study Rating: A measure of how well a study conforms to quality standards. The study rating uses a checklist system based on the comprehensive Strength of Evidence Report from the Evidence-Based Practice Center Program of the Agency for Healthcare Research and Quality (AHRQ). The rating checklists have been posted at www.alertpubs.com.

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