



BODYWEIGHT CHANGES ASSOCIATED WITH PSYCHOTROPIC MEDICATIONS

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and Renée Williard, PhD

Weight gain is an adverse side-effect common to many psychotropic drugs. While most weight gain is usually mild, drugs such as lithium, valproate and the atypical antipsychotics can produce clinically significant increases in bodyweight. This can lead to a number of health problems that are associated with obesity. In addition, patients may be less likely to adhere to a drug regimen that induces weight gain, even that which is mild with no significant effects on health. Such changes in bodyweight can have detrimental effects to a person's mood and self-esteem. The focus of this review is to provide a relative risk of weight gain or loss associated with antipsychotic, antidepressant, and mood stabilizing medications based upon their receptor pharmacology. A better understanding of the relationship between the neuropharmacology of obesity and the mechanisms of action of several psychotropic drugs may provide the clinician with information to design drug regimens that minimize or reverse drug-induced weight gain.

Body weight is regulated by central mechanisms controlling satiety, appetite and craving as well as peripheral mechanisms that control metabolism and energy utilization. Research has suggested that serotonin, dopamine, and histamine neurotransmitter systems, among others, appear to mediate central mechanisms regulating bodyweight (for reviews see ^{1,5,6}). Drugs that block or inhibit 5-HT_{2C}, D₂, and H₁ receptors are more likely to be associated with weight gain. In contrast, drugs that stimulate 5-HT_{2C}, D₂, or Beta₃-adrenergic receptors have been associated with weight loss.

Antipsychotic treatment frequently results in weight gain. Some of the newer atypical agents often produce a larger, more sustained change in weight compared to traditional agents. This can best be explained by their mechanisms of action.

Atypical antipsychotics inhibit all three receptors, 5-HT_{2C}, D₂, and H₁, which are associated with weight gain. Clozapine and olanzapine produce significant, prolonged weight gain while quetiapine, which has weaker inhibition at 5-HT_{2C}, and risperidone, which has weaker inhibition at H₁, produce more modest changes in weight. Typical agents such as the phenothiazines also produce modest weight gains with an inhibition profile similar to quetiapine. However, haloperidol (which primarily inhibits D₂ with weak inhibition at H₁) and molindone (which only inhibits D₂) produce mild weight gain and weight loss, respectively. The relationship between the neuropharmacology of antipsychotic drugs and bodyweight gain demonstrates an additive effect: those drugs which produce more inhibition at more receptor sites are likely to cause more weight gain.

Of the antidepressant medications, mirtazapine, the tricyclic antidepressants (TCAs), and the non-specific monoamine oxidase inhibitors produce the most gain. Mirtazapine is a strong inhibitor at 5-HT_{2C} and H₁ receptors with virtually no affinity for the D₂ receptor. The binding profiles of the TCAs and MAOIs are similar to that of mirtazapine, but with weak inhibition at D₂ receptors. While the pharmacological profile of mirtazapine is complex, its significant effects on weight gain can best be attributed to its potent inhibition at 5-HT_{2C} and H₁. The SSRIs are unique in that short-term treatment (<6 mos.) produces weight loss whereas long-term use (>6 mos.) can result in modest increases in weight.

Continued on page 5

TABLE 1. Relative risk of bodyweight changes associated with psychotropic drugs

Drug	Relative Risk *
Antipsychotics	
Clozapine	++++
Olanzapine	++++
Quetiapine	++
Risperidone	++
Phenothiazines	++
Haloperidol	+
Molindone	-
Ziprasidone	+
Antidepressants	
SSRIs	< 6 mos - > 6 mos +
TCAs/MAOIs	++
Fluvoxamine	0
Bupropion	-
Mirtazapine	++/+++
Nefazodone	0
Reboxetine	-
Venlafaxine	-
Mood Stabilizers	
Lithium	++++
Valproic Acid	++
Carbamazepine	+
Gabapentin	+
Lamotrigine	0/+
Topiramate	-

* Subjective judgement by the authors based upon the literature reviewed

- = mild weight loss;
0 = no change;
+ = low risk, smallest weight gain;
++++ = high risk, largest weight gain

Abbreviations:

MAOIs = monoamine oxidase inhibitors;
SSRIs = selective serotonin reuptake inhibitors;
TCAs = tricyclic antidepressants.

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CULTURAL COMPETENCY IN DIAGNOSIS AND PSYCHOPHARMACOLOGY

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Cultural competency has many definitions and there are many ways to implement the concept. Having bilingual/ bicultural culturally competent staff is ideal but is not always feasible. A staff psychiatrist should consider cultural factors when evaluating an individual. DSM IV addresses some of these issues in Appendix I Outline for Cultural Formulation and Glossary of culture-bound Syndromes and illustrates some of the cultural limitations of DSM IV.

Case Example

Mrs. A, a 28 y.o. Chinese woman experiencing significant social stressors, presented to a local clinic complaining of feelings of guilt, suicidal ideation, insomnia, anorexia, anergia, anhedonia, as well as chronic headaches, dizziness, tiredness, easy fatigue, weakness, and tinnitus. She would qualify for a diagnosis of major depression by DSM IV criteria. However, by ICD-10 criteria, she could be given a diagnosis of neurasthenia, with secondary depression, consistent with the Eastern view that many feelings can be explained by somatic causes. The Eastern health care provider would explain her basic problem as a "lack of energy" in the central nervous system, where a Western evaluator might emphasize the presence of unusual stress or conflicts. Thus, Mrs. A has one illness, but two valid diagnoses.

Mrs. A's illness is expressed through her culturally determined idioms and social relationships. Thus, she will tell her physician of her physical complaints and leave out the emotional distress. Further, Mrs. A knows about the syndrome of neurasthenia and will describe her symptoms in a way that is consistent with that syndrome. This helps her make sense of what is happening to her. (Lu et. al. 1995)

Here are a few other points to consider:

1. Somatic symptoms may be the chief presenting complaint not depression and patients may resist treatment if the somatic symptoms are minimized or recast as "just" depressive symptoms.
2. Axis II personality disorders are often difficult to diagnosis correctly in other cultures
3. Axis III diagnosis should be made in relationship to cultural norms.
4. Axis IV and V (Stressors and the GAF score) should be determined within the context of an assessment of patterns of acculturation and social, economic and cultural parameters

Mental Status Exam

5. Many questions asked in a traditional mental status exam are not easily translated into another language nor do they have any cultural relevance (i.e.proverb interpretation). Many questions may be offensive when asked.
6. Date and Orientation may not have the significance we place on it in Western culture.
7. When assessing calculations and the naming of objects (consider the patient's education and the cultural relevance of information).

Evaluation Questions

8. Patients presenting with somatic complaints should be evaluated as if they were presenting for a medical evaluation.
9. Look for somatic symptoms of depression & anxiety (e.g., sleep or appetite disturbance, weight change, decreased energy, irritability, shortness of breath, tremors, GI symptoms, etc.).

Diagnosis

10. Misdiagnosis frequently occurs, and the existence of culture-bound syndromes points to a lack of precise correspondence between indigenous labels and established diagnostic categories.
11. Consider cultural norms of behavior.
12. Consider ICD 10 and other cultural diagnoses.

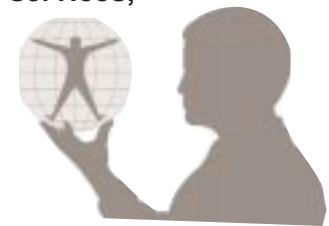
Medication Sensitivity

13. Certain ethnic groups may need lower medication dosage range because of sensitivity to medications (medications are metabolized differently in different ethnic populations, see the article by Barbara Liang in a previous edition).
14. There may be a need to prescribe something for the patient to "save face" after a doctor's visit.
15. Address somatic complaints as appropriate. Asian cultures often view the mind and body as unitary rather than dualistic, and patient's tend to focus more on physical discomforts than emotional symptoms, leading to over representation of somatic complaints.
16. Physical health liaison and communication is essential.
17. Consider using less sedating medications for individuals presenting with *low energy* as chief complaint.
18. Pay special attention to the signing of informed consent and to the explanation of the process of consent which may seem odd to patient's from cultures where the doctor makes all the decisions.

Beginning November 30th, Alameda County BHCS will host the seventh annual California Cultural Competence and Mental Health Summit. The theme for the two day symposium is "Inclusion by Design". Keynote speakers include Alvin Poussaint, MD, Theresa La Fromboise, Ph.D., and Stanley Sue, PhD. Over 35 different workshop topics will be presented, including creating cultural competent services, exploring client culture, and ethnicity & pharmacology.

The Summit will be held at the Oakland Convention Center, 1001 Broadway, Oakland, CA. November 30th & December 1st from 8:30 - 5pm.

For more information please call (916) 556-3480



ALAMEDA COUNTY BEHAVIORAL HEALTH CARE

ALL ANTIDEPRESSANTS DO NOT ACT THE SAME!

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A student of biochemistry might know S-adenosyl-l-methionine (SAME) as an important endogenous compound which participates in numerous metabolic pathways as a major source of methyl groups in the body. A customer of a natural products or vitamins outlet might know SAME as a nutritional supplement that has possible benefits in reducing the symptoms of maladies as varied as major depression and arthritis. If the two were to engage in conversation with each other and a bystander psychiatrist while all were traveling on a crowded BART train in Alameda County, what educational points might likely be shared?

While SAME seems to have gained recent notoriety in the lay press, its antidepressant effects have been reported in the medical literature since the late 1970s. Numerous open and double-blind studies have been conducted to formally test its efficacy in the treatment of depression, but most of them have involved small numbers of patients, and the results are often varied due to significant methodologic constraints. Nevertheless, most of the studies carried out in the United States have been encouraging. Initially, parenteral administration of SAME showed greater antidepressant efficacy over placebo, and perhaps with a more rapid onset of action than associated with a traditional antidepressant medication, imipramine^{1, 2}. Subsequent oral formulations of SAME have demonstrated similar therapeutic superiority over placebo and comparable efficacy with desipramine³. A meta-analysis of international clinical studies published through 1992 also suggests that SAME is efficacious in treating depressive target symptoms to an extent greater than placebo and equivalent to standard tricyclic antidepressants⁴.

SAME's actual mechanism of antidepressant action has not yet been identified. It is known that the process of methylation itself is critical

to the homeostatic regulation of hormones and neurotransmitters. SAME, in particular, participates in a key biochemical cycle whereby methyl groups are transferred to a wide variety of acceptor molecules, including biogenic amines, proteins, and phospholipids. The implication is that a disruption in the normal course of methylation by, for example, a deficiency of SAME, would result in a dysregulated system that could eventually manifest as a depressive disorder secondary to neurotransmitter imbalances, dysfunctional receptors, or

The interest in SAME stems in part from a longstanding search for an antidepressant medication that is relatively devoid of undesirable side effects.

damaged neuronal membranes. Interestingly, treatment with desipramine has also been shown to elevate endogenous SAME plasma concentrations to an extent that correlated with clinical improvement³. Although the evidence from this small cohort (n=6) is clearly circumstantial, this finding is consistent with the hypothesis that SAME is somehow associated with the normal regulation of mood.

The interest in SAME stems in part from a longstanding search for an antidepressant medication that is relatively devoid of undesirable side effects. While the introduction of the newer agents tend to have a more tolerable side effect profile compared to the tricyclic antidepressants, they are not without their own risks of adverse events that may negatively impact adherence to pharmacotherapy. Like other antidepressants, SAME has been reported to trigger a switch into mania^{3, 5}. Headache and mild nausea appear to be more commonly attributed to SAME. Daily dosages of SAME used as an antidepressant have ranged from

400 mg to 1,600 mg. However, as a "dietary supplement," SAME is not formally approved and indicated by the Food and Drug Administration for the treatment of depressive disorders - and is disallowed by law from making any therapeutic claims as such. As with other herbal medicines and nutritional supplements that are not regulated and can be purchased over-the-counter, there may be concerns about the standardization of active ingredient content and quality of manufacturing processes for any given product on the market. In addition, there is a current lack of knowledge regarding how SAME may interact with other medications, food, and concurrent medical conditions.

The BART train has entered the station at which the psychiatrist takes leave of the biochemist and nutritional outlet customer and disembarks. The pair suddenly asks the psychiatrist for a recommendation on whether one should use SAME in depression, but the doors close suddenly, and the reply is muffled. Likewise, without any large-scale, definitive clinical studies supporting SAME as a novel antidepressant, psychiatrists will probably be reluctant to give a clear recommendation based solely on the evidence presented in the literature to date. Clinicians of all disciplines should inquire whether their patients are using "alternative" products such as SAME, in order to effectively monitor for both therapeutic and adverse outcomes.

(1) Janicak PG, Lipinski J, Davis JM, Altman E, Sharma RP. S-Adenosylmethionine in depression: A literature review and preliminary report. *Ala J Med Sci* 1988; 25: 306-313.

(2) Bell KM, Plon L, Bunney WE, Potkin SG. S-Adenosylmethionine treatment of depression: A controlled clinical trial. *Am J Psychiatry* 1988; 145: 1110-1114.

(3) Kagan BL, Sultzer DL, Rosenlicht N, Gerner RH. Oral S-adenosylmethionine in depression: A randomized, double-blind, placebo-controlled trial. *Am J Psychiatry* 1990; 147: 591-595.

(4) Bressa GM. S-Adenosyl-l-methionine (SAME) as antidepressant: Meta-analysis of clinical studies. *Acta Neurol Scand* 1994; Suppl 154: 7-14.

(5) Carney MWP, Martin R, Bottiglieri T. The switch mechanisms in affective illness and SAM. *Lancet* 1983; 1: 820-821.

POSITIVE STATE DMH REVIEW

The California Department of Mental Health conducted its review of the Alameda County Behavioral Health Care Services Mental Health Plan during the week of 9/13/99. A wide variety of areas were examined for compliance with State regulations and with the operational policies and procedures established when private and public MediCal funding was consolidated and made the responsibility of counties to manage. Administrative staff was extensively interviewed as well as two focus groups of consumers and families. Broad areas audited included Plan Requirements, Access, Authorization Process, Beneficiary Protection, Contracts, Health and Safety, Interface with Physical Health Care (including pharmacy and laboratory services), Provider Appeals Process, Quality Management Activities (including Medication Monitoring) and Chart Review of Short Doyle/MediCal Hospital Services.



Performance Report

Other than a few minor deficiencies common to most counties, the team of reviewers not only found BHCS to be in excellent compliance, but offered complimentary

pharmacy with other counties for several years, it was gratifying to receive the compliments of the Department of Mental Health's reviewers who plan to share our materials, particularly with smaller counties. They were also told by both focus groups of consumers and family members that BHCS psychiatrists and other staff were just as concerned about the physical health of their patients as their mental health, and made the appropriate referrals and follow-ups on their behalf.

remarks in a number of areas as well. Of particular note from the standpoint of the Medical Director's Office and the BHCS medical staff were the positive comments made about two areas of our responsibility - our Pharmacy System and the interface between mental health and physical health care. While we have been providing information and consultation about our system of

While BHCS continues to provide excellent access for its patients to all psychotropic medications in cost-efficient ways and is working on further improving its access to physical health care resources, it is satisfying to have the State team of reviewers recognize our efforts and the achievements accomplished thus far.

PHARMACEUTICAL SETTLEMENT

A class action suit



brought against 19 pharmaceutical manufactures has resulted in a settlement structured to provide medications for the poor and indigent in California. A total of \$148 million in medications over a three year period will be distributed throughout California by both community and hospital organizations serving this population. The litigation, brought on by both consumers and independent pharmacies, contended that the manufacturers were engaged in illegal price fixing, forcing the independent stores to buy drugs at inflated prices. These prices were allegedly passed on to consumers.

The drug distribution plan created by the settlement will make available medications for asthma, hypertension, diabetes, infectious disease, as well as AIDS/HIV disease. Included in the over 120 medications provided by this settlement are many for psychiatric disorders:

ANTIPSYCHOTICS

olanzapine (Zyprexa)
quetiapine (Seroquel)

ANTIDEPRESSANTS

bupropion (Wellbutrin SR)
fluoxetine (Prozac)

ANXIOLYTIC

buspirone (Buspar)
gabapentin (Neurontin)
lamotrigene (Lamictal)

MISC

disulfiram (Antabuse)
nortriptyline (Aventyl)
sertraline (Zoloft)

There are drug company product-specific limits set for each agent, usually between \$500,000-\$750,000 annually. Once the annual limit for a particular product has been distributed, that product will be discontinued from distribution until the next year.

To participate, the clinic/hospital must be a non-profit, county/city-owned & operated program providing serves to the indigent. In addition, the program must have a pharmacy license or permit. No patient charge or third-party reimbursement can be attached to the product distribution. The Pharmaceuticals and Indigent Care (PIC) program will provide the ordering structure, distribution and training for any participating organization. The implementation timeline notes Year 1 distribution to commence 4/1/2000. For more information about the PIC program, please contact Kathryn Duke @ (510) 302-3300. For all Alameda County BHCS clinics, Douglas Del Paggio Pharm.D. will be co-ordinating the program.

WEIGHT GAIN ASSOCIATED WITH PSYCHOTROPIC DRUGS

Continued from page 1

Since the SSRIs have little or no affinity at dopamine or histamine receptors and either stimulate 5-HT_{2C} indirectly (increase 5-HT by blocking reuptake) or directly (e.g., fluoxetine), weight loss is expected. The mild to modest weight gain associated with long-term use may be due to down regulation of 5-HT_{2C} as a result of prolonged stimulation of this receptor⁴. Alternatively, the lifting of depressive symptoms may also play a role in increased appetite. It is interesting to note that paroxetine, which has the greatest 5-HT transporter affinity, produces more weight gain than fluoxetine or sertraline (based on preliminary uncontrolled comparative trials-for review see⁵). Those antidepressants with direct agonist effects at 5-HT_{2C} such as fluvoxamine, nefazadone (via its metabolite mCPP) and venlafaxine produce either weight loss or no changes in bodyweight during short-term use. In addition, bupropion, which has only agonistic effects at D₂ and the new specific norepinephrine reuptake inhibitor (SNRI) reboxetine, which has no affinity for serotonin, dopamine or histamine receptors, both produce mild weight loss.

The mechanism by which mood stabilizing drugs produce their weight gaining effects has not been well characterized. None of the mood stabilizers act on serotonin, dopamine, or histamine systems. These agents primarily increase gamma amino-butyric acid (GABA) levels. Their ability to induce weight gain based upon their neuropharmacology is unique, involving other systems (i.e., GABA) than those discussed above. A number of hypotheses regarding central and peripheral mechanisms have been proposed and include increased appetite and preference for high carbohydrate and fatty foods as well as metabolic changes such as increased carbohydrate/lipid storage and hypothyroidism. Lithium and valproic acid produce the most weight gain in this class of medications followed by carbamazepine and gabapentin with modest gains in weight. For the newer mood stabilizers, lamotrigine is associated with little or no weight gain and topiramate is associated with weight loss. Topiramate's novel mechanism of action as an inhibitor of carbonic anhydrase and glutamate transmission at the alpha-amino-3-hydroxy-5-methylisoxazole-4-propionate (AMPA) receptor as well as potentiating GABA transmission may provide clues to its ability to cause weight loss³.

In conclusion, the mechanisms by which psychotropic medications produce the unwanted side effect of weight gain differs among classes. Research has implicated the 5-HT_{2C}, D₂ and H₁ receptors to mediate central mechanisms involved in feeding behavior. Antipsychotic drugs, particularly the atypical antipsychotics, produce inhibition at all three of these receptors and are associated with substantial increases in weight gain. On the other hand, those drugs that stimulate 5-HT_{2C}, D₂ and H₁ receptors produce no increases in weight or mild weight loss. While the propensity of mood stabilizing agents such as lithium and valproic acid to produce significant gains in bodyweight has been well-documented, the pharmacologic mechanisms by which they produce this adverse effect is unknown, largely because the therapeutic mechanism of action of these agents remains unclear. Future research is warranted to elucidate the mechanisms by which these drugs exert changes in bodyweight. Nonetheless, understanding the neuropharmacology of psychotropic agents and the central and peripheral mechanisms of obesity will aid the clinician in choosing an effective drug regimen that minimizes the unwanted side effect of weight gain associated with these medications.

ACKNOWLEDGEMENTS

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References in the article can be requested by writing to the Psychopharmacology newsletter, 2532 Santa Clara Ave., PMB 219, Alameda, Ca 94501

PROFESSIONAL Advice

Clinical Tips for Managing Weight Gain Associated with psychotropic Drugs

- ◆ Reduce dose of current medication.
- ◆ Switch to another agent in the same therapeutic class that produces less weight gain (e.g., olanzapine to risperidone).
- ◆ Educate patient on exercise and dieting (e.g., decreasing caloric intake of foods high in fat).
- ◆ Possible use of low-dose topiramate to counteract weight gain associated with psychotropic medications (preliminary data)².
- ◆ Use of anorexic agents such as orlistat (lipase inhibitor), diethylpropion, phentermine (drug interactions with MAOIs, phenothiazines, TCAs) and sibutramine (drug interactions with SSRIs, MAOIs, lithium). These drugs (except orlistat) are contraindicated in patients with hyperthyroidism, moderate/severe hypertension, symptomatic cardiovascular disease, and glaucoma and should be used with caution in patients with diabetes mellitus.



CLINICAL TRIALS

MEDICATION TO TREAT METHAMPHETAMINE ABUSE

A controlled trial of the calcium channel blocking antihypertensive medication amlodipine (Norvasc®) in the treatment of methamphetamine abuse is underway at UCSF. This NIH-funded study is being conducted by substance abuse investigator Steven L. Batki, M.D.

Interested persons should call 415-502-5802
Monday through Friday, 8:30 AM - 4:30 PM.



CONTINUING MEDICAL EDUCATION

Doug DelPaggio, PharmD

Cerebral basis for Schizophrenia and Cognition, Scott Purdon, PhD
November 12, 1999

SFGH
1001 Potrero Ave., SF, CA
Room 7M30 (415) 206-4938

Transpersonal Aspect of Medication, Bruce Victor, MD
November 23, 1999

San Mateo Co MHS
225 W 37th Ave., San Mateo, CA
Multi-purpose Room (650) 573-2530

Cultural Competence & Mental Health Summit VII "Inclusion by Design"
November 30 - December 1, 1999

Oakland Convention Center,
1001 Broadway Oakland, CA
(510) 567-8126

Innovations in the Pharmacotherapy of Depression, Charles Debattista, MD
December 3, 1999

SFGH
1001 Potrero Ave SF, CA Room 7M30
(415) 206-49381

Venlafaxine in Depression, Alan Bott, MD
December 8, 1999

Alameda Co BHCS
2000 Embarcadero Cove, Oakland, CA
Alameda Room (510) 567-8106

The Borderline Child, Graeme Hanson, MD
December 14, 1999

San Mateo Co MHS
225 W 37th Ave., San Mateo, CA
Multi-purpose Room (650) 573-2530

Psychological Aspects of Using Medications, Victor Reus, MD
January 11, 2000

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225 W 37th Ave., San Mateo, CA
Multi-purpose Room (650) 573-2530

Developmental Model of Alcoholism, Stephanie Brown, MD
January 25, 2000

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225 W 37th Ave. San Mateo, CA
Multi-purpose Room (650) 573-2530

Update on Psychopharmacology of BAD
Lauren Marangell, MD
January 28, 2000

SFGH
1001 Potrero Ave., SF, CA
Room 7M30 (415) 206-49381

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